Swine growth physiology

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Abstract

Therefore, the aim of this literature review is to present the aspects related to the growth and development of muscle tissue and the animal as a whole, as well as to discuss the possible technical manipulations employed to improve the quantity and quality of meat produced. In recent years the pig industry has been focusing on the selection of high productivity sows, aiming at increasing the number of weaned pigs / sow / year. However, the emphasis on sow prolificity has resulted in a substantial increase in the number of piglets born / calving, having a direct effect on piglet quality, with reduced birth weight and unevenness of piglets. In addition to the quantitative aspects related to animal performance, it should be considered that the quality of the final product is also of extreme importance, being its main objective meat production, the factors that interfere in this process also deserve special attention. Small piglets reach slaughter weights lower than the heaviest ones and / or take longer days to reach their sales weight, which consequently costs the production cost.

Key-words: Pig Farming, Development, Animal Production

Introduction

In swine production, knowledge of the factors that characterize the growth and development of tissues and the organism as a whole, as well as their implications, are of fundamental importance for the development of any breeding program, knowledge of nutritional requirements, adequacy of nutritional and sanitary management, ambience and the definition of slaughtering age, alteration of carcass and the quantity and quality of meat of these animals are also determinants in pig farming.

Despite great genetic and management progress over the years, pre-weaning piglet mortality still represents a major economic constraint on modern pig farming (Leenhouders, 2002). This becomes a reality when we encounter pre-weaning mortality rates that remain relatively constant from the mid-1960s to the present day, at around 16% in the vast majority of herds (Quiniou, Dagorn and Gaudré, 2002).

According to Quiniou, Dagorn and Gaudré (2002) the weight of the piglet at birth is considered one of the main factors directly related to its survival, as well as its weight at weaning and subsequent performance, until the moment of slaughter. The growth of the animal is characterized by the rate of weight gain, length, height and circumference as a function of age, and this development implies changes in body conformation and organism functions. This process of animal growth and development takes place from conception until slaughter.

Nowadays, breeding swine strains are being genetically improved in order to become hyperprolific. In addition, changes inherent in management have made it possible to increase the
number of weaned piglets / female / year, from an average of 21 to 23 piglets (Merks et al., 2000), however, some genetics have already reached a total of 30 weaned piglets / female / year. As a consequence, there were problems related to the weight of the piglet at birth and the appearance of uneven litter, contributing to greater weight variability among piglets (Antunes, 2007). Another important intrinsic factor is the viability and vitality of less favored piglets, due to their low birth weight and possible exposure to stressful events during calving (Damgaard; Rydhmer; Lovendahl and Grandinson, 2003).

Thus, the objective of this literature review is to discuss the aspects related to the growth and development of the muscle tissue and the animal as a whole, as well as to discuss the possible technical manipulations aimed at improving the quantity and quality of meat produced.

**Maternal environment and prenatal growth in swine**

In swine production, the gilt represents the fundamental unit of production systems, and its good reproductive performance interferes not only in the immediate results of the herds, but also in their future productivity. Zangeronimo (2013).

According to Martin Rillo et al. (2001), piglets born from gilts are lighter at birth and weaning when compared to piglets from sows of more advanced calving. In addition, after weaning, piglet-descendent piglets are more susceptible to disease and have the highest mortality rates (Burkey et al., 2009). In more controlled breeding systems developed in collaboration with commercial producers, sows born from first calving sows invariably weigh less than 1.5 kg at birth and are not selected as replacement females because of the effects of low birth weight on subsequent performance (Foxcroft et al., 2004).

The phenomenon of fetal programming, in which early intrauterine malnutrition (resulting from inadequate maternal food intake or inadequate nutrient transfer) permanently alters body structure and function, is well documented in animals (Langley-Evans, 2006).

In practical terms, various body systems and tissues can be affected by prenatal programming, including cardiovascular, respiratory, endocrine, and immune systems, as well as skeletal muscle, bones, liver, and kidney (Godfrey, 1998). Some changes in maternal diet during pregnancy have shown permanent changes in offspring, including changes in blood pressure, cholesterol metabolism, insulin secretion, and especially tissue-specific effects, resulting in changes in muscle fiber numbers (Dwyer et al., 1995; Godfrey, 1998).

**CIUR** is defined as impaired growth and development of the mammalian embryo / fetus or its organs during pregnancy. It can be measured as fetal weight or birth weight less than two standard deviations from mean body weight for gestational age (WU et al., 2008). Among domestic animals, pigs represent the species in which IUGR occurs most severely (Wu et al., 2006).

Wooton et al. (1993) observed that the extremes of retarded intrauterine growth (CIUR) were identified within a discrete subgroup of fetuses. In addition, based on data from subsequent studies on the association between within-litter differences in prenatal development and postnatal survival and development, VAN Der Lende and Jager (1991) concluded that the lower pre-weaning growth of scrap
pigs. It could not be fully explained based on its low birth weight. The authors also suggested that CIUR would have a more complex effect on the developmental potential of those animals.

It has been confirmed that litter size affects the birth weight of piglets Bérard et al. (2008). In addition, the reduced growth of swine fetuses is exacerbated by the practice of feed restriction programs (e.g., 2.0 kg of feed/day) in the swine production routine throughout the gestation period in order to avoid gain of feed. Excessive weight of sow as reported by Kim et al. (2009). In some litter, almost all piglets have low birth weight (less than 1.10 kg), particularly when part or most of the gestation period is subjected to environmental stress (e.g., temperature extremes and/or disease).

**Postnatal Growth Rate**

The postnatal growth rate of pigs is determined by several factors, and the number of muscle fibers is the most discussed factor in the current literature. Prenatal events (such as genotype, nutrition, oxygen and nutrient flow through the placenta) can influence muscle fiber differentiation and determine the number of these fibers as well as their growth rate.

However, in addition to the number of fibers, an animal's growth depends in part on its ability to digest and assimilate ingested macromolecules, and the gastrointestinal tract is of fundamental importance for swine growth (King et al., 2000).

In pigs, IUGR is one of the main contributing factors to high neonatal mortality due to small bowel development abnormalities (Wang et al., 2008; Jiang et al., 2009). Compared to animals with normal intrauterine growth, animals affected by CIUR have lighter organs and have dysfunctions of the gastrointestinal tract. It is speculated that impairments in gastrointestinal development are the major causes by which animals with CIUR grow more slowly than those with normal intrauterine growth (Wang et al., 2010).

The small intestine is the main organ for terminal digestion and nutrient absorption. The gut is also a defense barrier against dietary pathogens, carcinogens and oxidants (Wu et al., 1998; Wu et al., 2009a).

The development of the GI system can be divided into three phases. The prenatal phase which is characterized by minimal stimulation of the gastrointestinal lumen; the perinatal phase that is associated with milk suction; and the post-weaning phase corresponds to the adaptation of the digestive system to use solid food components (Zabielski et al., 2008).

The rate of swine GI tract metabolism and maturation is extremely rapid during the neonatal phase, when the animal ceases to be parenterally fed and becomes completely dependent on enteral nutrition via the GI system (Reeds et al., 1993). Before birth, the gastrointestinal tract is exposed only to small amounts of complex nutrients via amniotic fluid intake, it is known that at this stage, the cellular turnover rate and probably the oxygen rate required by cellular metabolism is much lower before that after birth (Trahair and Sangild, 2002).

At birth, the flow of oxygen in the arterial blood increases dramatically (from 50 to 100% oxygen saturation) as a result of circulatory changes and onset of independent respiratory function (Sangild et al., 2000).

At birth the pig doubles its weight within the first week of life, however, the weight of the small intestine increases much faster, doubling in the first 2 to 4 days. Such drastic changes involve three main mechanisms: (a) increased local blood flow from the gastrointestinal tract in parallel with reduced basal vascular resistance; (b) accumulation of colostrum proteins in the enterocytes as a result of an open “intestinal barrier” and, finally, (c) changes in epithelial cell turnover, i.e., increased number of mitoses accompanied by inhibition of apoptosis, which results in a two-fold increase in the mitosis/apoptosis ratio in the first two days after birth (Nankervis et al., 2001).

Vacuolated fetal enterocytes have been observed in mammalian fetuses since the second trimester of pregnancy. They first appear at the top of the proximal small intestine villi and slowly enlarge to the rest of the intestine. A unique feature of the fetal enterocyte is the occurrence of cytoplasmic vacuoles of various dimensions that constitute the apical canalicular system (ACS) (Baintner, 1986). The characteristic of these cells is the ability to transport intact proteins from the gut lumen through the epithelium to circulation (subpopulation of cells with transport vacuoles) or to be digested within the cell (cell subpopulations with digestive vacuoles) (Baintner, 2002). Transport vacuole producing enterocytes are present throughout the small intestine and play a key role in the absorption of colostrum...
In piglets, these enterocytes are observed only during the first 2 to 3 days of postnatal life. The enterocytes that produce the digestive vacuoles are present in the lower small intestine and support the digestive process by intracellular enzymatic digestion of milk protein. Enterocytes with digestive vacuoles gradually disappear from the proximal jejunum to the ileum. In piglets, the whole process takes about 3 to 4 weeks. The rate of disappearance of the vacuole is closely associated with the maturation of the intestine, i.e., the shift to an adult type of digestion and absorption (Radberg et al., 2001). Adult enterocytes lack the ability of ACS and lose the ability to produce large vacuoles. The only mature epithelial cells where the ability to carry intestinal contents persists, as in vacuolated transport enterocytes, are the M cells that cover Peyer's plaque (Trahair and Sanglid, 2002).

A change in the balance between mitosis and apoptosis in epithelial cells is vital for maturation (Godlewski et al., 2005). Early in the postnatal period, development of the intestinal mucosa is associated with remodeling and modification of digestive and absorptive functions of the intestine (Skrzypek et al., 2007). The process of remodeling of the small intestine mucosa concerns the increased absorption and adaptability of brush edge enzymes, both improving food digestion and nutrient absorption. Remodeling can be achieved by modifying enterocyte functions and/or by replacing old epithelial cells (fetal enterocytes) with a new generation of enterocytes (adult ones) (Baintner, 2002). Modification of enterocyte structure and function (i.e., disappearance of lysosomal vacuoles) has a significant impact on intestinal barrier closure.

Currently, no artificial feeding system (milk, artificial milk powder, nor any feeding with other compositions such as lactose, glucose solutions) can reproduce the developmental characteristics obtained from ingestion of maternal colostrum. In addition, the high specificity of colostrum, especially regarding the composition of hormones and bioactives, precludes the use of colostrum from other species as substitutes (Zabielski et al., 2008).

The first intake of colostrum provides nutrients not only for growth and development, but also provides passive immunity through intestinal absorption of colostrum immunoglobulins. The ability of enterocytes to absorb large molecules by endocytosis is particularly important for farm animals (piglets, lambs, calves, foals, etc.) because the placenta of these species does not allow immunoglobulins to be transferred to the fetus via circulation before birth (Jensen et al., 2001).

After birth, villus height increases rapidly by the action of a large volume of blood distributed in the intestinal mucosa. Numerous transverse grooves allow villus stretching without extra energy cost. Within a few days after birth, the number of transverse grooves and their depth are dramatically reduced (Skrzypek et al., 2005).

After the first ingestion of colostrum, the volume of enterocytes is markedly increased due to the resumption of colostrum proteins and storage in large vacuoles. Vacuoles can occupy half the volume of the cell and sometimes even more. Skrzypek et al. (2007) observed that the height of duodenal enterocytes increased from 14.6 μm at birth to 20.0; 25.7 and up to 33.7 μm on days 3, 7 and 14 after birth, respectively. The changes observed in the jejunum and ileum after birth were not as drastic (Skrzypek et al., 2007).

The kinetics of intestinal epithelial reconstitution depends on a variety of hormones, growth factors and regulatory peptides that are present in colostrum/milk and/or are released locally in the gastrointestinal mucosa. These substances exert effects on proliferation, differentiation and programmed cell death. These include insulin, leptin, ghrelin, epidermal growth factor (EGF), insulin-like growth factors (IGFs) and tumor necrosis factor-α (TNF-α) (Zabielski, 1998).

Some studies have reported the influence of delayed intrauterine growth on IGF-I (insulin-like growth factor) expression and growth hormone and insulin receptor receptors in the intestinal mucosa. Growth hormone (GH), a protein secreted by the pituitary gland, acts directly and indirectly via IGF-I induction to promote tissue growth and regulate metabolism. GH induces IGF-I secretion by the liver and peripheral target tissues. Evidence suggests that locally secreted IGF-I is responsible for most GH-dependent growth effects. However, it is certain that GH and IGF-I act synergistically on target tissues (Laron, 2001). In this sense, Wang et al. (2005) observed that animals affected by CIUR (birth weight less than two standard deviations of the mean weight of the total population) presented lower IGF-I mRNA expression in the intestinal mucosa (P <0.05) and
tended to lower GH receptor and mRNA insulin expression in the intestinal mucosa.

Decreased IGF-I expression in the intestinal mucosa in cases of CIUR may be related to decreased growth hormone receptor expression in the intestinal mucosa. Therefore, it is reasonable to speculate that IGF-I expression would be regulated by nutritional and hormonal factors, however, these studies would need to be further investigated. Therefore, it can be said that changes in endocrine homeostasis contribute to lower growth rates in piglets affected by CIUR. In addition to the previously described consequences, restricted fetal growth in pigs is also associated with low birth weight and increased risk of fetal and neonatal death (Xu et al., 1994).

**Influence of birth weight on pigs**

Most low birth weight piglets die before weaning and those who survive suffer permanent growth retardation. In a study conducted between 2003 and 2009, Wu et al. (2009b) observed that piglets weighing less than 1.10 kg at birth would represent 76% of pre-weaning deaths.

Since high pre-weaning mortality is a major problem in pig production, optimizing embryonic-fetal development and consequently birth weight is of utmost importance to increase the efficiency of pork production.

It has been confirmed that litter size affects the birth weight of piglets (Bérard et al., 2008). With the increase in births, one of the main consequences of hyperprolificity is reduced birth weight. Under current conditions, the correlation between number of births and birth weight is inversely proportional, according to Quiniou et al. (2002) the average litter weight was reduced between 25 and 35 g for each additional total born piglet and the percentage of live births below 1 kg increased from 7.0 to 23.0% in litter over 16 piglets.

Besides resulting in smaller individual weights of piglets at birth, another consequence of the significant increase in the number of births is the increase in the coefficient of variation of weight within the litter, ie, greater unevenness. Milligan et al. (2002) found a positive correlation between the number of live births and the coefficient of variation, even in litter with an average of 10.4 live births and 11.8 total births. Thus, a higher percentage of piglets has been born in the low viability categories, increasing mortality rates in the low viability categories.

Some factors have a direct influence on birth weight, such as birth order. There is a linear increase in weight based on the first birth and a fall from the sixth birth onwards. Smith et al. (2007) pointed to the fourth parturition being the birth order where there is the peak birth weight, although some studies have shown that this peak can occur in the fifth parturition mothers.

According to Milligan et al. (2002), the increase in birth weight between the first and second parturition would be associated with the increase of the uterine space. Also according to these authors, the reduction in birth weight and the increase in weight variation in older mothers would be due to the increase in ovulation rate and litter size.

Additionally, birth weight is considered the most important factor for piglet survival (Kerr and Cameron, 1995; Roche and Kalm, 2000). Low birth weight piglets have a lower body energy level, high sensitivity to cold, take longer to breastfeed, and can hardly compete for the best teats, so they represent the category with the least chance of survival (Lay et al., 2008).

Another important birth weight interference is on colostrum intake. Devillers et al. (2008) concluded that the main factor determining colostrum intake was birth weight, as it is related to the vitality and ability to stimulate the mammary gland and extract colostrum. The same authors pointed out that the heterogeneity of piglet weight may also be determinant for the variation of individual colostrum intake. For every 100 grams more birth weight, there was an increase of 28 grams of ingested colostrum.

Mortality in low birth weight categories is high compared to piglets weighing more than 1.0 kg. In the work of Furtado et al. (2007), the category of births below 0.9 kg had mortality of approximately 30%. In research by Quiniou et al. (2002), only 15% of piglets weighing less than 0.6 kg at birth remained alive at weaning and 48% of those born between 0.6 and 0.8 kg. Recalling that the higher mortality in light animals may be more or less expressive, as it is influenced by the quality of care for these light piglets.

**Birth Weight Multiplier Effect**

In addition to the effect on survival / mortality of these piglets, birth weight also interferes with weaning weight and post-slaughter performance.
(Panzardi et al., 2009). According to the literature, small piglets at birth continue to have lower weights throughout the production phases (Quiniou et al., 2002) and require a larger number of days to reach slaughter weight than their larger siblings. (Wolter and Ellis, 2001; Gondret et al., 2006).

Studies on the effect of birthweight on future pig performance are quite old, dating back to the 1970s and 1980s (Powell and Aberle 1980; Campbell & Dunkin 1982). In more recent studies, several authors have confirmed the effect of birth weight on the performance of piglets in the nursery and finishing phases, which has been called the weight multiplier effect (Quiniou et al., 2002; Rehfeldt and Kuhn, 2006; Beaulieu et al., 2010).

All these studies showed that the multiplier effect of weights is real and consolidated, and the main biological explanation for this effect, according to the literature, is related to the development of muscle fibers in pig fetuses, which occurs differently between piglets, light and heavy at birth (Rehfeldt and Kuhn, 2006).

However, some reports did not find consistent variation in the total number of fibers in relation to birth weight (Dwyer et al., 1993). According to Gondret et al. (2006), piglets with lower birth weight had a lower total number of muscle fibers compared to their heavier siblings, a characteristic that would be defined before birth (Wigmore and Stickland, 1983), but presented hypertrophied slaughter fibers (Kuhn et al., 2002; Gondret et al., 2005).

Gondret et al. (2006) further observed that small piglets at birth also needed more days to reach slaughter weight and accumulated more fat in the carcass than their heavier siblings. However, the claim that birth weight would affect swine carcass characteristics remains quite controversial, and some studies associate low birth weight with lighter carcasses (Bee, 2004; Poore and Fowden, 2004; Gondret et al., 2005) and other studies show that neither birth weight nor number of muscle fibers would influence carcass parameters (Beaulieu et al., 2010; Bérard et al., 2008).

Dwyer et al. (1993) demonstrated that birth weight is a good indicator of growth rate during the early stages of postnatal development (from birth to approximately 70 days of age). However, birth weight does not necessarily determine growth potential until the age of slaughter, suggesting that other factors may be better indicators of growth during this period. According to England (1974), smaller piglets compete for food less effectively, so the relationship between chew weight and postnatal growth may be partly due to feed intake. Still, Azain et al. (1996) established that the magnitude of the correlation observed between post-weaning weight gain and slaughter weight is greater than the observed correlation between weight gain at birth and weaning weight. Such observation justifies the adoption of differentiated management capable of increasing gains in this phase.

In the study by Quiniou et al. (2002), the 0.6 kg (lighter) birth piglets took three weeks longer to reach 25 kg compared to the heavier (2.6 kg born) piglets. It was also observed that the difference between the lightest and the heaviest piglets was 5.4 kg at weaning, increasing to 11.9 kg at 63 days of age, evidencing the amplification of the effect on subsequent performance.

In the same vein, Heyer et al. (2004) also observed that higher birth weight was associated with better growth rates. Similar results were found by Nissen et al. (2004), who found that piglets with low birth weight (1.47 kg) had lower daily weight gain associated with a lower lean carcass deposition rate until slaughter, in relation to their medium-weight siblings (1.50 kg) and tall (1.71 kg) at birth.

In an experiment by Rehfeldt & Kuhn (2006), sixteen Landrace sows piglets were divided into three groups: (I) 25% of low weight (less than 1.20 kg) piglets, (II) 50% of medium weight and (III) 25% of high weight piglets (greater than 1.62 kg). In this study it was observed that the low weight piglets presented lower daily weight gain and live weight at 175 days of age, compared to the intermediate or high weight piglets, without significant differences between the last two groups.

The existence of the effect of birth weight on weaning weight was also observed by Furtado et al. (2007), when piglets born weighing less than 900 grams were found to be weaned at approximately 3.2 kg less than those born over 2.1 kg (3.9 and 7.1 kg, respectively). In the same sense, Smith et al. (2007) compared two categories of birth weight and concluded that piglets born with an average weight of 0.860 kg (0.66 kg - 0.94 kg) weighed 4.15 kg at weaning, while those born at 1.51 kg (1.43 kg - 1.58 kg) were weaned at 5.76 kg, with an average difference between piglets of 0.650 kg at birth and 1.61 kg at weaning.
In a recent study conducted by Beaulieu et al. (2010), it was observed that the advantage of body weight at birth in piglets divided into four different weight ranges (0.80 to 1.20 kg; 1.25 to 1.45 kg; 1.50 to 1.70 kg and 1.75 to 2.50 kg) was kept until slaughter, being 92.18 kg; 96.81 kg; 99.74 kg and 101.82 kg, respectively, according to the birth weight ranges given above. These authors also observed that animals born lighter did not achieve a daily average weight gain similar to those born heavier within seven weeks of weaning until slaughter.

In general, they propose that a lower growth rate could be due to the low number of myofibers in low birth weight piglets, due to the damage occurred during myogenesis, especially since the characteristics associated with postnatal development and Meat quality can be influenced by the number and size of muscle fibers, determined around 90-95 days of gestation (Gondret et al., 2006). It is observed that litter waste has the potential for reduced muscle growth. Dwyer et al. (1993) demonstrated that birth weight does not necessarily determine growth potential until slaughter age, so the number of muscle fibers may be the best indicator of growth in this period.

However, the possibility that birth weight would irreversibly affect muscle development remains controversial (Bee, 2004; Poore and Fowden, 2004; Gondret et al., 2005) due to external factors, such as competition between piglets, as well as the differences observed in subsequent consumption levels.

Studies have shown that lighter animals at birth had a higher frequency of giant fibers compared to heavier animals at birth (Gondret et al., 2006; Rehfeldt et al., 1999). According to Fiedler et al. (2004), observed changes in fiber morphological characteristics (increased size) may be associated with in vivo functional changes in muscle of animals and, consequently, their poor meat quality in swine and poultry.

Several studies have concluded that low birth weight is associated with lighter carcasses (Bee, 2004; Gondret et al., 2006; Rehfeldt et al., 2008), attributing these results to a lower total number of muscle fibers. Other scientific studies, in turn, showed that birth weight did not influence carcass characteristics (Bérard et al., 2008; Beaulieu et al., 2010).

According to Kuhn et al. (2002), the reduction in the number of fibers is associated with its limited hypertrophy observed during the rearing / termination period, when less muscle protein deposition and higher fat deposition occur in lighter born animals. Heyer et al. (2004) and Kuhn et al. (2002) found higher percentages of lean meat in animals with high birth weight compared to lighter-born piglets. According to Gondret et al. (2006), these variations in fat content are more related to differences in activity of lipogenic enzymes than to differences in duration of fat accumulation and / or physiological maturation among pigs.

Additionally, when considering the principles of muscle fiber growth, it is expected that in low weight animals there will be faster fiber growth due to the low number of fibers. In addition, a plateau in fiber growth is reached earlier in relation to high weight animals (Rehfeldt and Kuhn, 2006). Thus, it can be inferred that in low-weight piglets, the energy of nutrition may not have been used for muscle growth and was then used for lipogenesis.

Data indicative of inadequate functional development of the gastrointestinal tract involving low birth weight piglets may, to some extent, provide another physiological explanation for postnatal performance outcomes. According to Cranwell (1995), animals born lighter may present alterations in the gene expression of proteins related to gastrointestinal development and growth (Wang and Xu, 2005). It is further speculated that the harm from inadequate gastrointestinal development is the major cause why these animals grow more slowly than those with normal intrauterine growth (Wang et al., 2010).

Information involving the characteristics of gastrointestinal tract development and subsequent pig performance has not been presented in the literature. Thus, the current studies are limited to measurements of the gastrointestinal tract (weights and lengths), morphological, morphometric and immunohistochemical analyzes of the intestine, however, were not associated with postnatal performance results of the animal.

According to Adeola et al. (2006), the presence of a more developed intestinal mucosa in animals born with higher weight may be a determining factor for better postnatal performance. Recently, Wang et al. (2010) showed that nutrient utilization efficiency is reduced in newborn piglets that experienced retarded intrauterine growth (CIUR, n = 18, mean
weight of 730 grams) when compared to those with normal birth weight (PNN, n = 18, average weight 1.30 kg).

It is important to emphasize at this time that the morphological nature of the weight and length increase of the digestive and non-digestive organs, and their respective biological meanings are not yet fully explained, thus suggesting further studies on the various responses of the tract, gastrointestinal tract, in particular from swine from modern commercial strains (Gomes et al., 2007).

In the work of Rehfeldt and Kuhn (2006), previously mentioned, the high birth weight animals presented higher carcass weight, lean meat percentage and loin eye area, compared to the low birth weight animals, which due to once presented a higher percentage of internal fat. The absence of effects of birth weight on carcass fat deposition (based on fat thickness) is in agreement with Bérard et al. (2008) who reported a possible cause of the lack of response may have been the fact that in the other studies mentioned above, the piglets used were either male or female only. Thus, based on the findings of Poore & Fowden (2004), the effect of birth weight on carcass fat is more evident in females than in males.

Regarding carcass typification, the results showed that birth weight partially affects carcass cut weights. According to Handel and Stickland (1987), the higher weight of these cuts in the animals of the high birth weight group is in accordance with the greater number of muscle fibers present in the piglets of the AP group. Similarly, Gondret et al. (2005) compared low (0.75 to 1.25 kg) and high (1.75 to 2.05 kg) piglets at birth and reported lower ham, loin and belly weights in the carcasses of the animals born lighter. However, they did not find significant differences for the palette cut. In contrast to the results presented above, Beaulieu et al. (2010) observed that carcass cuts were not affected by the previously mentioned birth weight quartiles.

Rehfeldt et al. (2008) observed a lower pH value at 45 minutes postmortem, as well as a tendency for greater drip loss in low birth weight animals compared to medium and high birth weight animals. The small variations observed in pH can be explained by both speed and extent of post-mortem pH reduction, which may be influenced by intrinsic factors such as species, muscle type, variability between animals, and also by extrinsic factors, as room temperature (Lawrie, 2005).

As for the characteristic involving drip water loss, normal meat should have a water loss of less than 5% (Warner et al., 1997). Berard et al. (2008) reported that the effects of litter size and birth weight on meat quality characteristics were minimal. According to these authors, the differences observed between studies can be attributed to the differences in the genetic lines used. In agreement with these results, Heyer et al. (2004) and Oksbjerg et al. (2002) also observed that the birth weight of piglets had no influence on meat quality characteristics. The results obtained in the present study corroborate with Beaulieu et al. (2010) who also found no effects of birth weight on these variables. The objective and subjective analyzes performed in this study did not show any difference in meat quality attributable to birth weight, considering that the development of final product quality depends on lineage and may be affected by post-mortem refrigeration rate. In cases where genetic predisposition to PSE meat is not adequately controlled by refrigeration, differences in quality may occur. This is possibly due to differences in carcass fat or final weight resulting from initial differences in birth weight.

Gondret et al. (2006) observed that meat from low birth weight animals (0.75 to 1.25 kg) was classified with a low tenderness score, negatively correlated with fiber size (r = -0.34). According to several authors (Jiang, 1998; Kristensen and Purslow, 2001; Zhang et al., 2006; Bee et al., 2007), increased proteolysis of key myofibrils or protein associated with myofibrils positively affected retention capacity of water and tenderness of the meat. The degradation of these proteins causes the weakening of myofibrils and consequent increase of meat tenderness (Jiang, 1998).

Still in the work of Gondret et al. (2006) speculated that low-weight piglets would have low muscle protein turnover, which possibly resulted in a decrease in the amount and activity of proteolytic enzymes such as calpain and cathepsins (Kristensen et al., 2002), which in low birth weight animals negatively affected the final tenderness of meat.

Growth physiology of swine fed β-adrenergic diets

B-adrenergic agonists, such as ractopamine, are growth promoters that act as modifiers of animal
metabolism, being structural analogues of hormones called catecholamines (adrenaline and noradrenaline). They are used in animal production for better performance and promote the deposition of muscle tissue to the detriment of adipose tissue. In muscle cells, it stimulates the synthesis process (priority) and reduces degradation.

In pigs, as the animal grows, the proportion of C18: 0 and C18: 1 fatty acids increases in subcutaneous fat and the proportion of C18: 2 n-6 decreases. Comparing sex classes, whole males have lower subcutaneous fat thickness and higher proportion of polyunsaturated ones (Wood et al., 1984).

In swine the adipose tissue can still be quite manipulated, since the variations of its participation are high in the different deposition sites. Because pigs are monogastric, they can change the composition of fatty acids as a function of diet, aiming to improve the nutritional value of meat.

Diets rich in polyunsaturated fatty acids significantly increase the levels of linoleic and linolenic acids in longissimus dorsi muscle and subcutaneous fat and in parallel increases the polyunsaturated: saturated ratio, reducing the risks associated with cardiovascular disease in humans (Morel et al., 2006). However, increasing the percentage of unsaturated fatty acids results in greater susceptibility to lipid oxidation in meat and processed products (Mitchaothai et al., 2007).

In cellular metabolism, β-adrenergic agonists are associated with the GS protein, which when activated, alters the tertiary structure of adenyl cyclase by activating it and increasing the cAMP (adenosine 3’, 5’-monophosphate). CAMP acts on cAMP-dependent kinase proteins, activating them. Phosphorylation of various proteins occurs, resulting in increased catabolism (lipolysis) and a reduction in anabolism (fatty acid and triglyceride biosynthesis) (Mersmann et al., 1997). B-adrenergic agonists also inhibit adipocyte insulin sensitivity. In muscle tissue, β-adrenergic agonists may stimulate muscle growth by protein synthesis or by reducing protein degradation.

The increase in carcass meat content of ractopamine-supplemented pigs found by Crenshaw et al. (1987) was 3.4% and the reduction in fat was 3%. Warris et al. (1990) observed a 0.9% increase in carcass meat content and a 0.8% reduction in fat content. Rocha et al. (2013) found a 1.54 kg increase in hot carcass weight and 0.65 percentage points in carcass meat content for ractopamine-treated pigs.

The response of ractopamine supplementation to muscle growth depends on the pig's genetic ability to develop muscle tissue. In providing ractopamine to pigs, Bark et al. (1989) observed that animals with average genetic capacity for muscle tissue deposition presented higher muscle tissue deposition than animals with low muscle tissue deposition capacity. However, Warris et al. (1990) found that the β-adrenergic agonist salbutamol improved carcass quality (higher carcass yield and increased musculature) for both traditional and meat pig strains. However, fat reduction was less pronounced in meat strains. These studies indicate that pigs of different halothane genotypes may have different responses to ractopamine addition in the feed.

Controlling myogenesis to increase the number of muscle fiber myoblastosis is an important strategy when it comes to increasing muscle mass and meat production. The number of muscle fibers is characteristic in different breeds and genetic lines and is determinant in the growth rate of animals (Dwyer et al., 1993).

Recent studies have shown that it is possible to alter the number of muscle fibers through nutrition, hormone use and gene manipulation. According to Wigmore and Stickland (1993), primary fibers are resistant to the influence of the environment, not increasing their number due to nutrition or hormones, while secondary fibers are susceptible. However, Kim et al. (1994) found a significant increase in the number of primary cells in the Semitendinosus muscle of piglets whose mother received β-adrenergic.

Aiming to increase the number of muscle fibers of piglets through nutritional manipulation Dwyer et al. (1994), using β-adrenergic in the diet during the first 21 days of pregnancy, found better results when the best nutritional intake of the parent occurred before the appearance of secondary fibers (25 to 50 days of gestation), resulting in an increase of approximately 13 days. % of the number of muscle fibers of piglets.

Also Hoshi et al. (2005) observed that the supply of β-adrenergic ractopamine to pregnant sows in the prehyperplastic period (between 25 and 50 days of gestation) increased the number of offspring muscle cells by 6.8%.
When the nutritional contribution of the pregnant female improves, through nutrition or through the use of nutrient-sharing substances, there is a better supply of glucose and amino acids to the placenta and fetus. This fact stimulates the release of IGF, which is important in the regulation of muscle hyperplasia for stimulating myoblastic proliferation (Stickland, 1996).

Animals with low muscle fiber invariably grew less than animals with high fiber, indicating that higher numbers are a prerequisite for good growth (Dwyer et al., 1993).

Handel and Stickland (1988) evaluated birth weight, number of swine semitendinosus muscle muscle fibers, their relationship to growth rate and slaughter weight and concluded that low birth weight animals are not meant to be small slaughter, provided they have the same number of muscle fibers as animals of higher birth weight.

The authors considered the number of muscle fibers as an indicator of swine growth potential and concluded that birth weight does not appear to be a good indicator of the total number of muscle fibers and postnatal growth rate as low birth weight animals were able to reach slaughter weight and the same growth rate of animals with higher birth weight, provided they had the same number of muscle fibers.

For equivalent live weights, pigs with a high number of muscle fibers had a smaller diameter of their fibers compared to animals with a low number of fibers (Dwyer et al., 1993).

When Hegarty And Allen (1978) compared light and heavy piglets at birth up to 106 kg of live weight, they found that lightweight piglets had muscle fibers with larger diameters in two out of four studied muscles and smaller muscle weight, but with similar length, muscle and bone.

Studies with light-weight piglets (Powell and Aberle, 1980) and pigs with high fat deposition (Hausman et al., 1983) showed that both had lower numbers of muscle fibers, more carcass fat and grew less efficiently when compared with normal and lean animals. Fat deposition seems to be inversely correlated with the total number of muscle fibers in swine. This may explain the better growth efficiency of animals with higher number of muscle fibers.

Studying swine performance, Dwyer et al. (1993) found a positive correlation between daily weight gain and secondary / primary fiber ratio in the 25 to 80 kg bodyweight period, and there was also a correlation between feed conversion and number of muscle fibers. The authors concluded that birth weight was positively correlated with growth rate only at the earliest stages of swine growth, and at the later stage of growth after 70 days, it seemed to be determined by the number of muscle fibers, ie. by the genotype of the animal.

Restriction of fetal growth can be said to have permanent negative impacts on the neonatal phase until extraterine life: pre-weaning survival, postnatal growth, feed efficiency, life-long health of the animal, composition of body tissues (including protein, fat and minerals) and meat quality (Wu et al., 2006).

Final Considerations

The present work contributed to the knowledge of the importance of the study of the physiology of growth and development of animals, since they begin to be determined at the moment of conception and are influenced by genetic, environmental factors and the interactions between genetics and environment. The physiological and biochemical knowledge of how muscle tissue deposition occurs is a very important tool for growth manipulation, seeking better yields and higher quality carcass meat.

The weight of the piglet at birth is an extremely important factor, initially, for its survival, and later for a good performance until the moment of slaughter.

Hyperproliferous females produce a larger number of piglets born per litter, which results in lower average birth weight and, consequently, greater weight variability of these piglets.

Nutrition and feeding management should be carefully performed, always respecting the different reproductive stages in which females are in order to provide a high nutritional quality and thus a good weight of the piglet at birth. These two associated factors, genetics and nutrition, will certainly help to increase the survival of piglets during the lactation phase and, thus, contributing positively to the decrease of the maternal mortality rate and the increase of the economic gain of the production.

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