# Session 2 Animal growth and evaluation

### L 1 PREDICTION OF PORK QUANTITY AND QUALITY - BRIDGING THE GAP BETWEEN MYOGENSIS AND CONSUMER SCIENCE

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### Summary

It is generally recognized by the meat industry and the academia that a "status quo" has been reached between production of meat quantity and quality, and that the new directions for the industry are being clearly defined by the consumer trends. As a result, the industry needs more than ever before to be able to predict and control both meat quantity and quality. This is required to insure that continuous improvement is being achieved in efficient production of high-quality products designated for different end users/consumers. To achieve this goal the pork supply chain needs to develop and implement additional measurement tools and a different methodology to predict meat quantity and quality. Rapid advancement in life sciences; i.e., muscle biology, functional genomics, proteomics, and computerized electronic/robotic applications will generate new physiological, biochemical, molecular biology and engineering tools. Implementation of statistical process control systems that can utilize these new measurement tools, will dramatically help to control and optimize the supply chain factors which influence meat (lean) quantity and quality. As a consequence, the focus of the pork supply chain will dramatically shift towards economically balanced "best cost production of customer/consumer quality products", and the new production systems will merge the economic and marketing value of "Lean Quality". This feedback-based supply chain approach - from customized genetics/live animal production systems to customized consumer products, and back to customized genetics, will guarantee the sustainable future of the meat industry worldwide. Future production systems will constantly improve, control and monitor the entire production chain, which will ultimately reduce the necessity for the particular, meticulous and laborious measurements of different traits to a small, statistically justified, process control-based sample size.

### Introduction

The world meat industry, like many other agricultural and non-agricultural industries, is constantly undergoing changes. The most prominent change of the last few years is consolidation leading to bigger, and more complex, vertically integrated and/or coordinated meat industry entities. Also, in most parts of the world, the percentage of further processed meats is increasing at the expense of the fresh meat consumption. End users, like meat/food processing companies, although tending to view meat as "just" raw materials or more precisely as a protein source, are becoming more aware that these "raw" proteins must have consistent and specific quality/functionality characteristics. Retail and food service businesses are also becoming more interested in consistently-sized "case-ready" products, leaner and better tasting product varieties, and cuts of meat that are suited to today's consumer cooking demands, including demands for no-cooking at all. At the same time there is a growing consumer concern about the quality of meat and its production. There is no doubt that the consumer is now at the center of a considerable turmoil involving the entire food supply chain. Food safety crises and livestock epizooties have shaken both consumers and political confidence in animal/food science, and the meat chain at large. Harrington's (1994) list of consumer concerns: ethical, food safety, nutrition and fat, animal welfare, "third world", the environment and genetic engineering, remains as true today as it was a few years ago.

Meat production has been a commodity driven business. Meat quantity characteristics used to drive the major economic value for different meat production chains, due to the relatively easy means of measuring carcass weight and lean percentage/yield after slaughter (Diestre et al., 1989; Gresham et al., 1994; Madsen and Thodberg, 1994; Allen, 1995). However, latest consumer demands have led to product differentiation and greater pressure on the value of meat quality parameters (Hofmann, 1994). Meat processing companies are also becoming more aware of the necessity of understanding and controlling specific meat quality characteristics that benefit the economics of their production systems (Hoen, 1996; Swatland, 1992). These developments have increased the interest in measuring and predicting meat quality early and/or 24 hours postmortem. Food safety issues have accelerated the introduction of different quality assurance control systems in the meat industry, like Hazard Analysis of Critical Control Points (HACCP), General Manufacturing Practices (GMP), Total Quality Management (TQM), International Standards Organization (ISO), or Six-Sigma (Wood et al. 1998; www.6-sigma.com). It is important to note that several food safety critical control points apply to meat quality control processes (Van Logtestijn, 1993; Sosnicki et al., 1998). However, many of the tools that are necessary to achieve a continuous delivery of high quality meat/products fulfilling customer demands are still not available at the present time.

One of a set of very promising new tools that are becoming available is functional genomics and proteomics (analysis of the protein content of the sample) of muscle-foods animals. In addition, the rapidly growing knowledge of human and other species genome is also being utilized in farm animal research (comparative genomics), leading to applications in prediction and production of desirable lean quality. An example of this new, non genetic engineering DNA technology to control meat quality, is the identification of the melanocortin-4 receptor gene, which is associated with fatness, growth, and feed intake traits in pigs (Seeley et al., 1997; Marsh et al., 1999; Kim et al., 2000). Thus, the development in life sciences, including muscle biology and DNA technology, and automation will also increase the possibility of predicting meat quantity and quality ("lean quality") in life animals (De Vries et al., 1998; Rothschild and Plastow, 1999; Vissher et al., 2000) Integration of this knowledge into technological, engineering and robotic application-driven statistical process control systems aimed at constant monitoring and improvement of the entire production chain will ultimately reduce the necessity for the laborious measurement of different traits to a small, statistically required sample size.

This paper, although not intended to be all-inclusive, focuses on the pork supply chain, but the scientific and practical approaches apply also to other meat-animal species. We concentrate on discussing opportunities for future developments in life sciences and implementation of the results via process control systems. We also examine the implications of consumer, and to a much lesser extent, technological trends for prediction and efficient production of desirable meat lean quality.

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### Current situation in the pork industry

While analysing the current status of the pork industry, one must ask questions such as: does the industry consistently produce the quality products demanded by consumers? are we still thinking of production chains starting with the farmer producing the animals instead of focusing on the meat products first? how can the industry implement the strategy of "best cost production of consumer quality products" appreciating that several of the existing supply chains employ many profit centers that are not necessarly aligned around the quality of the final products?

As stated before, the worldwide pork industry has been mainly focused on production of lean meat. As a direct response to this particular need genetic companies have produced significant improvements in backfat depth, carcass lean percentage, daily gain, and feed conversion ratio. The basis for this improvement has been the development of increasingly sophisticated statistical methods to analyze performance test data based on the infinitesimal gene model, which assumes an infinite number of genes, each of small effect, but together additively affecting quantitative traits (McLaren and Schultz, 1992; Short et al., 1997; Knapp et al., 1997). It is also recognized that the industry has approached an apparent ceiling for carcass leaness; i.e., 56%-58% in the U.S at approximately 120 kg live weight; 58%-62% in Europe at approximately 100 kg live weight, but it is unclear if this will be a fixed end-point of carcass quantity or an evolving bottleneck. Economic benefits dictate continued priority for average daily gain (ADG) and feed conversion ratio (FCR), and the new physiological and genomic "tools" will only accelerate the rate of improvement of these traits. On the other hand, targeting muscle growth rate without understanding of its potential impact on meat quality and nutrient use could be detrimental.

It is commonly acknowledged that meat quality is a difficult characteristic to assess as many different aspects, both objective and subjective, make up the overall trait (Hofmann, 1994). Some systems/markets require specific quality characteristics such as high intra muscular fat content or dark color; i.e. dry cured ham production or the Japanese market. Several markets have started to demand different slaughter weights; e.g., in the UK to minimize boar taint of intact males, to market portion-controlled size of meat cuts. These demands have impacted the economics of production of lean quality. A further increase in product diversification will stimulate growth of niche markets with different requirements for raw materials and ready-to-cook and ready-to-eat branded consumer products. However, each of these markets will still be dedicated to produce a large proportion of saleable products per carcass that fulfill both niche and commodity market requirements.

So what is currently being done by the industry to address some of the questions under consideration? As a response to the growing meat quality awareness of the consumer, the pork industry has taken several steps to improve (or hold constant) meat quality, and still simultaneously improve production performance and carcass quality. There are several relatively inexpensive pig meat quality measurements, such as initial and ultimate muscle pH, that have been successfully included some breeding programs (Eikelenboom et al., 1995; Sosnicki et al., 1998). Genetic improvement in meat quality has also started being achieved via molecular genetic approach (marker assisted selection or MAS; see below; Webb, 1993; De Vries et al. 1998). In addition, as mentioned before, the rapidly developing field of domestic animal functional genomics and proteomics has already created, and will soon create more of very useful information enabling simultaneous improvement and better control of efficient growth of lean tissue and high meat quality (see below).

## Potential benefits of understanding the control mechanisms of skeletal muscle differentiation and growth for prediction of lean quality – "back to the future"

The main question that we address in this section is: how can the research in the field of skeletal muscle biology & meat science further help in developing new lean quality "control tools"? Before attempting to answer this question, a concise summary of this field is provided.

The control of muscle growth involves many genes and a complex array of transcription factors. Each step in myogenesis involves specific changes in gene expression. Terminal myogenic differentiation is characterized by expression of four transcription factors that are members of the myogenic determination factor (MDF) family: myogenin, MyoD, Myf5 and MRF4 (Mulvaney, 1994; Molkentin and Olson, 1996; Arnold and Winter, 1998; Te Pas et al., 2000). Individually these basic helix-loop-helix (bHLH) family members can activate myogenesis in non-muscle cells, but analysis of embryonic expression and knockout studies show that each has a unique function in myogenesis (Arnold and Braun, 1996). Additional transcription factors, particularly the MEF2 family, cooperate with the MDFs to activate muscle-specific gene transcription (Black and Olson, 1998). The MEF2C isoform appears to be most important in up-regulation of the MDF transcription factors (Ridgeway et al., 2000). A third group of transcription factors, termed NFAT (abbreviated for Nuclear Factor of Activated T cells) has also been found to affect the transcription of certain genes. At least five different NFAT isoforms have been identified (NFAT1-5), with NFAT2 and NFAT4 being present in larger amounts in skeletal muscle (Hoey et al., 1995).

Myogenesis is followed by fiber hypertrophy and maturation to yield adult muscle fibers (Swatland, 1973; Lengerken et al., 1994). The biochemical profile of adult muscle greatly influences its metabolic responses during pre-slaughter handling and, subsequently, postmortem conversion of muscle to meat and meat quality. One of the main factors determining muscle biochemical pathways is fiber type composition: skeletal muscle is composed of different types of fibers, which are the results of co-ordinated expression of distinct sets of structural proteins and metabolic enzymes (Pette and Staron, 1990; Musaro et al., 1995; Schiaffino and Reggiani, 1996). Fiber types are often defined by the isoforms of myosin heavy chain (MyHC) that are present. There are four major fiber types in postnatal pig muscle characterised by the expression of the slow/I/ $\beta$ , 2a, 2x and 2b MyHC gene isoforms. The slow/I/ $\beta$  and 2b fibers, also known as slow-oxidative and fast-glycolytic, respectively, represent two extreme metabolic profiles. The 2a and 2x fibers are intermediate fast oxidative-glycolytic fibers. (Chang and Fernandes, 1997; Greaser et al., 2001). In addition, fiber type is affected by several environmental factors as for example diet or physical activity (Karlsson et al., 1993; Klont et al., 1998; Petersen et al., 1998; Karlsson et al., 1999).

So what are the potential lean quality implications of the known muscle control mechanisms? It was reported that muscle fiber composition affects both growth and lean content of the pig and it is also breed/line specific (Essen-Gustavsson and Fjekjer-Modig; 1985; Warnants et al., 1993; Degens and Veerkamp, 1994; Lefaucheur et al., 1995; Ruusunen et al., 1996; Larzul et al., 1997; Tanabe et al., 1997). For instance, faster growing pigs appear to posses more, but smaller, fibers than slower growing pigs at the same weight. It was also established that the total number of muscle fibers in Longissimus dorsi (loin) muscle is more closely related to muscle mass than is fiber diameter; although a positive correlation was found between the percentage of Type IIb fibers and the cross sectional-area (CSA) of the Longissimus muscle. The Longissimus muscle CSA of Type I fibers being smaller and CSA of Type IIB fibers being larger in Landrace compared to Yorkshire pigs at a given live weight is an example of breed-specific muscle fiber composition (Lefaucheur et al., 1997).

The impact of muscle fiber type composition on lean quality is even less understood; although the relative high volume of Type IIb fibers was related to poor meat quality (Sosnicki, 1987; Brocks et al., 1998; Fiedler et al., 1999). For instance, PIC/Purdue University research showed that the abundance of Type IIb myosin is negatively related to pH at 45 minutes post-mortem (r=-.50) in Hal-gene positive and carrier pigs, and negatively related to pH at 24 hours post-mortem in the Hal-gene negative population (r=-.50; Gerrard, personal communication). It was also reported that Type IIb fiber percentage was negatively related to pH at 30-minutes post-mortem, negatively to pH at 24 hours post-mortem, positively to glycolytic potential, and positively to color lightness (Essen-Gustavsson and Fjekjer-Modig; 1985; Lefaucheur et al., 1995; Ruusunen et al., 1996; Larzul et al., 1997). Published heritabilities (h<sup>2</sup>) of muscle fiber traits are moderate to high; i.e. h<sup>2</sup> of Type I fiber CSA = .59; h<sup>2</sup> of Type I fiber percentage = .46; h<sup>2</sup> of Type IIb fiber percentage = .58 (Larzul et al, 1997). Published genetic correlations (rg) indicate that Type I and Type IIb fiber percentage are negatively related (rg = -.85; Gerrard and Sosnicki, 1997; Larzul et al., 1997). This particular genetic correlation indicates that breeding for higher percentage of Type I fibers would decrease the proportion of Type IIb fibers; thus directionally improving meat quality without negatively affecting Type IIa and IIx percentage (rg = .16) or mean fiber CSA (rg = -.15). This approach would enable selection for fast lean tissue growth rate without negatively affecting meat quality (Larzul et al., 1997; Klont et al., 1998).

The interaction between skeletal muscle and environmental stress before slaughter complicates even further the understanding, measuring and fully controlling major sources of variation in lean quality. The magnitude of stress response depends on the individual characteristics of the animal; i.e., the individual difference in behavior and physiology may have consequences for the ability of the pig to cope with unfamiliar stimuli such as preslaughter stress (Benus et al., 1987; Tarrant, 1989; Lawrence et al., 1991; Hessing et al., 1994). The two main neuro endocrine systems involved in physiological adaptation and metabolic regulation are the hypothalamic-pituitary adrenal axis (HPA) and the autonomic nervous system (Harbuz and Lightman, 1992). The known genetic differences in the basic functioning of these neuro endocrine systems or in their response to stress need to be fully explored (Benus et al., 1991).

The size of variation of the traits discussed above and their relation with production and lean quality traits remain to be quantified and implemented into "farm-to-table" customized production systems. It is conceivable, however, that the full understanding of the genetic, physiological and environmental interactions will enable, in the future, simultaneous genetic improvement in muscle growth, resistance to stress, and consequently lean quality.

### Implementation of DNA markers as a means of "designing", measuring and controlling lean quality

Meat quantity and quality are determined by a combination of genetic, nutritional and environmental factors and their interactions (for review see Cassens et al., 1975; Tarrant, 1989; Cameron, 1990; Sosnicki et al., 1998;). Genetic effects play a crucial role in "designing" pig carcass composition and quality; although pork quality is to a lesser extent influenced by genetic factors than meat quantity; i.e., generally between 10% and 30% of the variation in meat quality traits is determined by the genetic basis of the animal (De Vries et al., 1994; Sosnicki et al., 1998). The use of quantitative genetics, selection indexes, and estimated breeding values (EBVs) for lean quality have brought the pork industry where it is today. The basis for this improvement has been the development of increasingly sophisticated statistical methods. Progress is continuing in this area (Hill, 1999) fueled by the opportunities provided by biotechnology in terms of data collection and dissemination of genetic improvement. For example, the technique of "Optimal Genetic Contribution" can enhance genetic improvement by 10% to 20% (Woolliams et al, 1999, Hanenberg & Merks, 2000). The EBVs of some of the breeding organizations now include meat quality traits in addition to efficient production of carcass lean (De Vries et al., 1998).

Identification of genetic markers and candidate genes for meat quality characteristics in combination with Marker Assisted Selection (MAS) programs will also greatly enhance genetic improvement for meat quality whilst not compromising lean percentage (Meuwissen and Goddard, 1996). The pig industry is already actively using MAS strategies to improve swine production (Short et al., 1997, Rothschild and Plastow, 1999). It is anticipated that the developments in genomic technologies will increase the number of markers that can be used in MAS, so that selection for meat quality can be carried out on live animals. Table 1 shows some of the identified candidate genes and potential markers for pork quantity and quality.

Name	Description	Reference
Hal-1843® gene	Malignant hyperthermia – porcine stress syndrome: linked to PSE meat, carcass leanness and muscle mass	Fuji <i>et al.</i> (1991)
RN gene	Acid pork (Hampshire)	Milan <i>et al.</i> (2000)
MC4R	Appetite, fatness and homogeneity of pig carcasses	Kim et <i>al.</i> (2000)
BETTERgen™ / IGF2	Lean meat content of muscular breeds	Nezer <i>et al.</i> (1999), Jeon <i>et al.</i> (1999)
FABPs	Level of intramuscular fat (Duroc)	Gerbens et al. (1997, 1998)
Unidentified (QTL)	Level of intramuscular fat and backfat thickness (Iberian pig)	Ovilo et al. (2000)
Unidentified (QTL)	Level of intramuscular fat (Meishan)	Janss et al. (1994, 1997)
Unidentified (QTL)	Level of intramuscular fat (Duroc)	Monin et al. (1998)
Unidentified (QTL)	Level of intramuscular fat (Meishan)	Renard & Mourot (2000)
Calpain	Level of calpain – pork tenderness	Parr et al. (1999)
MyoG (myf4)	Muscle yield	Soumillion et al. (1997)
MyHC	Fiber type – pork tenderness	Beuzen et al. (2000)
CAST	Level of calpastatin – pork tenderness	Ernst et al. (1998)
Unidentified (QTL)	Level of androsterone – boar taint	Fouilloux et al. (1997)
Unidentified (QTL)	Linoleic acid content of pork fat	Pérez-Enricso et al. (2000)

### Table 1. Potential Genetic Markers for Carcass and Meat Quality

Knowledge of the genome and the establishment of genetic maps are essential in order to isolate and characterize genes of interest. In recent years the linkage and physical maps of the pig genome have developed considerably (for review see Rothschild and Plastow 1999). These maps have been exploited to search for genes influencing variation in commercially important traits. Several quantitative trait loci (QTL) scans and candidate gene analyses have identified important chromosomal regions and major genes associated with traits of economic interest in the pig. These include QTL for growth and backfat (chromosomes 1, 2, 3, 4, 5, 6, 7, 8,

13, 14), meat quality traits (chromosomes 2, 3, 4, 6, 7, 12, 15) and reproduction (chromosome 4, 6, 7, 8). The causative mutations for porcine stress syndrome (HAL or CRC1) and coat color have been identified. Results obtained with candidate genes are also very encouraging (e.g. ESR and PRLR for litter size, heart and adipocyte FABP for meat quality and FUT1 for disease resistance).

The use of DNA markers in breeding programs will also help to reduce variation in carcass composition and meat quality traits. In addition, the exploitation of new markers (both physiological and DNA) in combination with controlled environmental conditions will allow for customization of breeding programs and, therefore, pig/carcass differentiation for specific markets. For example, in certain types of dry cured ham high intramuscular fat content (IMF) is required, whereas other products such as a variety of cooked ham require low amount of IMF. Thus, the future will see processors and retailers specifying a whole series of genes that have to be present or absent in each product/product line. The scientific emphasis will be to discover multiple genes that determine the specific quality trait. Technologies such as functional genomics using cDNA micro arrays (containing thousands of genes) and proteomics (analysis of protein content of samples) is making it possible to analyze gene expression and gene products in the muscle/meat and relate this knowledge to meat yield, eating and processing quality.

### Conclusions

What immediate steps should the pork industry take to guarantee that pork is not only lean, but that this lean has a fresh appearing reddish-pinkish color, is high in water holding capacity, and it is consistently tender and juicy? Please keep in mind that to compete with other animal proteins in global markets, producers must deliver quality pork at least cost (Tubbs, 1997). In order to achieve this, (1) breeding companies need to fully understand the economic value of these quality attributes to optimally select for best cost of high quality pork; this includes a research approach to fully understand skeletal muscle differentiation, growth and protein deposition, and functinal genomics and proteomics; (2) guidelines should be established and implemented to insure acceptable on-farm production management and welfare procedures at all times; (3) pork processing companies should implement statistical process control procedures for pre-slaughter handling and post-slaughter processing to minimize quality variation and develop more robust equipment for on-line measurement of lean quality; (4) procedures should be put in place to electronically identify and evaluate individual groups of pigs slaughtered for carcass weight, leanness, and quality; this information should be included in quality assurance reports to continually monitor quality variations so that the appropriate steps can be made to further improve breeding stock, production and processing environment. Finally, the total value paid for market pigs should reflect accurate value differentials (as dictated by supply-demand forces) between desirable and undesirable pork quantity and quality.

We envision that it will no longer be necessary in the near future to measure lean quality on every single carcass. Processors and retailers will specify a whole series of genes, along with environmental controls, that have to be present or absent in each product. This set of customized genes will be the starting point of the process/system enabling for prediction of meat quality and quantity of specified products. Measurements of lean quality will be minimized to a small, statistically established sample size that will be sufficient to insure that each product complies with the quality specifications demanded by the end user and/or by the consumer.

### References

Allen, P. 1995. In: Conf. Proc. International developments in process efficiency and quality in the meat industry. November 16-17, Dublin, Ireland. Pp 33-45.

Arnold, H. H. and B. Winter. 1998. Curr. Opin. Genet. Dev. 8:539-44.

Arnold, H. H., and T. Braun. 1996. Int. Dev. Biol. 40:345-363.

Benus, R.F., J.M. Koolhaas, and G.A. Van Ootmerssen. 1987. Behav. 100: 105-122.

Benus, R.F., B. Bohus, J.M. Koolhaas, and G.A. Van Ootmerssen. 1991. Experientia 47: 1008-1019.

Beuzen, N.D., M.J. Stear, and K.C. Chang. 2000. The Veterinary Journal, 160: 42-52.

Black, B. L., and E. N. Olson. 1998. Annu. Rev. Cell Dev. Biol. 14:167-96.

Brocks, L., B. Hulsegge, and G. Merkus. 1998. Meat Sci. 50:411-20.

Cameron, N.D. 1990. Meat Sci. 27: 227-247.

Cassens, R.G., D.N. Marple, and G. Eikelenboom. 1975. Adv. Food Res. 21: 71-155.

Chang, K.C. and K. Fernandes. 1997. DNA Cell Biol. 16, 1429-1437.

De Vries, A.G, P.G.van der Val, T. Long, G. Eikelenboom, and J.W.M. Merks. 1994. Livestock Prod. Sci. 40: 277-289.

De Vries, A.G., A.A. Sosnicki, J.P. Garnier, and G.S. Plastow. 1998. Proc 44th ICoMST, Barcelona, Spain, 1998, Vol 1: 66.

Degens, H., and J.H. Veerkamp. 1994. Int. J. Biochem. 267: 871-878.

Diestre, A., M. Gispert, and M.A. Oliver. 1989. Anim. Prod. 48:443-448.

Eikelenboom, G., P.G. van der Wal, A.G. de Vries. 1995. Proc. 41st ICoMST, San Antonio, USA . pp. 654-655.

Ernst, C.W., A. Robic, M. Yerle, L. Wang, and M.F. Rothschild. 1998. Anim. Gen., 29: 212-215.

Essen-Gustavsson, B., and S. Fjelkner-Modig. 1985. Meat Sci. 13: 33-42.

Fiedler, I., K. Ender, M. Wicke, S. Maak, G. V. Lengerken, and W. Meyer. 1999. Meat Sci. 53:9-15.

Fouilloux, M.N., P. Le Roy, J. Gruand, C. Renard, P. Sellier, and M. Bonneau. 1997. Genetics Selection Evolution, 29: 357-366. Fuji, J., K. Otsu, F. Zorzato, S. De Leon, V.K. Khanna, J.A. Weiler, P.J. O'Brien, and D.H McLennan. 1991. Science, 253: 448-451.

Gerbens, F., G. Rettenberger, J.A. Lenstra, J.H. Veerkamp, and M.F.W. Te Pas. 1997. Mammalian Genome, 8: 328-332.

Gerbens, F., A. Jansen, A.J.M. Van Erp, F. Harders, T.H.E. Meuwissen, G. Rettenberger, J.H. Veerkamp, and M.W.F. Te Pas. 1998. Mammalian Genome, 9: 1022-1026.

Gerrard, D.E., and A.A. Sosnicki. 1997. Purdue University (unpublished information).

Greaser, M.L., H. Okochi, and A.S. Sosnicki. 2001. Proc. ICoMST, 2001.

Gresham, J.D., S.R. McPeake, J.K. Bernard, M.J. Riemannn, R.W. Wyatt, and H.H. Henderson. 1994. J. Anim. Sci. 72: 1409-1416.

Hanenberg E.H.A.T., and J.W.M. Merks. 2000. J. Anim. Sci. 78 S1, 68.

Harbuz, M.S., and S.L. Lightman. 1992. J. Endocrinol. 134: 327-339.

Harington, G. 1994. Meat Sci. 36:5-18.

Hessing, M.J.C., A.M. Hagelsø, W.P.G. Schouten, P.R. Wiepkema, and J.A.M. Van Beek. 1994. Physiol. Behav. 55: 39-46.

Hill, W.G. 1999. In: J.C.M. Deckers, S.J. Lamont, M.F. Rothschild, Eds. From J.L. Lush to Genomics: Visions for Animal Breeding and Genetics, Iowa University Press, USA : 35-46.

Hoen, O. 1996. In: Allen D. Leman Swine Conference Proceedings, pp. 45-50.

Hoey T, Sun YL, Williamson K, Xu X. 1995. Immunity 2:461-72.

Hofmann, K. 1994. Meat Focus Int. 2:73-82.

Janss, L.L.G., J.A.M. Van Arendonk, and E.W. Brascamp. 1994. Proc. 5th Congr. of Genetics Applied to Livestock Prod. 18: 361-364.

Janss, L.L.G., J.A.M. Van Arendonk, and E.W. Brascamp. 1997. Genetics, 145: 395-408.

Jeon, J.T., O. Carlborg, A. Tornsten, E. Giuffra, V. Amarger, P. Chardon, L. Andersson-Eklund, K. Andersson, I. Hansson, K. Lundstrom, and L. Andersson. 1999. Nature Genetics 21: 157-158.

Karlsson, A., A.C. Enfält, B. Essén-Gustavson, K. Lundström, L. Rydhmer, S. Stern. 1993. J.Anim. Sci. 71, 930-938.

Karlsson, A.H., R.E. Klont, and X. Fernandez. 1999. Livestock Prod. Sci. 60:255-269.

Kim, K.S., N. Larsen, T. Short, G. Plastow, and M.F. Rotschild. 2000. Mammalian Genome 11: 131-135.

Klont, R.E., L. Brocks, and G. Eikelenboom. 1998. Meat Sci. 49: S219-S229.

Knapp, P., A. Willam, and J. Solkner. 1997. Livest. Prod. Sci. 52: 69-73.

Larzul, C., L. Lefaucheur, P. Ecolan, J. Gogue, A. Talmant, P. Sellier, and G. Monin. 1997. J. Anim. Sci. 75: 3126-3137.

Lawrence, A., E. Terlouw, and A. Illius. 1991. Appl. Anim. Behav. Sci. 30: 73-86.

Lefaucheur, L., F. Edom, P. Ecolan, and, G.S. Butler-Browne. 1995. Develop. Dynamics. 203-27-41.

Lengerken, G. Von, S. Maak, M. Wicke, I. Fiedler, and K. Ender. 1994. Arch. Tierz. Dummerstorf, 37: 133-143.

Madsen, N.T., and H.H. Thodberg. 1994. Proc. 40th Int. Congr. Meat Sci. Technol., The Hague, The Netherlands.

Marsh, D.J., G. Hollopeter, D. Huszar, R. Laufer, K.A. Yagaloff, et al. 1999. Nature Genetics 21: 119-122.

McLaren, D.G. and C.M. Schultz. 1992. Proc. Reciproc. Meat Conf. 45: 115-121.

Meuwissen, T. and M.E. Goddard. 1996. Genet. Sel. Evol. 28: 161-176.

Milan, D., J.T. Jeon, C. Looft, V. Amager, M. Thelander, A. Robic, C. Robel-Gaillard, S. Paul, N. Iannuccelli, L. Rask, H. Ronne, K. Lundström, N. Reinsch, J. Gellin, E. Kalm, P. Le Roy, P. Chardon, and L. Andersson. 2000. Science 288: 1248-1251.

Molkentin, J.D., and E. Olson. 1996. Curr. Opin. Genet. Dev. 6:445-453.

Mulvaney, D.R. 1994. Reciproc. Meat Conf. Proc. 47:119-131.

Musaro, A., M.G. Cusella de Angelis, A. Germani, C. Ciccarelli, M. Molinaro, and B.M. Zani. 1995. Exp. Cell Res. 221: 241-248.

Nezer, C., I. Moreau, L. Karim, B. Brouwers, W. Coppieters, J. Detilleux, R. Hanset, A. Kvasz, P. Leroy, and M. George. 1999. Nature Genetics, 21: 155-156.

Ovilo, C., M. Pérez-Enciso, C. Barragan, A. Clop, C. Rodriguez, M.A. Oliver, M.A. Toro, and J.L. Noguera. 2000. Mammalian Genome 11: 344-346.

Parr, T., P.L. Sensky, G.P. Scothern, R.G. Bardsley, P.J. Buttery, J.D. Wood, and C. Warkup. 1999. J. Anim. Sci., 77: 661-668.

Pérez-Enciso, M., A. Clop, J.L. Noguera, C. Óvilo, A. Coll, J.M. Folch, D. Babot, J. Estany, M.A. Oliver, I. Díaz, and A. Sánchez. 2000. J. Anim. Sci. 78: 2525-2531.

Petersen, J.S., P. Henckel, N. Oksbjerg, and M.T. Sorensen. 1998. Animal Sci. 66, 733-740.

Pette, D., and R.S. Staron. 1990. Rev. Physiol. Biochem. Pharmacol., 116: 1-76.

Renard, C., and J. Mourot. 2000. INRA Productions Animales, numéro hors-serie «Génétique Moléculaire: Principes et Application aux Productions Animales» : 161-163.

Ridgeway A.G., S. Wilton, and I.S. Skerjanc. 2000. J. Biol. Chem. 275:41-46.

Rothschild, M.F., and G.S. Plastow. 1999. AgBiotechNet, 10: 1-8.

Ruusunen, M., M. Sevon, M. Aimonen, and E. Poulanee. 1996. Agr. Food Sci. Finl. 5: 593-600.

Schiaffino, S. and C. Reggiani. 1996. Physiol.Rev. 76, 371-423.

Seeley, R.J., K.A. Yagaloff, S.L. Fisher, P. Burn, T.E. Thiele, et al. 1997. Nature, 390: 349.

Short, T.H., M.F. Rothschild, O.I. Southwood, D.G. McLaren, A. de Vries, H. van der Steen, G.R. Eckardt, C.K. Tuggle, J. Helm, D.A. Vaske, A.J. Mileham, and G.S. Plastow. 1997. J. Anim. Sci. 75:3138-3142.

Sosnicki, A.A. 1987. J. Anim. Sci. 64:1412-1422.

Sosnicki, A.A., E.R. Wilson, E.B. Sheiss, and A. de Vries. 1998. Proc. Reciproc. Meat Conf. 51:11

Soumillion, A., J.H. Erkens, J.A. Lenstra, G. Rettenberger, and M.F.W. Te Pas. 1997. Mammalian Genome, 8: 564-568.

Swatland, H.J. 1973. J. Anim. Sci. 37 : 526-535.

Tanabe, R., S. Murroya, K. Chikuni, and H. Naki. 1997. ICoMST, G2:31: 606-607.

Tarrant, P.J.V. 1989. Ir. J. Food Sci. Technol. 13: 79-107.

Te Pas, M.F.W., F.J. Verburg, C.L.M. Gerritsen, and K.H. de Greef. 2000. J. Anim. Sci. 78:69-77.

Van Logtestijn, J.G. 1993. Meat Focus Internat. 2:123-128.

Vissher, P., R. Pong-Wong, C. Whittemore, and C. Haley. Livest. Prod. Sci. 65:57-70.

Warnants, N., W. Eeckhout, and C. Boucque. 1993. J. Anim. Breed. Genet. 110: 357-362.

Webb, J. 1993. Meat Focus Int. Feb. pp. 78-85.

Wood, J.D., J.S. Holder, and D.C.J. Main. 1998. Proc. 44<sup>th</sup> Inter. Congr. Meat Sci. Technol. Vol. I, Barcelona, Spain, 206-215. Woolliams, J.A., P. Bijma, and B. Villanueva. 1999. Genetics. 153: 1009-1020.