

EFFECT OF SOW PROLIFICACY AND NUTRITION ON PRE- AND POSTNATAL GROWTH OF PROGENY – A REVIEW*

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Abstract

Weakened growth and development of embryos as well as of fetuses or their organs (IUGR) show a relationship with increasing sow fertility. When aiming to increase birth weight in piglets and reduce within-litter variation in piglet body weight, efforts should be made to maintain a favourable maternal environment (uterus-placenta-embryo). Intrauterine undernutrition can be limited through the hormonal and/or nutritional treatment of pregnant sows. This has an effect on prenatal myogenesis, resulting in better development of skeletal muscles, higher birth weight of piglets, and progression in postnatal growth rate.

Key words: sows, prolificacy, feeding, IUGR

Intrauterine crowding in mammals

The causes of low birth weight in piglets are determined using two terms: intrauterine crowding (IUC) and intrauterine growth retardation (IUGR). The latter is defined as impaired growth and development of the mammalian embryo/fetus or its organs during pregnancy (Wu et al., 2004; Wu et al., 2006).

Intrauterine fetal growth is a complex biological process influenced by genetic, epigenetic and environmental factors. These factors affect placental size and functional efficiency, placental blood flow, delivery of maternal oxygen and nutrients to the fetus, nutrient availability to the fetus, hormonal balance, and the balance of metabolic pathways. Alterations in fetal nutrition and endocrine status may influence adaptive and developmental changes, structural, physiological and metabolic changes, and postnatal growth (Chmurzyńska, 2010). The impairment of fetal growth potential can be primary or secondary (Zhu et al., 2010). The primary causes are of

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genetic, vascular, autoimmune and infective origins, and the secondary cause is inadequate fetal nutrition. Of great importance in IUGR induced by fetal undernutrition is weakened placental synthesis of nitric oxides (NO), which has an effect on vascular efficiency. Another important factor is the synthesis of polyamines, which serve vital functions in regulation of DNA and protein synthesis in fetal endothelium (Wu et al., 2004). IUGR was induced by experimental inhibition of NO synthesis through inactivation of nitric oxide synthases in rats. The lack of the enzyme caused IUGR to occur in mice. Studies with sheep showed that weakened nitric oxide synthesis at placental and fetal levels is paralleled by decreases in distensibility of blood vessels and blood flow between them.

Wu et al. (2004) believe that maternal nutritional status can change the epigenetic state of the fetus, while DNA methylation and histone modification alter DNA expression. It follows from the description of fetal programming that nutritional stimuli during fetal development may leave a permanent imprint by changing the epigenetic state of the fetal genome and gene expression. The identified mechanisms of epigenetic effects include changes in DNA methylation (replacement of cytosine within CpG dinucleotides in the mammalian genome) and histone modifications (acetylation and methylation). CpG methylation can affect chromatin conformation, while DNA methylation and histone modifications may modify amino acid and nutrient availability. In mice and rats, amino acid deficiency was found to influence genomic DNA methylation and reduce the expression of some genes important for fetal development.

Fetal undernutrition is quite common in the animal world. The peri-implantation period and the period of rapid placental development are particularly important with regard to IUGR. Ishida et al. (2002) attributed a key role in fetal development to arginine, which is a common substrate for the synthesis of nitric oxide and polyamines, the role of which was described by Wu et al. (2004). They are key regulators of angiogenesis and embryogenesis, being responsible for cell proliferation and differentiation (Ishida et al., 2002). Arginine deficiency hinders fetal development and increases perinatal mortality. The arginine-supplemented diet has positive effects by improving fetal growth and development and reducing the mortality rate. Fetal development is positively affected by a diet that meets 100% of the requirement for folic acid, vitamin A, iron, zinc and magnesium. In pregnant females, a vital role in fetal development is also played by lipid and carbohydrate metabolism. Diets enriched with omega-3 long-chain fatty acids were found to be particularly beneficial during both pregnancy and the nursing period (Ishida et al., 2002).

IUGR is a cognitively interesting research problem of utilitarian and health importance not only to animals but also humans (Wu et al., 2004). In the western USA intrauterine growth restriction (IUGR symptoms) is found in 5% of the children, which may predispose them to metabolic, endocrine and cardiovascular diseases in adulthood. The intrauterine environment is a more important determinant of fetal growth than the fetal genome. Embryo transfer studies show that it is the recipient mother rather than the genetic (donor) mother that more strongly influences fetal growth (Wu et al., 2004).

Prolificacy and intrauterine growth retardation

High prolificacy of sows and increased fetal survival lead to greater incidence of intrauterine crowding (IUC), which may affect pre- and postnatal development of the progeny.

Selection for prolificacy, creation of lines with very good reproductive traits, and the common use of maternal heterosis shown by two-breed cross sows have increased litter size at birth. Increases in sow fertility and also in piglet mortality were noted in many countries, including Switzerland, France, Great Britain, and Poland (Bee, 2007; Boulot et al., 2010; Orzechowska and Mucha, 2010).

The results of selection experiments indicate that there is still room for improvement in reproductive traits of the sows (Johnson et al., 1999; Ruiz-Flores and Johnson, 2001). Johnson et al. (1999) found that ovulation rate and corpora lutea increased by 6.5 during 11 generations. However, at 50 days of gestation, the number of fetuses was greater by only 2.8, and the total number of pigs per litter by only 0.8. According to Ruiz-Flores and Johnson (2001), one should not expect litter size to improve by more than 8–10% when using multigenerational selection.

Prenatal survival is correlated to uterine space. Fetuses that develop in a crowded intrauterine environment with limited space are at a greater risk of perinatal and postnatal mortality compared to piglets that develop under normal conditions (Chen and Dziuk, 1993; Ryan and Vandenberg, 2002). Pig fetuses require at least 36 cm of uterine length on day 50 of gestation to develop normally (Wu et al., 1999), but this condition is not always met with improved fertility.

According to German and Canadian researchers, production efficiency of sows of modern genotypes is determined by a large number of piglets born and weaned per litter, per year and during the breeding lifetime (Quinton et al., 2006; Müller, 2011). Brüssow et al. (2011) indicate that breeding progress in reproductive traits, which are characterized by low heritability and repeatability, is small. Expression of even the best genotype can be constrained by adverse effects of the external and internal environment that occur continuously (Onteru, 2011). The environment influences the animal through feeding, housing and reproductive technique. Reproductive performance is ultimately determined by the age of females, embryonic and fetal nutrition during pregnancy, intrauterine position, uterine capacity, fetal crowding and litter size, as well as the endocrine and hormonal processes in maturing and mature healthy females.

Piglet weight at birth and its within-litter variation are important in productive and economic terms. Increased litter size decreases mean birth weight ($r = -0.46$) (Milligan et al., 2002). When litter size increases, within-litter birth weight variation also increases (Quiniou et al., 2002; Tribout et al., 2003; Boulot et al., 2010), making the litter less uniform (Milligan et al., 2002; Bee, 2007; Beaulieu et al., 2010). In a study with a large number of litters ($n = 965$), Quiniou et al. (2002) found that an approx. 45% increase in litter size reduced mean birth weight of piglets by over 20% and increased the proportion of piglets weighing <1 kg by 16%. According to Quiniou et al. (2002) and Boulot et al. (2010), mortality in the group of piglets weighing <1 kg is 3- to 5-fold higher than in the group of piglets weighing >1.0 kg. Low body weight

and high within-litter variation at birth are negatively correlated with piglet survival (Herpin et al., 2002) and may affect productive traits such as growth rate during growth and fattening, and carcass meatiness and fatness. Therefore, preference in reproduction is given to sows born to females of high uterine capacity, because this increases the chance for improved litter size (Vallet et al., 2002). Koczanowski et al. (2006) found that the uterine horn length and uterine weight are positively correlated with the number of fetuses and their survival. However, the presence of runt piglets in litters is directly proportional to litter size, which is generally determined around 30 days of gestation (Foxcroft et al., 2006).

When investigating the effect of intrauterine crowding, Pere and Etienne (2000) showed that uterine blood flow decreased with increasing number of fetuses per uterine horn, while the increasing competition for food may reduce fetal growth due to undernutrition. Foxcroft et al. (2006) express the view that inadequate transfer of nutrients and oxygen from mother to the fetus reduces its growth. As a consequence, newborn piglets are light and have limited capacity for compensatory growth. Studies by Rehfeldt et al. (2011, 2012 a, b) suggest, however, that lower birth weight of piglets, lower muscle weight, and differences in chemical composition of the body and histological structure of muscles may be compensated during the maternal nursing period and during the later growth period. The potential for increasing the number and diameter of fibres was described by Rehfeldt and Kuhn (2006). From birth to 5 weeks of age, the number of muscle fibres in piglets may increase by over 60%, from approx. 0.4 to 0.65 million. In subsequent weeks, the scale of change in the number of fibres is much lower. Finally, at the age of around 32–33 weeks, the number of fibres is approx. 0.75 million. Meanwhile, fibre diameter clearly increases, from approx. 25 μm at 5–6 weeks of age to 70–75 μm at slaughter. These changes may form the basis of compensatory growth in growing pigs.

Canario et al. (2010) estimated the coefficients of genetic correlation to be $r = -0.40$ between the number of live born piglets and their weight on day 21, and $r = +0.59$ between neonatal weight and body weight on day 21. They found heritability of homogeneity in body weight of piglets at birth and on day 21 to be low at $h^2 = 0.10$ and $h^2 = 0.08$, respectively. However, based on a thorough analysis of the results obtained, the same authors believe that genetic selection for equalized piglet weight is possible because of a high genetic correlation ($r = +0.51$) between standard deviations for piglet weight at birth and on day 21.

Influencing fetal intrauterine conditions falls within the scope of fetal developmental programming. Based on literature data, Foxcroft et al. (2006) concluded that IUGR-induced lower birth weight, poorer postnatal growth performance, smaller muscle development as fatteners, and failure of compensatory growth to equalize inadequate body weight are determined not only by their low birth weight but also by conditions of myogenesis during fetal life. The same authors distinguished five stages in the life of pigs, important for the rate of growth and development of skeletal muscles:

Stage 1 – from conception to 35 days of gestation – uterine crowding,

Stage 2 – from 35 to 55 days of gestation – primary muscle fibre formation,

Stage 3 – from 55 to 90 days of gestation – secondary muscle fibre formation,

Stage 4 – from 90 to 95 days of gestation – establishment of muscle fibre number,

Stage 5 – after birth – hypertrophic growth and maturation of muscles.

Foxcroft et al. (2006) concluded that giving preference to hyperprolific sows, without influencing intrauterine crowding, will not contribute to increasing pork production efficiency.

Secondary fibre myogenesis in pigs (Dwyer et al., 1994) and ruminants (Du et al., 2010; Funston et al., 2010) can be influenced through nutrition. Increased nutrition in mid-gestation sows (55–90 days) increases the number of secondary fibres in the progeny (Dwyer et al., 1994), which may improve the rate of their growth and feed conversion efficiency in the later stages of growth. Embryo developmental programming in farm animals accounts for traits such as growth, muscling and fatness. Skeletal muscles develop through myogenesis and adipogenesis. Both forms of cells are derived from mesenchyma and their differentiation depends on beta-catenin signalling. Up-regulation promotes myogenesis and down-regulation enhances adipogenesis. Interactions between myogenesis and adipogenesis may affect intramuscular fat content, and thus meat quality.

The increasing sow fertility is associated with weakened growth and development of embryos as well as of fetuses or their organs (IUGR). If we aim to increase birth weight in piglets and reduce within-litter variation in piglet body weight, efforts to this end should focus on maintaining a favourable maternal environment. Intrauterine undernutrition can only be limited through the hormonal and/or nutritional treatment of pregnant sows. Well-balanced diets that meet the nutritional requirements have a positive effect on prenatal myogenesis and adipogenesis. Better growth and development of skeletal muscles is found in fetuses.

Dietary and hormonal treatments that reduce IUGR

The achievement of proper prenatal myogenesis and body weight increase in piglets, especially the low within-litter variation of body weight, can be influenced in two ways:

- through genetic selection, and
- by contributing to a favourable state of the intrauterine environment.

For fetal development and weight gain, it is essential that primary fibres are formed from myoblasts between 25 and 50 days of gestation, the number of which is genetically determined (Wu et al., 2006). Secondary fibres begin to form at 50–55 days and their number is established at around 90–95 days of gestation. In different species of animals, this process is largely affected by the environment, notably nutrition (Ashton et al., 2005; Nissen et al., 2005; Rehfeldt, 2005). The fibres, characterized by high plasticity, are finally transformed to type I, IIA and IIB muscle fibres. During fetal life, not only the total number of muscle fibres is established, but also adipocytes – which determine the content of intramuscular fat, a determinant of meat flavour and juiciness – are also formed (Du et al., 2010). Therefore, there is more and more research concerning the effect of feeding pregnant sows not only on reproductive parameters, but also on the development of progeny, and carcass and meat quality (Nissen et al., 2005; Wu et al., 2006; Du et al., 2010; Hill et al., 2010).

Research provides no conclusive results (Musser et al., 2004; Cerisuelo et al., 2009). While some report a positive effect of more intensive feeding of pregnant sows on the number of muscle fibres, the birth weight of piglets, and the rate of their growth (Gatford et al., 2003; Markham et al., 2009; Rehfeldt et al., 2012 a, b), others fail to confirm a positive effect of feeding pregnant females on the number of muscle fibres, fetal weight, piglet weight gains after birth, or meat quality (Nissen et al., 2003; McNamara et al., 2011).

Weakened prenatal growth associated with the course of prenatal myogenesis has a residual effect on muscling, fatness and quality of pig meat (Powell and Aberle, 1981; Dwyer et al., 1994; Gatford et al., 2003; Lefaucheur et al., 2003; Bérard et al., 2008). This happens because a small number of secondary fibres generated during the fetal period, followed by a rapid increase in their size, contribute to the unused energy being used for deposition of fat. During gestation, undernourished sows give birth to lighter piglets that die more often, grow more slowly and are less efficient in feed conversion. During the later growth and fattening period, they produce carcasses with a lower weight of primary cuts, lower meat content, and poorer meat quality (Bee, 2004; Gondret et al., 2005; Rehfeldt, 2005).

Therefore, efforts are made to limit intrauterine undernutrition through nutritional and hormonal treatment of pregnant mothers. Nutrition research looks at the problem comprehensively (Brown et al., 2011; McNamara et al., 2011; Campos et al., 2012; Rehfeldt et al., 2012 a, b). It accounts for differences in feed supply during the formation of primary and/or secondary fibres in fetuses of pregnant sows (Cerisuelo et al., 2009; McNamara et al., 2011), protein supply (Rehfeldt et al., 2012 a, b) and L-carnitine supplementation of feed (Lösel et al., 2009; Nissen and Oksbjerg, 2009, cited from Oksbjerg et al., 2013), as well as the role of arginine in prevention of IUGR (Mateo et al., 2007, cited from Oksbjerg et al., 2013; Wu et al., 2010, 2013). In a study by Wu et al. (2013), piglet weight and proportion of muscle tissue in newborn animals were highest when pregnant sows received a feed with optimal protein content (12.1%). These parameters were poorer when sow diet was rich (30%) or deficient in protein (6.5%), which confirms the role of protein in the diet of pregnant females with regard to IUGR as well as slaughter traits of the offspring. It is also interesting to note the antagonistic and synergistic interaction of different compounds, including arginine and carnitine (Oksbjerg et al., 2013). Wu et al. (2013) emphasize the role of arginine in stimulating the synthesis of placental and uterine proteins. When supplemented to the diet of pregnant sows (0.83%), it increases litter size and weight at birth. The researchers (Remaekers et al., 2006; De Blasio et al., 2009; Cambell, 2009; Bérard and Bee, 2010; Gao et al., 2012) cited by Wu et al. (2013) demonstrated that arginine supplementation also reduces embryo mortality in pregnant sows. Other authors (Greiner et al., 2012; Zier-Rush et al., 2012) cited by Wu et al. (2013) hold the view that litter size remains unchanged after excessively increasing the level of arginine to 1.23% or 28 g/sow/day in early and late pregnancy.

The results of several experiments show that the action of the growth hormone (GH), somatotropin (ST) and insulin-like growth factor (IGF-1) is related to fetal nutrition as well as birth weight of piglets, their growth and postnatal development (Rehfeldt et al., 2001; Sterle et al., 2003; Gatford et al., 2010). In an experiment by

Rehfeldt et al. (2001), giving porcine somatotropin (pST) to pregnant sows improved the nutrient supply of embryos, as evidenced by changes in biochemical and physiological parameters, increased RNA concentration in the endometrium (showing a higher potential for protein synthesis) and increased glucose and protein content in amniotic and allantoic fluids. The beneficial effects on the weight of lightest piglets led the authors to conclude that the beneficial effect of pST can be selective in nature. In another study, Rehfeldt et al. (2004) analysed the results of research investigating the administration of the growth hormone to pregnant sows. The authors claim that the nutrition of mothers and the somatotropic axis are interrelated. Sow nutrition determines the concentration of nutrients, while the somatotropic axis has an effect on IGF-1 and IGFBP that regulate their use by fetuses. However, the more rapid fetal growth after somatotropin administration in early and mid-gestation does not persist until the end of gestation. Experiments with growing pigs suggest that somatotropin administration stimulates the synthesis of IGF-1 in the liver and significantly increases the plasma levels of glucose and free fatty acids. While the supplementation of nutrients for fetal development can be regarded as direct, the effect of the maternal growth hormone is indirect. Maternal somatotropin exerts a large effect on nutrient transfer through placenta, which improves the growth conditions for piglets in litter. The maternal somatotropic axis plays a significant role in “coordinating” the distribution of nutrients among maternal, placental and fetal tissues.

Effect of birth weight of piglets on postnatal growth

Neonatal body weight has often been studied in terms of its effect on rearing and fattening performance as well as quantitative and qualitative slaughter traits (Lawlor et al., 2007; Bérard et al., 2008, 2010; Schinckel et al., 2010). Beaulieu et al. (2010) determined a relationship between birth weight of piglets and growth rate until weaning and from weaning to 120 kg of body weight. Piglets from the groups with low birth weight had significantly lower daily gains from weaning and during fattening compared to animals from the groups with higher birth weights (Table 1), which caused them to reach slaughter weight 7 to 9 days later. Studies by different authors confirmed a significant effect of piglet birth weight on growth rate from birth to weaning (Bee, 2004; Nissen et al., 2004; Lawlor et al., 2007; Bérard et al., 2008, 2010), during fattening (Nissen et al., 2004; Lawlor et al., 2007; Bérard et al., 2008, 2010) and collectively during growth and fattening (Gondret et al., 2005, 2006).

When investigating feed conversion by growing animals differing in birth weight, Bee (2004) found no effect of piglet birth weight on feed conversion during fattening (Table 1). The findings of Gondret et al. (2006) showed 8.4% poorer ($P > 0.05$) feed conversion (kg/kg gain) in lighter piglets at birth compared to heavier piglets (Table 1). Rehfeldt and Kuhn (2006) showed that piglet birth weight had an effect on lifetime daily gains and body weight at 175 days of age, while Quiniou et al. (2002) confirmed the relationship and effect of variation in piglet birth weight on body weight during the later growth and fattening period. When studying the effect of body weight in neonates born over 8 successive litters on weaning weight at 28 days and on survival with mothers, Milligan et al. (2002) obtained best results for the heaviest piglets (Table 1). The results obtained after 4 weeks of growth for

piglets with different birth weights did not differ significantly. The presence in litters of piglets with low body weight was the principal reason for the lack of uniformity in neonatal weight, and the differences in body weight persisted at weaning.

Table 1. Effect of birth weight on body weight gains and feed conversion by young pigs (according to different sources)

	Source
Effect of birth weight on:	
daily gain until weaning on day 28 (or day 21*)	Bee (2004) – $P \leq 0.05$ Bérard et al. (2008, 2010) – NS Beaulieu et al. (2010) – $P \leq 0.05$ Gondret et al. (2005, 2006) – $P \leq 0.05$ Lawlor et al. (2007) – NS Wolter et al. (2002) – $P \leq 0.01^*$
daily gain during fattening (or from day 21 to 110 kg b.w.)*	Bee (2004) – NS Bérard et al. (2008, 2010) – $P \leq 0.05$ Beaulieu et al. (2010) – $P \leq 0.05$ Gondret et al. (2005, 2006) – $P \leq 0.05$ Wolter et al. (2002) – $P \leq 0.01^*$
age at slaughter weight	Bérard et al. (2008, 2010) – $P \leq 0.05$ Beaulieu et al. (2010) – $P \leq 0.05$ Gondret et al. (2005, 2006) – $P \leq 0.01$ Wolter et al. (2002) – $P \leq 0.01$
feed conversion during fattening	Bee (2004) – NS Powell and Aberle (1980) – exp. I – NS, exp. II – $P \leq 0.05$
feed conversion from weaning to slaughter	Bérard et al. (2008, 2010) – $P \leq 0.05$ Gondret et al. (2005, 2006) – NS
body weight at weaning on day 28	Lawlor et al. (2007) – $P \leq 0.05$ Milligan et al. (2002) – NS Nissen et al. (2004) – $P \leq 0.01$
body weight at 157 days of age	Lawlor et al. (2007) – $P \leq 0.05$
weight gain from 28 to 157 days of growth	Lawlor et al. (2007) – NS
weight gain from weaning at 28 days	Milligan et al. (2002) – NS Nissen et al. (2004) – NS
weight gain during fattening to 104 kg b.w.	Nissen et al. (2004) – $P \leq 0.01$

The coefficients of correlation calculated by Dwyer et al. (1993) confirm the relationship between birth weight and growth rate in young pigs. Between birth weight of piglets and growth rate to 6 kg body weight, which the piglets reached by 4 weeks of age, the correlation was $r = +0.36$, while the correlation between birth weight and growth weight after weaning (from 6 to 26 kg b.w.) was $r = +0.58$.

Nissen et al. (2004) estimated the correlation between birth weight and body weight at weaning ($r = +0.53$), body weight at slaughter ($r = +0.29$) and mean weight gain until slaughter weight ($r = +0.24$). According to the authors, the differences in the correlation coefficients resulted from the differences in muscle fibre growth.

They were due to the number and size of muscle fibres, which result from differences in satellite cell proliferation and protein turnover. DNA and RNA content are the indicators of the protein turnover and synthesis of muscle-specific proteins. Therefore, changes in the growth rate of littermates with different weight can be attributed to both the number and growth of fibres. The smaller number of muscle fibres found in lighter piglets allows a conclusion that it is due to intrauterine undernutrition.

In an experiment by Lawlor et al. (2002), suckling piglets were fed a milk replacer with a higher nutritive value. Piglets differing in birth weight were assigned to two groups in the experiment. When both groups received a high-density diet, the difference in body weight between the lighter and heavier piglets increased from 0.20–0.25 kg at birth to 3.0 kg at 30 days of age. When a low-density diet was used, the difference was smaller and amounted to only 1.7 kg after 30 days of feeding. The higher energy density of the diet during intensive growth did not limit, during the later growth (fattening) period, the negative effect of the lower birth weight on the rate of growth in growing piglets. The growth rate of marginal piglets, i.e. those weighing an average of 0.79 kg in the experiment (marginal piglets – those with a body weight <1.0 kg or reaching <65% of the weight of other littermates), was slower. To improve the nutritional status, some of marginal piglets were moved to foster sows. During growth to 26 kg body weight, the growth rate of lighter and heavier piglets was similar, but during fattening it clearly slowed down ($P \leq 0.05$) in relation to the heavier littermates. Feed conversion by marginal piglets reared by foster dams was significantly less efficient. The lower growth rate of marginal piglets was the result of their behavioural status (the inability to compete against stronger littermates) and physiological factors. To evaluate the effect of birth weight and method of supplementary feeding on weaning weight and fattening performance of piglets, Wolter et al. (2002) assigned piglets differing in body weight to two treatments. The use of milk replacer significantly increased weaning weight of piglets compared to their birth weight ($P < 0.01$) but had no effect on weight gains from weaning to slaughter. There was a birth weight by supplemental feeding method interaction, but only for weight gains between 14.5 and 25 kg of body weight. It resulted from the fact that light piglets that received no milk replacer to 21 days of growth grew much more quickly than piglets fed milk replacer (700 vs. 624 g). According to the same authors, birth weight has a greater effect than milk replacer on growth rate from weaning to slaughter, which is consistent with the results obtained by Powell and Aberle (1980) and the conclusions made by Lawlor et al. (2002).

Schinckel et al. (2010), who used regression analysis to study the effect of birth weight on weight gain in different growth and fattening periods, found that they were related. However, the importance of birth weight decreased with increasing body weight and age. Birth weight accounted for 12–13% of variation in daily gain obtained until 47 kg, for 8–9% of gain until 64 kg of body weight, and for only 2.0–2.4% of gain until 102.5 kg of body weight. The coefficients of regression for daily gain on birth weight depended on the weight of piglets. The coefficient of regression for daily gain to 102.5 kg per g of birth weight was 20–27 g for a piglet weight of around 1.0 kg, 9–10 g for a birth weight of around 1.5 kg, and only 3 g for a weight of 2.0 kg. The authors of the study reported the expected differences in daily

gains of piglets whose birth weight was 1.0 and 2.0 kg. Piglets weighing 1.0 kg grow more slowly: by 82 g/day up to 46.7 kg body weight, by 71 g/day up to 64.6 kg, by 46 g/day up to 83.5 kg, and by 43 g/day up to 102.5 kg of body weight.

The results obtained by all the researchers cited above indicate that piglets with low birth weight grow more slowly and reach slaughter weight later than their heavier littermates.

Conclusions

Analysis of the literature dealing with determinants and characteristics of IUGR in animals has pointed to high fetal survival and nutritional and hormonal deficiencies as the reasons for weaker growth and development of embryos as well as fetuses or their organs during pregnancy. The key role of arginine in placental development and fertility, as well as the importance of nutrition research in terms of prenatal growth and development of fetuses, and postnatal growth and development of progeny were emphasized. The effect of mother nutrition on myogenesis and adipogenesis was examined. Attention was given to the balancing of rations and the need to meet the requirement of pregnant sows for protein and some amino acids by changing their proportion in the diet. Special consideration should be given to research results that can be used in an interdisciplinary manner. One possibility is prevention of IUGR in different species of mammals, including humans; another is to manipulate the neonatal weight of farm animals, especially pigs, as well as their postnatal growth and development expressed as fattening parameters. In summing up this literature review, it should be pointed out that not all the results of the experiments are consistent, which shows that research in the area under discussion should be continued.

References

- Ashton C., Bayol S., McEntee G., Maltby V., Stickland N. (2005). Prenatal influences on skeletal muscle development in mammals, birds and fish. *Arch. Tierz., Dummerstorf*, 48: 4–10.
- Beaulieu A.D., Aalhus J.L., Williams N.H., Patience J.F. (2010). Impact of piglet birth weight, birth order, and litter size on subsequent growth performance, carcass quality, muscle composition and eating quality of pork. *J. Anim. Sci.*, 88: 2767–2778.
- Bee G. (2004). Effect of early gestation feeding, birth weight, and gender of progeny on muscle fiber characteristics of pigs at slaughter. *J. Anim. Sci.*, 82: 826–836.
- Bee G. (2007). Birth weight of litters as source of variation in postnatal growth, and carcass and meat quality. *Adv. Pork Prod.*, 18: 191–195.
- Bérard J., Kreuzer M., Bee G. (2008). Effect of litter size and birth weight on growth, carcass and pork quality, and their relationship to postmortem proteolysis. *J. Anim. Sci.*, 86: 2357–2368.
- Bérard J., Pardo C.E., Bethaz S., Kreuzer M., Bee G. (2010). Intrauterine crowding decreases average birth weight and affects muscle fiber hyperplasia in piglets. *J. Anim. Sci.*, 88: 3242–3250.
- Boulot S., Quesnel H., Quiniou N. (2010). Management of high prolificacy in French herds: can we alleviate side effects on piglet survival? *Pig Industry, Engormix*.
- Brown L.D., Green A.S., Limesand S.W., Rozance P.J. (2011). Maternal amino acid supplementation for intrauterine growth restriction. *Front. Biosci. (Schol Ed)*, 1, 3: 428–444.
- Brüssow K.P., Wähler M., Jaśkowski J.M. (2011). Biological limit of fecundity in sows – do they exist? *EJPAU*, 14, p. 3.
- Campos P.H., Silva B.A.N., Donzele J.L., Oliveira R.F.M., Knol E.F. (2012). Effects

- of sow nutrition during gestation on within-litter birth weight variation: a review. *Animal*, 6: 797–806.
- Canario L., Lundgren H., Haandlykken M., Rydhamer L. (2010). Genetics of growth in piglets and the association with homogeneity of body weight within litters. *J. Anim. Sci.*, 88: 1240–1247.
- Cerisuelo A., Baucells M.D., Gasa J., Coma J., Carrion D., Chapinal N., Sala R. (2009). Increased sow nutrition during midgestation affects muscle fiber development and meat quality, with no consequences on growth performance. *J. Anim. Sci.*, 87: 729–739.
- Chen Z.Y., Dziuk P.J. (1993). Influence of initial length of uterus per embryo and gestation stage on prenatal survival, development, and sex ratio in the pig. *J. Anim. Sci.*, 71: 1895–1901.
- Chmurzyńska A. (2010). Fetal programming – link between early nutrition, DNA methylation and complex diseases. *Nutr. Rev.*, 68 (2): 87–98.
- Du M., Tong J., Zhao J., Underwood K.R., Zhu M., Ford S.P., Nathanielsz P.W. (2010). Fetal programming of skeletal muscle development in ruminant animals. *J. Anim. Sci.*, 88: E51–E60.
- Dwyer C.M., Fletcher J.M., Stickland N.C. (1993). Muscle cellularity and postnatal growth in the pigs. *J. Anim. Sci.*, 71: 3339–3343.
- Dwyer C.M., Stickland N.C., Fletcher J.M. (1994). The influence of maternal nutrition on muscle fiber number development in the porcine fetus and subsequent postnatal growth. *J. Anim. Sci.*, 72: 911–917.
- Foxcroft G.R., Dixon W.T., Novak S., Putman C.T., Town S.C., Vinsky M.D. (2006). The biological basis for prenatal programming of postnatal performance in pigs. *J. Anim. Sci.*, 84: E105–E112.
- Funston R.N., Larson D.M., Vonnahme K.A. (2010). Effects of maternal nutrition on conceptus growth and offspring performance: implications for beef cattle production. *J. Anim. Sci.*, 88: E205–E215.
- Gatford K.L., Ekert J.E., Blackmore K., De Blasio M.J., Boyce J.M., Owens J.A., Campbell R.G., Owens P.C. (2003). Variable maternal nutrition and growth hormone treatment in the second quarter of pregnancy in pigs alter semitendinosus muscle in adolescent progeny. *Br. J. Nutr.*, 90: 283–293.
- Gatford K.L., Smits R.J., Collins C.L., Argent C., De Blasio M.J., Roberts C.T., Nottle M.B., Kind K.L., Owens J.A. (2010). Maternal responses to daily maternal porcine somatotropin injections during early-mid pregnancy or early-late pregnancy in sows and gilts. *J. Anim. Sci.*, 88: 1365–1378.
- Gondret F., Lefaucheur L., Louveau I., Lebret B. (2005). The long-term influences of birth weight on muscle characteristics and eating meat quality in pigs reared and fed during fattening. *Arch. Tierz.*, 48: 68–73.
- Gondret F., Lefaucheur L., Juin H., Louveau I., Lebret B. (2006). Low birth weight is associated with enlarged muscle fiber area and impaired meat tenderness of the longissimus muscle in pigs. *J. Anim. Sci.*, 84: 93–103.
- Herpin P., Damon M., Le Dividich J. (2002). Development of thermoregulation and neonatal survival in pigs. *Livest. Prod. Sci.*, 78: 25–45.
- Hill R.A., Connor E.E., Poulos S.P., Welsh T.H., Gabler N.K. (2010). Growth and development symposium: fetal programming in animal production. *J. Anim. Sci.*, 88 (E Suppl.): 38–39.
- Ishida M., Hiramatsu Y., Masuyama H., Mizutani Y., Kudo T. (2002). Inhibition of placental ornithine decarboxylase by DL-[alpha]-difluoro-methyl ornithine causes fetal growth restriction in rat. *Life Sci.*, 70: 1395–1405.
- Johnson R.K., Nielsen M.K., Casey D.S. (1999). Responses in ovulation rate, embryonal survival, and litter traits in swine to 14 generations of selection to increase litter size. *J. Anim. Sci.*, 77: 541–557.
- Koczanowski J., Kopyra M., Orzechowska B., Tyra M., Żak G., Nowicki J. (2006). Effect of feeding level after mating on embryo survival in purebred and crossbred gilts. *Ann. Anim. Sci.*, Suppl., 2/2: 357–361.
- Lawlor P.G., Lynch P.B., Caffrey P.J., O'Doherty J.V. (2002). Effect of pre- and post-weaning management on subsequent pig performance to slaughter and carcass quality. *Anim. Sci.*, 75: 245–256.

- Lawlor P.G., Lynch P.B., O'Connell M.K., McNamara L., Reid P., Stickland N.C. (2007). The influence of over feeding sows during gestation on reproductive performance and pig growth to slaughter. *Arch. Tierz.*, 50: Special Issue, pp. 82–91.
- Lefaucheur L., Ecolan P., Barzic Y.M., Marion J., Dividich J. (2003). Early postnatal food intake alters myofiber maturation in pig skeletal muscle. *J. Nutr.*, 133: 140–147.
- Lösel D., Kalbe C., Rehfeldt C. (2009). L-carnitine supplementation during suckling intensifies the early postnatal skeletal myofiber formation in piglets of low birth weight. *J. Anim. Sci.*, 87: 2216–2226.
- Markham T.C.W., Latorre R.M., Lawlor P.G., Ashton C.J., McNamara L.B., Natter R., Rowlerson A., Stickland N.C. (2009). Developmental programming, of skeletal muscle phenotype/metabolism. *Anim. Cambridge Univ. Press*, 3: 1001–1012.
- McNamara L.B., Giblin L., Markham T., Stickland N.C., Berry D.P., O'Reilly J., Lynch P.B., Kerry J.P., Lawlor P.G. (2011). Nutritional intervention during gestation alters growth, body composition and gene expression patterns in skeletal muscle of pig offspring. *Animal*, 5: 1195–1206.
- Milligan B.N., Fraser D., Kramer D.L. (2002). Within-litter birth weight variation in the domestic pig and its relation to pre-weaning survival, weight gain, and variation in weaning weights. *Livest. Prod. Sci.*, 76: 181–191.
- Müller J. (2011). 32 piglets per sow and year – development of costs in piglet production under conditions of high production intensity (in German). *Schweinezucht aktuell*, 37: 21–23.
- Musser R.E., Davis D.L., Dritz S.S., Tokach M.D., Nelssen J.L., Minton J.E., Goodband R.D. (2004). Conceptus and maternal responses to increased feed intake during early gestation in pigs. *J. Anim. Sci.*, 82: 3154–3161.
- Nissen P.M., Danielsen V.O., Jørgensen P.F., Øksbjerg N. (2003). Increased maternal nutrition of sows has no beneficial effect on muscle fiber number or postnatal growth and has no impact on the meat quality of the offspring. *J. Anim. Sci.*, 81: 3018–3027.
- Nissen P.M., Jørgensen P.F., Øksbjerg N. (2004). Within-litter variation in muscle fiber characteristics, pig performance, and meat quality traits. *J. Anim. Sci.*, 82: 414–421.
- Nissen P.M., Vestergaard M., Øksbjerg N. (2005). The influence of increased maternal nutrition on offspring muscle development, growth and meat quality and endocrine concentration of IGFs and IGFBPs in both sow and fetuses. *Arch. Tierz., Dummerstorf*, 48: Special Issue, 40 pp.
- Øksbjerg N., Nissen P.M., Therkildsen M., Møller H.S., Larsen L.B., Andersen M., Young J.F. (2013). In utero nutrition related to fetal development, postnatal performance and meat quality of pork. *J. Anim. Sci.*, 91: 1443–1453.
- Onteru S.K., Fan B., Nikkila M.T., Garrik D.J., Stalder K.J., Rothschild M.F. (2011). Whole-genome association analyses for lifetime reproductive traits in the pig. *J. Anim. Sci.*, 89: 988–995.
- Orzechowska B., Mucha A. (2010). Results of reproductive performance of breeding sows. In: Report on pig breeding (in Polish). IZ-PIB, Kraków, Poland, XXVIII: 3–19.
- Pere M.Ch., Etienne M. (2000). Uterine blood flow in sows: effects of pregnancy stage and litter size. *Reprod. Nutr. Dev.*, 40: 369–382.
- Powell S.E., Aberle A.D. (1980). Effects of birth weight on growth and carcass composition of swine. *J. Anim. Sci.*, 50: 860–867.
- Powell S.E., Aberle A.D. (1981). Skeletal muscle and adipose tissue cellularity in runt and normal birth weight swine. *J. Anim. Sci.*, 52: 748–756.
- Quiniou N., Dagorn J., Gaudre D. (2002). Variation of piglets birth weight and consequences on subsequent performance. *Livest. Prod. Sci.*, 78: 63–70.
- Quinton V.M., Wilton J.W., Robinson J.A., Matur P.K. (2006). Economic weights for sow productivity traits in nucleus pig populations. *Livest. Sci.*, 99: 69–77.
- Rehfeldt C. (2005). Prenatal events that determine the number of muscle fibers are important for lean growth and meat quality in pigs. *Arch. Tierz., Dummerstorf*, 48: Special Issue, pp. 11–22.
- Rehfeldt C., Kuhn G. (2006). Consequences of birth weight for postnatal growth performance and carcass quality in pigs as related to myogenesis. *J. Anim. Sci.*, 84 (E Suppl.): 113–123.
- Rehfeldt C., Kuhn G., Nürnberg G., Kanitz E., Schneider F., Beyer M., Nürnberg K., Ender K.R. (2001). Effects of exogenous somatotropin during early gestation on maternal performance, fetal growth, and compositional traits in pigs. *J. Anim. Sci.*, 79: 1789–1799.

- Rehfeldt C., Nissen P.M., Kuhn G., Vestergaard M., Ender K., Oksbjerg N. (2004). Effects of maternal nutrition and porcine growth hormone (pGH) treatment during gestation on endocrine and metabolic factors in sows, fetuses and pigs, skeletal muscle development, and postnatal growth. *Domest. Anim. Endocrinol.*, 27: 267–285.
- Rehfeldt C., te Pas M.F.W., Wimmers K., Brameld J.M., Nissen P.M., Berri C., Valente L.M.P., Power D., Picard B., Stickland N.C., Oksbjerg N. (2011). Advances in research on the prenatal development of skeletal muscle in farm animals in relation to the quality of muscle-based food I. Regulation of myogenesis and environmental impact. *Animal*, 5: 703–717.
- Rehfeldt C., Lefaucheur L., Block J., Stabenow B., Pfuhl R., Otten W., Metges C.C., Kalbe C. (2012 a). Limited and excess protein intake of pregnant gilts differently affects body composition and cellularity of skeletal muscle and subcutaneous adipose tissue of newborn and weaning piglets. *Eur. J. Nutr.*, 12, 51: 151–165.
- Rehfeldt C., Stabenow B., Pfuhl R., Block J., Nürnberg G., Otten W., Metges C.C., Kalbe C. (2012 b). Effects of limited and excess protein intakes of pregnant gilts on carcass quality and cellular properties of skeletal muscle and subcutaneous adipose tissue in fattening pigs. *J. Anim. Sci.*, 90: 184–196.
- Ruiz-Flores A., Johnson R.K. (2001). Direct and correlated responses to two-stage selection for ovulation rate and number of fully formed pigs at birth in swine. *J. Anim. Sci.*, 79: 2286–2297.
- Ryan B.C., Vandenberg J.G. (2002). Intrauterine position effects. *Neurosci. Biobehav. Rev.*, 26: 665–678.
- Schinckel A.P., Einstein M.E., Jungst M.E., Booher S., Newman S. (2010). Evaluation of impact of pig birth weight on grow-finish performance, backfat depth and loin depth. *Prof. Anim. Sci.*, 26: 193–205.
- Sterle J.A., Cantley T.C., Matteri R.I., Carroll J.A., Lucy M.C., Lamberson W.R. (2003). Effect of recombinant porcine somatotropin on fetal and placental growth in gilts with reduced uterine capacity. *J. Anim. Sci.*, 81: 765–771.
- Tribout T., Caritez J.C., Gogué J., Gruand J., Billon Y., Bouffaud M., Lagant H., Le Dividich J., Thomas F., Quesnel H., Guéblez R., Bidanel J.P. (2003). Estimation par utilisation de semence congelée, du progrès génétique réalisé en France entre 1977 et 1998 dans la race porcine Large White: résultats pour quelques caractères de reproduction femelle. *J. Rech. Porc. Fr.*, 35: 285–292.
- Vallet J.L., Leymaster K.A., Christenson R.K. (2002). The influence of uterine function on embryonic and fetal survival. *J. Anim. Sci.*, 80 (E Suppl. 2): E115–E125.
- Wolter B.F., Ellis M., Corrigan B.P., DeDecker J.M. (2002). The effect of birth weight and feeding of supplemental milk replacer to piglets during lactation on preweaning and postweaning growth performance and carcass characteristics. *J. Anim. Sci.*, 80: 301–308.
- Wu M.C., Chen Z.Y., Jarell V.L., Dziuk P.J. (1999). Effect of initial length of uterus per embryo on fetal survival and development in the pig. *J. Anim. Sci.*, 67: 1767–1772.
- Wu G., Bazer F.W., Cudd T.A., Meininger C.J., Spencer T.E. (2004). Maternal nutrition and fetal development. *J. Nutr.*, 134: 2169–2172.
- Wu G., Bazer F.W., Wallace J.M., Spencer T.E. (2006). Board-invited review: Intrauterine growth retardation: implications for the animal sciences. *J. Anim. Sci.*, 84: 2316–2337.
- Wu G., Bazer F.W., Burghardt R.C., Johnson G.A., Kim S.W., Li X.L., Satterfield M.C., Spencer T.E. (2010). Impacts of amino acid nutrition on pregnancy outcome in pigs: mechanisms and implication for swine production. *J. Anim. Sci.*, 88 (E Suppl.): E195–E204.
- Wu G., Bazer F.W., Satterfield M.C., Li X., Wang X., Johnson G.A., Burghardt R.C., Dai Z., Wang J., Wu Z. (2013). Impacts of arginine nutrition on embryonic and fetal development in mammals. *Amino Acids*, 4, Published online: 04 June 2013.
- Zhu M.J., Ford S.P., Means W.J., Hess B.W., Nathanielsz P.W., Du M. (2010). Maternal nutrient restriction affects properties of skeletal muscle in offspring. <http://jp.physoc.org/content/575/1/241.full>