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Physiological Changes in Sows that may Cause to Higher Risk for Mortality and Prolapse?

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Mini Review

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ABSTRACT

Long-term profitability of swine farms is highly related to maintaining a healthy sow farm that consistently improves production efficiency. In recent years, genetic selection techniques have successfully reversed the trend of increased pre-wean pig mortality with increased litter size by improving litter weight at birth. However, the genetics modifications to reduce pre-wean pig mortality may have exerted more physiological pressure on the sow to deliver a heavier litter birth weight because sow mortality and incidence of Pelvic Organ Prolapse (POP) has increased over the past few years. Recent production records have reported an annualized sow mortality of 12.7% in North American herds, with POP contributing to 21% of the sow mortality. Some factors associated with risk for POP include poor sow body condition, sows with greater protrusion of the perineal region, higher stillborn rate, constipation, inadequate pre-farrow feed intake, and poor water quality. Similar factors associated with POP in humans have been reported and include other metabolic triggers like anemia, oxidative stress, and inflammation as likely contributing factors. Recent data indicates about 50% of sows have anemia with a higher prevalence in older parity sows and in sows during late gestation and lactation. Other metabolic factors like elevated serum glucose in sows with high risk for POP may indicate a possible somatotropic relationship and sensitivity of maternal organs to growth hormone on regulation of glucose homeostasis and transplacental nutrient supply to fetuses. Herein, we review the physiological changes during gestation and lactation, propose how these changes may be contributing to POP, and briefly discuss other potential factors associated with sow mortality.

INTRODUCTION

Historically, genetic selection for increased litter size led to lower average pig birth weight due to limited uterine capacity and higher pre-wean mortality [1-3]. However, within the last few years modified genetic selection techniques have reduced pre-wean pig mortality by increasing uterine capacity and moderately increasing average gestation days by 2-3 more days resulting in higher average pig birth weight along with continued improvement in total born litter size [1]. Unfortunately, these genetic selection modifications used to successfully reduce pre-wean pig mortality may exert more physiological pressure on the sow to deliver a larger and heavier litter at birth, so selection criteria including sow longevity should also be considered [4].

LITERATURE REVIEW

In recent years as genetic advancements have been implemented at commercial farms, production records from 104 farms (385,000 sows) have reported a total annualized sow mortality of 12.7% [5]. Causes of mortality were recorded as unknown (39%), lameness (29%), pelvic organ prolapse (21%), farrowing difficulty (6%), disease (2%) and ulcers (3%). Factors associated with Pelvic Organ Prolapse (POP) include poor sow body condition, sows with greater protrusion of perineal region, inadequate pre-farrow feed intake, higher number of stillborn pigs, constipation, pelleted feed, low fiber sow feed, and poor water quality. Similar factors related to POP in humans have been reported. Conditions that cause excessive pressure on the pelvic floor like obesity, persistent coughing, heavy weight-lifting and chronic constipation contribute to the development of POP in humans [6]. Interestingly, anemia was also identified as a major contributing factor for prolapse. Human epidemiology studies reported that moderate to severe anemia was found in 52% of the patients, and that anemia was proposed as one of the biggest risk factors for the development of prolapses [6,7]. However, a moderate degree of anemia is physiologically normal during human pregnancy and should be distinguished from iron deficient anemia. Iron deficient anemia in humans reflects depleted iron stores and is usually defined in clinical practice using serum markers like ferritin or percentage of transferrin saturated by iron [8]. As in humans, some degree of anemia can be expected in sows. Physiologically, blood serum volume and packed red blood cell volume undergo significant changes in gestating and lactating sows. Past research has shown that serum volume as a percentage of body weight increases by 25%, while packed red cell volume decreases 22% during late gestation compared with early gestation [9]. Changes in serum and red cell volume makes biological sense because sows during late gestation divert more nutrient supply to their rapidly developing fetuses and to the mammary glands for colostrum production derived from the circulating plasma. The immunological protein profile in colostrum is remarkably like that in blood plasma or serum, therefore the sow in late gestation may be limiting her own immune defense needs to provide more passive immunity through colostrum to support neonatal pig immunity. Genetic selection focused on birth weight and pre-wean survivability, may unintentionally further increase serum volume for colostrum production, reduce red blood cell volume, and therefore result in more severe anemia for sows. A more thorough understanding of changes in blood volume and nutrient supply to various organ systems throughout gestation and lactation is needed to develop appropriate strategies to support health and life-time productivity of the modern sow.

DISCUSSION

Recent studies in sows indicate variable degrees of anemia at different stages of pregnancy or lactation with a greater prevalence of anemia in older parity sows and during lactation [10-12]. Approximately 50% of 2,683 sows sampled had serum hemoglobin (Hb) at <10 g/dL as an indicator of anemia [10]. However, simply supplementing

sows with more dietary iron or injecting sows with iron may not eliminate anemia [12]. Absorption of dietary iron is highly regulated and excess iron intake is excreted.

Sow anemia can affect body metabolism. Hemoglobin is a well-known scavenger of nitric oxide. Nitric oxide produced in the endothelial cells of blood vessels relaxes the muscle cells surrounding the vessel. Decreased concentration of circulating red blood cells and hemoglobin can lead to more nitric oxide production and cause pelvic organ muscles to relax and thereby contribute to POP. Sows at higher risk of POP also have higher serum lactic acid and increased abundance of 2-aminobuanoic acid, a derivative of butyric acid [13]. Serum lactic acid is inversely related with circulating hemoglobin [14] and butyric acid is positively correlated with inflammation [15,16]. Inflammation can induce anemia and further reduce red cell and hemoglobin level, contributing to POP incidence [17]. Also, oxidative stress has been proposed as a trigger for POP in humans. Oxidative stress disrupts normal cellular signaling pathways thereby interfering with collagen and elastin synthesis [18]. Therefore, the natural changes in serum and packed cell volume of sows near parturition may contribute to anemia, inflammation, and oxidative stress which collectively increases the risk for higher incidence of POP. Basic research is needed to fully understand the physiological changes of serum and red cell volume and concentration changes of modern sows with large litter size and litter weight to better understand how it could potentially be related to higher POP incidence.

Although the fetal genome is an important factor for growth potential, maternal uterine capacity and nutrient supply from the uterus has the greater impact on pig birth weight [1]. Selection for maternal growth rate instead of fetal growth rate resulted in the increased litter size and birth weight that we have observed over the last decade. Increased blood flow into the placenta can enhance uterine capacity and nutrient supply. Increased placenta blood flow is an important and primary mechanism for increased transplacental exchange throughout gestation [19,20]. The large increase of blood flow to the uterus during late pregnancy results primarily from the formation and growth of the placental vascular bed [19]. Altered placental growth and vascular development has been associated with altered expression of the genes for the major angiogenic factors, including vascular endothelial growth factor and endothelial nitric oxide synthase, which produces nitric oxide, an important regulator of both angiogenesis and vasodilation [21]. Genetic selection for increased litter size and weight is associated with increased placental vascularity and vascular endothelial growth factor expression in Meishan and Yorkshire pigs [22-24]. Unfortunately, the trade-off of enhanced placental growth and increased nitric oxide produced in the endothelial cells of blood vessels may contribute to increased pressure on the body cavity organs and relaxation of surrounding muscle cells which may potentially contribute to POP.

Increased or altered nutrient supply in blood can also enhance transplacental exchange and subsequentially increase intrauterine fetal growth. For example, glucose can be transported by facilitated diffusion transporters of the solute carrier 2A (SLC2A; GLUT) in pigs [25]. Concentrations of glucose in fetal blood are normally 40% to 70% lower than concentrations of glucose in maternal blood and this promotes glucose transfer [26]. Creating a higher concentration of nutrients in blood may further increase nutrient transport and fetal growth. Serum D-glucose (19.7 *vs.* 0.794 mmol/L) was 25 times greater in high risk (perineal score 3) POP sows than for low risk (perineal score 1) POP sows [13]. This large increase in blood glucose for high-risk POP sows was likely derived from maternal reserves considering the tightly controlled feed allowance typically provided during gestation. Glucose levels in blood are tightly controlled under normal health and environmental conditions. A significant change in metabolic status is needed to mobilize glucose from maternal reserves.

Metabolic changes associated with exogenous growth hormone can affect circulating glucose [27,28]. Growth hormone has somatotropic effects that act on the liver and adipose tissue to influence gluconeogenesis and lipolysis to help ensure an adequate glucose supply to the fetal pig [29]. Maternal growth hormone levels rise profoundly during late gestation but do not increase in the fetal circulation. Increased maternal growth hormone level and/or sensitivity to growth hormone may be one of the reasons why we see higher placental nutrient supply and increased litter size and birth weight. Indeed, researchers have demonstrated that growth hormone infused in pregnant sows can increase placental and fetal growth [30,31]. However, further investigations about the somatotropic relationship and sensitivity of maternal organs to growth hormone are needed to better understand if it is associated with sows at a higher risk of POP and mortality.

Past studies have investigated the use of exogenous recombinant Porcine Somatotropin (pST) to increase fetal growth and sow milk production. More sows treated with pST died compared to non-treated sows with 10 of the 14 deaths of the pST-treated sows occurring just before, during, or within 2 days after farrowing [27]. Of the sows that died, postmortem findings included lesions that were indicative of cardiac failure resulting from hyperthermia. Lesions reported included degenerative cardiomyopathy, hepatic lipidosis, and centrilobular degeneration and renal tubular lipidosis and hypoxia was suspected to cause these lesions. This study was done during the summer and many of the sows treated with pST had increased respiration rates suggestive of heat stress. A diet with higher fat was suggested for pST-treated sows to reduce heat increment and potentially mitigate some of the mortality related to pST treatment. Others have reported that pST increased heat production which could increase sow susceptibility to heat stress [32]. These metabolic effects of pST show some similarity in terms of the current pattern of sow POP and mortality in North America.

Sow mortality associated with pST injected immediately before or during parturition was attributed to acute respiratory distress [33] while others reported that a dose-related incidence of mortality in sows treated with pST during lactation was attributed to gastric ulcers [34]. Furthermore, pST treated sows failed to consume their feed allowance and had a higher rate of hemorrhagic gastric and intestinal ulcers in sows that died [27]. As we discussed previously, considering that blood volume with a reduced hematocrit increases in late gestation to support an increased flow of blood towards the uterine and mammary systems, the trade-off may be a reduction of blood flow and nutrients to the GI tract. If so, this shift in blood volume away from the GI tract may be related to a higher incidence of gastric ulcers, especially for sows under heat stress or other factors that affects metabolism causing reduced feed consumption and subsequent nutrient uptake and utilization. Field experiences have supported that spray dried plasma proteins are an effective nutritional tool to help pigs recover from gastric ulcers. Spray-dried plasma proteins have been used in drinking water to help grower pigs recover from gastric ulcers [35]. Also, performance horses provided a nutritional supplement containing plasma-based proteins had a reduced incidence of gastric ulcers [36]. It would be worthy to investigate the application of plasma proteins in feed or water for sows with a high risk for POP and mortality.

CONCLUSION

Although genetic selection techniques have increased litter size and birth weight, the trade-off in physiological changes in the sow may have unwanted consequences. Further research is needed to investigate the potential relationship of these physiological changes in the sow to develop management, health, nutrition, and genetic strategies to enhance overall sow resilience against POP and mortality. Dietary and health management strategies that could reduce anemia, constipation, respiratory pressure, inflammation, and oxidative stress status of the sow around parturition should be further investigated.

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