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EFFECT OF DIETARY VITAMIN E AND SELENIUM ON PIG MEAT QUALITY

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INTRODUCTION

Dietary deficiency of vitamin E and associated deficiency of selenium give rise to lesions of the reproductive system, of skeletal muscle and liver and of the nervous and cardiovascular system. Vitamin E which is located within biological membranes and associated with the highly peroxidizable PUFAs of cell membrane phospholipids (Diplock and Lucy, 1973) functions as an in vivo chain-breaking antioxidant that protects tissue lipids from free radical attack. Vitamin E does not function alone as a cellular antioxidant, but rather is an integral part of a network of antioxidants including glutathione peroxidase (Wayner *et al.*, 1987). Combs and Scott (1979) concluded that both nutrients have cooperative roles in the membrane integrity, vitamin E functioning as a free radical quencher and Se functioning via the Se-containing enzyme, glutathione peroxidase as a preventative antioxidant in the aqueous phase by eliminating hydrogen peroxide as a potential source of hydroxy radicals.

Considerable interest has been expressed in the use of antioxidants incorporated in the diet, and their effects on meat quality (Gray and Pearson, 1984). Dietary supplementation with α-tocopherol improves the oxidative stability of pork (Monaghan *et al.*, 1990) and chicken (Sheehy *et al.*, 1993). Little work has been done on the effect of dietary Se on muscle food quality.

The objectives of the present study were to investigate the effect of dietary α-tocopherol and/or selenium on lipid oxidation in pork.

MATERIALS AND METHODS

Animals and Diet

Forty-eight Landrace X Large White pigs (24 males, 24 females) and averaging 45kg in weight were divided into eight groups of six. Pigs were allocated to receive a barley-based diet or a maize-based diet containing either basal or supplemented levels of vitamin E (30 or 200mg α-tocopherol acetate/kg feed) and basal or supplemented levels of selenium (0.1 or 0.5mg Se/kg feed). The pigs were given feed and water *ad libitum*. The average weight of the pigs at slaughter was 85kg.

Sampling Procedure

Samples were taken from all pigs post-slaughter. Blood samples were taken at the point of slaughter in 10ml

heparinized tubes and the plasma was separated and stored at -20°C until required. Lung, heart and kidney were taken at the point of evisceration. The carcasses were chilled overnight and brain and *longissimus dorsi* samples were removed. All samples were vacuum packaged and stored at -20°C until required.

Analyses

α-tocopherol concentrations were determined by HPLC (Buttriss and Diplock, 1984).

The susceptibility of tissues to lipid oxidation during incubation with iron-ascorbate was measured by a modification of the method of Kornbrust and Mavis (1980). After incubation, thiobarbituric acid-reacting substances (TBARS) were quantified by the method of Beuge and Aust (1978).

Statistical analysis

The statistical significance of the difference between means was determined by the t-test using the Minitab Statistical Package (Ryan *et al.*, 1986).

RESULTS AND DISCUSSION

α-tocopherol concentrations

α-tocopherol concentrations in muscle of pigs fed high levels of α-tocopherol acetate (200mg/kg diet) were significantly higher ($P \geq 0.01$) than those of pigs fed the basal (30mg/kg diet) levels. The results suggest that replacement of barley with maize did not alter the absorption or deposition of α-tocopherol in muscle. In addition, selenium supplementation did not influence the deposition of α-tocopherol. While the responsiveness of different tissues (heart, liver, lung, brain, kidney) and plasma to vitamin E supplementation varied as previously observed (Monahan *et al.*, 1990), selenium did not influence the deposition of α-tocopherol in these tissues (results not shown).

Iron ascorbate induced lipid peroxidation

The time course of lipid peroxidation for muscle and kidney as induced by iron ascorbate are shown in Figure 2. The TBARS at zero, which are indicators of in vivo lipid peroxidation, did not differ significantly between the pigs fed the various diets. However the rate of peroxidation of all tissues fed the high α-tocopherol diets was less than that of tissues fed the basal diet.

In addition, high dietary selenium reduced the rate of peroxidation in muscle, but the selenium effect was not as pronounced as in the other tissues studied (data for kidney presented, Figure 2). Dietary α-tocopherol supplementation significantly increased ($P>0.01$) the stability of muscle and kidney tissue to lipid peroxidation after 15 minutes incubation (Fig. 2). Muscle from pigs fed high Se/low vitamin E was significantly ($P<0.01$) more stable to peroxidation, after 15 minutes incubation, than muscle from pigs fed low Se/low vitamin E. High dietary Se combined with high vitamin E further increased the oxidative stability of muscle. The data in Figure 2 shows that the effects of dietary Se and vitamin E are additive.

Recently, a glutathione peroxidase specific to phospholipid hydroperoxides has been described (Maiorino *et al.*, 1989). In muscle systems, it is possible that this enzyme may prevent depletion of α-tocopherol in muscle membranes by reducing phospholipid hydroperoxides before they can be converted to chain breaking altoxyl radicals by ferrous iron. Further studies are ongoing to define the mechanisms of selenium and vitamin E interaction in muscle and other tissues.

In conclusion, the present results show that the oxidative stability of muscle tissue homogenates is enhanced by vitamin

E and that selenium complements the stabilising effect of vitamin E.

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