

# PORCINE MALIGNANT HYPERTHERMIA: NEW INSIGHTS INTO THE PATHOPHYSIOLOGY AS IT MAY RELATE TO MEAT QUALITY

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## INTRODUCTION

The malignant hyperthermia syndrome (MHS) is probably related to the inability of cellulare membranes to handle signal transduction in an appropriate manner.

The identification of MHS in pigs normally involves in-vivo the application of halothane, where only the homozygous animals react as positive. When the in-vitro contraction test for muscle biopsies is applied, also the heterozygous animals will be identified as positive (SEEWALD et al., 1989). It is generally assumed that MHS-positive pigs show a reduced stress resistancy and a higher incidence of pale, soft and exsudative (PSE) meat. Therefore Halothane testing is widely applied in European pig breeding. Measurements of CK and pH1 monitore differences in muscle metabolism, which largely modulates the cellulare permeabilities, and in return modify postmortem meat quality. Due to the discrepancies in MHS-reaction the physiological basis of Halothane testing and the selection for MHS-negative animals has to be reevaluated.

## MATERIAL AND METHODS

18 German Landrace pigs were Halothane tested at 20kg body weight, also the CK activity was enzymatically evaluated in the peripheral blood 8 hours after Neostigmin-stimulation. Under anesthesia samples were removed for immediate MHS contracture tests and also for further biochemical analysis. 1 hour post mortem the intramusculare pH-value

was measured and 24h postmortem the water binding capacity (GRAU/HAMM) was determined. The contents of phospholipids, e.g. Phosphatidylinositol and Phosphatidylserine and Phosphatidylethanolamine, as well as the pattern of their esterified fatty acids were analysed by a combined method including High Performance Liquid Chromatography and Gas Chromatography (SEEWALD and EICHINGER, 1989). Data were analysed by analysis of variance including linear correction for body weighth using the SAS statistical package.

## RESULTS AND DISCUSSION

There were no significant differences in carcass length, muscle conformation or in the weight-corrected heart dimensions between the animal types determined by contracture tests (Tab. 1). MHS-positive animals split into nearly equal groups according to the Halothane tests, but these two sub-groups showed in all physiological and anatomical criteria virtually the same dimensions. The MHS-negative animals came very close to the MHS-positive values, the ham circumference being nearly identical. There was a slight, not significant better score for ham conformation in the positive animals. The anatomical heart parameters are nearly identical for the two groups. The CK values differed as expected in favour of the negative animals. PH values and waterbinding capacity also indicated the poorer meat quality for the MHS-positive group. Crude protein and intramuscular fat content confirmed a leaner carcass for the MHS-positive animals (Tab. 2).

Phosphatidylinositol (PI) is the common origin of two secondary messengers, i.e. inositoltrisphosphate and diacylglycerol. Phosphatidylserin (PS) must be present to couple proteinkinase C to the membrane before it can be activated, and Phosphatidylethanolamin (PE) can intracellularly be transformed to PS. These in cellular activities involved phospholipids were reduced in the heart muscle of MHS-positive animals.

PI-content was also significantly lower in the skeletal muscle of MHS-positive animals (Tab. 3). Arachidonic acid (AA) is an integrated part of most membranal phospholipids where it can modify the structure of the membrane, especially their fluidity, and from where it can also serve with or without hydroxylation in other signalling functions. The AA contents of the PI- and PE-fractions of MHS-positive pigs were significantly reduced in the heart musculature. Likewise, skeletal muscle showed significantly reduced AA contents in the PS- and PE-fractions of muscle from the MHS-positive animals (Tab. 4).

#### CONCLUSION

We conclude that anatomic differences including properties of the heart need not to be very different between the normal and MHS animals within the DL (German Landrace) breed. Our data provide evidence, that MHS-negative animals can show reasonable carcass characteristics, and that MHS-positive animals can have almost the same anatomical heart parameters compared to their MHS-negative counterparts. We also found the usual differences

in post mortem muscle metabolism, but our data provide increasing evidence for the fact, that already small differences in signal transmitting properties of muscle membranes can be associated with extraordinary metabolic reactions and CK-permeability, resulting in postmortem meat quality problems. These data also raise questions as to the reliability of the in-vivo Halothan test.

#### REFERENCES

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- Seewald, M., H. Eichinger, F. Lehmann-Horn, and P. A. Iaizzo (1989): Characterization of Homozygous and Heterozygous Pigs susceptible to Malignant Hyperthermia by in Vivo, in Vitro and Post-Mortem Techniques. *Brit. J. Anesthesiology*, *subm.*

Table 1: Halothane reaction, carcass traits and anatomical criteria of the heart from MHS-negative and MHS-positive pigs (n = 18, weight-corrected)

		MHS (contracture tested)		S.E.
		neg. LSM	pos. LSM	
<u>Halothanreaction</u>				
positive, homozyg.	n=	0	4	-
negative, homozyg.	n=	8	0	-
negative, heterozyg.	n=	0	6	-
<u>Carcass traits</u>				
length	cm	80.3	80.3	2.2
conformation of ham	pts.	1.9	2.3	0.1
circumference of ham	cm	55.2	55.6	1.6
<u>heart parameters</u>				
weight	g	177	176	9
left ventricle	g	110	110	5
right ventricle	g	38	37	2
Atria	g	28	30	2

no significant differences between MHS-positiv and MHS-negativ in all criteria shown above, L.S. Means and S.E.

Table 2: CK-values and postmortem muscle properties (m. semi-membranosus) of MHS-negative and MHS-positive pigs (n = 18)

		MHS (contracture tested)		Diff.
		neg.	pos.	
Creatine kinase (CK)	$\bar{X}$	2.66	3.45	.79**
	SD	0.42	0.44	
<u>muscle properties (m. semim.)</u>				
pH1 (U)	$\bar{X}$	6.73	6.25	.48**
	SD	0.31	0.28	
waterbinding cap. (wet area, cm <sup>2</sup> )	$\bar{X}$	8.8	10.8	2.0**
	SD	1.6	1.5	
crude protein (%)	$\bar{X}$	21.2	22.4	1.2**
	SD	0.5	0.6	
total fat (%)	$\bar{X}$	1.52	1.40	.12
	SD	0.19	0.16	

\* = P < 0.05, \*\* = P < 0.01,  $\bar{X} \pm$  SD

Table 3: Phosphatidylinositol, Phosphatidylserine and Phosphatidylethanolamine content of total lipid extracts of musculus longissimus dorsi and left ventricle of MHS-negative and MHS-positive pigs (n = 18)

		MHS (contracture tested)		Diff.
		neg. %	pos. %	
<u>P-inositol</u>				
m. long. dorsi	$\bar{X}$	11.9	8.9	3.0*
	SD	1.5	2.2	
left ventricle	$\bar{X}$	8.5	6.1	2.4*
	SD	1.2	1.5	
<u>P-serine</u>				
m. long. dorsi	$\bar{X}$	8.0	8.4	0.4
	SD	2.0	3.9	
left ventricle	$\bar{X}$	7.0	4.9	2.1*
	SD	2.0	1.6	
<u>P-ethanolamine</u>				
m. long. dorsi	$\bar{X}$	10.5	11.3	0.8
	SD	3.5	3.4	
left ventricle	$\bar{X}$	18.7	16.3	2.4
	SD	4.3	5.4	

\* = P < 0.05,  $\bar{X} \pm SD$

Table 4: Arachidonic acid (AA) contents of Phosphatidyl-inositol, Phosphatidylserine and Phosphatidylethanolamine in skeletal (m. long. dorsi) and heart (left ventricle) muscle of MHS-negative and MHS-positive pigs

		MHS (contracture tested)		Diff.
		neg. %	pos. %	
<u>P-inositol-AA</u>				
m. long. dorsi	$\bar{X}$	21.2	19.8	1.4
	SD	10.6	9.2	
left ventricle	$\bar{X}$	25.8	16.2	9.6**
	SD	5.2	3.4	
<u>P-serine-AA</u>				
m. long. dorsi	$\bar{X}$	14.5	7.7	6.8*
	SD	2.9	3.8	
left ventricle	$\bar{X}$	11.7	13.2	1.5
	SD	4.1	4.5	
<u>P-ethanolamine-AA</u>				
m. long. dorsi	$\bar{X}$	19.4	15.4	4.0*
	SD	4.3	2.8	
left ventricle	$\bar{X}$	33.0	25.9	7.1**
	SD	4.4	4.6	
* = P < 0.05,    ** = P < 0.01, $\bar{X} \pm SD$				