FEEDING AND MEAT QUALITY

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Introduction

Traditionally, the term 'meat quality' covers inherent properties of meat decisive for the suitability of the meat for eating, further processing and storage including retail display. The main attributes of interest are safety, nutritional value, flavour, texture, water-holding capacity, colour, lipid content, lipid composition, oxidative stability and uniformity. However, the dramatic changes at the international market over the past decade also require high standards of quality assurance regarding diversity and of aspects related to environmental, ethical and animal welfare problems in the production of meat. Consequently, quality is now to be considered a complex and multivariate property of meat, which is influenced by multiple interacting factors including the conditions under which the meat is produced. Production conditions include management system, breed, genotype, feeding, pre-slaughter handling and stunning, slaughter method, chilling and storage conditions.

Feeding strategy is the management factor, which is most actively used as a quality control tool in the production of meat and in relation to improvement and/or control of performance, animal welfare, safety, nutritional value, and eating and technological quality. Most feeding strategies tested and implemented in meat production have until recently been based on 'passive' effects, i.e. the uptake and incorporation of specific feeding components or chemical/structural behaviour in the intestinal tract, e.g. contribution to lipid content and composition in relation to nutritional value (Wood, Richardson, Nute, Fisher et al., 2004), technological quality (Allen & Foegeding, 1981; Sheard, Enser, Wood, Nute, Gill & Richardson, 2000) and/or anti-oxidative status (vitamin E) in relation to storage life and technological quality (Faustman & Cassens, 1989; Morrissey, Buckley & Galvin, 2000; Granit, Angel, Akiri, Holzer et al., 2001) and also to minimize pathogen growth in the intestinal tract (Engberg, Hedemann & Jensen, 2002; Hansen, Bach Knudsen, Jensen & Kjærsgaard, 2001a,b) and off-flavour formation by regulation of microbial fermentation in the intestinal tract (Jensen, Cox & Jensen, 1995). However, the use of feeding to optimise livestock performance and lean meat percentage has also produced a considerable biological understanding of the influence of diet composition on muscle deposition.

In view of our present knowledge of the effect of feeding on meat quality, it might be relevant to ask whether additional focus on individual feedstuffs in relation to meat quality is an area where we can expect significant breakthroughs in the years to come? The answer might be ***no***, if the traditional thinking of feed *versus* meat quality continues to dominate. On the other side, if the focus changes towards an understanding of how feeding influences biological mechanisms and the outcome of these in relation to specific meat quality parameters, a picture begins to emerge of a huge potential for future production of diverse and specific meat qualities.

The present paper will pinpoint a few critical areas where a strengthened emphasis on a basic understanding of the influence of specific feeding strategies on biological responses may help to revolutionise future management practices aiming at the production of meat of the required quality. A fundamental understanding of muscle physiological and physical processes, and their interactions in relation to gene expression and environmental stressors, will be fundamental to exploiting future meat science and production through a systems biology[1] line of thought. Considering the already extensive knowledge of feed and meat quality, feeding seems the optimal tool in the further elucidation of physiological and physical events of importance for demanded meat qualities. Feeding can hereby become a key parameter together with the impending presence of genomes of most of the significant livestock in establishing a systems biology line of thought in the future quality assurance of meat. Simultaneously, the understanding obtained in employment of the discrete feeding strategies necessary for data accumulation to a systems biology approach will ensure temporary short-term solutions for specific meat quality problems.

In the following a few examples of feeding-dependent meat quality aspects of high priority from both an economic and/or socioeconomic point of view will be highlighted in relation to their potential as quality assurance tools. Furthermore, their contribution to the knowledge of importance for a coming systems biology line of thought in meat science will be discussed.

Feeding-regulated muscle protein turnover

Basic principles of muscle growth

Muscle growth is the major determinant of the performance of meat-producing animals. The amount of meat produced is related to the number of muscle fibres and the growth of the individual muscle fibre. Muscle fibres are formed during the embryonic and foetal stages, and the number is fixed around birth in most mammals. Thus postnatal growth is related to growth in the cross-sectional area (hypertrophy) and length in the fibres by adding additional sarcomeres. Postnatal growth is determined by the difference between two dynamic processes; i) the rate of protein synthesis and ii) the rate of protein degradation, referred to as the protein turnover. Thus, during postnatal muscle growth the rate of synthesis exceeds the rate of degradation. With increasing age both the rate of synthesis and degradation decrease and become equal in adult animals (for review see Oksbjerg, Gondret & Vestergaard, 2004). The rate of protein turnover is also related to the fibre type frequency, being higher in slow-twitch fibres than in slow-twitch fibres (Garlick, Maltin, Baillie et al., 1989, Goldspink, 1996).

Influence of feeding-regulated muscle protein turnover on specific meat quality parameters

The growth rate of farm animals is related to muscle protein turnover. Thus the more positive the muscle protein balance becomes the better is the growth performance of farm animals, hereby making this parameter economically essential in meat production. Moreover, maintenance of maximal positive muscle protein balance throughout the life of farm animals with minimal feed intake (superior feed conversion rate) is decisive for the environmental load during production. The proteolytic potential in the muscle at the time of slaughter has long been regarded as an important factor in the tenderisation process in meat, which claims

[1] Systems biology is an emerging field that aims at system-level understanding of biological systems. What is meant by a "system level"? In contrast to molecular biology, which has its focal point on molecules, systems biology focuses on systems that are composed of molecular components. Although systems are composed of matters including diversities and functionalities of components, the core of systems lies in dynamics and it cannot be described merely by enumerating components of the system. Moreover, both structure and components of the system play indispensable roles in forming the symbiotic state of the system. Within this context, *i*) understanding of structure of the system, such as gene regulatory and biochemical networks, as well as physical structures, *ii*) understanding of quantitative and qualitative dynamics of the system, and construction of theory/models with powerful prediction capability, *iii*) understanding of control methods of the system, and *iv*) understanding of design methods of the system, are the landmarks to assess to exploit how much we understand the system. For a further definition of systems biology, see Ideker, Galitski & Hood (2001) and Kitano (2002).

high muscle protein turnover in healthy animals at the time of slaughter. Consequently, management of muscle protein turnover may enable control of the three important meat quality attributes - *price*, *tenderness* and *sustainability*.

Relationship between muscle protein degradation in vivo and tenderisation post mortem

Several reports suggest that a relationship between the rate of muscle protein degradation and the rate and extent of tenderness in meat exists (Table 1). Thus, in situations where the rate of protein degradation is decreased, this may lead to increased muscle growth but decreased tenderness, e.g. *i*) treatment with β-adrenergic agonists (for review see Beermann, 1993), *ii*) restrictive feeding (Kristensen, Therkildsen, Riis, Sørensen, et al., 2002), *iii*) bulls versus steers (Morgan, Wheeler, Koohmaraie, Crouse & Sawell, 1993) and *iv*) animals possessing the callipyge gene (Lorenzen, Koohmaraie, Shackelford, Jahoor et al., 2000). In contrast, treatment with porcine growth hormone results in increased muscle growth by stimulating both the rates of synthesis and degradation without change in tenderness, which most probably can be explained by simultaneous increase in lean tissue (Sevé, Ballèvre, Ganier, Noblet et al., 1993; Oksbjerg, Petersen, Sørensen, Henckel et al., 1995). Finally, short term fasting for 5 days leads to increased rate of muscle protein degradation resulting in increased tenderness in lambs (McDonagh, Fernandez & Oddy, 1999). The link between the rate of muscle protein degradation and tenderness development may be coupled to the calpain system, which is known to be the rate-limiting proteolytic system disassembling the myofibrillar proteins to their individual constitutive proteins (Goll, Thompson, Taylor & Ouali, 1992; Koohmaraie, Kent, Shackleford, Veiseth & Wheeler, 2002). Having established a link between the rate of protein degradation and postmortem tenderisation, the challenge becomes to implement this into a feeding strategy.

Table 1. Factors affecting protein degradation parameters and shear force in muscle and meat, respectively, of meat producing mammals.

	Muscle Growth	FDR[a]	Calpastatin activity	Shear Force
β-adrenergic agonist	⇑	⇓	⇑	⇑
pGH[b]	⇑	⇑		⇔
Bull *vs* Steer	⇑	⇓	⇑	⇑
Fasting (short time)	⇔	⇑	⇓	⇓
Fasting (long time)	⇓	⇓	⇑	⇑
Callipyge gene	⇑	⇓	⇑	⇑

[a]Fractional Degradation Rate; [b]porcine Growth Hormone

For decades it has been recognised that compensatory growth may occur after a period of feed restriction in most farm animals, and the phenomenon has continuously been supported to take place in pigs (McMeekan, 1940; Critser, Miller & Lewis, 1995), cattle (Abdalla, Fox & Thonney, 1988; Rossi, Loerch, Keller & Willett, 2001), broilers (Washburn & Bondari, 1978; Acar, Petterson & Barbato, 2001) and sheep (McManus, Reid & Donaldson, 1972; Kabbali, Johnson, Johnson, Goodrich & Allen, 1992). The degree of compensatory growth or the index is calculated as: Index = (A-B)/A, where A is the difference in weight between feed-restricted animals and *ad libitum* fed animals following the restriction period and B is the difference between *ad libitum* fed animals and compensatory animals at the end (at slaughter) of the period (Hornick, Van Eenaeme, Gerard, Dufrasne, & Istasse, 2000). The degree of compensatory index may dependent on;

- Degree of restriction
- Length of the restriction period
- Length of the period of compensatory growth
- Sex
- Genotype.

During compensatory growth both the rate of protein synthesis and degradation are elevated according to findings in rat (Milward, Garlick, Stewart & Nnanyelugo, 1975) and cattle (Jones, Starkey, Calkins & Crouse, 1990). Consequently, implementation of a compensatory growth strategy in production of meat-producing animals could be a means to improve tenderness of meat.

However, the increase in protein turnover during compensatory growth is dynamic. Thus, initially during compensatory growth increased protein synthesis is evident while protein degradation remains low as a consequence of the former restricted feeding regime. Later on also the rate of protein degradation increases gradually and eventually exceeds the rate of protein degradation of control *ad libitum* fed animals. Thus, one of the goals to successfully implement a compensatory feeding approach in the production of meat of high quality is to establish the length of the compensatory period which results in highest muscle protein degradation potential at the time of slaughter. Recently, Therkildsen, Riis, Karlsson et al. (2002) showed that the optimal length of the compensatory period in pigs was between 42 to 50 days with regard to elevated protein degradation rate at slaughter. This was verified by measuring the activity of μ-calpain, m-calpain and their inhibitor calpastatin as indicators of protein degradation and total RNA and elongation factor-2 (eEF-2) as indicators of protein synthesis.

Having established the optimal length of the compensatory period of pigs in relation to elevated protein degradation rate at slaughter we have continued studies on the effect of compensatory growth response on muscle protein turnover and tenderness (Therkildsen, Vestergaard, Busk, Jensen et al., 2004; Kristensen et al., 2002; Kristensen, Therkildsen, Aaslyng et al., 2004). In general these studies showed that both castrated male pigs and female pigs exerted compensatory growth, while tenderness was only improved in meat from female pigs, see Figure 1 (Page 15). The reason for this is unknown at present; however, the compensatory growth response resulted in decreased intra-muscular lipid in castrated male, which may counteract the increase in tenderness by elevated protein degradation. In female pigs no effect of compensatory growth response on intra muscular-lipid was observed (Kristensen et al., 2004).

Use of a compensatory growth approach has also been verified to increase tenderness in meat from cattle (Allingham et al., 1998), and studies including young bull calves are now in progress in our laboratory, where the compensatory growth response may prove to be of even larger significance regarding the tenderness of beef compared to pork. The preliminary data show that both muscle protein degradation and synthesis reach a maximum level in bull calves exhibiting compensatory growth that exceeds the level found in continuously *ad libitum* fed calves. Thus there may be a time during compensatory growth, which would be the optimal time of slaughter with respect to tenderness development *post mortem* (Therkildsen, 2004).

Relationship between feed regulated muscle protein turnover and sustainability
The compensatory growth model used by Oksbjerg, Sørensen & Vestergaard (2002) and Therkildsen et al. (2004) resulted in a more efficient production in terms of feed, as the feed conversion ratio was improved by 5% in pigs exerting compensatory growth over the total period of growth. This will result in a lower environmental load with nitrogen and phosphorus. Similar results have been found in ruminants with

improved feed conversion ratio in periods involving compensatory growth, whereas the overall feed conversion ratio of both the restrictive period and the realimentation period is likely to be the same as in ruminants fed *ad libitum* throughout the experimental period (Abdalla et al., 1988; Carstens, Johnson, Ellenberger & Tatum, 1991; Sainz, De la Torre & Oltjen, 1995; Rossi et al., 2001).

Feed-induced manipulation of muscle energy levels

Basic principles of post mortem muscle metabolism

Glycogen plays the leading role as substrate for energy metabolism of living muscle as well as in the *post mortem* metabolism during which muscle converts to meat. The *post mortem* conversion of muscle to meat is an energy-demanding process, which requires ATP. After exsanguination, the energy needed for the *post mortem* process is mainly obtained from ATP produced by anaerobic metabolism and partly from phosphorylation of ADP by creatine phosphate (Henckel, Karlsson, Jensen, Oksbjerg & Petersen, 2002). The anaerobic metabolism of glycogen results in formation of lactate and a simultaneous decline in pH. Both the degree (Hamm, 1960; Bendall, 1973) and rate (Offer, 1991; Offer & Cousins, 1992) of *post mortem* metabolism are known to influence critical meat quality attributes, which is why control of glycogen and creatine phosphate levels at the time of slaughter may enable control of the vital technological quality parameters, e.g. water-holding capacity and sensory characteristics including colour and juiciness.

Influence of feeding-regulated glycogen levels on specific meat quality parameters

Sugar feeding, e.g. supplementation of high levels of sucrose or other digestible carbohydrate sources, a few days prior to slaughter or during overnight lairage has for decades been known to increase muscle glycogen stores and thus reduce $pH_{24\ h}$ (Briskey, Bray, Hoekstra, Phillips & Grummer, 1959; Fernandes, Smith & Armstrong, 1979). This is a short-term effect (Fernandez, Tornberg, Mågård & Göransson, 1992), where glucose is transported from the gastrointestinal tract via the circulation to the liver and the muscle, and subsequently incorporated into the glycogen stores. Such an increase in glycogen stores does not call for changes in the expression of any of the key enzymes of glycogenesis, as the increase is solely of a regulatory nature.

In contrast, other diet-induced regulations of the glycogen pools in monogastrial animals require changes in metabolic pathways and the glycogen synthesis apparatus. In this context muscle glycogen stores are reduced in rats (Lapachet, Miller & Arnall, 1996) and rabbits (Gierus & Rocha, 1997) using diets with a high fat content and digestible carbohydrate content. Similar results have been obtained after feeding standard diets with increased fat content to pigs from 25 kg live weight to slaughter (Lauridsen, Nielsen, Henckel & Sørensen, 1999). Recently, feeding diets high in fat (approx. 17-18%) and protein (22-24%) in combination with a low content of digestible carbohydrate (< 5%) to pigs during the last three weeks of finishing has been shown to reduce muscle glycogen stores in *M. longissimus dorsi* without influencing the overall growth performance (Rosenvold, Lærke, Jensen, Karlsson et al., 2001a; Rosenvold, Petersen, Lærke, Jensen et al., 2001b; Rosenvold, Lærke, Jensen, Karlsson et al., 2002). The combination of low digestible carbohydrate and high fat content in the diet causes a muscle glycogen-reducing effect (Figure 2, Page 16). The reduction in muscle glycogen content improved the WHC of *M. longissimus dorsi*, *M. biceps femoris* and *M. semimembranosus* (Rosenvold et al., 2001a; 2002). This reduction in muscle glycogen level did not result in higher $pH_{24\ h}$, but a higher $pH_{45\ min}$, suggesting a diet-induced delayed *post mortem* conversion of glycogen to lactate.

Bee (2001) has shown that only a minor reduction in digestible carbohydrate content (~20%) with a simultaneous iso-energetic supplement in fat has no influence on *post mortem* glycogen stores in porcine *M. longissimus dorsi* and the red part of *M. semitendinosus*. Hence, feed-induced reduction in muscle glycogen content, which affects pH_{45} and WHC in the porcine muscle, as observed by

Rosenvold et al. (2001a; 2001b; 2002b) seems to claim a critical ratio between fat and digestible carbohydrate. In support of this theory, Leheska, Wulf, Clapper, Thaler, and Maddock (2002) found no effect on *post mortem* glycolytic potential or pork quality attributes upon feeding a low digestible carbohydrate/high protein diet during the last two weeks prior to slaughter.

In ruminants several studies too indicate that muscle glycogen levels are at least to a certain extent responsive to finishing composition (for review see Immonen, 2000), despite the general belief that muscle glycogen stores are hardly affected by diet composition, as the ruminal fermentation is expected to dominate the availability of gluconeogenic precursors for hepatic glucose production. In an experiment with intensive (concentrate) or extensive (pasture) feeding with or without finishing feeding with concentrate of Friesian bulls, Vestergaard, Oksbjerg & Henckel (2000) found a decreased concentration of glycogen in *M. longissimus* and *M. semitendinosus* from bulls fed with pasture (E) compared with bulls fed on concentrate (I). However, when the E-bulls were finished on a concentrate ration for 10 weeks and had a compensatory feed intake compared with the I-bulls, the glycogen concentration increased and exceeded the level seen in the I-bulls.

Although muscle glycogen stores at the time of slaughter have long been recognised to be decisive for meat quality (Briskey, 1964), the physiological nature of the regulation of muscle glycogen stores is poorly understood (Graham & Adamo, 1999; Roach, 2002). This applies especially in *post mortem* muscle as existing knowledge is mainly based on findings in *in vivo* muscle. However, the recent identification of a key enzyme, glycogenin, and renewed interest in the existence of the two forms of glycogen, proglycogen and macroglycogen, may remedy this fact.

Proglycogen and macroglycogen are distinguished on the basis of size and protein content. The existence of two pools of glycogen was first studied extensively forty years ago (Stetten & Stetten, 1960). However, Lomako, Lomako, Whelan, Dombro, Neary & Norenberg (1993) were the first to describe the two forms in detail. The pool with a high ratio of carbohydrate to protein (maximum M_w ~10^4 kDa) is termed macroglycogen. The pool with relatively high protein content to carbohydrate (M_w<400 kDa) is termed proglycogen. It is known that the macroglycogen pool increases with high muscle glycogen concentrations (Jansson, 1981; Adamo & Graham, 1998; Hansen, Derave, Jensen & Richter, 2000; Graham, Adamo, Shearer, Marchand & Saltin, 2001). Moreover, dietary energy favours the *post* exercise synthesis of proglycogen over that of macroglycogen (Adamo, Tarnopolsky & Graham, 1998; Derave, Gao & Richter, 2000) and the metabolism of the two pools appears to depend on the type of exercise (Asp, Daugaard, Rohde, Adamo & Graham, 1999; Graham et al., 2001).

Proglycogen is degraded in favour of macroglycogen during the first 45-60 min *post mortem* in porcine muscle (Charpentier, 1966; Rosenvold, Essén-Gustavsson & Andersen, 2003). Moreover, total glycogen and the concentration of proglycogen have been found to be higher in pigs showing a rapid pH decline *post mortem* and subsequent development of inferior meat quality (pale, soft and exudative meat) (Wismer-Pedersen & Briskey, 1961). Reduced muscle glycogen stores as a result of a 3-week strategic finishing period are reflected in a reduction in the macroglycogen pool (Rosenvold et al., 2003). Interestingly, the subsequent reduction in the rate of *post mortem* glycolysis observed in muscle from these animals was due to reduced metabolism of the proglycogen pool. Recently, it was found that the increased glycogen level in pigs carrying the RN^- gene is reflected in larger macroglycogen stores (B. Essén-Gustavsson, unpublished observations), which might explain why early *post mortem* glycolysis is not enhanced in pigs carrying the RN^- gene compared with that of non-carriers. These preliminary data clearly show that a complete understanding of how feeding influences glycogen pools might be critical in setting up quality assurance schemes for the production of high quality meat.

Significance of vitamin E supplementation on muscle glycogen level

Vitamin E stripped diets have been reported to reduce liver glycogen stores in rats (Schroeder, 1974) and muscle glycogen stores in pigs (Lauridsen et al., 1999). Recently, we found that supra-nutritional vitamin E supplementation on the day prior to slaughter in both a strategic finishing diet known to reduce muscle glycogen stores and control diets gave rise to significantly higher muscle glycogen levels and tended to decrease WHC of meat from supplemented pigs (Rosenvold et al., 2002). Schroeder (1974) suggested that metabolic changes caused by vitamin E depletion were controlled by cyclic AMP, which is central in the co-ordinated control of glycogen synthesis and breakdown (Stryer, 1988). The hypothesis was rationalised on the fact that the result of vitamin E deficiency (e.g. increased catabolic activity) can be mimicked by the administration of glucagon, which stimulates glycogenolysis, lipolysis and the activity of certain catabolic enzymes through an increase in the cellular concentrations of cyclic AMP (Jost & Rickenberg, 1971 in Schroeder, 1974). It has also been noted that adenylate cyclase, the enzyme responsible for the synthesis of cyclic AMP, is localised mainly in the cellular membranes where vitamin E could play a major role (Srivastava, Robin, & Thakur, 1992). Moreover, vitamin E possibly inhibits protein kinase C (Mahoney & Azzi, 1988), which catalyses the phosphorylation and inactivation of glycogen synthase - the rate-limiting enzyme for glycogen synthesis (Ahmad, Lee, DePaoli-Roach, & Roach, 1984). However, the mechanism behind this effect of vitamin E on protein kinase C is still debated (Azzi et al., 2001). Thus, a vitamin E-induced inhibition of protein kinase C would increase the activity of glycogen synthase and stimulate glycogen synthesis, as found in the studies by Schroeder (1974), Lauridsen et al. (1999) and Rosenvold et al. (2002). Even though none of these studies can identify the mechanism for the significance of vitamin E supplementation on muscle glycogen levels, they do indicate that vitamin E has a direct effect on the mechanisms controlling glycogen metabolism. Considering the positive effect of vitamin E supplementation of farm animals on other meat quality parameters, e.g. colour and oxidative stability (Jensen, Lauridsen, & Bertelsen, 1998) and the negative effect on WHC in the study of Rosenvold et al. (2002), one could speculate whether an optimal level of vitamin E supplementation exists, where improved colour and oxidative stability are obtained before the possible elevation of muscle glycogen stores and increased drip formation overrides these positive effects. However, this calls for a better understanding of the influence of vitamin E supplementation on muscle glycogen stores.

Creatine

In contrast to many years of general belief, dietary supplementation with creatine monohydrate in humans, has been found to increase intramuscular creatine load by 20% (Balsom, Söderlund, Sjödin & Ekblom, 1995; Greenhaff, 1996). Moreover, it has been suggested that creatine supplementation increases the muscle energy stores as phosphocreatine (Balsom, Söderlund & Ekblom, 1994; Casey Constantin-Teodosiu, Howell, Hultman & Greenhaff, 1996). Supplementation with creatine monohydrate is known to increase weight gain (Maddock, Bidner, Carr, McKeith, Berg & Savell, 2000; Berg & Allee, 2001; Stahl, Allee & Berg, 2001; Young, Bertram & Oksbjerg, 2004a), probably by increased water retention in lean tissue (Balsom et al., 1995; Juhn, 1999). Moreover, muscle protein may also increase as the synthesis of myosin heavy-chain probably increases upon creatine supplementation in chicken skeletal muscle cells (Ingwall, Morales & Stockdale, 1972). A feed-induced increase in energy stores, as a consequence of elevated muscle phosphocreatine level should according to the generally accepted theory delay the glycogen metabolism in *post mortem* muscle and thus slow down the pH decline in the muscle, which minimize potential protein denaturation during the conversion of muscle to meat. A reduction in protein denaturation will consequently increase water-holding properties of the meat (Offer, 1991; Bertram, Dønstrup, Karlsson et al., 2001). Creatine monohydrate supplementation for five days has been shown to reduce the rate of the early *post mortem* pH decline and decrease cooking loss (Maddock et al., 2000; Berg & Allee, 2001; Stahl et al., 2001). Moreover, the incidence of PSE was significantly reduced in supplemented pigs (Maddock et al., 2000). However, extending the supplementation

period to 10 or 15 days had no (O'Quinn et al., 2000) or negative (Stahl et al., 2001) effect on technological pork quality, and high concentrations of creatine had even adverse effects on chicken meat quality (Young, Karlsson & Henckel, 2004b). The PSE-reducing effect of creatine monohydrate supplementation observed by Maddock et al. (2000) could be explained by the fact that half the pigs were carriers of the Halothane gene, and that the effect of creatine monohydrate supplementation was most pronounced in meat from pigs carrying the gene. However, in a consecutive study Maddock et al. (2002) found no significant effects of creatine monohydrate supplementation on technological pork quality. Effects of dietary creatine on meat quality traits are thus very diverse, and recent results by Young et al. (2004a) also demonstrate how differently two breeds of pigs are affected by creatine supplementation. Young et al. (2004a) showed that pure Duroc pigs had an increased pH both early *post mortem* and after 24 h, as well as reduced drip loss after dietary creatine treatment. Similar dietary creatine addition, however, had no systematic affect on *post mortem* pH in pure Landrace pigs, and drip loss was not significantly affected, although two independent drip loss methods indicated increased drip loss upon creatine supplementation (Figure 3, Page 17). These opposing results indicate that genetics play a major role for meat quality attributes related to dietary creatine treatment of pigs, and could be used in the elucidation of underlying mechanisms.

Genomics versus feeding - Nutrigenomics

The genetic influence on meat quality comprises differences among breeds as well as differences among animals within the same breed. With the exception of a few monogenic effects (major genes) in the different farm animals, e.g. the calipyge gene in sheep, the myostatin gene in cattle, the halothane, RN^-, 'intramuscular fat' and 'androstenone' genes in pigs, the heritability of most attributes relating to the quality of meat is low to moderate (0.15–0.30) (Sellier & Monin, 1994; Sosnicki, Wilson, Sheiss, & Vries, 1998). These differences in quality attributes are caused by a large number of genes with small effects, polygenic effects. The best approach to genetically improve meat quality is to identify relevant DNA-markers directly in populations under selection (de Vries, Faucitano, Sosnicki & Plastow, 2000). This calls for continuous meat quality measurements on the nucleus population of breeding organisations. However, this is a very long-term approach, which is extremely expensive, and as full assessment of meat quality can only be done after slaughter, the data have to be collected on culled animals and cannot be obtained on potential breeding animals. Another way to utilize the genetic potential of farm animals, considering the emerging development of molecular biological techniques, is the introduction of a molecular approach, e.g. DNA therapies, for altering biological processes of importance for meat quality (Grant & Gerrard, 1998). However, such an approach will hardly become ethically acceptable for conventional exploitation.

Alternatively, a fundamental understanding of the biological processes, which are directly linked to meat quality attributes, will rationalise a marker-assisted DNA approach, as this allows sampling on live breeding animals. Considering that most traits of interest for meat quality have a multifactorial background, i.e. meat quality attributes are due to interaction between the genetic potential and the environment, with feeding being the most decisive factor in meat production renders that a nutrigenomic approach seems most optimal in the exploitation of biological processes of direct importance for vital meat quality attributes. A nutrigenomic approach in relation to meat quality will in this way kill two birds with one stone, as it will support both a marker-assisted DNA approach and include a basic understanding of the most pronounced interaction of importance for meat quality, namely the interaction between the genome and the feed.

Nutrigenomics – an emerging strategy in modern meat science?

As illustrated in the above paragraphs, feeding has a regulatory effect on biological processes in muscle, which is directly reflected in the quality of the meat from the fed animals. This is in accordance with the fact that specific diet components *inter alia* are known to regulate gene expression within the cells. The knowledge of the complex interaction between the individual nutrients and their interaction on the genome of farm animals, which represent 30-40,000 genes within each species, is practically an unexplored area. The major reason for this is that the technical resources for such an understanding have only become accessible within the past few years. The ongoing mapping of the genomes of the principal farm animals together with the progress within information technology and molecular biological techniques will undoubtedly accelerate this process in the future.

The key element that distinguishes nutrigenomic from nutrition research is that the observable response to diet, or phenotype, is analysed or compared in different genotypes (or individuals). Classical nutrition research essentially treats all test individuals as genetically identical, and hereby misses a considerable part of the potential differentiation, which is essential in the future production of meat. Likewise, molecular biology and biochemical approaches show the same limitations as classical nutrition. Consequently, to include the environments, here represented by feeding, in relation to the expression or activity of variant forms (within groups or individuals defined by Single Nucleotide Polymorphisms (SNPs), haplotypes, and other polymorphism) of normal genes will allow a new essential understanding, which will have significant implications on future strategies to control meat qualities.

Conclusions

Considering a pro-active quality control system in the future production of high quality meat, the traditional way of using feeding as a quality control tool has outlived its usefulness. However, as emphasized by above results regarding fed induced control of muscle protein turnover and muscle energy levels and subsequent influences on important meat quality attributes of importance for the industry and/or consumers, feeding seems the optimal tool to explore biological processes of importance for meat quality development. Via a nutrigenomic approach feeding could be the base to initiate a systems biology line of thought in meat science, which subsequently could change from the present qualitative strategy to a quantitative strategy enabling development of optimal decision support systems for future meat quality control.

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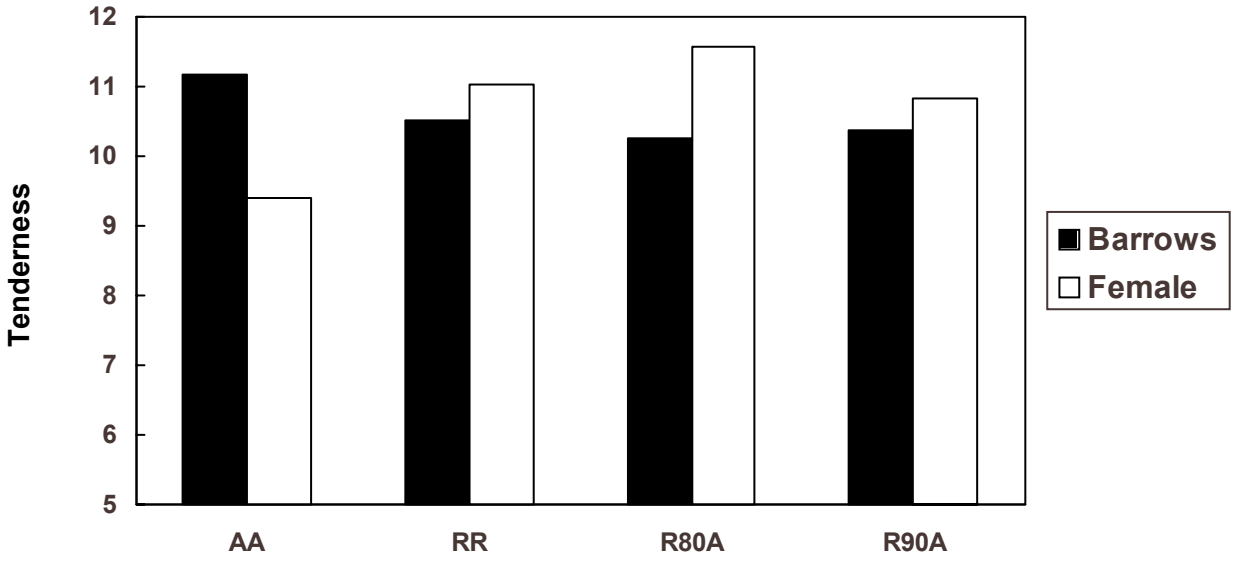

Figure 1. Tenderness (scored at a scale from 1-15, with 15 being extremely tender and 1 being extremely tough) of muscle longissimus dorsi from female and castrate pigs fed different from weaning (day 28) to slaughter (day 140). AA: Pigs fed *ad libitum* throughout the experiment; RR: Pigs fed restrictively (60% of *ad libitum*) throughout the experiment; R80A: Pigs fed restrictively (60% of *ad libitum*) from weaning to day 80 followed by *ad libitum* feeding to slaughter; R90A: Pigs fed restrictively (60% of *ad libitum*) from weaning to day 90 followed by *ad libitum* feeding to slaughter.

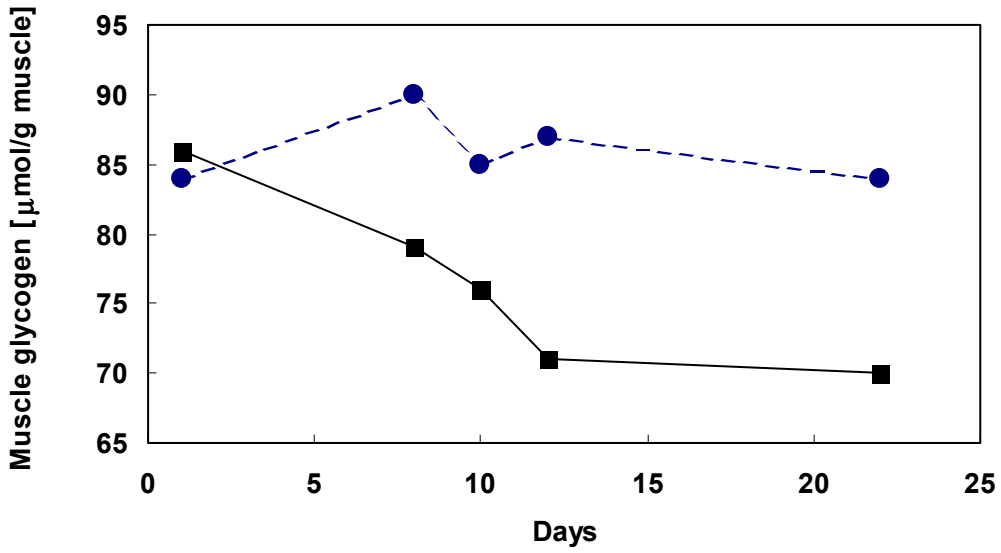

Figure 2. Muscle glycogen levels in muscle longissimus dorsi from slaughter pigs fed control diet (■—■) and after change to experimental diet (●- -●). Control diet: 19.5% soybean meal; 58% barley; 21% wheat; 1% animal and vegetable fat; 0.2% vitamin mineral mixture. Experimental diet: 50% rape seed cake; 10% soybean meal; 33% raw potato starch; 1% sugar beet molasses; 6% animal and vegetable fat; 0.2% vitamin mineral mixture.

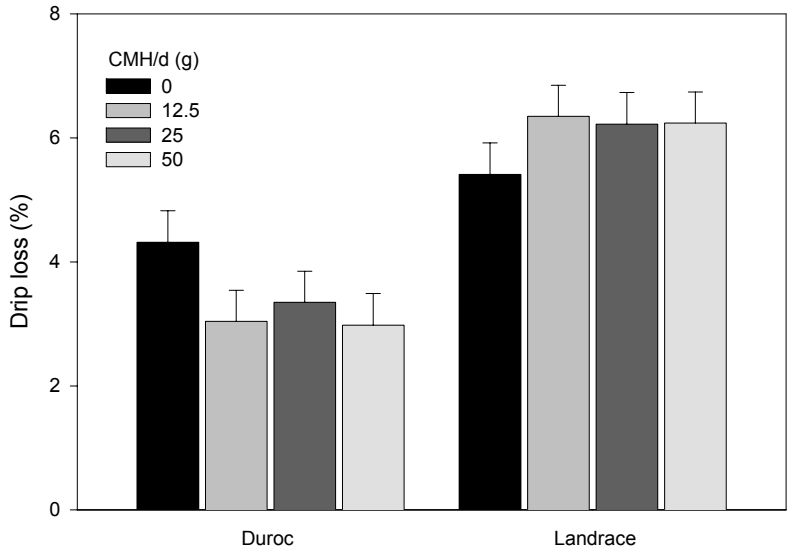

Figure 3. Water-holding capacity determined as drip loss (%) from muscle longissimus dorsi of creatine monohydrate (CMH) supplemented Duroc and Landrace pigs. Values are given as LSmeans.