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Brief Communication

MALIGNANT HYPERTHERMIA IN PIGS MODIFIED BY LOW-POTASSIUM DIETS

In a recent investigation, malignant hyperthermia susceptible pigs fed purified diets, developed a delayed and modified malignant hyperthermia (MH) syndrome during halothane anaesthesia (*Jørgensen* 1982). Compared with the control feed used the purified diets contained different levels of protein and low amounts of potassium and lipids.

Based on these findings a preliminary investigation on the effect of dietary levels of potassium and lipids was carried out on malignant hyperthermia susceptible Danish Landrace pigs. Two pigs were fed a conventional diet containing 5.3 g K/FU_s (feed unite for swine) and 36 g crude fat/FU_s, and six pigs were fed a casein diet containing 1.2 g K/FU_s and 5 g fat/FU_s (*Jørgensen* 1982). Two of these pigs were supplied with KCl, and two pigs with oleum arachidis to raise the daily intake of potassium and lipids to those of the controls. The investigation also comprised two pigs fed a protein-deficient diet. The pigs were subjected to weekly episodes of halothane anaesthesia during which venous blood acid-base balance was measured on a Radiometer BGA3 blood-gas analyzer. Estimation of base-excess was done according to *Siggaard-Andersen* (1963). After a period of four weeks (five weeks in the controls) the animals were killed by

Table 1. M. longissimus dorsi potassium (mmol/kg fresh muscle), average anaesthetic time for development of malignant hyperthermia (min.), and base-excess (mmol/l) in venous blood at 2, 5, and 10 min. after start of halothane anaesthesia.

Pig no.	Diet	K	Duration of anaesthesia	Base-excess, min.		
				2	5	10
711	Control	102.4	4.3	—6.1	—12.4	—16.3
712	Control	108.7	2.0	—11.1	—15.0	—15.8
715	Casein	97.3	11.5	—8.6	—8.8	—10.0
716	Casein	68.5	11.4	—0.3	—3.0	—4.0
717	Casein + KCl	106.1	3.0	—7.4	—14.9	—19.3
718	Casein + KCl	105.0	3.8	—7.2	—12.1	—13.9
719	Casein + lipids	72.8	7.5	—2.3	—3.4	—6.7
720	Casein + lipids	67.2	10.5	+1.8	+0.8	—3.1

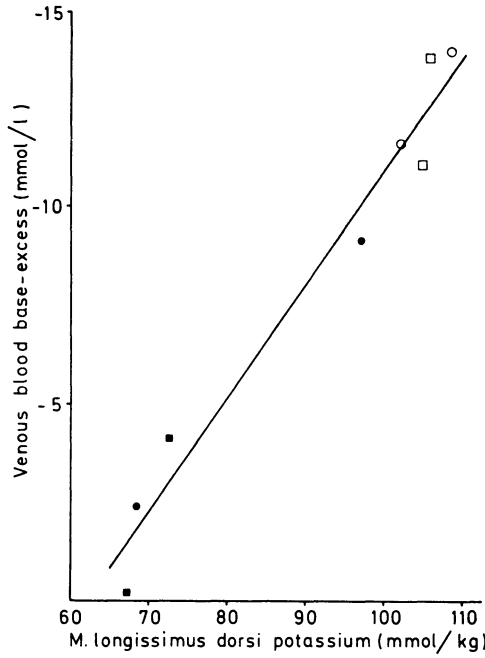

Figure 1. Base-excess (average at 2, 5, and 10 min. after start of anaesthesia) and m. longissimus dorsi potassium in malignant hyperthermia susceptible pigs. Controls (○). Casein diet (●). Casein diet + KCl (□). Casein diet + lipids (■). ○ and □ symbolizes normal, ● and ■ low potassium supply.

bleeding in thiomebumal anaesthesia and m. longissimus dorsi potassium determined according to *Jørgensen* (1981).

The average duration of anaesthesia before appearance of MH during the third and fourth week (third to fifth week in controls) and base-excess in relation to anaesthesia of protein-supplied pigs are given in table 1. M. longissmius dorsi potassium levels are also included in the table. Three of the four pigs fed low potassium diets had low muscle potassium levels, and in all of the four pigs MH was delayed. In pigs fed normal levels of potassium the concentration of potassium in m. longissimus dorsi was comparable to that observed in conventionally fed pigs (*Aitken* 1976, *Jørgensen* 1981). The correlation between base-excess at 2, 5, and 10 min. after anaesthetic start and m. longissimus dorsi potassium is highly significant ($r^2 = 0.88$, 0.95, and 0.89 respectively, $P < 0.001$). Figure 1 further illustrates the relationship between muscle potassium and base-excess (average value at 2, 5, and 10 min.). Low muscle potassium levels, caused

by a low dietary content and a possible increased renal excretion of the potassium element during repeated anaesthetic episodes, are accompanied by a less pronounced metabolic acidosis during halothane anaesthesia.

With respect to anaesthetic time the regression on muscle potassium is less clear ($r^2 = 0.53$, $0.05 < P < 0.1$). Furthermore MH is delayed in protein-deficient animals with normal M. lonsimus dorsi potassium levels (99 mmol/kg), indicating that other factors than potassium depletion may be responsible for a delayed MH.

The specific mechanisms of a modified metabolic acidosis during MH in potassium depleted animals are unclarified. They may in part be secondary to an extracellular metabolic alkalosis in depleted animals (*Welt et al.* 1960), or they may be a primary effect of potassium depletion on cell membrane characteristics and intracellular amino acid metabolism.

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