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Characteristic of selected genes controlling meat quality in pigs. A review*

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ABSTRACT

For many years, pig breeders have concentrated their pig improvement efforts on producing a population characterized by adequate meatiness. Development of molecular genetics techniques resulted in the detection of genes with major effects in quantitative traits. Segregation analysis of numerous microsatellite markers, evenly distributed throughout the genome, and variation of a productive trait in the reference family, may help to identify the chromosome region in which the major gene locus probably occurs. This analysis has led to the identification in pigs of over 2 262 DNA microsatellite markers, and 1 381 genes, amoung others *FAT1, RYR1, RN-, LEP, LEPR GH IGFs, MyoD* family genes presented in this articile which are significantly correlated to meat quality, carcass quality or growth rate.

KEY WORDS: pigs, genes, QTLs, meatiness, carcass quality

INTRODUCTION

Early breeding efforts at genetic improvement of pigs were based on selection for traits that could be determined on the basis of phenotype or reproductive value of animals. The main emphasis in pig improvement was placed on body muscling or fatness, and on the number of piglets per litter and sow. The next stage of breeding work was to search for traits responsible for animal performance.

Methods used until today were the basis of breeding work whose aim was to improve the genetic value of pigs. This work was carried out with large

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populations of animals. Genetic merit of animals was analysed by comparing their phenotypic traits with mean values of their contemporaries in individual herds (Różycki, 2001).

An important stage in pig breeding was the introduction of artificial insemination, which enabled better use of the genetic potential of herds or populations being improved. It also made possible the application of the BLUP (Best Linear Unbiased Prediction) - Animal Model program, which enables more accurate analysis of the genetic value of individual animals based on larger data sets that also account for traits of low heritability.

However, this type of genetic improvement of animals has reached a ceiling, making it necessary to supplement the analysis of pig breeding value with studies on molecular structure of genes whose expression is associated with the acquisition of specified levels of productive traits.

In the early 1990s in the European Union and Poland alike, special research projects (PiGMaP) were launched in order to gather as much information on the pig genome as possible (Archibald et al., 1991; Komisarek et al., 1998; Korwin-Kossakowska et al., 1998). The aim of these projects was also to make gene maps that pinpoint the structure (DNA nucleotide sequences) and, more importantly, the location of genes on specific chromosomes. Many genes and DNA sequences have been mapped and sequenced in pigs until today. The information obtained from brief summary of the Pig Genome Coordination Program for 2003 (http://www.genome.iastate.edu/newsletter/update2003.html) showed that in total, there were over 1 381 genes and 2 262 DNA markers in the database. The physical map is also growing quickly and there are now nearly 1 315 genes and anonymous markers.

Molecular genetics techniques resulted in the detection of genes with major effects in quantitative traits (QTL). Segregation analysis of numerous microsatellite markers, evenly distributed throughout the genome, and variation of a productive trait in the reference family, may help to identify the chromosome region in which the major gene locus probably occurs. This analysis has led to the identification in pigs of many loci (such as *GH*, *RYR1* or *RN*$^{-}$) localized on different chromosomes, which are significantly correlated with meat quality, carcass quality, fatness or growth rate (Andersson et al., 1994; Wilkie et al., 1996; Miller et al., 2000).

FAT1 GENE

The first major gene of a quantitative trait (*FAT1*) for fatness and growth in pigs was localized on chromosome 4 by using a wild boar intercross (Andersson et al., 1994). This gene region on chromosome 4 in the pig is homologous to parts

of human chromosome 1 and 8. The latest comparative mapping results between humans and pigs indicate that the QTL is located in a region homologous to HSA1q (q arm of human chromosome 1 pair) (Berg et al., 2002).

RYANODINE RECEPTOR (*RYR1*) GENE

The ryanodine receptor (*RYR1*) gene, responsible for pig sensitivity to stress, has received the most study. Pig sensitivity to stress is due to C-1843-T transition (resulting in the conversion of amino acid arginine into cysteine) in the *RYR1* gene. The product of a gene showing such mutation leads to calcium release unit in the endoplasmic reticulum of skeletal muscles. An analysis of meat quality made by MacLennen and Phillips (1992) showed that under intense stress conditions, a rapid glycogen disintegration leads to increase of lactic acid content in the muscle cells of the mutated gene carriers. In consequence the level of muscle acidification increases. At slaughter, such animals are a source of PSE (pale, soft, exudative) pork (Essen-Gustavsson et al., 1992). On the other hand, studies of pigs heterozygous for the *RYR1* genotype (Pedersen et al., 2001) demonstrated that they were characterized by 4-5% higher meat content and 14% lower fat content in carcass compared to mutation-free pigs. These studies indicate that the *RYR1* gene exerts an important influence on parameters of meat quality and carcass meatiness. For this reason, this gene is regarded as one with a major effect on these two traits. Analysis of the pig genome physical map made it possible to localize the *RYR1* gene on chromosome pair 6 in the q1.1→1.2 region.

ACID MEAT (HAMPSHIRE) GENE (*RN- RENDEMENT NAPOLE*)

Another gene controlling pig meat quality, regarded as a major effect gene, is the dominant acid meat (Hampshire) gene (*RN*$^{-}$). Unfavourable *RN*$^{-}$ gene increases the glycogen level as assessed by glycolytic potential and branching enzyme activity in the myofibres, giving rise to a lowered protein content, ultrastructural abnormalities and resulting in decreased technological abilities associated with high lactic acid levels postmortem (Monin and Sellier, 1985; Lebret et al., 1999).

The presence of a mutated allele in the genome causes meat processors even greater losses than PSE meat. The meat of pigs with *RN*$^{-}$/- genotype is also observed to contain less protein than the meat of animals without the mutated gene (Monin et al., 1992). Previous studies showed that the *RN* gene is located between the markers SW120 and SW936 on the porcine chromosome 15 (Milan et al., 1995; Mariani et al., 1996; Reinsch et al., 1997). Further studies showed that the incidence of acid (Hampshire) meat is conditioned by a point mutation

(G→A) at codon 200 (changing arginine to glutamine) of the *PRKAG3* gene. This gene was localized on chromosome pair 15 in the q2.4→2.5 region, between microsatellite sequences S1006 and S1007 (Milan et al., 2000). It should be noted, however, that the *RN*$^{-}$ gene has so far been identified only in European and American Hampshire pigs (Miller et al., 2000).

OBESITY GENE *(LEP)* AND LEPTIN RECEPTOR *(LEPR)* GENE

The obesity gene codes for protein named leptin (*LEP*) that is produced by adipocytes (fat-storage cells) and circulates in blood (Zhang et al., 1994). Its physiological function is still obscure, but it is thought that this hormone maintains stability of body fat mass (Friedman, 1997). The effect of leptin on feed intake has led to the hypothesis that leptin has a role in the feedback regulation of adipose mass on feed intake and energy output (Barash et al., 1996). Segantini do Nascimento Borges and Goulart (2002) showed that in pigs the obesity gene had a significant effect on shoulder weight and meat quality (texture). For this reason it represents a very interesting candidate gene for meat production.

Neuenschwander et al. (1996) were the first to report a partial cDNA sequence for pig leptin. Ramsay et al. (1998) presented the full length coding region of porcine *LEP* gene. Next, Čepica et al. (1999) mapped the *LEP* gene to SSC18q1.3→2.1. In this gene several different polymorphisms *(*i.e. A2845T; T3996C; G2728A) were described (Stratil et al., 1997; Jiang and Gibson, 1999; Kennes et al., 2001) and evaluated for association with economically important traits in Yorkshire, Landrace and Duroc pigs (Kennes et al., 2001), as well as in Duroc, Hampshire, Landrace and Large White pigs (Jiang and Gibson, 1999). A significant difference was noticed in the frequency of *LEP* alleles between the high- and low-fat groups of pigs. In the Large White pigs a significant effect was observed of the T/C polymorphism at nucleotide 3469 (T3469C) in the *LEP* gene on the percentage of backfat, lean in dissected shoulder as well as loin and ham (Jiang and Gibson, 1999) and on the mean daily weight gain in Landrace pigs (Kennes et al., 2001). Similar studies were carried out in Poland (Kurył et al., 2003) with pigs of the Pietrain, Zlotnicka Spotted and Landrace breeds and Torhyb and Stamboek lines. The authors also showed the T3469C polymorphism, identified with enzyme *HinfI*, which was detected in exon 2 of the *LEP* gene and was shown to influence meat quality.

In the literature some data suggest that the leptin receptor (*LEPR*) gene (localized on chromosome pair 6 in the q3.3→3.5 region) is correlated with backfat thickness and intermuscular fat content in pig carcasses (Ernst et al., 1997; Ovilo et al., 2002). However, the studies of Szydłowski et al. (2003) with Polish Landrace, Polish Large White and line 990 pigs demonstrated a haphazard nature of the correlation between polymorphism in exon IV of the *LEPR* gene and

backfat thickness and loin eye area. The same authors suggested that the detected associations may be specific to some other breed of pigs.

GROWTH HORMONE *(GH)* GENE

The developing pig is particularly sensitive to treatment with growth hormone (*GH*), which increases the average daily weight gain (Etherton et al., 1987), enhances protein accretion and reduces fat deposition (Campbell et al., 1989).

The growth hormone gene is localized on chromosome pair 12 (in the p1.4 region) (Larsen et al., 1995). In the 1990s several experiments were carried out to identify polymorphic variants of this gene and to determine the most favourable haplotype controlling carcass fatness (Schellander et al., 1994; Pierzchała et al., 1999). Slightly different nature were the studies of Kopchik and Cioffi (1991) showing that introduction of an exogenous growth hormone gene into pig organisms results, among others, in increased intensity of protein synthesis, which in turn makes it possible to increase weight gains by 10-20% while reducing fat in tissues by 30-40%, and allows a 15-30% higher fattening efficiency compared to the control animals.

In the 1990s some experiments were carried out to study the influence of the bovine growth hormone gene (*bGH*) transferred to the genome of the pig on growth and development of the animal (Solomon et al., 1994; Różycki et al., 1999). Gene transfer in animals is required to evaluate the potential for improving production efficiency, carcass quality and disease resistance of livestock. It is established that administration of exogenous porcine somatotropin (*pST*) to pigs at different stages of growth and development results in alteration of body composition (Campbell et al., 1988; Smith and Kasson, 1990). The studies of Pursel and Rexroad (1993) showed that the bovine growth hormone gene (*bGH*) transferred to the genome of pig results in stimulation of muscular development. The transgenic pigs showed muscular hypertrophy at approximately 3 months of age, as well as longer hams. Furthermore, Solomon et al. (1994) showed that carcass tissue from *bGH* - transgenic pigs reflected much more favourable levels of fatty acids as compared with control pigs.

INSULIN-LIKE GROWTH FACTORS *(IGFS)* GENES

In addition to its direct influence on tissue growth, the growth hormone acts indirectly through insulin-like growth factors (somatomedins) *IGF1* and *IGF2*. These peptides are built of 70 and 67 amino acids, respectively, and synthesized by myoblasts during prenatal growth and postnatally in the muscle tissue (Montarras et al., 1996).

The *IGF1* gene in pigs was localized on chromosome pair 5 in the neighbourhood of the microsatellite sequence S0005 (Wintero et al., 1994). However, the study carried out by Lamberson et al. (1995) did not show that *IGF1* concentration affected backfat thickness, area of the *M. longissimus dorsi* and percentage of lean. Biereder et al. (1999) reported a relationship between the *IGF1* level in blood and pig leanness.

Molecular genetics studies also led to the discovery of parental imprinting, whereby only genes transmitted by father (paternal imprinting) or mother (maternal imprinting) are expressed in the progeny. The very nature of imprinting makes it a highly valuable element of selection programmes, for it offers an efficient way of using differences between female and male lines.

The first gene detected in pigs to be characterized by expression in the form of parental imprinting, was the *IGF2* gene, localized on chromosome pair 2 in the p1.7 region, in the close neighbourhood of the microsatellite sequence SWC9. The *IGF2* gene is expressed only within the line of boars, which makes possible restrictive selection for carcass lean (Nezer et al., 1999). Moreover, recent Swedish studies showed that (G→A) transition in intron 3-G3072A of the gene (in conservative region of CpG islet) is related to an increase of about 10-20% in the muscle weight in pigs. This mutation was transmitted to the F_1 generation (created for experimental purposes) by sires - homozygous or heterozygous carriers of the changed gene (Van Laere et al., 2003).

The human and mouse *IGF2* is expressed during embryo development, and its activity is regulated by maternal imprinting. This monoallelic expression is important for normal development of muscular tissue (Pedone et al., 1994). On the contrary, the corresponding receptor gene *IGF2R* is paternally imprinted and expressed when transmitted by the mother.

The *IGF* system consists also of six binding proteins (*IGFBP-1* to *-6)*. The *IGFBPs* can modify *IGF* activity by binding *IGFs* and preventing *IGF* receptor activation. Up to now, the best known is the inhibiting influence of *IGFBP-2, IGFBP-3* and *IGFBP-5* expression on muscle differentiation in pigs (Green et al., 1994; James et al., 1996; Dunaiski et al., 1999).

MYOD FAMILY GENES

Based on a knowledge of molecular mechanisms controlling myogenesis (muscle development) several candidate genes may be selected as potentially affecting carcass meat content. In this regard the *MyoD* family genes encoding muscle regulatory factors are adequate genes for carcass meat deposition. The *MyoD* family consists of several structural and functional genes: myogenin – *MYOG* - located on 9q2.1→2.6 of pig chromosome (Ernst et al., 1998), myogenic

factor 3 - *MYF3 (MYOD1)* - located in the 2p1.4→1.7 region (Čepica et al., 1999), myogenic factor 5 - *MYF5* - located in the 5q2.5 region (Soumillion et al., 1997), and myogenic factor 6; herculin *MYF6* (previously named *MRF4*) - located in 5q2.4→2.5 between microsatellite DNA sequence SW378 and the most distal marker SW967 (Vykoukalova et al., 2003). The PCR-RFLP analysis showed the point mutations in *MYOG* in intron 2*, MYF3* in intron 1 and exons 2 and 3 and *MYF5* in intron 2. Application of the SNP-PCR technique (Single Nucleotide Polymorphism - Polymerase Chain Reaction) revealed the polymorphism of *MYF6* gene in intron 1 and exons 2 and 3 (Knoll et al., 1997; Soumillion et al., 1997; Stratil and Čepica 1999; Te Pas et al., 1999; Vykoukalova et al., 2003).

The results of the studies performed on different breeds and lines (Cieślak et al., 2002; Kurył et al., 2002) lead us to suggest that the mutation in coding as well as non-coding regions of *MYOG*, *MYF3* and *MYF5* genes is the mechanism changing the value of meat quality. For this reason it is very interesting to find out if the polymorphism in the regions of herculin (*MYF6*) is connected with carcass meat and fat deposition traits.

CONCLUSIONS

From the time when the first major genes controlling quantitative traits were discovered, researchers asked how to use molecular studies in pigs to advance genetic progress. Although the gene polymorphism studies explain only part of the genetic variation in pigs, knowledge of it may be used to analyse the genotype of individual animals. This is why information contained in quantitative trait genes should be definitely included in the evaluation of the productive value of farm animals.

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STRESZCZENIE

Charakterystyka wybranych genów warunkujących jakość mięsa świń

Przez wiele lat prace prowadzone przez hodowców świń skoncentrowane były przede wszystkim na tworzeniu populacji charakteryzującej się odpowiednią mięsnością. Rozwój technik stosowanych w genetyce molekularnej zaowocował odkryciem genów, dających główny efekt w cechach ilościowych. Analiza segregacji licznych, równomiernie rozmieszczonych w genomie markerów mikrosatelitarnych, oraz zmienność określonej cechy produkcyjnej w rodzinie referencyjnej może prowadzić do wskazania regionu chromosomowego, w którym prawdopodobnie występuje *locus* genu głównego. Poprzez taką analizę oznaczono u świń około 2 262 markery mikrosatelitarne oraz 1 381 genów, pośród których geny *FAT1, RYR1, RN, LEP, LEPR, GH, IGFs* oraz geny należące do rodziny *MyoD* wykazują istotne zależności z jakością mięsa, tuszy czy z tempem wzrostu.