

## Short communication

# The effect of the malignant hyperthermia gene on carcass characteristics of commercial crossbred pigs in the Western Cape

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Malignant hyperthermia (MH) genotype, as expressed by the halothane genotype, was determined on a random sample of 100 pigs originating from the Western Cape. The pigs were slaughtered to investigate the effect of MH genotype on certain carcass characteristics and meat quality traits. Genotypes were determined by restriction endonuclease assay. No significant differences in back-fat thickness and percentage predicted lean yield were observed between genotypes. However, between genotypes,  $\text{pH}_1$  values differed ( $P < 0.001$ ), with NN being the highest (6.3), nn the lowest (5.4), and the Nn intermediate (5.9). There was a difference ( $P < 0.05$ ) in ham yield (as percentage of carcass mass) between the NN (23.7%) and Nn (24.7%) genotypes. The percentage lean in the ham showed a significant difference ( $P < 0.05$ ) between NN (71.1%) and Nn (72.7%), and between NN and nn genotypes (73.6%).

Die genotipe vir kwaadaardige hipertermie, soos deur die halootaan genotipe tot uitdrukking kom, is op 100 ewekansig gekose varke, afkomstig uit die Weskaap, bepaal. Die varke is geslag om die effek van genotipe op sekere karkaseienskappe en vleiskwaliteitseienskappe te bepaal. Die genotipes is bepaal met behulp van 'n beperkende endonuklease toets. Geen betekenisvolle verskille in rugvettidte en voorspelde persentasie maervleis is waargeneem nie. Die  $\text{pH}_1$  waardes het verskil ( $P < 0.001$ ), met die NN-genotipe die hoogste (6.3), die nn-genotipe die laagste (5.4), en die Nn-genotipe intermedieër (5.9). Die hamopbrengs (as 'n persentasie van karkasmassa) het verskil ( $P < 0.05$ ) tussen die NN- (23.7%) en Nn- (24.7%) genotipes. Die persentasie maervleis in die ham het verskil ( $P < 0.05$ ) tussen die NN- (71.1%) en Nn- (72.7%) genotipes, sowel as tussen die NN- en nn- (73.6%) genotipes.

**Keywords:** Carcass characteristics, genotype, halothane, malignant hyperthermia, meat quality, pigs.

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Malignant hyperthermia is an inherited myopathy which, in pigs, causes pale, soft exudative pork (PSE). A single point mutation

in the porcine gene for the skeletal muscle ryanodine receptor (*ryr1*) was found to be correlated with MH in certain major breeds of lean, heavily muscled pigs (Fujii *et al.*, 1991). Two of the three halothane genotypes are associated with MH: Nn (heterozygous halothane non reactor) and nn (homozygous halothane reactor), while NN (homozygous halothane non reactor) is not associated with MH. Stressful situations such as transportation can trigger MH in nn and possibly Nn pigs resulting in enhanced glycolysis pre- or post-slaughter with a rapid drop in the pH post mortem. With the high energy content of the muscle at sticking, the carcasses of such pigs will develop PSE (Barton-Gade *et al.*, 1988). Certain inhalation anaesthetics, such as gaseous halothane (2-bromo-2-chloro-1,1,1-trifluoroethane), and skeletal muscle relaxants can precipitate MH (Mitchell & Hefron, 1982). An increase in lean yield as well as the assumption that meat quality is not adversely affected by the mutant *ryr1* gene in Nn pigs, prompted studies in mating NN and nn genotypes to produce terminal Nn progeny (Webb & Simpson, 1986). Recent studies however indicate that the Nn pigs tend to produce inferior quality meat with higher drip loss and lower soluble protein (Sather *et al.*, 1991). The object of the present study was to determine the effect of genotype on carcass characteristics.

Over a five-day period, 100 pigs were randomly selected at a Western Cape abattoir. The selected pigs were Landrace x Large White crosses and originated from the Western Cape. Pigs were kept in lairage for 2 h prior to slaughter and no prodders or restrainers were used. Commercial slaughter procedures (Stunning: 90 V AC, ear to ear for 3 – 5 sec; sticking within 30 sec; scalding at 60°C (shower) and mechanical removal of hair) were adhered to and initial carcass measurements were taken 60 min after sticking and exsanguination. Back-fat thickness was determined between the second and third last ribs, 45 mm from the midline, with an Intrascope and the percentage lean calculated (Government Notice No. R. 1748, 26 June 1992). Warm carcass mass and  $\text{pH}_1$  (on the *M. longissimus thoracis et lumborum*) were also determined one hour post mortem. The carcasses were then chilled for 24 h at 2°C whereafter cold carcass mass was determined. Factory specifications and techniques were used to divide the carcasses into commercial joints. The right-hand side back and ham (without shank) were then removed from the carcasses on the processing line. The back was used to determine  $\text{pH}_{24}$  at the same position as  $\text{pH}_1$ . The ham was weighed and expressed as a percentage of the carcass mass. The ham was subsequently separated into meat, fat and bone and the mass of each determined separately. A sample of marrow was extracted from the femur for genotyping using the technique described by Fujii *et al.* (1991). Chilling loss was determined by subtracting the cold carcass mass from the warm carcass mass and was expressed as a percentage of warm carcass mass. The data was analysed using a one way classification analysis of variance and the LSD method was used to test for significant differences (Snedecor & Cochran, 1967). The results are presented in Table 1.

There were no significant differences in the back-fat thickness between the three genotypes although a tendency for the nn pigs to have a lower back-fat thickness (17.4 mm), and the NN pigs a higher value (18.7 mm) was observed. This is consistent with the results of previous studies (Jones *et al.*, 1988), which reported no significant difference between nn, Nn and NN pigs for most of the back-fat thickness measurements. Based on the back-fat

**Table 1** Average values (with standard deviations) of carcass characteristics of different genotypes

	Genotype		
	NN (n = 45)	Nn (n = 48)	nn (n = 7)
Warm carcass mass (kg)	65.6 (±4.31)	66.9 (±5.41)	67.2 (±4.86)
Cold carcass mass (kg)	63.2 (±4.20)	64.3 (±5.28)	64.9 (±4.95)
% Chilling loss	3.7 (±0.83)	3.8 (±1.68)	3.5 (±0.61)
Fat thickness (mm)	18.7 (±3.81)	17.6 (±4.27)	17.4 (±3.40)
% Predicted lean	65.9 (±2.05)	66.5 (±1.92)	67.3 (±1.73)
Ham as % of carcass	23.7 <sup>d</sup> (±1.17)	24.7 <sup>c</sup> (±1.49)	24.0 <sup>d,c</sup> (±1.34)
% Lean in ham	71.1 <sup>d</sup> (±3.33)	72.7 <sup>c</sup> (±3.19)	73.6 <sup>c</sup> (±2.44)
pH <sub>1</sub>	6.3 <sup>a</sup> (±0.31)	5.9 <sup>b</sup> (±0.28)	5.4 <sup>c</sup> (±0.13)
pH <sub>24</sub>	5.5 (±0.19)	5.5 (±0.17)	5.6 (±0.19)
% pH <sub>1</sub> > 6.00	84.6	35.5	0
% pH <sub>24</sub> > 6.00	4.0	0	0

<sup>a-c</sup> Values in the same row with different superscripts differ ( $P < 0.001$ )

<sup>d-f</sup> Values in the same row with different superscripts differ ( $P < 0.05$ )

thickness, the predicted percentage carcass lean yield showed no significant differences, although the nn pigs had the highest predicted lean yield (67.3%). However, it could be that the back-fat measurements do not identify the superior lean yield of the Nn and nn carcasses (Pommier *et al.*, 1992). The Nn pigs had a value (66.5%) intermediate to the nn and NN (65.9%) pigs. Sather *et al.* (1989) reported lean yield, as defined by the Canadian carcass grading system, of Nn carcasses as approximately equal to the average of the NN and nn carcasses with no significant differences between them. This was confirmed by the results of Sather *et al.* (1991) where no differences were found between Nn and NN pigs.

The proportion of ham as a percentage of carcass mass showed a significant difference between the NN (23.7%) and Nn (24.7%) pigs ( $P < 0.05$ ) which is not in agreement with Pommier *et al.* (1992). The incorporation of the mutant *ryr1* gene may therefore have a positive effect on the yield of ham as a percentage of carcass mass. A significant difference ( $P < 0.05$ ) in the percentage lean in the ham was observed between the NN (71.1%) and Nn (72.7%) pigs, and between the NN and nn (73.6%) pigs. The tendency towards a higher percentage lean in the ham in Nn and nn pigs is in agreement with Jones *et al.* (1988). It must be kept in mind that the pigs in this study were randomly selected at an abattoir and did not originate from one farm with controlled management and nutrition and therefore may not have realized full genetic potential.

A significant difference in pH<sub>1</sub> was observed between all the genotypes ( $P < 0.001$ ). The pH<sub>1</sub> values were highest for the NN

(6.3) pigs and lowest for the nn (5.4) pigs which supports the results of Murray *et al.* (1989). The pH<sub>24</sub> values showed no significant differences between genotypes which also supports Murray *et al.* (1989). Only two borderline DFD (pH<sub>24</sub> > 6.0) cases were observed in the NN genotype, which indicates near optimal pre-slaughter conditions (Barton-Gade *et al.*, 1988).

The results in Table 1 suggest that the mutant *ryr1* gene does not hold any real advantage for the producer or the meat processor, except for the higher percentage ham yield. There were no significant differences between the genotypes regarding carcass traits such as back-fat thickness and percentage predicted lean. As these criteria (back-fat thickness and percentage predicted lean) are used in the current classification system to determine the financial return to the producer, there seems to be no advantage in the incorporation of the mutant *ryr1* gene. Previous studies (Murray *et al.*, 1989; Sather *et al.*, 1991) suggest that the presence of the mutant *ryr1* gene in Nn and nn pigs causes a decline in traits such as colour, driploss and pH<sub>1</sub>.

These results suggest that the deliberate incorporation of the mutant *ryr1* gene in pig production systems in the Western Cape may be of little or no benefit to the producer since no significant differences were observed in either back-fat thickness or predicted percentage lean yield. Elimination of the mutant *ryr1* gene may, on the other hand, result in higher pH<sub>1</sub> values (Table 1) with a lower risk of development of the porcine stress syndrome.

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