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Health Quiz

NADIS Pig Health – November 2008

Vitamin E / Selenium Deficiency

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The mammalian body is in a constant state of turnover with cell replacement occurring as a natural renewal mechanism. Old, dying cells are broken down and reabsorbed into the body but have a tendency to undergo a process called oxidation – the same process that makes butter go rancid – with the production of toxic peroxides or free radicals. These chemicals are highly damaging to other tissues and in the healthy body are avoided by the presence of anti-oxidants usually provided in the diet. There is a wide range of these chemicals involved in metabolism with each animal species having its own priorities. In the pig, the three main groups providing anti-oxidant protection are:

- a. Bio-flavonoids
- b. Vitamin C
- c. Vitamin E and selenium

When deficiency occurs, metabolic pathways are disturbed and damage results. In the pig, Vitamin E and Selenium – working in conjunction – are the primary limiting anti-oxidants and as such it is deficiency of one or both of these which is associated with typical disease problems. In this paper, the effects of deficiency in young growing pigs will be discussed; the role of Vitamin E in sow breeding efficiency is outside the scope.

Clinical Presentation

The most obvious effect of Vitamin E/Selenium deficiency in the pig is sudden death, typically in young fast growing weaners, although it can be seen in piglets still on the sow beyond the 3rd week of life.

Two principle presentations can be seen at post mortem examination:

1. **Mulberry Heart Disease (MHD)**. Here the toxic peroxides target heart muscle leading to myocardial failure. The heart will generally be enlarged with white streaks throughout the muscle. There is usually an accumulation of 'jelly' around the outside of the heart within the pericardium – which must be distinguished from pericarditis. Additionally, the lungs will show signs of right sided heart failure with fluid accumulation and interlobular oedema. (Fig 1.) Affected pigs will often be the best pigs in the group – the fastest growing pigs having the highest requirement for Vitamin E/selenium. It is suggested that MHD is more a manifestation of Vitamin E deficiency than selenium shortage.

2. **Hepatos dietetia**. Here the damage is done to the liver. Whilst standard text book descriptions suggest an enlarged mottled appearance to the liver, most commonly in practice, the liver is enlarged and engorged with blood and has split, resulting in haemorrhage into the abdomen. (Such damage must be

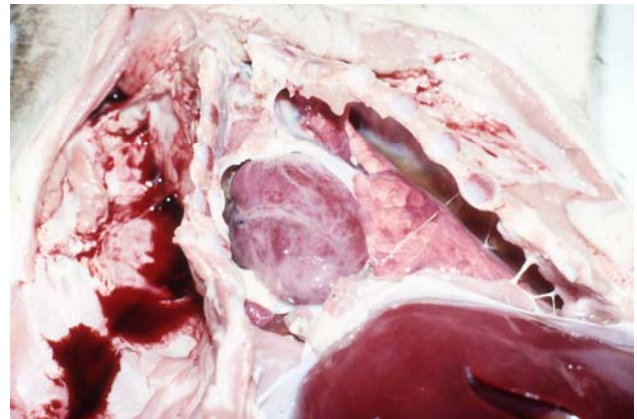


Fig 1: Typical Mulberry Heart Disease showing oedema and streaking of the heart.



Fig 2: Acute Porcine Stress Syndrome can be mitigated by raising anti-oxidant provision; note here the 'just cooked' appearance of affected muscle.

differentiated – particularly in the sucking pig – from trauma as a result of crushing.)

It is quite possible to see both manifestations in the same pig along with some degree of skeletal muscle damage – which is the predominant manifestation of Vitamin E/Selenium deficiency in ruminants. Selenium deficiency may be implicated as the predominant issue where liver damage is the primary presenting sign.

Suspected deficiency of either Vitamin E or selenium can be verified by laboratory testing involving analysis of tissue levels (especially in the liver) and histopathology of affected organs. Furthermore, where a population is suspected of ongoing deficiency, cross-sectional blood sampling for levels of Vitamin E and glutathione peroxidase – the enzyme which contains Selenium – can reveal falling levels over the post-weaning period.

Sources of Vitamin E/Selenium

The transfer of Vitamin E across the placenta to the developing foetus is generally poor (but better for Selenium) with the piglet relying on Vitamin E transfer in fat particles within colostrum. (Transfer via milk is minimal.) The 'dose' of Vitamin E received in this way must last the piglet until such a time as it is eating sufficient food to supplement its resources. Serial blood testing has shown that in modern pig keeping, there can be a dramatic drop in serum Vitamin E levels after weaning and thus it is critical that:

a) Sow colostrum contains the highest possible levels of Vitamin E by ensuring high levels in sow

Notwithstanding the classic presentations of MHD and Hepatosis dietetia, the role of Vitamin E in the functioning of the immune system should not be underestimated. Challenge to the pig by any disease process triggers an immune reaction, which is highly expensive in terms of nutrients and micronutrients. Adequate levels of Vitamin E and Selenium are necessary for such a reaction, but conversely are consumed in the process. Thus, a challenge to the immune system can have a Vitamin E/Selenium depleting effect- which can be demonstrated by blood sampling - exposing the pig either to acute deficiency or a compromised immune system. As vaccination involves challenging the immune system, vaccination of young pigs will only be successful if the pigs have adequate Vitamin E/Selenium but can also act to deplete reserves, particularly where multiple vaccination occurs. (Some vaccines now contain Vitamin E as part of the adjuvant offsetting this risk.)

diet in the month prior to farrowing, possibly supplemented with Vitamin E injections which might be included in vaccines.

b) Every pig receives its full dose of Vitamin E within the first 6 hours of life from colostrum.

c) Following weaning, diets must be adequately supplemented with Vitamin E/Selenium and feed intake must increase rapidly to fulfil requirements.

It should also be mentioned that certain dietary constituents can have a Vitamin E depleting effect – particularly saturated fats and propionic-acid treated corn – whilst wheat offal can act to bind selenium.

Susceptibility

Whilst fast growing pigs are most susceptible to MHD/Hepatitis dietetia, a range of other factors should be considered as possible trigger factors:

1. Excessive disease challenge post weaning.
2. Excessive vaccinal challenge post weaning – ensure, where multiple vaccination is needed that veterinary guidance is followed with respect to separation of different doses.

3. Highly susceptible breed types. Landrace animals are claimed to be more vulnerable but also Halothane gene (stress gene) carriers are known to have a higher anti-oxidant requirement which can be met by Vitamin E supplementation. (This issue is of less significance now that the stress gene has been largely bred out of the UK pig population.) (Fig 2.)

Levels of Vitamin E

This is a highly contentious area with recommended levels frequently found to be inadequate in the field. Typical compound diets nowadays may have the following levels of Vitamin E:

- Sow diets (lactator and dry sow) 100 iu/kg
- Creep diets 250 iu/kg
- Weaner diets 100-150 iu/kg
- Grower and finisher diets 40-100 iu/kg

Selenium levels are normally limited to no more than 0.3 ppm of the diet as high levels of Selenium can be toxic.

Vitamins, in particular, decay in storage and thus, use-by dates and correct storage provision must be observed. This is particularly important for creep diets, which on smaller farms can take time to use, and are frequently stored in warm rooms.

Treatment and Control

Where a problem is identified, additional antioxidants must be provided. Whilst supplementing the diet with additional Vitamin E is the obvious solution, it will take time to work through the system – at least 2 weeks of supplemented feed is needed to restore Vitamin E levels – and the escalating costs of Vitamin E has triggered interest in alternatives. In the acute disease situation, Vitamin C can be successfully supplemented via water for 5-7 days. Alternatively, injectable Vitamin E can be given around weaning. Vitamin C can be included in creep diets in addition to Vitamin E but only where diets are prepared as meal or are

pelleted using cold water – hot pelleting will destroy Vitamin C.

The increasing use of organic acid supplements – especially in young pig diets – can provide additional bio-flavenoids to support the Vitamin E antioxidant system.

Minimising disease challenge, minimising stress and review of vaccination regimes, particularly where multiple vaccination occurs around weaning in the young pig will all have a Vitamin E/Selenium sparing effect.

Costs

Whilst sporadic deaths associated with Vitamin E/Selenium deficiency may be commercially tolerable if growth rates are high, particularly given the escalating costs of supplemental Vit E, there is an obligation on pig keepers to the wellbeing of their charges and to take all reasonable measures to ensure survival. In specific cases of severe problems, post-weaning mortality levels can reach

4% of production associated with acute antioxidant production. This can equate to additional cost per pig produced of £1.50.

Furthermore, where anti-oxidant levels are marginal, triggering secondary infectious disease such as Porcine Respiratory Disease Complex, the cost per pig measured by the mixture of lost growth, mortality and medication can more than double.

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