Copper Nutrition in Pasture-Fed New Zealand Thoroughbreds, and its Role in Developmental Orthopaedic Disease

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ABSTRACT

This thesis reports studies to test the hypothesis that "dietary supplementation of pasture-fed New Zealand Thoroughbreds with copper will reduce the incidence and severity of developmental orthopaedic disease". Thoroughbred foals were raised based on New Zealand Thoroughbred industry standards at AgResearch's Equine Research Stud. Thus the first stage required the development of methods to define indices of copper status and growth plate development. Techniques were developed and evaluated for *in vivo* sampling of liver from the mare and neonatal foal and the distal radial physis of the neonatal foal.

Pregnant Thoroughbred mares (n=24) were divided into either copper supplemented or control groups. Live foals born to each group of mares were also divided into copper supplemented or control groups. Supplementation was administered orally with aqueous copper sulphate at a rate of 0.5 mg Cu/kg liveweight (LW), and controls received a placebo of equivalent fluid volume. Mares were supplemented for the final 13 to 25 weeks of gestation until parturition. Foals were supplemented from 21 days of age with 0.2 mg Cu/kg LW increasing to 0.5 mg Cu/kg LW by 49 days remaining at that level until euthanasia at approximately 150 days. All animals grazed tall fescue pasture containing 4.4 to 8.6 mg Cu/kg dry matter (DM) for the duration of the experiment. This experimental design allowed independent investigation of both the effect of mare treatment during gestation and foal treatment on the evidence of developmental orthopaedic disease in the foals. Randomisation was stratified based on mare age, sire of the foal, last date of service and in the case of the foals only, sex of the foal.

Indices of copper status were measured in both the mares and foals throughout the experiment, and at postmortem. The foals were examined regularly for evidence of developmental orthopaedic disease (DOD) which included clinical, conformational and radiological examinations. At euthanasia, an exhaustive postmortem examination was performed which included investigation of all limb and cervical spine articulations, and examination of the physes from the proximal humerus, proximal and distal radius and tibia, and distal femur, third metacarpus and third metatarsus. Physes were examined after cutting bone ends into slabs of approximately 6 mm widths using a bone saw. All abnormalities in cartilage or bone found at postmortem were submitted for histological examination. A histomorphometric study was also performed on the growth plate and metaphyseal primary spongiosa of standard sections cut through the distal radius.

Plasma copper concentration of mares declined throughout the final trimester, and was not affected by copper supplementation. There was a trend toward increased copper concentration in the livers of supplemented mares, and a significant increase in the liver copper concentration of foals born to supplemented mares as determined by liver biopsies of mares and foals shortly after parturition,. Plasma and blood cell copper concentration, and plasma caeruloplasmin oxidase activity in the foals were not affected by copper supplementation, but liver copper concentration was significantly greater in copper-supplemented foals compared with controls.

There was no effect of copper supplementation on the evidence of DOD *in vivo*, in the physes examined at postmortem, or on the histomorphometry in the distal radius. However there was an effect of mare supplementation during gestation on indices of physitis in the foals assessed from postmortem radiographs and on mild abnormalities found in the articulations of foal limbs. Copper supplementation of the foal had no effect on these indices. The low incidence and severity of lesions found in foals grazing a pasture based diet containing 4.4 to 8.6 mg Cu/kg DM, combined with the failure to demonstrate an effect of foal copper supplementation on the evidence of DOD is contrary to published and anecdotal reports within New Zealand and internationally. This prompted the investigation of a possible dietary constituents which may affect the requirement for copper.

Molybdenum was identified as a possible antagonist which under certain circumstances may be found in high concentrations in a pasture diet. A pasture was prepared containing 8 to 15 mg Mo/kg DM. Weanlings were grazed on this pasture for 70 days and then supplemented with oral copper sulphate at a rate of 1.0 mg Cu/kg DM for a further 14 days (84 days total). There was no effect of high dietary molybdenum on plasma copper, trichloroacetic acid insoluble plasma copper, or blood cell copper concentration, or on the activities of caeruloplasmin oxidase or red blood cell superoxide dismutase. There was also no effect of dietary molybdenum on the liver copper concentration after 70 or 84 days. It was therefore concluded that at concentrations that might be expected in a pasture diet molybdenum is unlikely to increase copper requirements of horses.

The possible implications of this work to the New Zealand Thoroughbred industry, and suggestions for further research are discussed.

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I dedicate this thesis to my parents, Pam and Martin,
with my love and respect.

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