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GENETICS AND MOLECULAR BIOLOGY AND PIG MEAT QUALITY IMPROVEMENT

GENETICA ȘI BIOLOGIA MOLECULARĂ ÎN IMBUNĂTĂȚIREA CALITĂȚII CĂRNII DE PORC

BULLA, J.¹, OMELKA, R.², JASEK, S.³, ČURLEJ, J.¹, BENCSIK, J.⁴,
FILISTOWICZ, A.³

¹Faculty of Biotechnology and Food Sciences, SAU Nitra, Slovakia

²Univestiy of Constantin Philosopher, Nitra, Slovakia

³Wroclaw University of Environmental and Live Sciences, Wroclaw, Poland

⁴Faculty of Animal Sciences and Biotechnologies, Timisoara, Romania

The main goals in pig breeding have for many years been to improve growth rate, feed conversion and carcass composition. There have been less efforts to improve meat quality parameters (WHC, pH, tenderness, colour etc.) but the main contribution has been a reduction of stress susceptibility and PSE meat. Unfortunately, the quantitative genetic approach has yielded few clues regarding the fundamental genetic changes that accompanied the selection of animal for superior carcass attributes. While mapping efforts are making significant major effects on carcass and his quality composition DNA test would be available to detect some positive or negative alleles. There are clear breed effects on meat quality, which in some cases are fully related to the presence of a single gene with major effect (RYR1, MYF4, H-FABP, LEPR, IGF2). Molecular biology methods provides excellent opportunities to improve meat quality in selection schemes within breeds and lines. Selection on major genes will not only increase average levels of quality but also decrease variability (ei increase uniformity). The aim of this paper is to discuss there genetic and non-genetic opportunities.

Key words: meat quality, pigs, marker genes, selection

Introduction

The quality of pig meat is influenced by a large number of genetic and non-genetic factors. Some several studies have performed a substantial amount of research on these factors, which has led to considerable quality improvement. Most traits of economic importance in pig breeding are complex environmental factors i. g. farm, transport, slaughter and processing conditions. Large single gene effects can be detected as segregating QTL's (quantitative trait loci) with genetic markers. The identification of genes or genetic markers associated with production traits included pig meat quality could have a great economic impact on pork production. In pigs, for example selection for increased lean muscle mass has led to selection for animals highly susceptible to stress with reduced meat quality. After stress or exposure to halothane animals develop the malignant hyperthermia syndrome (MHS), intuited as a monogenic recessive traits. The mutation causing this syndrome is characterized by an abnormality in the ryanodine receptor – RYR 1 (Fuji et al., 1991). Meat quality traits, such as water holding capacity, meat colour and pH 24 are already negatively

affected in heterozygous carrier animals (Hamilton et al., 2000). Similarly in Hampshire pigs, selection for high growth and meat content in the carcass has led to a reduced water – holding capacity and reduced yield of cured cooked ham measured as rendement Napole (RN). Muscle glycogen content is increased by 70 % at the RN⁻ allele. Complete association with a mutation in the PRKAG3 (protein kinase, AMP – activated, gamma 3 non – catalytic subunit) gene has been shown (Milan et al., 2000).

MHS/RYR1

The ryanodine receptor 1 (RYR1) gene codes for the calcium release channel of the sarcoplasmic reticulum of muscle cells. Skeletal muscle contraction is initiated by a release of Ca²⁺ from the sarcoplasmic reticulum, resulting in an increase in Ca²⁺ concentration in the myoplasm. The mutated calcium release channel is associated with malignant hyperthermia susceptibility (MHS), but under normal conditions the muscle of MHS pigs functions undisturbed.

Once opened (e.g. under the influence of stress or slaughter), the mutated channel is unresponsive to Ca²⁺ and Mg²⁺ induced closing and thereby induces muscle contracture, hypermetabolism and hyperthermia. The missense mutation in the porcine calcium-release channel was shown to increase Ca²⁺ ion concentration in myoblastic cells after exposure to clinical doses of halothane (Otsu et al., 1994). Gallant et al. (2001) induced an enhanced Ca²⁺ release from the sarcoplasmic reticulum via calcium release channels (RYR1) by a short peptide representing a functional domain of the dihydropyridin receptor (DHPR). DHPR serves as voltage sensor for incoming action potentials and is located directly opposite the calcium release channels. Calcium release channels of MHS muscle were up to fourfold more strongly activated than normal calcium release channels. In addition, Ca²⁺ release is activated at lower depolarisation potentials (Dietze et al., 2000). Also post mortem, Küchenmeister et al. (1999) observed differences in the calcium-release channel of MHS pigs in Ca²⁺ transporting proteins. The ability of the sarcoplasmic reticulum to accumulate calcium decreases with time post mortem and this decrease occurs at a higher rate in MHS muscle.

It is generally accepted that the tenderness of the meat of carriers of the RYR1 gene mutation is less than that of non-carriers. Sensky et al. (1999) have shown that the levels of m-calpain, but not μ -calpain and calpastatin, are lower in heterozygous carriers than in homozygous non-carriers. The calpain –calpastatin system is involved in meat tenderization after slaughter. From the study by Sensky et al. (1999) it is not possible to separate the effect of altered pH and Ca²⁺ concentrations during postmortem storage, induced by leakage of Ca²⁺ from the mutated calcium release channel of the sarcoplasmic reticulum, from an adaptive response at gene level involving a change in m-calpain expression.

RN/PRKGA3

The RN gene, with two major segregating alleles RN⁻ and rn⁺, has a major effect on meat quality in pigs. The RN⁻ allele is associated with leaner carcasses, a considerably higher muscle glycolytic potential and a detrimental effect on processing yield (Le Roy et al., 2000).

The dominant missense mutation in the PRKGA3 gene causes a dramatic increase in muscle glycogen content. The PRKGA3 gene encodes a muscle-specific isoform of the regulatory γ subunit of adenosine monophosphate-activated protein kinase (AMPK). Loss-of-function mutations in a homologous gene in yeast (SNF4) cause defects in glucose metabolism, including glycogen storage. AMPK is activated by an increase in the ratio of adenosine monophosphate to adenosine triphosphate. Activated AMPK turns on ATP-producing pathways and inhibits ATP-consuming pathways. AMPK can also inactivate

glycogen synthase, the most important regulatory enzyme of glycogen synthesis, by phosphorylation. The muscle-specific expression of PRKAG3 is consistent with the fact that RN⁻ animals show high glycogen content in skeletal muscle but not in liver. Milan et al. (2000) found that AMPK activity in muscle extracts was about 3 times higher in normal rn⁺ pigs than in RN⁻ pigs. Whether the mutation in the PRKGA3 gene inhibits AMPK activation and thus glycogen degradation or constitutively activates the holoenzyme leading to an increased glucose transport and/or glycogen synthesis remains to be investigated.

LEPTIN (LEP)

Leptin, the product of the LEP gene is secreted mainly by adipose tissue and acts as a safety signal on the hypothalamus, thereby regulating body weight and energy expenditure (Campfield et al., 1995). In swine leptin mRNA levels are greater in adipose tissue from obese pigs than lean pigs (McNeel et al, 2000). Furthermore, injection of recombinant porcine leptin reduces feed intake and increases growth hormone (GH) secretion in swine (Barb et al., 1998). These observations suggested that LEP may be a candidate gene for economically important production and qualitative traits such as backfat tickness, feed intake and growth rate in swine. Seven polymorphism in the pig leptin gene (LEP) were described (Stratil et al., 1997, Jiang and Gibson, 1999, Kennes et al., 2001) and evaluated for association with economically important production 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. A significant effect was observed of the T/C polymorphism at nucleotide 3469 in the LEP gene on the percent of backfat and directed lean in shoulder, loin and ham of Large White pigs (Jiang and Gibson, 1999), on the mean daily weight gain in Landrace (Kennes et al., 2001). Genotype TT at locus Lep Proved more advantageous for decreasing both fat weight and fat content of ham in PIC pigs than genotype CT (Kuryl et al., 2003).

H-FABP

An example for a genetic marker for meat quality is provided by the gene for heart fatty acid binding protein (H-FABP). Gerebens et al., (1997) identified polymorphism in this gene and found these to be associated with variation in intramuscular fat in the Duroc. H-FABP maps to pig chromosome G and not to the QTL regions identified by De Kouring et al., (1998). More recently this group has found a larger effect on intramuscular fat with the related gene adipoc cyte FABP.

IGF2

The IGF2 gene is also paternally expressed in the pig (Jeon et al, 1999) and large QTL on pig chromosome 2 is very likely to be a direct effect of an IGF2 variant affecting complex traits such as growth and fitness traits in pigs.

Discussion

Improving meat quality is not just about changing levels of traits like tenderness of marbling, but it is also about increasing uniformity. The existence of major genes or genetic markers provides excellent opportunities of improving meat quality, since it allows large steps to be made in the desired direction (e.g. improving technological yield of ham process by selection against RN⁻ gene in pigs). Secondly, it will help to reduce variation, since we can fix relevant genes in our products. Another aspect is that major genes allow differentiation for specific markets. For example, in certain types of dry cured ham a high

intramuscular fat is required, whereas other products like cooked ham require a low amount of intramuscular fat.

Conclusion

There are clear breed effects on meat quality, which in some cases are fully related to the presence of a single gene with major effect

Within breeds, there is considerable genetic variation in important meat quality traits, which again is partly caused by major genes

Molecular biology and molecular genetic provides excellent opportunities to improve meat quality in selection schemes within lines.

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³*Wroclaw University of Environmental and Live Sciences, Wroclaw, Poland*

⁴*Faculty of Animal Sciences and Biotechnologies, Timisoara, Romania*

Principalele deziderate în creșterea suinelor au fost îmbunătățirea ratei de creștere, de conversie a furajelor și a compoziției carcaselor. Pentru îmbunătățirea parametrilor de calitate (WHC, pH, frăgezime, culoare, etc.) a cărnii au nu fost făcute eforturi suficiente, dar cea mai mare îmbunătățire s-a înregistrat în reducerea sensibilității la stres carne PSE. Din păcate, prin genetică cantitativă s-au găsit puține indicii legate de modificările genetice fundamentale care pot duce la selecția animalelor pentru carcase superioare. În timp ce experimentele de cartare au dus la efecte majore asupra calității și compoziției carcaselor, în curând vor fi disponibile teste de ADN care vor permite identificarea unor alele pozitive sau negative. Este cunoscut faptul că calitatea carcasei depinde de rasă iar în unele cazuri este legată strâns de prezența unei singure gene cu efect major (RYR1, MYF4, H-FABP, LEPR, IGF2). Metodele folosite în biologia moleculară oferă posibilități excelente de îmbunătățire a calității cărnii prin scheme de selecție aplicate raselor și liniilor. Selecția genelor majore nu va avea ca efect numai creșterea nivelelor medii de calitate dar și o descreștere a variabilității (creșterea uniformității). Scopul acestui studiu a fost de a discuta aceste posibilități genetice sau negetice.

Cuvinte cheie: calitatea cărnii, porci, markeri genetici, selecție