

Birth Weight of Litters as a Source of Variation in Postnatal Growth, and Carcass and Meat Quality

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■ Introduction

Both birth weight of the individual piglet and the intra-litter variation of birth weight are of considerable economic interest for pork production. Not only the survival rate within the first week after birth, but also postnatal growth (pre-weaning, nursery and grow-finish period) is impaired in low compared with high birth weight pigs (Quiniou et al., 2002). The phenotype of the newborn piglet is determined by prenatal growth and development, which is affected by a variety of environmental and other factors. These include maternal nutrition, maternal intestinal malabsorption, inadequate provision of amniotic and allantoic fluid nutrients, the ingestion of toxic substances, disturbances in maternal or fetal metabolic and homeostatic mechanisms, insufficiency or dysfunction of the uterus, endometrium, or placenta, environmental temperature and stress, and poor management (reviewed by Wu et al., 2006).

Over the last decade, selection for improved prolificacy has resulted in an increase of litter size at birth. In Switzerland, the number of piglets born alive has increased in the last 10 years from less than 10.9 to 11.8 per litter (Annual Report Suisag, 2005). In France the improvement in litter size has been even more impressive, increasing from 10.9 in 1992 to a mean litter size of 12.2 piglets in 2001 (Gondret et al., 2005). The selection for sow's ability to give birth to a higher number of piglets has led to an increased within-litter variation in piglet birth, as well as to an overall decrease in birth weight. A possible cause for these observations is the increased competition among littermates for maternal nutrients in utero, because fetal weight and birth weight have been shown to be inversely related with litter size.

This short review will focus on: 1) the consequences of impaired prenatal growth on myogenesis (prenatal skeletal muscle development) and 2) the impact of differences in birth weight on postnatal development, carcass characteristics, and meat quality traits.

■ General Considerations on the Significance of Myogenesis for Postnatal Growth Performance

Embryonic Muscle Development

In the pig, myogenesis is a biphasic phenomenon and involves determination, migration, proliferation, differentiation, and fusion of myoblasts to form myotubes. In the first phase, lasting from d 35 to d 55 of gestation, a primary generation of myotubes (the so-called primary myofibers) develop. In the second phase, which lasts until d 90 of gestation, the formation of a second generation of myotubes (secondary myofibers) occurs. Over 20 secondary myofibers appear around each primary myotube, using them as a scaffold. Considering the fact that in the pig hyperplasia (increase in myofiber number) ceases by around d 90 of gestation, the number of formed primary and secondary myofibers ultimately determines the total number of myofibers at birth.

As reported by Wigmore and Stickland (1983), the number of secondary myofibers, as well as the total number of myofibers, formed is lower at birth in smaller compared with larger fetuses. Furthermore, primary myofibers in small fetuses are smaller than in large foetuses. The authors hypothesized that the small size of the primary myofibers may restrict the available surface area for secondary myofiber formation.

Postnatal Muscle Development

The increase in skeletal muscle weight during postnatal growth results from muscle fibre hypertrophy (increase in the size and the length of the individual myofibers). Furthermore, the extent of myofiber hypertrophy and, thus, the capacity of the muscle to grow depends also on the total number of myofibers within a muscle, which is fixed at birth. It has been shown that myofiber size is inversely correlated ($r = -0.3$ to -0.8) with myofiber number, which means that growth rate of the individual myofiber is lower when there are high numbers of myofibers and higher when there are low numbers of myofibers (Rehfeldt et al., 2000). On the other hand, both number and size of myofibers are positively correlated ($r = 0$ to 0.6 and $r = 0.3$ to 0.5 , respectively) with the cross-sectional area of the muscle. This raises the question whether hypertrophy or total number of myofibers is more important for lean tissue growth. As reviewed by Rehfeldt et al. (2000) it seems that the potential for

lean tissue growth depends primarily on the number of the prenatally formed myofibers, because myofiber hypertrophy is limited by genetic and physiological constraints. Based on the aforementioned findings, one can conclude that impaired postnatal growth can be expected in low birth weight piglets displaying low myofiber numbers.

■ **Low Birth Weight Impairs not only Postnatal Growth Performance but also Carcass and Meat Quality**

Recent results from various experiments demonstrate the close relationship between birth weight, carcass characteristics, and meat quality traits. Rehfeldt et al. (2004) determined that at birth, the lightest piglets exhibited the lowest percentages of muscle tissue, total protein, total fat, the lowest semitendinosus muscle weight and total number of myofibers, whereas the percentages of internal organs, skin, bone, and total water were highest, compared to their heavier littermates. In finishing pigs slaughtered at d 182 of age, the pigs of low birth weight were lighter, had lower meat percentages, and loin area was smaller compared to pigs of high birth weight, whereas the percentage of omental fat tended to be higher. The pigs of low birth weight exhibited the lowest myofiber numbers, the largest myofiber size, and the highest percentages of abnormal "giant" myofibers in both muscles under investigation. With respect to meat quality, higher drip losses were determined in the longissimus muscle of low birth weight pigs.

Consistent with these earlier results, we reported larger myofibers and fatter carcasses in low birth weight pigs slaughtered at 105 kg body weight (BW) (Bee, 2004). In a recent study Gondret et al. (2006) reported that compared to heavy birth weight pigs, low birth weight pigs reached the same slaughter weight of 112 kg 12 days later. Not only was growth rate impaired, but feed conversion ratio was also inferior in low birth weight pigs. Accordingly, low birth weight pigs exhibited a fatter carcass, associated with markedly higher activity of enzymes involved in lipogenesis, such as fatty acid synthase and malic enzyme. Again, the total myofiber number was lower in the semitendinosus muscle, and the myofibers were larger in both the semitendinosus and longissimus muscles of low compared to high birth weight pigs. Of great importance with respect to consumer's satisfaction with pork, was the finding that the low birth weight pigs exhibited a lower score for loin meat tenderness compared with high birth weight pigs. The present results also indicate that the birth weight effect on meat tenderness could be partly attributed to its influence on myofiber hypertrophy, because tenderness score was negatively correlated ($r = -0.34$) with myofiber size of the longissimus muscle.

Collectively, available results indicate that pigs of low birth weight develop lower carcass and meat quality. There is increasing evidence that this is related to the low number of myofibers that undergo accelerated hypertrophy during postnatal growth. A possible explanation for the differences in adipose tissue accretion observed between low and high birth weight pigs, is that in low birth weight pigs the increase in myofiber size is faster because of the low myofiber number and the plateau of myofiber growth is attained earlier compared to high birth weight pigs. Consequently, in low birth weight pigs, nutritional energy can no longer be used for muscle accretion and is therefore used for lipogenesis.

■ Can specific feeding strategies overcome the negative impact of low birth weight pigs?

The question arises whether using specific feeding strategies in the growing-finishing period could help to overcome the negative impact of low birth weight. Such feeding strategies, developed to reduce the intra-litter variation in muscle growth, would benefit production efficiency and improve the quality and consistency of pork products. Therefore, in a recent study, our aim was to evaluate the effect of three different feeding regimens applied during the growing-finishing period on growth performance, carcass characteristics, and meat quality in low (**Lwt** = 1.12 kg) and high birth weight barrows (**Hwt** = 1.94 kg). For the present experiment we selected the lightest and heaviest barrows from 21 litters. At the beginning of the experimental period (27 kg BW) the barrows were randomly assigned to one of the following dietary treatments; **AA**: ad libitum feed access from 27 to 102 kg BW, **RR**: restricted feeding from 27 to 102 kg BW, and **RA**: restricted feeding from 27 to 63 kg and ad libitum feed access from 63 to 102 kg BW.

By design, the ADFI differed among barrows of the three dietary treatments and was highest in AA- (2.12 kg), intermediate in RA- (2.00 kg), and lowest in RR-barrows (1.78 kg). As expected, AA- and RA-barrows grew faster than RR-barrows (0.77 vs. 0.68 kg/d). For these traits, no significant differences between birth-weight groups were found. Nevertheless, due to the numerically lower ADWG of the Lwt- compared to Hwt-barrows (0.72 vs. 0.75 kg/d), Lwt-barrows consumed more feed (204 vs. 193 kg) and were less efficient (G/F: 369 vs. 382 g/kg) than Hwt-barrows, which is in agreement with results presented by Gondret et al. (2006). Percentage lean meat and adipose tissue of carcasses in treatment RR, RA, and AA of high birth weight barrows, as well as in treatment RR of low birth weight barrows, were similar. In contrast, carcasses of low birth weight barrows in treatments AA and RA were fatter. These findings illustrate that lean tissue deposition is limited in low birth weight pigs. This is probably a consequence of accelerated myofiber hypertrophy due to the lower myofiber number and because the plateau of

myofiber growth is attained earlier in low- compared to high-birth weight pigs. Thus, in low birth weight pigs, higher dietary energy and protein intake provided by *ad libitum* feed access in treatments AA and RA is utilized for lipogenesis rather than for protein synthesis. In contrast to previous results (Rehfeldt et al., 2004; Gondret et al., 2006), meat quality traits such as colour, drip loss, and tenderness were not affected by birth weight. Interestingly, the longissimus muscle of RA- was more tender than in RR-barrows, as indicated by lower shear force values. In line with the results for shear force, calpain activity at 30 min and 24 h postmortem in the longissimus muscle of barrows with *ad libitum* feed access in the finishing period (treatment RA and AA) was higher than in restrict-fed barrows, suggesting higher proteolytic capacity of the muscle at the time of slaughter. In conclusion, the present results revealed that regardless of the applied feeding strategy, low birth weight was associated with impaired carcass quality. Furthermore, compensatory growth positively affected meat tenderness.

■ Conclusion

In pigs, low birth weight results from fetal growth retardation and is associated with low myofiber numbers due to impaired myogenesis. Not only survival rate but also postnatal growth performance, carcass characteristics and meat quality can be compromised by low birth weight, because myofiber number and size are related with these traits. The results from a recent feeding trial suggest that in low birth weight pigs, *ad libitum* feed access favored adipose tissue accretion, resulting in fatter carcasses at market weight. Thus, it seems unlikely that postnatal feeding strategies are effective in overcoming the demonstrated effects of low birth weight. Because maternal feeding during gestation has also not been shown to be very effective in improving fetal growth, genetic selection for litter homogeneity seems to be the only suitable tool for improving fetal muscle growth and ultimately pork quality.

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