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Institute of Genetics and Animal Breeding, Jastrzębiec, Poland

The influence of *CAST* and *RYR1* genes polymorphism and their interactions on selected meat quality parametres in four-breed fatteners*

Jolanta Kurył[1], Elżbieta Krzęcio[2], Maria Koćwin-Podsiadła[2], Gabriel Monin[3]

[1] Polish Academy of Sciences, Institute of Genetics and Animal Breeding, Jastrzębiec, 05-552 Wólka Kosowska, Poland

[2] Chair of Pig Breeding and Meat Science, University of Podlasie, 08-110 Siedlce, Prusa 14, Poland

[3] Station de Recherches sur la Viande, INRA, 63122 Saint Genes Champanelle, Theix, France

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Investigation was carried out on 96 [(Polish Large White × Polish Landrace) × (Duroc × Pietrain)] fatteners. The results concerning the influence *CAST* × *RYR1* genes interaction on some meat quality parametres suggest that incidence of faulty, exudative meat appearing in some stress-resistant fatteners (*CC* genotype in relation to *RYR1 locus*) and also normal meat in some stress-sensitive fatteners (*TT* genotype in relation to *RYR1 locus*) may be due to simultaneous modifying effect of genotype in relation to *CAST* gene on *post mortem* changes in muscle tissue.

KEYWORDS: fatteners /gene polymorphism / *CAST* / meat quality / *RYR1*

The quality of porcine meat is important both for meat processing industry, and consumers who purchase so-called culinary-fresh meat. Among factors influencing formation of meat quality traits there are two well-known major genes *RYR1* (*HAL*) and *RN*. Besides, investigations are still carried out on identification and effect(s) of candidate genes [Emnett *et al*. 2000, Malek *et al*. 2001].

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Calpastatin (CAST) is an endogenous inhibitor of calpains (calcium dependent cysteine proteases). Activity of CAST from skeletal muscle is highly related to the rate of meat tenderization and protein turnover after slaughter [Goll *et al.* 1998]. Thus, *CAST* represents an excellent candidate gene for studying variation in pork quality [Ernst *et al*. 1998].

Post mortem proteolysis of myofibrillar proteins is associated with activity of the calpain system (-μ and -m calpain) and their inhibitor calpastatin [Sensky *et al*. 1999, Hao *et al* 2000]. It was documented that calcium channel activity is regulated by domain L of calpastatin [Hao *et al*. 2000]. Ca^{2+} level in skeletal muscle is also regulated by *RYR1* gene. *RYR1* codes subunit of Ca^{2+} release channel of sarcoplasmatic reticulum in skeletal muscles, called ryanodine receptor. The point C1843T mutation in *RYR1* gene is one of the reasons for the disturbed regulation of intracellular Ca^{2+} in pigs skeletal muscle.

The aim of this study was to analyse the effect of polymorphism in *CAST* and *RYR1* genes (identified by digestion of PCR products with restriction endonuclease *Hin*P1 for *RYR1*, and *Hinf*I, *Msp*I and *Rsa*I for *CAST*) on selected meat quality traits and also to determine the respective effect of interaction between *RYR1* and *CAST* gene variants.

Material and methods

Investigations were carried out on a group of 96 [(Polish Large White × Polish Landrace) × (Duroc × Pietrain)] fatteners. The animals were kept under the same environmental conditions and fed balanced diet. Slaughtering was preceded by electrical stunning (INARCO system), and performed 4-5 hours after transportation over a distance of 300 km. Mean hot carcass weight of pigs was 80.50 ±3.02 kg.

The following meat quality traits were determined.

35 min *post mortem* pH of *Longissimus lumborum* (LL) directly in the carcass, after last rib (pH_{35}) and at 45 min (pH_{45}) in water homogenate of muscle tissue and also R_1 (coefficient of energetic changes expressed as IMP/ATP ratio) according to Honikel and Fischer [1977]. At 24 h *post mortem* meat lightness (measured with Minolta CR310 in L*a*b* system), RTN – laboratory yield of curing meat in cooking [Naveau *et al*. 1985], water holding capacity (WHC) according to Grau and Hamm [1952] using Pohja and Niniivaara [1957] modification, and losses of weight of meat in cooking process. Drip loss from muscle tissue at 48, 72 and 96 h *post mortem* was determined according to Prange *et al*. [1977]. Besides, the basic chemical composition of muscle tissue was determined with standard methods. The above mentioned traits were measured in LL samples obtained just after last rib. Moreover, meat lightness and pH_{24} were measured in *Semimembranosus* (SM) muscle.

At 45 min *post mortem* LL samples were collected for determination of glycogen [Dalrymple and Hamm 1973] and lactate [Bergmeyer 1974]. On this basis the gly-

colytic potential (GP) was calculated according to formula proposed by Monin and Sellier [1985].

The *RYR1* genotypes were established according to Fujii *et al*. [1991]. Polymorphism in *CAST* was identified for 87 animals with *Hinf*I, *Msp*I and *Rsa*I enzymes, according to Ernst *et al*. [1998].

Statistical evaluation of results was performed using two-way non-orthogonal analysis of variance. Detailed comparison of means was made with Tukey test (STATISTICA PL 5.1).

Analysing the effect of genes interaction, groups of fatteners with *AA* genotype in relation to *CAST/Hinf*I and *CAST/Msp*I *loci* as well as of *BB* genotype in relation to *CAST*/*Rsa*I *locus* were excluded because of very low number of animals in subgroups differentiated by *RYR1* genotype.

Results and discussion

In population analysed a highly significant effect of *RYR1* gene polymorphism was noted on: pH_{35} and pH_{45}, R_1 value as coefficient of energetic changes in muscle tissue and also on lactate level in LL muscle tissue (P≤0.001). Significant effect of *RYR1* genotype (P≤0.05) was noted on dry matter and water content of muscle tissue (Tab. 1). For dry matter and water content a significant effect (P≤0.05) of polymorphism in *CAST* gene identified with *Hinf*I restriction endonuclease (*CAST/Hinf*I) and close to significant (P=0.051) with *CAST /Rsa*I was also noted. No significant effect of *CAST* gene polymorphism identified with *Msp*I enzyme (*CAST/Msp*I) on analysed meat quality traits was found. It should be stressed that when analysing the influence of *CAST/Hinf*I gene on investigated traits, animals of *AA* genotype were excluded because of their small number (2 individuals only).

Among fatteners analysed a good quality of meat was noted after slaughter. Nevertheless, significant differentiation between means for genotypes *RYR1* appeared for traits that are basic for meat quality immediate post-slaughter diagnosis – pH_{35}, pH_{45} and R_1. Best values of these parameters were found in stress resistant (*CC*) group of fatteners. Mean values of pH_{35}, pH_{45}, R_1 and lactate level in LL muscle of heterozygous animals (*RYR1 CT*) did not differ significantly from means for homozygotes *TT.* Moreover, among *CT* animals the highest water and the lowest dry matter contents of LL muscle were noted (73.50, 72.67 and 72.57% water, in *CT, CC* and *TT* animals, respectively).

In LL tissue of fatteners of *BB* genotype at the *CAST/Hinf*I *locus* the water content was higher than in tissue of *AB* animals at this *locus*. An expected, the inverse tendency was confirmed for dry matter content. Analysing *CAST/Rsa*I *locus*, water content of LL muscle of *AB* was similar to that of *BB* genotype animals.

Significant effects of interaction *RYR1* × *CAST* genes were noted for WHC (*RYR1* × *CAST/Hinf*I; P≤0.001) and drip loss at 48 h *post mortem* (*RYR1* × *CAST/Msp*I; P≤0.001

Table 1. A relationship between polymorphisms in RYR1 and CAST genes and meat quality traits in pigs

Trait	CAST/HinfI genotypes AB (n=[illegible])	CAST/HinfI BB (n=[illegible])	signif.	CAST/MspI genotypes AA (n=[illegible])	AB (n=[illegible])	BB (n=[illegible])	signif.	CAST/RsaI genotypes AA (n=[illegible])	AB (n=[illegible])	BB (n=[illegible])	signif.	RYR1 genotypes CC (n=[illegible])	CT (n=[illegible])	TT (n=[illegible])	signif.	Significance of interaction between RYR1 and CAST/HinfI	CAST/MspI	CAST/RsaI
Lactate [illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.
Glycogen [illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.
Glycolytic potential [illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	[illegible]	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	n.s.	n.s.
[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	[illegible]	[illegible]	[illegible]	n.s.	n.s.	n.s.	n.s.

Table 1 Continued

Item[1]	CAST/HinfI genotype AB (n=49)	CAST/HinfI genotype BB (n=81)	sign.	CAST/MspI genotype AA (n=61)	CAST/MspI genotype AB (n=58)	CAST/MspI genotype BB (n=11)	sign.	CAST/RsaI genotype AA (n=29)	CAST/RsaI genotype AB (n=48)	CAST/RsaI genotype BB (n=41)	sign.	RYR1 genotypes CC (n=58)	RYR1 genotypes CT (n=42)	RYR1 genotypes TT (n=13)	sign.	Significance of interaction between RYR1 and CAST/HinfI	CAST/MspI	CAST/RsaI
Drip loss at 48 h (%)	[illegible]	[illegible]	ns	1.50	[illegible]	[illegible]	ns	4.81	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	ns	0.001	0.01
	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]				
Drip loss at 72 h (%)	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	ns	ns	ns
	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]				
Drip loss at 96 h (%)	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	ns	ns	ns	ns
	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]				
Total protein content (%)	23.41	[illegible]	ns	23.56	[illegible]	[illegible]	ns	[illegible]	[illegible]	23.16	ns	23.44	[illegible]	[illegible]	ns	ns	ns	ns
	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]				
Water content (%)	[illegible]	[illegible]	0.05	[illegible]	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	0.01	[illegible]	[illegible]	[illegible]	0.05	ns	ns	ns
	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]				
Dry matter content (%)	[illegible]	[illegible]	0.05	[illegible]	[illegible]	[illegible]	ns	[illegible]	[illegible]	[illegible]	0.01	[illegible]	[illegible]	[illegible]	0.05	ns	ns	ns
	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]				

[1]Unless otherwise indicated all parameters concern m. longissimus lumborum muscle
[a,b]—Within rows means bearing different superscripts differ significantly: small letters – P ≤ 0.05; capitals – P ≤ 0.01
ns – nonsignificant

and *RYR1* × *CAST/Rsa*I; P≤0.01). Besides, interaction between *RYR1* and *CAST/Msp*I genotypes was close to significance (P=0.055) for ultimate pH (pH_{24}) of LL muscle.

It can be concluded that in population analysed the presence of allele *A* in heterozygotes (*AB*) at *CAST/Hinf*I *locus* is related to highly significant lowering of WHC (P≤0.001) among fatteners with genetically conditioned stress resistance (*CC*) (Fig. 1). Among animals of *CT* and *TT* genotype at *RYR1 locus*, significant differentiation of WHC value between animals of *AB* and *BB* genotypes in relation to *CAST/Hinf*I *locus* was not found.

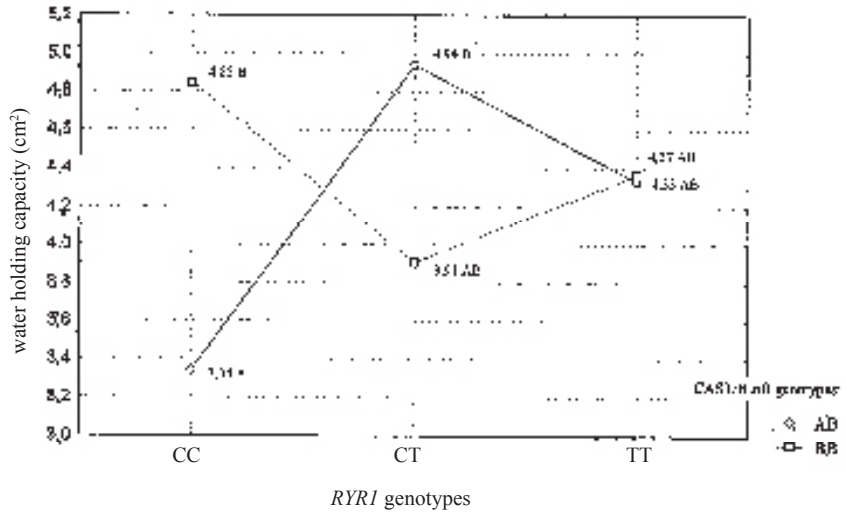

Fig. 1. Interactive effect of *CAST/Hinf*I × *RYR1* genotypes for WHC.

Koćwin-Podsiadła *et al*. [2003] in analogous scheme but on crossbreeds [(Polish Large White × Polish Landrace) × (Hampshire × Pietrain)] showed significant interaction between *CAST/Hinf*I and *RYR1* genotypes for drip loss from LL muscle. In the present study such interaction was not confirmed. Besides, significant for drip loss of LL tissue at 48 h appeared interactions *RYR1* × *CAST/Rsa*I (Fig. 2) as well as *RYR1* × *CAST/Msp*I genotype (Fig. 3).

Differentiation of drip loss between stress resistant (*CC*) animals with different *CAST/Rsa*I genotype (*AA* and *AB*) was noted when analysing *CAST/Rsa*I × *RYR1* interaction. Meat of stress-resistant animals (*CC*) heterozygous at the *CAST/Rsa*I *locus* (*AB*) was characterized by drip loss at 48 h *post mortem* two times lower than that in fatteners of the same *RYR1* genotype and of genotype *AA* at *CAST/Rsa*I l*ocus* (2.74 *vs* 5.12%) – Figure 2.

Interesting for the drip loss at 48 h *post mortem*, interaction was noted between *RYR1* and *CAST/Msp*I *loci*. Data presented on Figure 3 show that drip loss from LL

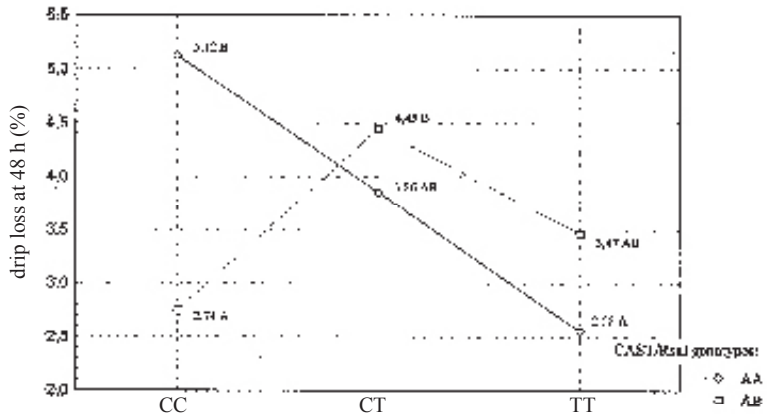

Fig. 2. Interactive effect of *CAST/Rsa*I × *RYR1* genotypes for drip loss at 48 h.

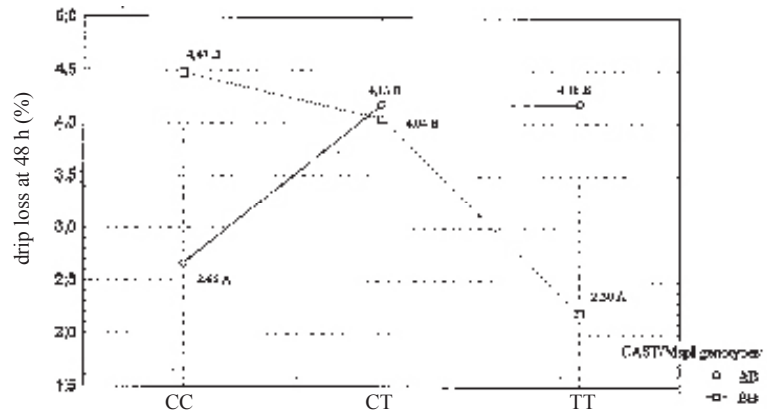

Fig. 3. Interactive effect of *CAST/Msp*I × *RYR1* genotypes for drip loss at 48 h.

muscle tissue 48 h *post mortem* in group of heterozygotes as regards *RYR1 locus* (*CT*) was very close in subgroups differentiated by *CAST/Msp*I genotype.

Drip loss at 48 h *post mortem* in heterozygotes as regards *RYR1 locus* was similar to that obtained in stress-resistant group (*CC*) simultaneously homozygous (*BB*) at *CAST/Msp*I *locus* and also similar to stress-sensitive animals simultaneously *AB* heterozygous at *CAST/Msp*I *locus*.

Summing up relationships presented in Figure 3 one should conclude that the mag-

nitude of drip loss from LL muscle tissue is a result of cumulating effects of genotypes in relation to both *loci* analysed.

Close to significance (P=0,055) was the effect of interaction between *RYR1* and *CAST/Msp*I *loci* for ultimate pH (pH_{24}) of LL muscle (Fig. 4). Fatteners of *AB* genotype at the *CAST/Msp*I *locus* and simultaneously of *CC* genotype as regards *locus RYR1* and also of *TT* and *BB* genotype at the *loci RYR1* and *CAST/Msp*I, respectively,

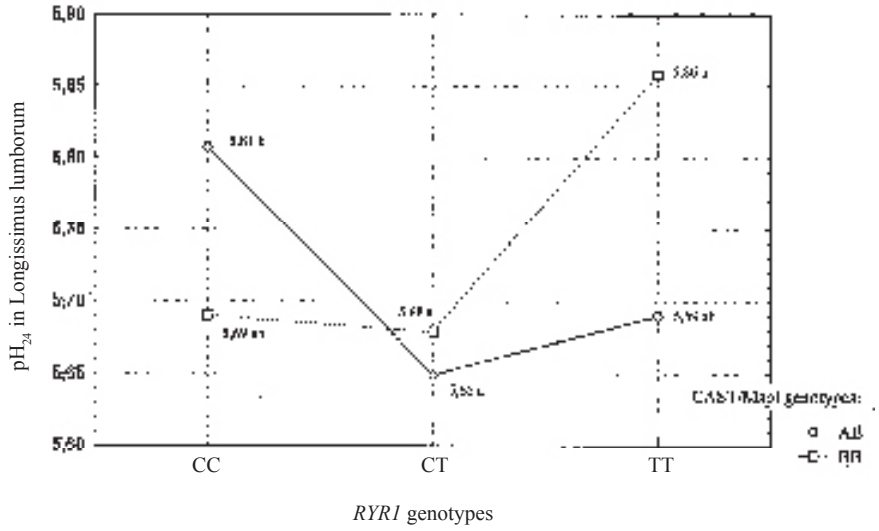

Fig. 4. Interactive effect of *CAST/Msp*I × *RYR1* genotypes for pH_{24} in LL muscle.

showed significant increase in pH_{24} value in comparison to remaining combinations of *loci* analysed. It is known that stress-susceptible animals of *TT* genotype at *locus RYR1* show higher level of Ca^{2+} ions released from cells as a result of malfunction of calcium channels under stress conditions. Next, proteolytic activity of calpain system (calpains and their inhibitor – calpastatin) is significantly dependent on Ca^{2+} ions level. One should suppose that different variants of calpastatin determined by polymorphism in *CAST* gene could have different sensibility to level of Ca^{2+} ions, and the same different activity stopping proteolytic activity of calpain. This may explain the differences in drip loss and pH values noted between fatteners of the same *RYR1* genotype but differentiated by *CAST* genotype. Moreover, it was shown that calcium channel activity might be regulated through domain L of calpastatin [Lee *et al*. 1992]. It is also known that phosphorylase responsible for glycogenolysis is a substrate for calpain [Lametsch *et al*. 2002]. Degradation of glycogen in muscles *post mortem* may run with diverse speed in dependency on activity of these proteases regulated by calpastatin – it may explain influence of *CAST/Msp*I × *RYR1* interaction on pH_{24} value. Claeys *et al*. [2001] found wide differences in the activities of several proteolytic and lypolytic

enzymes in meat samples from two pig lines differing in stress susceptibility. These differences were strongly related to the rate of pH fall. Moreover, it was shown that following an increase in intracellular Ca^{2+}, calpastatin is released from its association, becoming a soluble protein and this change in cell localization may be correlated with the regulation of the overall mechanism of calpain activation [Averna *et al*. 2001]. The polymorphisms in the *CAST* gene genotyped in this study were located in intron 6 and it is difficult to conclude about their effect on calpastatin level or activity. The effect of analysed mutations on meat quality traits might be due to the linkage to any other mutation (causal) within the coding or regulatory regions of the *CAST* gene.

Summing up the results presented here and concerning the influence *CAST* × *RYR1* genes interaction on some meat quality parametres it can be concluded that incidence of faulty exudative meat appearing among some stress resistant fatteners (*CC* genotype in relation to *RYR1 locus*) and also normal meat among those being stress-sensitive (*TT* genotype in relation to *RYR1 locus*) may be a result of combined effect of genotype in relation to *CAST* gene on *post mortem* changes in muscle tissue.

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Jolanta Kurył, Elżbieta Krzęcio,
Maria Koćwin-Podsiadła, Gabriel Monin

Wpływ polimorfizmu genów *CAST* i *RYR1* oraz ich interakcji na wybrane parametry jakości mięsa wieprzowego czterorasowych mieszańców

Streszczenie

Celem badań była ocena istotności wpływu polimorfizmu genów *CAST* i *RYR1* oraz ich interakcji na wartość cech jakości wieprzowiny. Badaniami objęto 96 tuczników – czterorasowych mieszańców [(wielka biała polska × polska zwisłoucha) × (duroc × pietrain)]. Uzyskane wyniki wskazują, że występowanie mięsa o znacznym naturalnym wycieku i niskiej wodochłonności wśród zwierząt odpornych na stres (czyli o genotypie *CC* względem *locus RYR1*), jak również mięsa o prawidłowych parametrach jakości wśród zwierząt wrażliwych na stres (czyli o genotypie *TT* względem *locus RYR1*) może być wynikiem modyfikującego wpływu genotypu *CAST* na zmiany zachodzące w tkance mięśniowej *post mortem*.