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GLYCOGEN METABOLISM IN MUSCLE AND ITS EFFECTS ON MEAT QUALITY IN PIGS – A MINI REVIEW*

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The review is aimed at presenting the state of current knowledge into the structure of a glycogen molecule as well as its synthesis and degradation during *post mortem* changes in muscle tissue.

Currently-accepted model of the structure of glycogen molecule is that suggested by Whelan [Immonen, 2000; & Pösö Puolanne. 2005]. It has been demonstrated that the structure of a molecule is very well adjusted to the functioning of glycogen in muscles through strong packing of glucose particles and that glucose is well available for glycogenolytic enzymes due to strongly branched structure. The review provides results of research at pigs referring to the analysis of a variety of interactions of factors (mainly genetic ones, as HAL^n/RYR^T and RN^- genes) on the activity of enzymes related with synthesis and degradation of glycogen in muscles. Recent results of research into factors affecting the activity of glycogen debranching enzyme have shown that fast chilling of carcass is likely to limit the occurrence of PSE defect. Another aspect described in the review is the effect of residual glycogen on meat quality, whose occurrence has been reported in both pigs and cattle. It has been shown to lower the yield of meat in cooking, to affect a decrease in protein content of muscle tissue and to influence the sensory quality of meat.

INTRODUCTION

Glycogen constitutes 3–5% of liver weight. It is the main source of blood glucose. Muscle glycogen (constituting 0.5–1.5% of muscle weight) is an important source of energy for contraction of a muscle [Bendall, 1973]. Glycogen breakdown plays a major role in changes occurring in muscle tissue *post mortem* and has a considerable effect on meat quality. The paper presents the current knowledge on muscle glycogen synthesis and degradation, and their effects on meat quality in pigs.

STRUCTURE OF A GLYCOGEN MOLECULE

The glycogen molecule has a weight of nine to ten million Da and contains about 55000 glucose residues. The diameter of the molecule is about 40 nm. Glycogen molecule is constructed from two linear glucose chains named A and B. Each chain contains 13 units of glucose bound together with α-1,4-glycosyl bonds. At the fourth and eighth glucosyl units of the B-chain is bound with α 1,6-bond new A-chain and a total number of chains is $1+2^{12}$, *i.e.* about 4100 [Meléndes--Hevia *et al*., 1993; Pösö & Puolanne, 2005] (Figure 1). The structure of the glycogen molecule has been adopted to function as a source of energy that may by quickly accessible by: smallest possible volume (maximally packed glucose molecules) and maximum number of non-reducing ends accessible for the first glycogenolytic enzyme – phosphorylase [Meléndes-Hevia *et al*., 1993; Pösö & Puolanne, 2005]. Adamo & Graham [1998] showed that glycogen molecules

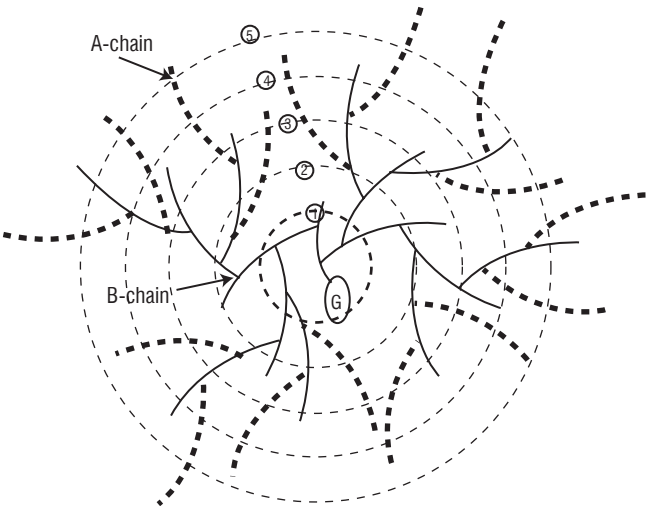

FIGURE 1. The structure of a glycogen molecule as stated in Whelan's model [Immonen, 2000; Pösö & Puolanne, 2005].

exist as two forms referred to as: macro- and proglycogen, respectively. The two forms differ in protein content, size and solubility. Proglycogen contains 10% of protein, is acid insoluble and has a MW of 400000 Da. Macroglycogen has a much higher MW (10^7 Da), contains less than 1% of protein and is acid soluble [Lomako *et al*., 1991; Van Laack *et al*., 2001]. Adamo & Graham [1998] suggested that proglycogen is the precursor of macroglycogen.

The glycogen molecules in muscle are bound with water and enzymes that are responsible for synthesis and degrada-

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tion. One gram of glycogen would bind 2–4 g of water but the binding of water depends on the molecule size [Estrade, 1994]. Each glycogen molecule is bound to glycogenin, a protein that plays an initiating role for starting the synthesis. This protein has a molecular weight of 37300 Da. It catalyses the synthesis of an 8 glucosyl-unit-long primer necessary for the activity of glycogen synthase [Pösö & Puolanne, 2005]. Glycogen synthase (α 1-4 glucano α 4 glucosyl transferase) and branching enzyme (α-1,4-gluco α-1,4 gluco 6-glucosyl transferase) are the two enzymes responsible for the synthesis. The enzymes involved in the hydrolysis of glycogen are phosphorylase and debranching enzyme that catalyse two reactions as transferase and amylo-1,6-glucosidase [Lehninger, 1993]. The mechanism of synthesis and degradation of glycogen is very precise and more and more known but not totally elucidated [Meléndes-Hevia *et al*., 1993; Estrade, 1994; Pösö & Puolanne, 2005; Nelson & Cox, 2005].

CONCENTRATION OF GLYCOGEN IN MUSCLE

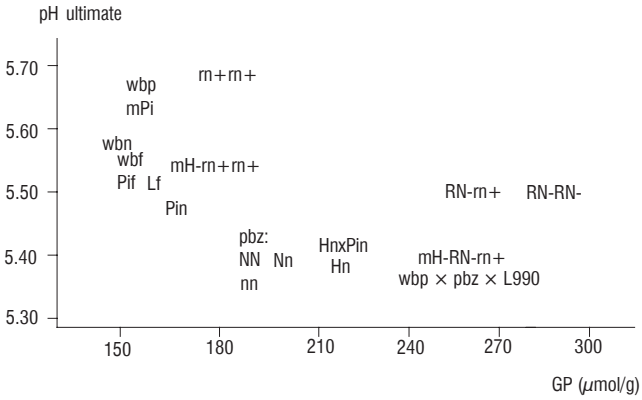
The amount of glycogen in muscle tissue depends on animal species and type of muscle [Przybylski *et al*., 1994; Monin *et al*., 2003; Pösö & Puolanne, 2005] (Table 1).

TABLE 1. Glycolytic potential in different animal species and muscles [Przybylski *et al*., 1994].

Species	Muscle	Glycolytic potential (μmol/g fresh tissue)
Pigs	*Semimembranosus*	116 ± 39
	Semispinalis capitis	54 ± 24
Calves	*Longissimus*	154 ± 53
	Psoas major	133 ± 38
	Rectus abdominis	107 ± 55
Sheep	*Supraspinatus*	102 ± 37
	Pectoralis profundus	102 ± 30
	Semitendinosus	100 ± 33
	Rectus abdominis	70 ± 18

The difficulty in the determination of the level of muscle glycogen in live farm animals lies in the sampling technique. During sampling on live animals, glycogen metabolism is more or less modified depending on the operating conditions. During slaughter the glycogenolysis is strongly activated, hence glycogen values obtained from *post mortem* samples are noticeably lower compared to the actual level before death. Monin & Sellier [1985] proposed an indicator of glycogen level in the living animal, calculated as the sum of the main compounds susceptible to conversion into lactate and referred to as "glycolytic potential" (GP). Glycogen level at slaughter time in the muscle is responsible for ultimate pH value of meat and the relationship between the two traits is not linear as shown by Przybylski *et al*. [1994]. The GP level varies also among pig breeds. Modern pig breeds can be divided into three groups according to the level of glycogen and meat quality (Figure 2). The breeds Large White (or Yorkshire), Piétrain and Duroc can be classified as having a low level of glycogen and good meat quality when they do not carry the $HAL^n/RYR1^T$ gene (very frequent in Piétrains and scarce in both other breeds). Landrace pigs show medium levels of GP with satisfying pork quality when they are not carriers of the $HAL^n/RYR1^T$ gene. Hampshire pigs exhibit frequently an abnormally high level of glycogen (about 70% more than the Large Whites in the white muscles) and give the so-called "acid meat" [Sellier, 1998; Monin & Sellier, 1985; Lundström *et al*., 1996; Przybylski *et at*., 1996]. Acid meat (also referred to as "Hampshire type meat") is mainly characterised by low ultimate pH, reduced water holding capacity, low protein content, and reduced yield of cured cooked ham [Le Roy *et al*., 1995, 1996; Lundström *et al*., 1996; Enfält *et al*., 1997; Josell *et al*., 2003]. Regarding the sensory quality, controversial results were obtained by the French and Swedish researchers: for the first acid meat is tougher while more tender for the latter. The acid meat defect was attributed to a dominant gene called RN^- by Naveau [1986]. Milan *et al*. [2000] identified the mutation associated with the excessive glycogen content in RN^- carrier pigs, *i.e.* R200Q in the *PRKAG3* gene on chromosome 15. This gene encodes a muscle-specific isoform of the regulatory γ subunit of adenosine monophosphate-activated protein kinase (AMPK). Activated AMPK is expected to inhibit glycogen synthesis and stimulate glycogen degradation. The RN^- mutation was found only in the Hampshire breed. Milan *et al*. [2000] described a V199I mutation which was identified in Hampshire, Large white, Landrace, Berkshire and Duroc [Milan *et al*., 2000; Ciobanu *et al*., 2001]. Recently, three functionally significant alleles have been identified at *PRKAG3* (RN) locus: 199V-200R (wild type, rn+), 199V-200Q (RN^-) and 199I-200R denoted rn* by Lindahl *et al*. [2004]. The allele rn* is associated with a lower level of glycogen, lactate and glycolytic potential and with higher *post mortem* ham/loin pH compared with the rn+ allele [Ciobanu *et al*., 2001] (Table 2).

The level of glycogen in muscle may also be modified by feeding and exercise of animals [Fernandez & Tornberg, 1991; Immonen, 2000], and in this way affect meat quality. This was frequently studied in cattle or pigs.



Explanation: wbp-PolishLlarge White; mPi-crossbreed with Pietrain; mH-crossbreed with; pbz-Polish Landrace; NN. Nn. nn – HAL genotypes; RN-rn+. rn+rn+- RN genotypes; wbn – Deutch Large White; Pin – Deutch Pietrain; Hn x Pin -crossbreed; wbf – French Large White; Pif – French Pietrain; Lf – French Landrace; wbp x pbz x L990 – crossbreed with 990 line

FIGURE 2. Glycolytic potential and ultimate pH in *Longissimus dorsi* muscle of various pig genetic types [Przybylski, 2002].

TABLE 2. Least square means and pooled standard errors for carcass traits and meat quality traits of m. *longissimus dorsi* from different RN genotypes [Josell *et al*., 2003].

Traits	Genotypes				
	RN-RN-	RN-rn+	RN-rn*	rn+rn+	rn+rn*
Lean meat (%)	59.4	58.4	58.6	58.0	57.9
pH_1	6.49	6.44	6.47	6.51	6.55
pH_3	6.15^{b}	5.90^{a}	6.12^{b}	6.18b	6.15^{b}
pH_{24}	5.46^{ab}	5.41^{a}	5.44^{ac}	5.50^{bc}	5.52^{b}
pH_{48}	5.35^{a}	5.35^{a}	5.35^{a}	5.38^{ab}	5.45^{b}
Glycogen (μmol/g dry weight)	297.0^{ac}	310.8^{a}	243.6^{c}	80.0^{b}	68.4^{b}
Protein (%)	21.0^{a}	21.6^{a}	21.4^{a}	22.9^{b}	22.7^{b}
Water (%)	76.4^{a}	76.0^{a}	76.4^{a}	75.3^{b}	75.3^{b}
IMF (%)	0.83	0.96	0.86	0.98	0.98
Shear force (N/cm^2)	65.3^{ab}	61.1^{a}	67.2^{ab}	77.6^{b}	75.8^{b}
Cooking loss (%)	30.7^{a}	30.2^{a}	30.6^{a}	26.2^{b}	26.5^{b}

Means in a row with different letters are significantly different $p<0.05$

EFFECT OF GLYCOGENOLYSIS ON MEAT QUALITY

A significant effect of *post mortem* glycogenolysis has been shown on meat quality. After the death of an animal when oxygen is no longer available, the *post mortem* breakdown of muscle glycogen yields lactic acid, the accumulation of which contributes to the change in meat pH. Two enzymes are responsible for degradation of glycogen: glycogen phosphorylase and debranching enzyme. The first one exists in two forms, active phosphorylase a or inactive phosphorylase b [Immonen, 2000]. For the action of this enzyme only 34.6% of total glycogen molecule is available since the limit for its action is about 4 glucose residues and next the glycogen must by degraded by the debranching enzyme [Pösö & Puolanne, 2005]. Glycogenolysis can be accelerated by hormonal mechanisms (during stress) and allosteric activation of phosphorylase by calcium ions and/or by AMP [review by Pösö & Puolanne, 2005]. Stress before slaughter (transportation and manipulation) or adrenalin injection deplete glycogen level before slaughter and increase the ultimate pH [Monin, 2003; Fernandez *et al*., 1992]. A rapid fall of pH as an effect of accelerated glycogenolysis and ATP breakdown gives a PSE defect. Mainly the stress-susceptibility gene is responsible for this defect. This acceleration of glycogenolysis in stress-susceptible pigs has been shown in ample studies [*e.g.* Monin & Sellier, 1985; Lundström *et al*. 1989; Kocwin-Podsiadła *et al*., 1995, 2001]. At 45 min after the slaughter, the stress susceptible pigs or heterozygotes are characterised by a lower glycogen content and a higher lactate level. Monin *et al*. [1986] showed a markedly higher activation of phosphorylase expressed as the ratio of phosphorylase a over a +b in stress susceptible Pietrain pigs in comparisons to Large White and Hampshire pigs in muscle 1 h after slaughter. However Fernandez *et al*. [2002], comparing the 3 genotypes at the HAL locus (NN normal, nn stress-susceptible, and Nn), demonstrated a higher activity of phosphorylase a in biopsy muscle samples from NN (stress-resistant) pigs whereas the a/a+b ratio did not differ significantly between the three genotypes (Table 3). This apparent discrepancy can be explained by the fact that sarcoplasmic free calcium concentration increases much faster *post mortem* in stress-susceptible pigs than in normal pigs [Cheah *et al*., 1993, 1995], which is likely to induce the change of phosphorylase b to a. Other studies by Klont *et al*. [1993] and Kocwin-Podsiadła *et al*. [2001] showed that the consumption of glycogen in stimulated muscles before slaughter is similar in all three genotypes at the HAL locus (Table 4). Kocwin-Podsiadła *et al*. [2001] and Fernandez *et al*. [2002] reported that the consumption of glycogen (expressed as GP) during pre slaughter holding is similar in NN and Nn pigs and reaches *ca*. 42-43 μmol/g and 36–38 μmol/g, respectively, but it is higher in the nn pigs (*ca*. 60 μmol/g).

TABLE 3. Effect of halothane genotype on the activities of glycogen phosphorylase determined in biopsy samples and glycogen at different times before and after slaughter in the muscle *Longissimus lumborum* [Fernandez *et al*., 2002].

Enzyme	Genotype		
	NN	Nn	nn
Phosphorylase a	14.5 ± 1.5^{a}	11.1 ± 1.0^{ab}	9.6 ± 1.1^{b}
Phosphorylase a + b	29.3 ± 1.3	28.6 ± 1.6	24.5 ± 2.9
Ratio a / a + b (%)	0.51 ± 0.06	0.40 ± 0.04	0.45 ± 0.05
Four days before slaughter:			
Glycogen (μmol/g)	90.3 ± 2.5	87.4 ± 2.6	83.0 ± 2.8
Lactate (μmol/g)	5.1 ± 0.8^{a}	9.2 ± 0.5^{b}	13.8 ± 0.5^{c}
Glycolytic potential (μmol/g)	186 ± 5	184 ± 5	180 ± 6
Immediately before stunning:			
Glycogen (μmol/g)	62.2 ± 4.5^{a}	69.6 ± 3.1^{a}	52.2 ± 4.7^{b}
Lactate (μmol/g)	8.0 ± 1.0^{a}	8.9 ± 0.8^{a}	15.7 ± 0.9^{b}
Glycolytic potential (μmol/g)	133 ± 9	148 ± 7	120 ± 10
45 min *post mortem*:			
Glycogen (μmol/g)	54.7 ± 5.2a	46.8 ± 4.0^{a}	26.7 ± 3.1^{b}
Lactate (μmol/g)	34.3 ± 4.4^{a}	52.0 ± 4.7^{b}	83.3 ± 4.7^{c}
Glycolytic potential (μmol/g)	144 ± 10	146 ± 6	120 ± 10

a, b – different letters indicate significant differences between means at $p<0.05$

When phosphorylase a cleaves the glucosyl units to the level of four units from the 1,6-branching point, the bifunctional glycogen debranching enzyme transfers maltotriosyl groups from the 1,6-branching point (transferase function) and then cleaves the remaining 1,6-glucosyl unit as free glucose (1,6-glucosidase function) [Pösö & Puolanne, 2005]. Kylä-Puhju *et al*. [2005] showed that the activity of this enzyme is not very dependent on pH range of 5.5–7 but it is sensitive to temperature. *i.e.* it is close to zero at a temperature below 15°C. They showed also that in red muscle (*Masseter*) a decrease in the activity is faster than in the white muscle (*Longissimus dorsi*) (Figure 3). This enables explaining the positive effect of fast chilling on reduction of PSE in pork [Pösö & Puolanne, 2005].

TABLE 4. The comparison of RYR1(HAL) genotypes for biopsy parameters and meat quality traits [Koćwin-Podsiadła *et al*., 2001].

Traits	Genotype	
	CC(NN)	CT(Nn)
Glycolytic potential-biopsy (μmol/g)	182.82 ± 52.40	178.31 ± 43.74
Lactate-biopsy (μmol/g)	8.90 ± 3.91[a]	13.64 ± 4.23[b]
Glycolytic potential-45 *post mortem* (μmol/g)	139.95 ± 29.02	142.85 ± 23.74
Lactate-45 *post mortem* (μmol/g)	54.50 ± 19.71[a]	68.95 ± 16.14[b]
pH_1	6.32 ± 0.31[a]	6.03 ± 0.25[b]
pH_{24}	5.54 ± 0.14	5.52 ± 0.07
R1 (IMP/ATP)	0.94 ± 0.10[a]	1.02 ± 0.11[b]
Meat lightness	17.05 ± 2.13	17.77 ± 2.40
WHC (cm^2/g)	5.51 ± 1.28	5.74 ± 1.40
% of PSE meat with pH_1 <5.8	10	25

Means with different letters differ significantly at $p<0.05$

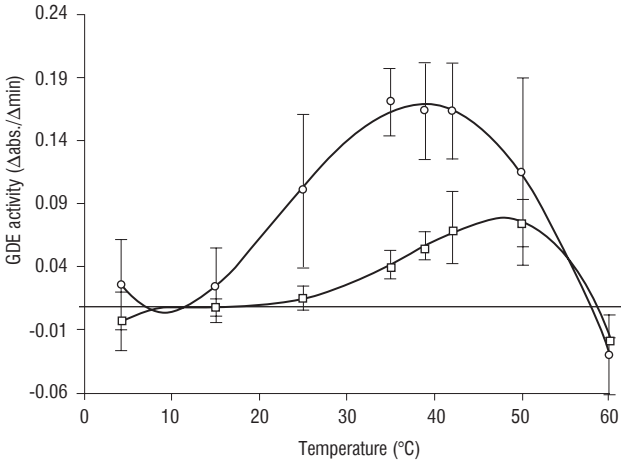


FIGURE 3. The activity of glycogen debranching enzyme in porcine *Longissimus dorsi* and Masseter muscles in relation to temperature [Kylä-Puhju *et al*., 2005].

EFFECT OF RESIDUAL GLYCOGEN ON MEAT QUALITY

The ultimate pH depends on glycogen concentration at time of slaughter, and the relationship between these two traits is not linear. According to Przybylski *et al*. [1994], the relationship between ultimate pH and glycolytic potential at slaughter is best described in most muscles by a quadratic segmented model with a plateau. The ultimate pH decreases following a curvilinear regression when glycolytic potential increases, until a plateau value, dependent on the animal species and muscle. The glycolytic potential corresponding to the convergence point between the quadratic part of the curve and the plateau depends also on muscle and animal species. This implies that when the glycolytic potential at slaughter is higher than the value at the convergence point, the ultimate pH remains constant and some glycogen (referred to as "residual glycogen") is not degraded. The residual glycogen has been found by many authors in pigs [Estrade *et al*., 1992; Fernandez *et al*., 1991; Przybylski, 2002] and in cattle [Immonen & Puolanne, 2000; Immonen *et al*., 2000]. Fernandez *et al*. [1991] and Przybylski [2002] reported residual glycogen values of 0 to 78 μmol/g fresh tissue and 0 to 50 μmol/g fresh tissue, respectively. Enfält *et al*. [1997] observed residual glycogen contents of 33 μmol/g in rn+ Hampshire pigs, 63 μmol/g in RN- Hampshire pigs, 20 μmol/g for Yorkshire pigs and 27 μmol/g for Landrace pigs (Yorkshire and Landrace being presumably rn+). Using rat muscle Monin *et al*. [1987] demonstrated that the level of residual glycogen could influence directly the technological yield of cured-cooked meat processing independently of its effect on ultimate pH. They suggested that the water bound by glycogen molecules was likely to be released during *post mortem* glycogenolysis as well as during the processing of the meat. This suggestion was supported by the results of Fernandez *et al*. [1991] and Przybylski [2002] who found a negative relationship between residual glycogen and technological yield in cured-cooked pork. Moreover, Monin *et al*. [1992], Enfält *et al*. [1997] and Przybylski [2002] found more water and less protein in pig muscle containing the residual glycogen.

CONCLUSIONS

Ample studies demonstrated that glycogen breakdown plays a major role in changes proceeding in muscle tissue *post mortem* and has a tangible effect on meat quality. The amount of glycogen in muscle tissue depends on animal species, breed within a given species, and muscle type. In pigs, the RN^- gene is responsible for the accumulation of glycogen in white muscles by about more than 70% in comparison to normal pigs. Meat from pigs carrying the RN^- gene is characterised by low ultimate pH, low protein content, reduced water holding capacity and reduced yield of cured cooked ham. The glycogen level in muscle may also be modified by feeding and exercise. It has been shown that stress or adrenalin injection before slaughter deplete glycogen level and increases the ultimate pH.

A rapid fall of pH as an effect of accelerated glycogenolysis and ATP breakdown gives rise to the PSE defect. The stress susceptibility gene (HAL/RYR1) is mainly responsible for this defect . The well-known positive effect of fast chilling, which decreases PSE frequency, on pork quality can be partly explained by the strong effect of temperature on the debranching enzyme activity.

Relationships between residual glycogen, water and protein contents and technological yield of meat processing have been demonstrated, but they still need to be investigated.

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REFERENCES

1. Adamo K.B., Graham T.E., Comparison of traditional measurements with macroglycogen and proglycogen analysis of muscle glycogen. J. Appl. Physiol., 1998, 84, 908–913.
2. Bendall J.R., *Post mortem* changes in muscle. 1973, *in*: Structure and Function of Muscle (ed. G.H. Bourne). Academic Press, New York, pp. 243–309.

3. Cheah K.S., Cheah A.M., Krausgrill D.I., Variations in meat quality in live halothane heterozygotes identified by biopsy samples of m. *longissimus dorsi*. Meat Sci., 1995, 39, 293–300.
4. Cheah K.S., Cheah A.M., Lahucky R., Mojto J., Kovac L., Prediction of meat quality in live pigs using stress-susceptible and stress-resistant animals. Meat Sci., 1993, 34, 179–189.
5. Ciobanu D., Bastiaansen J., Malek M., Helm J., Woollard J., Plastow G., Rothschild M., Evidence for new alleles in the protein kinase adenosine monophosphate-activated γ_3-subunit gene associated with low glycogen content in pig skeletal muscle and improved meat quality. Genetics, 2001, 159, 1151–1162.
6. Enfält A.C., Lundström K., Karlsson A., Hansson I., Estimated frequency of the RN^- allele in Swedish Hampshire pigs and comparison of glycolytic potential, carcass composition, and technological meat quality among Swedish Hampshire, Landrace, and Yorkshire pigs. J. Anim. Sci., 1997, 75, 2924–2935.
7. Estrade M., Bazin C., Monin G., Vignon X., Influence du gène RN- sur la localisation intracellulaire et l'évolution post mortem du glycogene dans le muscle de porc. Journées Rech. Porcine en France, 1992, 24, 71–76.
8. Estrade M., Etude de l'expression métabolique du gène RN^-. 1994, Thèse de Doctorat, Université Blaise Pascal, Clermont-Ferrand, p. 132.
9. Fernandez X., Forslid A., Mågård M., Möller B.M., Tornberg E., Effect of time between adrenaline injection and slaughter on rate and extend of *post-mortem* metabolism in porcine skeletal muscle. Meat Sci., 1992, 31, 287–298.
10. Fernandez X., Lefaucheur L., Guéblez R., Monin G., Paris ham processing: Technological yield as affected by residual glycogen content of muscle. Meat Sci., 1991, 29, 121–128.
11. Fernandez X., Neyraud E., Astruc T., Sante V., Effects of halothane genotype and pre-slaughter treatment on pig meat quality. Part 1. *Post mortem* metabolism, meat quality indicators and sensory traits of m. *longissimus lumborum*. Meat Sci., 2002, 62, 429–437.
12. Fernandez X., Tornberg E., A review of the causes of variation in muscle glycogen content and ultimate pH in pigs. J. Muscle Foods, 1991, 2, 209–235.
13. Immonen K., Bovine muscle glycogen concentration in relation to diet, slaughter and ultimate beef quality. 2000, PhD Dissertation. University of Helsinki, p. 64.
14. Immonen K., Puolanne E., Variation in residual glycogen-glucose concentration at ultimate pH values below 5.75. Meat Sci., 2000, 55, 279–283.
15. Immonen K., Ruusunen M., Puolanne E., Some effects of residual glycogen concentration on the physical and sensory quality of normal beef. Meat Sci., 2000, 55, 33–38.
16. Josell A., Enfält A.C., Von Seth G., Lindahl G., Hedebro-Velander I., Anderson L., Lundström K., The influence of RN genotype, including the new V199I allele, on the eating quality of pork loin. Meat Sci., 2003, 65, 1341–1351.
17. Klont R.E., Lamboy E., Van Logtestijn J.G., Effect of preslaughter anesthesia on muscle metabolism and meat quality of pigs of different halothane genotypes. J. Anim. Sci., 1993, 71, 1477–1485.
18. Koćwin-Podsiadła M., Przybylski W., Fernandez X., Monin G., The comparison between $RYR1^CRYR1^C$ and $RYR1^CRYR1^T$ pigs for meat quality and glycolytic potential measured before and after slaughter. Ann. Anim. Sci., 2001, 2, 31–36.
19. Koćwin-Podsiadła M., Przybylski W., Kurył J., Talmant A., Monin G., Muscle glycogen level and meat quality in pigs of different halothane genotypes. Meat Sci., 1995, 40, 121–125.
20. Kylä-Puhju M., Ruusunen M., Puolanne E., Activity of porcine muscle glycogen debranching enzyme in relation to pH and temperature. Meat Sci., 2005, 69, 143–149.
21. Le Roy P., Caritez J.C., Billon Y., Elsen J.M., Talmant A., Vernin P., Lagant H., Larzul K., Monin G., Sellier P., Etude de l'effet du locus RN sur les caractères de croissance et de carcasse. Premier résultats. Journees Rech. Porcine en France, 1995, 27, 165–170.
22. Le Roy P., Juin H., Caritez J.C., Billon Y., Lagant H., Elsen J.M., Sellier P., Effet du génotype RN sur le qualités sensorielles de la viande de porc. J. Rech. Porcine en France, 1996, 28, 53–56.
23. Lehninger A.L., Nelson D.L., Cox M.M., Principles of Biochemistry. 2nd edit. 1993, Worth Publishers, USA, p. 1013.
24. Lindahl G., Enfält A.Ch., Von Seth G., Josel Å. Hedebro-Velander I., Andersen J.H., Braunschweig M., Anderson L., Lundström K., A second mutant allele (V199I) at the *PRKAG3 (RN)* locus-I. Effect on technological meat quality of pork loin. Meat Sci., 2004, 66, 609–619.
25. Lomako J., Lomako W.M., Whelan W.J., Proglycogen: a low-molecular-weight form of muscle glycogen. FEBS Lett., 1991, 279, 223–228.
26. Lundström K., Anderson A., Hansson I., Effect of the RN gene on technological and sensory meat quality in crossbred pigs with Hampshire as terminal sire. Meat Sci., 1996, 42, 145–153.
27. Lundström K., Essén-Gustavsson B., Rundgren M., Edfors-Lilja I., Malmfors G., Effect of halothane genotype on muscle metabolism at slaughter and its relationship with meat quality: a within-litter comparison. Meat Sci., 1989, 25, 251–263.
28. Meléndes-Hevia E., Waddel T.G., Shelton E.D., Optimization of molecular design in the evolution of metabolism: the glycogen molecule. Biochem. J., 1993, 295, 477–483.
29. Milan D., Jeon J.T., Looft C., Amarger V., Robic A., Thelander M., Rogel-Gaillard C., Paul S., Iannuccelli N., Rask L., Ronne H., Lundström K., Reinsch N., Gellin J., Kalm E., Le Roy P., Chardon P., Andersson L., A Mutation in PRKAG3 associated with excess glycogen content in pig skeletal muscle. Science, 2000, 288, 1248–1251.
30. Monin G., Abattage des porcs et qualités des carcasses et des viandes. INRA Prod. Anim., 2003, 16, 251-262.
31. Monin G., Brard C., Vernin P., Naveau J., Effect of the RN^- gene on some traits of muscle and liver in pigs. 1992, 38th ICoMST Clermont-Ferrand, France, pp. 391–394.
32. Monin G., Przybylski W., Koćwin-Podsiadła M., Glycolytic potential as meat quality determinant. Anim. Sci. Pap. Rep., 2003, 21, 109–120.
33. Monin G., Sellier P., Pork of low technological quality with a normal rate of muscle pH fall in the immedi-

ate *post-mortem* period: The case of the hampshire breed. Meat Sci., 1985, 13, 49–63.
34. Monin G., Talmant A., Laborde D., Zabari M., Sellier P., Compositional and enzymatic characteristics of the *Longissimus dorsi* muscle from Large White, halothane-positive and halothane-negative Piétrain, and Hampshire pigs. Meat Sci., 1986, 16, 307–316.
35. Monin G., Talmant A., Valin C., A possible relation between muscle residual glycogen and yield of meat processing by curing and cooking. 1987, *in*: Proc. 33rd Int. Congr. Meat Sci. Technol., Helsinki, pp. 6–21.
36. Naveau J., Contribution l'etude du déterminisme génétique de la qualite de viande porcine. Héritabilité du Rendement Technologique Napole. Journées Rech. Porcine en France, 1986, 18, 265–276.
37. Nelson D.L., Cox M.M, Lehninger Principles of Biochemistry. 2005, Worth Publishers, New York, p. 1152.
38. Pösö A.R., Puolanne E., Carbohydrate metabolism in meat animals. Meat Sci., 2005, 70, 423–434.
39. Przybylski W., Koćwin-Podsiadła M., Kaczorek S., Krzęcio E., Naveau J., Monin G., Effect of the RN^- gene on meat quality traits in pigs originated from crossing of Large white breed, Polish Landrace 23 with P-76 line and Hampshire boars. Przegl. Hodowl., 1996, Zesz.y Nauk., 26, PTZ, 143–149 (in Polish).
40. Przybylski W., The application of glycolytic potential of *Longissimus dorsi* muscle in research on conditioning of selected pork quality traits. 2002, Habilitation Thesis, Agricultural University, Warszawa, p. 84 (in Polish).
41. Przybylski W., Vernin P., Monin G., Relationship between glycolytic potential and ultimate pH in bovine, porcine and ovine muscles. J. Muscle Foods, 1994, 5, 245–255.
42. Sellier P., Genetics of meat and carcass traits. 1988, *in*: The Genetics of the Pig (eds. M.F. Rotshild, A. Ruvinsky). CAB Int., Wallingford, UK, pp. 463–510.
43. Van Laack R.L.J.M., Kauffman R.G., Greaser M.L., Determinants of ultimate pH of meat. 2001, *in:* Proceed. 47th ICoMST, Kraków, Poland, Session 3, L1, 22–26.

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METABOLIZM GLIKOGENU MIĘŚNIOWEGO I JEGO WPŁYW NA JAKOŚĆ MIĘSA WIEPRZOWEGO

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Celem pracy było przedstawienie aktualnego stanu wiedzy w zakresie budowy cząsteczki glikogenu oraz jego syntezy i rozkładu podczas przemian zachodzących w tkance mięśniowej *post mortem*.

Obecnie przyjmuje się model budowy cząsteczki glikogenu zaproponowany przez Whelan [Immonen, 2000; Pösö & Puolanne. 2005]. Jak wykazano budowa granuli jest bardzo dobrze dostosowana do funkcji jaką glikogen spełnia w mięśniach poprzez silne upakowanie cząsteczek glukozy a jednocześnie dzięki silnie rozgałęzionej strukturze jest dobrze dostępna dla enzymów glikogenolitycznych. Podano wyniki badań u świń związane z analizą oddziaływania różnorodnych czynników (przede wszystkim genetycznych – gen HAL^n/RYR^T i RN^-) na aktywność enzymów związanych z syntezą i degradacją oraz ilością glikogenu w mięśniach. Najnowsze wyniki badań nad czynnikami warunkującymi aktywność enzymu usuwającego rozgałęzienia w cząsteczce glikogenu wykazują, iż szybkie wychładzanie półtusz ma swoje uzasadnienie w możliwości ograniczenia występowania wady PSE. Opisano również oddziaływanie tzw. glikogenu resztkowego (residual – nie rozłożonego podczas przemian zachodzących w tkance mięśniowej *post mortem)*. Jego występowanie stwierdza się zarówno u świń jak i u bydła. Stwierdzono, iż glikogen resztkowy obniża wydajność mięsa w gotowaniu, wpływa na zmniejszenie zawartości białka w tkance mięśniowej oraz oddziaływuje na cechy sensoryczne mięsa.