The effect of vitamin E and/or selenium depletion on tissue peroxidation and peroxidisability in ruminant cattle

Dominic M. Walsh, Seamus Kennedy, W. John Blanchflower and D. Glenn Kennedy

Veterinary Research Laboratories, Belfast, BT4 3SD

It is believed that nutritional degenerative myopathy in cattle results from lipid peroxidation induced by dietary imbalances of vitamin E, selenium and polyunsaturated fatty acids [1,2]. This study was undertaken to determine: (i) if lipid peroxidation and peroxidisability were increased in calves depleted of vitamin E and selenium and (ii) if a single deficiency of either micronutrient could increase tissue peroxidation or peroxidisability.

Four groups of 4 calves each, were fed diets that varied only in \( \alpha \)-tocopherol (\( \alpha -T.P. \)) and selenium content. The basal diet [3] used for each group contained 3 ug \( \alpha -T.P./g \) and 0.02 ug Se/g. The diet of each group was supplemented as follows: 0.2 ug Se/g and 200 ug \( \alpha -T.P. \) (group I); 0.2 ug Se/g (group II); 200 ug \( \alpha -T.P. \) (group III). No supplement was added to the diet of group IV.

The animals were blood-sampled weekly. Whole blood glutathione peroxidase (GPx) activity and plasma \( \alpha -T.P. \) concentration were determined [4,5]; Figure 1 and 2.

The calves were slaughtered after 147 days and tissue samples collected for biochemical analyses. Concentrations of thiobarbituric acid reactive substances (TBARS) were measured as an indicator of lipid peroxidation [6]. Tissue peroxidisability (ATBARS) after activation with ascorbic acid [7] and also by gas chromatographic determination of hexanal by modification of a published method [8].

Mean concentrations of ATBARS and hexanal were significantly greater in the heart, biceps and supraspinatus of vitamin E depleted animals than in vitamin E controls, irrespective of Se status. ATBARS results were also significantly greater in the masseter of vitamin E depleted animals than in vitamin E sufficient controls. Similarly, hexanal concentrations in masseter of vitamin E deficient animals were greater than those in the vitamin E sufficient controls, although this only reached statistical significance in the animals fed the doubly deficient diet. Mean concentrations of TBARS were only significantly greater in the heart of vitamin E depleted animals when compared to controls.

**Figure 1**

**Figure 2**