ABSTRACT

GUO, JIYAO. Nutrition and Management Strategies for Improving Performance and Health of Sows and Nursery Pigs. (Under the direction of Dr. Sung Woo Kim).

The research hypotheses are: (1) management of litter size and lactation length can affect sow and litter performance in a subsequent lactation, (2) nutritional management of reducing dietary cation-anion difference can enhance the Ca homeostasis of lactating sows, and (3) milk supplementation can improve the intestinal health and subsequently improve the growth performance of nursery pigs.

The first study was conducted to evaluate the effects of suckling intensity (litter size and lactation length) on sow performance and litter growth in a subsequent parity. Upon parturition, 115 primiparous sows (222.7 ± 20.0 kg) were initially allotted to 1 of 4 treatments according to a 2 × 2 factorial arrangement with varied litter sizes: 10 and 13 piglets; and varied lactation lengths: 21 and 27 d in the first lactation. Sixty-six sows were successfully rebred and used in the second lactation with the same litter size (10 piglets) and the same lactation length (21 d). This study found that increasing suckling intensity during the first lactation did not negatively affect the sow and litter performance, but reduced BW loss from 3.7 to 1.7% of BW and reduced backfat loss from 12.1% to 5.2% of backfat in the second lactation.

The second study evaluated the effects of suckling location and suckling history (suckling condition in the previous lactation) of mammary glands on piglet growth in a subsequent lactation. In the first experiment, 27 primiparous sows were used in the first lactation, and all were reused in the second lactation. Mammary glands were allotted in 1 of 3 suckling locations (anterior, middle, and posterior). In the second experiment, the suckling condition of glands (not suckled or suckled) in the first lactation was considered as the

suckling history of glands at the second lactation. Results showed that piglets at anterior and middle locations had 13.2 and 41.4 g greater ADG at the first and second lactations, respectively, as compared with those at posterior location. Suckling history of the mammary glands did not affect the weight gain of piglets at either suckling location during the second lactation.

The third study estimated the effects of dietary cation-anion difference (DCAD) on acid-base balance and Ca homeostasis in sows during late gestation and lactation. Twenty-two multiparous sows were fed 1 of 2 dietary treatments: (1) a control diet with a DCAD of 67 and 78 mEq/kg or (2) an anionic diet with a DCAD of -122 and -148 mEq/kg, from d 94 of gestation to d 18 of lactation. The third study found that reducing DCAD during late gestation and lactation could induce a mild metabolic acidosis at farrowing, and increase serum and colostrum Ca by 4.6% and 20.0%, respectively during the lactation, possibly due to increased bone resorption.

The fourth study was conducted to evaluate the effects of supplemental milk (10% of estimated postweaning feed intake) during the first 4 d postweaning on growth performance, nutrient digestibility, intestinal integrity, diarrhea, and economic returns of nursery pigs. A total of 644 crossbred pigs (3 wk of weaning age, 6.4 ± 1.2 kg of BW) were randomly assigned to 1 of 2 dietary treatments in a randomized complete block design with sex and initial BW as blocks. Pigs were fed pellet feed either with or without milk supplementation from d 1 to 4 postweaning in a liquid feeding trough. All pigs had free access to another nursery metal feeder for the pellet feed during the 3-phase nursery period (49 d). Results showed that supplemental milk for the first 4 d postweaning improved intestinal integrity and reduced the severity of diarrhea during phase I, increased feed efficiency by 5.65% during

phase I and II, and decreased mortality from 4.35% to 1.55%. However, supplemental milk did not increase the economic returns during the entire nursery period.

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Nutrition and Management Strategies for Improving Performance and Health of Sows and Nursery Pigs

by
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DEDICATION

To my family, Fuchang Guo, Xiuling Yang, Jipeng Guo; mentor, Dr. Sung Woo Kim; and friends. Without your support, it was almost impossible for me to complete my graduate program.

BIOGRAPHY

Jiyao Guo, son of Fuchang Guo and Xiuling Yang, was born in on October 8th, 1987 in Youyu, Shanxi, located in the North of China. From childhood, Jiyao was interested in fiction, animals, outdoor activities, and TV. Jiyao graduated from Shuocheng No. 1 High School in 2006. At age 19, after talking with his Uncle Qingping Liu and his father, he decided to study animal science. At that time, he had never thought of being a scientist. Jiyao was accepted by China Agricultural University in Beijing, China and received the Bachelor of Science degree in Animal Science in 2010. During the 4 years in China Agricultural University, Jiyao had a better understanding to animal science through taking courses and participating researches in beef cattle, poultry, and swine. With a desire to know and learn swine nutrition better, Jiyao was accepted in the Department of Animal Science of North Carolina State University in 2011, under the direction of Dr. Sung Woo Kim. With the guide from Dr. Sung Woo Kim, he overcame the obstacles and improved himself. He received his Master of Science degree in Animal Science and Poultry Science at North Carolina State University in 2013. At that time, he thought 2-year study was too short to view a full picture of swine nutrition and to think deeply about what he had researched, therefore, he decided to pursue his Ph.D. program in the Department of Animal Science of North Carolina State University under the direction of Dr. Sung Woo Kim in 2013.

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LIST OF ABBREVIATIONS

ADG: average daily gain

ADFI: average daily feed intake

AOAC: Association of Official Analytical Chemists

BW: body weight

CP: crude protein

d: day

DM: dry matter

g: gram

G:F: gain to feed ratio

h: hour

ME: metabolizable energy

min: minute

NASS: National Agricultural Statistics Service

NRC: National Research Council

s: second

SEM: standard error of the mean

SID: standard ileal digestibility

USDA: United States Department of Agriculture

wk: week

CHAPTER 1

LITERATURE REVIEW

Introduction

Swine operations commonly include units from farrowing to nursery, grower-finishing and breeding. Each unit requires unique management and feeding strategies to meet animal requirements at different stages. Effective strategies for managing sows and nursery pigs could improve growth performance, health, reproductive performance, and longevity, which benefit the producers as well. This literature review is focused on evaluating different farrowing management and nutritional strategies for sows and nursery pigs and corresponding outcomes on their performance and health.

The average numbers of farrowing sows and pigs per litter in the United States from 2014 to 2015 were 3,004,000 head and 10.28 piglets, respectively, according to NASS-USDA (2016). The average weaning age was 20.8 d. According to the National Animal Health Monitoring System of USDA, from 2011 to 2012, the total born per litter was 11.3 piglets, of which 10.3 piglets were born alive and 9.4 were weaned. The mortality of weaned pigs was as low as 3.6%. The mortality of sows and gilts in average was 4.3% and the culling rate was 25.1% on average. The reason for culling is mainly due to old age (35.2%), reproductive failure (25.4%), low performance (13.5%, indicated with small litter size, high preweaning mortality, or low birth weight), lameness, injury, and other reasons.

The data provided by USDA represent the average level of pig production nowadays. A deep understanding of pig production efficiency is critical. According to See (2000), efficiency of sow production can be characterized by: 1) maximized number of pigs per year per litter size; 2) optimal pig birth and weaning weights, 3) maximized litters per year; 4)

maximized lactation yield; and 5) optimized sow longevity. The continuous genetic selection has led to leaner, faster-growing, and more prolific pigs than years ago. However, genetic selection based on above production and reproduction traits have resulted in limited feed intake capacity and reduced body reserves (Ten Napel et al., 1995; Whittemore, 1996). It has been well documented that the selection for leaner and lower-feed intake pigs adversely affects reproductive performance (Rydhmer et al., 1992; Gaughan et al., 1995; Kerr and Cameron, 1995), and even adversely affects sow longevity since the reproductive performance is tightly associated with sow longevity.

It is critical to improve sow longevity because it can benefit pig producers by reducing the expense of gilt replacement and associated management cost. Sow longevity is influenced by various factors, such as gilt development, gilt pool management, the age of puberty and first farrowing, nutrition, lactation length, body condition, repeat breeding, season, housing and sow behavior, feet and leg soundness, and management (Stalder et al., 2004). Improving the efficiency of sow productivity is complex and requires comprehensive approaches in nutrition and management. Some sow management strategies aim at maintaining body reserves such as backfat and body weight during reproductive cycle, balancing dietary nutrients, and eliminating the heat and oxidative stresses (See, 1996; Flowers and Day, 1990; Zhao et al., 2013). For example, the maintenance of body reserves can be achieved by encouraging feed intake and reducing litter size during late lactation using the strategy of “split weaning”. Meanwhile, heat stress can be reduced by maintaining an optimally functioning ventilation system with an adequate flow rate and by reducing heat

increment of feeding. Diets with low CP and high fat are considered to reduce the heat production and to assistant animals be more tolerant under the heat stress situation (Renaudeau et al., 2001; Le Bellego et al., 2002). The purpose of maintaining health sows good-body condition is to produce enough milk and for supporting fetal survival to achieve optimal litter weight gain. Insufficient milk production can directly contribute 6 to 17% of all pre-weaning mortality in commercial pig farms (Alonso-Spilsbury et al., 2007). Therefore, it is necessary to understand the physiological characteristics of milk production and mammary growth, as well as the nutrient requirements and balance in sows.

Weaning is accompanied by dietary, environmental, social, and psychological stressors (Hampson, 1986). Development of optimal feeding programs serve as an important approach to attenuate the weaning stress. The immediate period following weaning is characterized by low feed intake, slow growth rate, and depressed immune system (Bilko et al., 1994a; McCracken et al., 1995; Spreeuwenberg et al., 2001b). Poor performance of newly weaned pigs may be due to the inadequate feed intake and insufficient dietary nutrients utilization during the first 7 d postweaning (Braude et al., 1977; Bark et al., 1986). The feed intake and nutrient digestibility of newly weaned pigs are influenced by various factors such as health status, creep feeding, weaning age, environment, dietary nutrient levels and balance, sources of ingredients, water supply and quality (Leibbrandt et al., 1975; Hampson, 1986; Tokach et al., 1995). A comprehensive approach is required to overcome the problem of low postweaning feed intake. The major dietary strategies to increase feed intake of newly weaned pigs include using highly digestible and palatable ingredients,

formulating diets with balanced nutrients, adopting growth-promoting agents, and flavors and taste enhancers (Dong and Pluske, 2007; de Lange et al., 2010). Feed represents about 60 to 70% of the total cost of pork production. Pig producers also need to take serious consideration about balancing feed cost, the use of highly digestible but expensive ingredients, the overall health status, and total revenue of nursery pigs.

Milk Production

Milk especially colostrum contains nutrients that are essential for the survival and substantial growth of neonatal piglets until weaning. Mature sow's milk (mean value for 4 wk of lactation) contains 18.8% dry matter, 4.9% protein, 5.2% lactose, 7.6% fat, and 1,208 kcal/kg gross energy; whereas colostrum has a higher dry matter and nutrients than mature milk: 26.6% dry matter, 16.4% protein, 3.4% lactose, 5.3% fat, and 1,555 kcal/kg gross energy (Vadmand et al., 2015). The nutrient composition of sow's colostrum can vary significantly within the herd. Lin et al. (2009) reported a nutrient composition of cow's colostrum with 21.9% dry matter, 14.9% protein, and 4.8% fat. The protein concentration in mature milk is about one-third of that in colostrum. Sow colostrum production varies from 1.9 to 5.3 kg/d with a mean of 3.5 kg/d (Devillers et al., 2007); the milk production on d 4 of lactation varies from 4.6 to 9.6 kg/d with a mean of 8 kg/d (Theil et al., 2002). In general, the mature milk production is about twice of colostrum production daily. Milk protein and free amino acids concentrations decrease with the lactation period (Vadmand et al., 2015). According to a previous study (Csapó et al., 1996), sow milk at d 4 of lactation contains 0.84% ash, 1,965 mg/kg Ca, 1,510 mg/kg P, 6.49 mg/kg Zn, 2.44 mg/kg Fe, and 1.34 mg/kg

Cu, which are all greater than the concentrations of these in cow milk. Moreover, the concentrations of Ca and P in sow milk increase throughout lactation.

Blood components are the major sources for milk synthesis. The milk protein mainly includes α-, β-, γ-, and κ-casein, α-lactalbumin, β-lactoglobulin, blood serum albumin, immunoglobulins, and a protease-peptone fraction (Rose et al., 1970). The protein synthesis in the mammary epithelial cells is similar to the process in other cells, which is accompanied by the transcription of genetic information to mRNA and subsequent translation into protein from an mRNA template. Casein is the dominating protein in milk and the major milk proteins are synthesized from the free amino acids in blood. Milk lactose is a disaccharide derived from galactose and glucose, which is originally from blood glucose. Galactose can be synthesized in mammary glands or other tissues through hexoneogenesis. Milk fat consists of triglycerides, very low abundance amount of phospholipids, cholesterol, fat-soluble vitamins, squalene, free fatty acids, monoglycerides, and other compounds (Swenson, 1984).

How is milk produced by mammary glands? Milk synthesis occurs in epithelial cells of mammary glands. In general, when piglets suckle or massage the mammary glands, the behavior induces neuroendocrine reflex and stimulates the oxytocin production from the posterior pituitary. Oxytocin travels through the blood stream of the mammary glands and causes myoepithelial cell contraction, which will cause milk to be actively eject out of alveoli and pinched into the alveolar lumen. The milk will be forced down the ducts and go into gland cistern (McManaman and Neville, 2003). The duration of milk flow during each suckling bout is only 10 to 20 s. The suckling frequency of piglets is usually more than 24

times per day with the average suckling interval less than 1 h. However, milk injection can be stopped by stress, fear, and pain induced inhibition of oxytocin. This is because stress can inhibit the milk ejection by stimulating the release of epinephrine and cortisol from the adrenal gland.

Milk removal from the lactating mammary glands is considered as the major factor in maintaining milk secretion (Hurley, 2001). Maintenance of milk production relies on milk removal because the process removes a protein named Feedback Inhibitor of Lactation, which inhibits milk production (Rennison et al., 1993). Incomplete milk removal reduces milk production in dairy animals (Wilde and Knight, 1990). Except for milk removal, milk production is affected by several other factors such as maternal nutrition, genetics, management, body condition, environment, and characteristics of litter (King, 2000; Farmer and Quesnel, 2009). Moreover, milk production is also associated with paracellular and tight-junction permeability (Stelwagen et al., 1994; Stelwagen et al., 1995). In addition, milk production is also affected by the endogenous hormones before farrowing. For instance, the colostrum production is positively correlated with prolactin concentration and negatively correlated with progesterone concentration (Farmer and Quesnel, 2009; Foisnet et al., 2010). The negative relationship between progesterone and milk production is resulted from the inhibition of lactose secretion by progesterone (Banchero et al., 2006).

The traditional method for estimating milk production is based on the litter weight gain during suckling (weigh-suckle-weigh technique) (Perisse and Salmon-Legagneur, 1960;

Mahan et al., 1971). In addition, water turnover of piglet body (dilution of deuterium oxide) is used as another approach to estimate milk production (Pettigrew et al., 1985).

Mammary Growth

Anatomy of Lactating Mammary Glands

The mammary glands of sows are located along the ventral surface of both thorax and abdomen. The lactating mammary glands are composed of branches of ducts formed lobulo-alveolar cluster (McManaman and Neville, 2003). As the basic component of the lactating mammary glands, alveolus is composed of a single layer of epithelial cells within the cluster, which is the site of milk secretion (Figure 1). Myoepithelial cells are attached outside of the secretory epithelial cells, with the main functions of milk ejection and vascularized connection for adipocytes and fibroblasts.

Mammary gland growth can be indicated by the mammary tissue weight and DNA content of mammary glands. The number of milk secretory cells in mammary glands is the main limiting factor for milk production. The DNA content per mammary cell is a constant (Simpson and Schmidt, 1969), therefore, total DNA content per gland is directly related to the number of mammary cells. Total mammary DNA is also correlated with the size and total mammary area of animals. In the rat, 11% of the mammary development occurs before pregnancy, 41% of the mammary development occurs during pregnancy, and 48% of the mammary development occurs during lactation (Tucker, 1969). The graph of mammary gland development (indicated as total mammary DNA) in rats from birth through lactation is presented in Figure 2.

Gestation

In prepubertal gilts, mammary tissue and DNA accumulation rate increases slowly with age until 3 months of age, and then increases 4 to 6 folds (Sorensen et al., 2002). The development and growth of mammary glands is slow during the first two-thirds of gestation, however, almost all the mammary tissue and DNA accumulation during pregnancy occurs during the last third of gestation (Sorensen et al., 2002). During early gestation, mammary ducts are elongated and branched (Hurley and Bryson, 1999). The major differentiation and proliferation of alveolar cells occurs during the second half of the gestation. During this period, there is a remarkable increase in the number of lobular-alveolar structures and there is a great loss of the fat pad histologically (Ji et al., 2006). The mammary glands undergo functional reorganization and changes from adipose storage to the secretory functions. Ji et al. (2006) indicated that the wet weight gain and protein accretion accelerated from d 75 of gestation. The DNA content reaches to a maximum value by 90 d of gestation (Kensinger et al., 1982). From d 90 to 105 of gestation, the lactogenesis starts with an abundant accumulation of epithelial secretion cells (Kensinger et al., 1982; Farmer and Sorensen, 2001). During gestation, dietary nutrients can affect the mammary development. For instance, Weldon et al. (1991) reported that dietary energy had a great impact on mammogenesis. In addition, hormones such as progesterone and combined with estrogen triggers the mammary growth during the gestation (Ash and Heap, 1975). Farmer and Sorensen (2001) stated that prolactin also involves in mammogenesis, especially associated with the parenchymal tissue weight as well as DNA, RNA, and protein contents.

Lactation

The mammary glands undergo a substantial growth during the lactation. Hurley (2001) concluded that mammary gland development associated with milk production can occur in 2 ways: 1) proliferation of mammary cells, which results in greater numbers of milk secretory cells; and 2) enhanced differentiation of mammary cells, which results in greater milk production per secretory cell. Unlike dairy cows, the involution of milk secretory cells during lactation can be ignored, because lactation of modern lactating sows is normally terminated at 3 to 4 wk postpartum. Now, the milk production just reaches the peak and the main mammary involution has not fully started (Hartmann and Holmes, 1989).

Limited literature is found related to sow mammary growth during lactation. Kensinger et al. (1982) firstly indicated that DNA concentration of mammary tissues increased from farrowing to d 4 of lactation. Kim et al. (1999b) reported that lactating mammary glands had a 55% increase in wet weight and a 100% increase in total mammary DNA from d 5 to 21 of lactation, suggesting that the total number of milk secretory cells increased during lactation. Kim et al. (1999b) also indicated that the percentage of protein, dry fat-free tissue, and DNA per wet tissue weight also increased along with lactation, which means that the cell density in the mammary tissue increased as well. Therefore, Kim et al. (1999b) concluded that both hyperplastic and hypertrophic growth occurred during lactation.

Postweaning

Weaning causes the cessation of milk removal, which initiates the mammary involution or regression (Ford et al., 2003). During lactation, mammary glands that are not

regularly suckled undergo a quick regression. Regression of unsuckled mammary glands occurs rapidly during the first 7 to 10 d of lactation (Kim et al., 2001). In addition, Kim et al. (2001) indicated that the regression of mammary glands was highly associated with dietary energy and nutrient levels. Increasing dietary energy and protein levels from 12 to 17.5 Mcal ME/d and 32 to 65 g lysine/d, respectively, reduced the regression of unsuckled mammary glands by 91%. Mammary glands that are not regularly suckled had no further loss of parenchymal tissue during d 0 to 7 postweaning (Ford et al., 2003). In addition, mammary glands that are regularly suckled during lactation also undergo a quick regression after weaning. Ford et al. (2003) indicated that wet weight and DNA of suckled mammary glands were 485.9 and 838.2 g on the day of weaning, and decreased to 151.5 and 278.4 g, respectively, by d 7 postweaning. Therefore, mammary glands lost about two-thirds of the total mass or total mammary cells by d 7 postweaning. Results from Ford et al. (2003) also proved that the substantial growth of mammary glands during lactation could carry over to d 7 postweaning, because the suckled mammary glands still had a greater wet weight of parenchymal tissue than the unsuckled mammary glands. The graph of mammary gland development (as wet weight of the glands) in sows from birth through lactation is presented in Figure 3.

Subsequent Lactation

Pitkow et al. (1972) reported that alveolar cells formed in the first lactation did not involute faster during the second lactation, as compared to alveolar cells newly formed in the second lactation, suggesting that alveolar cells can be functional in 2 consecutive lactations.

Fraser and Thompson (1986) concluded that association between piglet weight gains and mammary locations (ADG of piglets that suckled anterior glands was the greatest) did not exist in the first-parity sows but existed in the second-parity sows. Fraser et al. (1992) attributed the different effects of mammary locations on piglet weight gain between 2 lactations to the suckling stimulus to mammary glands during the first lactation. Moreover, results from Fraser et al. (1992) and Ford et al. (2003) implied that the retention of a greater mass of suckled mammary glands might contribute to a greater mammary productivity in the subsequent lactation.

Farmer et al. (2012) indicated that lactating mammary glands that were suckled in the first lactation contains greater amount of parenchymal tissue (800.4 vs. 641.6 g/gland), greater DNA content (1.80 vs 1.41 g/gland), and greater RNA content (3.99 vs. 3.37 g/gland) at d 17 of the second lactation, as compared with lactating mammary glands that were not suckled during the first lactation. However, weight of piglets was not different at d 14 of the second lactation. At d 37 postweaning during the second lactation, piglets who suckled mammary glands that were suckled during the first lactation weighed heavier (1.12 kg greater), as compared with piglets who suckled mammary glands that were not suckled during the first lactation (Farmer et al., 2012). However, Fraser et al. (1992) and Hurley (2001) suggested that substantial variability exists in the total mass of glands and piglets weight gain, especially when the effects of mammary location are involved.

Factors Affecting Milk Production and Mammary Growth

Maternal Nutrition and Sow Body Condition

Adequate nutrients in the diets are critical for producing sufficient milk, maintaining piglet survival, and supporting piglet growth. In addition, sows can mobilize body reserves to fulfill the nutrient requirements of milk production (King and Williams, 1984; King and Dunkin, 1986; Dourmad et al., 1998). Therefore, the dietary nutrition is interacting with the sow body tissue mobilization in regards to the milk production. Nelssen et al. (1985) reported that sow weight and backfat loss linearly decreased and litter weight increased at weaning, as dietary energy intake increased from 10 to 14 Mcal of ME. Moreover, the breakdown of lean tissue also increases during lactation, to fulfill the requirement of amino acids for milk production, especially when the dietary amino acids are restricted (Jones and Stahly, 1999). Sows, that do not consume adequate amounts of amino acids, produce less milk, and subsequently have a reduced litter weight gain (King and Williams, 1984; King and Dunkin, 1986). King and Williams (1984) and King and Dunkin (1986) also reported that primiparous sows, which received diets with high digestible energy and CP concentration, were in a positive nitrogen balance, had a reduced body weight loss, and increased milk production. Adequate dietary energy and protein intake is critical for maximizing the litter weight gain during late lactation as well. Sows cannot provide enough milk to maintain the maximum litter weight gain (Hartmann et al., 1984; Harrell et al., 1993) during late lactation when their piglets grow bigger. However, unlike multiparous sows, primiparous sows may partition more nutrients into body growth than into milk production. Pluske et al. (1998) evaluated the

milk production of primiparous sows with varied feed intake levels: 1) restricted feeding (50% of estimated *ad libitum* intake); 2) *ad libitum* feeding; 3) superalimented feeding (25 to 30% greater than estimated *ad libitum* intake). The results showed that milk production was similar among treatments, whereas, the primiparous sows in superalimented feeding accumulated a greater live weight and a higher backfat thickness during the lactation compared with others.

Dietary energy and protein intake affects the mammary growth of primiparous sows during lactation (Kim et al., 1999b). The weight of suckled mammary glands, amount of mammary tissue protein, dry fat-free tissue, and DNA were maximal on d 27.5 of lactation, as sows consumed 16.9 Mcal of ME and 55 g lysine per day during lactation. Kim et al. (2009) reviewed the ideal ratios of amino acids for fetal growth, mammary growth, and maternal gain during both gestation and lactation. The true ileal digestible Lys was suggested as 5.57 and 8.78 g from d 0 to 60 and d 60 to 114 of gestation, respectively. The ideal ratios for Lys:Thr:Val:Leu was 100:59:77:115 and 100:69:78:123, respectively, with lactational BW loss of 0 and 33 to 45 kg, respectively. In addition, the nutrient demand for mammary and litter growth increases with increased litter size (Kim et al., 1999b). An addition of 0.96 g lysine per day was suggested to the diets when litter size increases by one pig, to account for the additional mammary growth.

Body condition of sows is tightly relevant to milk production when sows do not have adequate feed intake (Kim et al., 2000). Milk that is produced by sows with a lower feed intake is synthesized directly from nutrients mobilized from their body tissues. Klaver et al.

(1981) reported that sows that have a normal body condition produced 24% more milk than sows that have a thin body condition. Except for the effects of sows' body condition on milk yield, O'Grady et al. (1973) and Klaver et al. (1981) indicated that sows in a better body condition produced milk with greater contents of fat and energy than sows in a thinner body condition. However, fat sows had a reduced milk production and a reduced litter growth rate as compared to lean sows (Revell et al., 1998). King (2000) speculated the reason was that lean sows had a greater body protein reserves until farrowing as compared to fat sows.

Litter Management of Suckling Intensity: Litter Size

Litter size is the major factor determining milk production (King, 2000). This is mainly because increasing litter size increases the number of functional mammary glands and increases the number of milk secretory cells (Tucker, 1966; Knight and Peaker, 1984). Milk production is linearly associated with litter size (Elsley, 1971). Increasing litter size per additional piglet increased 0.6 kg milk production per day (Noblet et al., 1998). Previous literature also reported that sows with a 12-piglet litter produced 50% more milk than sows with a 6-piglet litter (King et al., 1989; King, 1991). However, increasing litter size decreases the milk production of each individual gland. Increasing litter size from 6 to 14 piglets decreased ADG of piglets from 283 to 202 g from birth to 28 d of age (Auldist et al., 1998). Collectively, Auldist et al. (1998) and King (2000) commented that increasing the number of functional mammary glands by increasing litter size could compensate for any decrease in milk output of individual glands, and improve the overall milk production subsequently. Milk composition is relatively stable in response to the increased litter size; whereas, suckling

frequency increased linearly during early lactation (d 10 to 14 of lactation) in response to the increased litter size (Auldist et al., 1998).

Sows with a larger litter have a greater amount of total mammary tissue and total mammary cells than sows with a smaller litter. Kim et al. (1999) reported that increasing litter size from 6 to 12 piglets increased the wet weight of total mammary tissue, the amount of total protein, and the amount of total DNA of glands by 75%, 63%, and 67% on d 21 of lactation, respectively. The change of mammary growth in response to the increased litter size showed the same pattern as the change of milk production. Individual glands were smaller in sows having a larger litter size as compared with those in sows having a smaller litter size, which was indicated by that wet weight, and the amounts of protein, dry fat-free tissue, DNA, and ash per gland linearly decreased as litter size increased from 6 to 12 piglets. Even through the size of individual glands decreased with increased litter size, litter weight gain until d 21 of lactation increased by 96% (18.1 kg) in response to increasing litter size from 6 to 12 piglets (Kim et al., 1999a). Therefore, mammary glands of sows with a larger litter size may be more efficient to produce milk as compared with those of sows with a smaller litter size.

Litter Management of Suckling Intensity: Age and Size of Piglets

Heavier or older piglets can stimulate a greater milk flow (King, 2000). Fraser (1984) suggested that heavier piglets could massage the glands more vigorously. Because of that, mammary blood flow increases and subsequently milk flow in the glands increases. In addition, the more vigorous massage of heavier piglets on glands can affect the secretion of

lactogenic hormones, such as prolactin (Algers et al., 1991). The different lactation responses to age and size of piglets can be observed by cross-fostering the litters either in different ages or in different weights. Marshall et al. (2006) cross-fostered litters of 1-d-old of age with litters of 7-d-old of age, the results showed that the older litters had a greater accumulated weight gain during early lactation (d 3 to 9 of lactation), but had a similar accumulated weight gain by d 15 of lactation, as compared to the younger litters. King et al. (1997) cross-fostered 14-d-old piglets onto newly farrowed sows, and the results showed that the milk yield enhanced by 20% during the first 14 d of lactation. In addition, Van der Steen and De Groot (1992) and Boyce et al. (1997) proved that piglets with heavier birth weights grow faster during the lactation, suggesting that heavier piglets can remove a greater amount of milk from sows.

Mammary growth is stimulated by an increased demand for milk production (Kim et al., 1999c). Heavier piglets or older piglets have a greater demand on milk during lactation. Results from Kim et al. (1999c) revealed that individual piglets within a smaller litter size had a larger weaning weight; individual mammary glands suckled by these heavier piglets had a greater growth as indicated by greater wet weight, greater percentage of dry fat-free tissue and protein, and greater content of DNA at d 21 of lactation. In addition, Kim et al. (2000) summarized that size of glands is positively associated with the weight gain of piglets.

Litter Management of Suckling Intensity: Suckling Frequency

The typical suckling interval varies from 30 to 70 min for sows during the first wk of lactation (Jensen et al., 1991), whereas the suckling interval could be varied among sows.

Spinka et al. (1997) suggested that the milk secretion is commonly completed in 35 min after the previous suckling bout. The suckling interval between nursing tends to be prolonged during the night and in later period of lactation (Niwa et al., 1951). Auditory disturbance and social facilitation can potentially increase the frequency of suckling bout and milk production. About 35% all nursing is initiated by the piglets, 27% by the sows, and 36% by the social facilitation (Whatson and Bertram, 1980). Therefore, the social facilitation such as artificially reproduced nursing call has been used to increase the suckling frequency. Stone et al. (1974) indicated that playing back the record of nursing sounds could increase the suckling frequency and milk production at early lactation. Fisette et al. (2004) also suggested that playing recorded nursing grunts at a shorter-time interval could reduce the nursing interval. Moreover, Petrie and Gonyou (1988) suggested that piglet and sow nursing vocalization could increase the number of drinking bouts and time of feeding during the early postweaning period. In addition, the suckling frequency could be increased by using a cross-suckling treatment with 2 groups of litters (Auldist et al., 2000).

Increasing suckling frequency or shortening suckling intervals increases milk production (Sauber et al., 1994; Auldist et al., 1995). Changing suckling frequency has a greater impact on sows than on ruminant, because sows have little non-alveolar milk storage volume (Hurley, 2001). Suckling frequency is related to the milk removal frequency and the milk accumulation in mammary glands. High suckling frequency means that milk accumulation after a suckling bout can be diminished rapidly and further milk secretion rate will not be influenced. Spinka et al. (1997) suggested that milk secretion was largely

determined by the volume of alveoli and by the frequency and completeness of emptying these alveoli. The same study extended the suckling interval from 35 to 70 min and found that although milk intake per suckle increased by 30%, overall daily milk production of sows decreased by 21%. Auldist et al. (1995,2000) reported a similar result that milk yield increased in response to the reduction of suckling interval from 44 to 35 min during the early lactation (d 10 to 14 of lactation).

Increasing suckling frequency had a positive feedback on the development of alveoli through decreasing the inhibitory effects of milk accumulation, as speculated by Spinka et al. (1997). Wilde et al. (1995) identified a milk protein, termed Feedback Inhibitor of Lactation (FIL), which regulates the milk secretion through autocrine feedback inhibition in goat. Undoubtedly, increasing frequency and completeness of milk removal from alveoli reduce the inhibitory effects of this FIL protein. Moreover, Wilde and Knight (1989) reported that the frequency of milk removal influenced both cellular differentiation and proliferation in mammary tissue. Subsequently, increasing the suckling frequency increased mammary cell numbers in rats (Tucker et al., 1967). In addition, Auldist et al. (2000) reported that induvial gland weight increased by 26%, when suckling interval decreased from 45 to 35 min during early lactation (d 10 to 14 of lactation) and decreased from 49 to 42 min during late lactation (d 24 to 28 of lactation).

Physiological Demands of Sows and Corresponding Strategies

The reproductive success of genetic selection has accelerated the reproductive cycles, increased the pigs per sow per year, increased pig growth rate, and increased lean tissue

development and feed conversion (Canario et al., 2007). Therefore, sows nowadays demand greater and faster nutrient supplies to support fetal growth and milk production than before. However, the body fat reserve of modern sows reduces due to the genetic selection for leaner pigs, which implies modern sows have less body fat to mobilize. Therefore, sows must cope with the high physiological demands when the external (dietary feed intake) and internal nutrients (body reserves and mobilization) are limited. In addition, modern sows should utilize the limited nutrients to encounter the detrimental effects from the environment.

Energy and Protein Demand of Sows

Lactating sows require adequate energy and protein to maintain body tissues, milk production, and maternal growth. Dietary energy and protein deficiency during lactation increases the mobilization of fat and protein body reserves (Noblet and Etienne, 1986). The demand of energy and protein of lactating primiparous sows can be more serious, because the primiparous sows during lactation lose greater amount of body weight and backfat than multiparous sows (Mahan, 1998). Eissen et al. (2000) considered the lower appetite, lower feed intake during lactation, and higher demand for nutrients for body growth of primiparous sows as the major reasons for the greater body weight losses. Insufficient energy and protein intake may further impair the reproductive performance of sows. Quesnel (2009) reviewed the nutritional effects on follicular development and reported that dietary energy and protein deficiency resulted in a delayed weaning-to-estrus interval, a reduced embryonic survival, and an increased culling rate.

As Meisinger (2010) suggested, the negative energy and nitrogen balance can be minimized by increasing sow dietary energy and protein intake. The nutrient requirements of lactating sows are influenced by factors such as parity, their body weight and body condition, milk yield and composition, and environment conditions. Therefore, the energy and protein in diets are expected to be adjusted based on the above factors. In addition, increasing the fat and protein body reserves at farrowing could help sows to prepare for the milk production and catabolic status during lactation (Clowes et al., 2003; Quesnel et al., 2005). Management strategies, aimed at reducing milk production during late lactation without impairing litter growth, can reduce body tissue mobilization and catabolism. The related strategies include cross-fostering, anticipated- or split-weaning. These management strategies can reduce the body weight loss of lactating sows by removing heavier litter or piglets from the farrowing batch and weaning these piglets earlier than others.

Introduction of Dietary Minerals in Sows

Minerals are required for fetal growth, mammary secretion, bone growth and development, and maintenance. The mineral requirements are the largest during late gestation and lactation (Mahan, 1990). Kornegay and Kite (1983) and Maxson and Mahan (1986) suggested that dietary requirements minerals, such as Ca and P, during pregnancy increases in proportion to the demand for fetal growth. The requirements of Ca and P are largely influenced by the milk production during lactation (NRC, 1998). When dietary minerals are deficient, sows use their body mineral pool primarily to compensate the fetal mineral requirements. Most of the negative mineral balance can be compensated by the

increased bone mineral mobilization (Mahan, 1990). Severe mineral deficiencies can cause various symptoms depending on the deficiency of certain element. Collectively, if the mineral demand for reproductive performance is greater than the mineral supply from body reserves and dietary intake, the reproductive performance may be impaired and culling rate can be increased (Mahan, 1990). For example, inadequate Se supplementation during late gestation could cause a smaller litter size (Mahan et al., 1974; Chavez and Patton, 1986). Failure to maintain the mineral balance and failure to rapidly mobilize mineral reserves from bones can also cause a series of skeletal problems such as lameness, which is one of the major reasons for sow culling (Jones, 1967; Penny, 1973,1980). In addition, the mineral requirements in nutrition guides are based on a certain level of feed intake of sows. Therefore, voluntary of feed intake is also a critical determinate factor that influences the mineral balance of sows. Strategies for reducing the incidence of lameness include formulating balanced diets with adequate minerals and vitamins, and stimulating the feed intake of lactating sows. Likewise, manipulating the dietary cation-anion difference is one of the solutions to balance the dietary minerals.

Cation-anion Difference

Cation-anion difference is one of the indicators of mineral balance. Cation-anion difference (anion-cation balance, electrolyte balance, strong ion difference) can be defined quantitatively by the difference between strong cations (positive) and strong anions (negative) in solution. The dietary cation-anion difference (DCAD) is used to determine the relationships between strong cations and anions and to predict whether the diet will evoke an

acidic or alkaline response to animals. Cations such as Na, K, Ca, and Mg and anions such as Cl, S, and P, are commonly used in the calculation of dietary cation-anion difference. Among these, Na, K, S, and Cl, which have the strongest ionic effects on acid-base balance, are regarded to as the "strong ions" or "fixed ions". Dietary cation-anion difference mainly affects acid-base status, blood minerals, renal function, and even milk production of lactating animals (Delaquis and Block, 1995). In detail, effects of DCAD on Ca homeostasis include influencing milk Ca content, bone Ca accretion, and incidence of hypocalcemia (Oetzel et al., 1988; Oetzel et al., 1991; Liesegang et al., 2007).

The most common DCAD equation is commonly expressed as milliequivalents (mEq) of (Na + K – Cl) (Mongin, 1981). Patience et al. (1987) reported that growth performance of growing pigs did not change when DCAD ranged from 0 to 341 mEq/kg but decreased at -85 mEq/kg. Other studies (Golz and Crenshaw, 1990; Haydon and West, 1990) subsequently suggested an optimal DCAD value for postweaning and growing pigs to be 250 mEq/kg. Moreover, Dove and Haydon (1994) and DeRouchey et al. (2003) reported that decreasing DCAD at gestation and lactation from 250 to130 mEq/kg or from 500 to 0 mEq/kg did not affect the ADFI, BW change, litter weight gain, and weaning-to-estrus interval of sows. These 2 studies also reported that decreasing DCAD tended to decrease the piglet weight gain and increase the piglet survivability during lactation (Dove and Haydon, 1994; DeRouchey et al., 2003).

The effects of DCAD on acid-base balance physiology have been evaluated in ruminant (Tucker et al., 1988; West et al., 1992) and nonruminant species (Cohen and

Hurwitz, 1974; Hamilton and Thompson, 1980; Patience et al., 1987). Urine cation-anion difference and serum cation-anion difference are positively associated with DCAD (Tucker et al., 1988; West et al., 1992). Decreasing DCAD linearly or quadratically decreases blood pH, decreases the concentration of blood HCO_3, and increases base excess in swine (Haydon and West, 1990; Budde and Crenshaw, 2003; DeRouchey et al., 2003) and dairy cows (Tucker et al., 1988; West et al., 1991). Maintaining blood pH constantly is critical for normal physiological function. Therefore, the physiological acidic or alkaline response evoked by changing DCAD need to be regulated. The physiological regulation includes adjusting respiration rate to control blood pCO_2 and adjusting rental excretion to control the concentration of blood HCO_3 (Tucker et al., 1988). Decreasing DCAD not only induces the metabolic acid load in blood but also results in the acidification of urine. Previous studies showed that decreasing DCAD could stimulate the renal compensation, increase the urine H, Cl, and NH_4 excretion, and reduce the urine HCO_3 excretion (West et al., 1992; Patience and Chaplin, 1997; Budde and Crenshaw, 2003). Consequently, it has been shown that urine pH decreased linearly or quadratically with the decreased DCAD (Canh et al., 1998; DeRouchey et al., 2003; Razzaghi et al., 2012).

Dietary cation-anion difference equation expressed as mEq of (Na + K – Cl – S) is most commonly used in studies related to Ca homeostasis (Oetzel et al., 1988; Oetzel et al., 1991; Horst et al., 1997). Anionic or acidifying diets are formulated with supplementation of anionic salts in diets, which results in a decreased DCAD. As we reviewed previously, changing DCAD alters the acid-base balance through a series of adjustments by kidney and

blood buffer systems. The acid-base balance, especially acidosis also alters Ca metabolism in swine (Budde and Crenshaw, 2003), dairy cows (Goff et al., 1991; Abu Damir et al., 1994), and goat (Horst and Jorgensen, 1974), through influencing bone resorption (Block, 1984; Goff et al., 1991), intestinal Ca absorption (Lomba et al., 1978), and renal Ca excretion (Braithwaite, 1972). Abu Damir et al. (1994) reported that acidifying diet to a DCAD of -35 mEq/kg increased Ca absorption and Ca mobilization rate in dairy cows during 14 d pre-parturition. Results from Budde and Crenshaw (2003) showed that Ca intake, serum Ca, and urine Ca excretion increased with decreased DCAD from 212 to -35 mEq/kg, whereas, Ca retention was not different among treatments with different levels of DCAD. Similarly, Darriet et al. (2009) also reported that decreasing DCAD from 33 to -216 mEq/kg increased mobilization of body Ca pools through increasing urinary and fecal Ca excretion for gestating sows. In addition, several studies suggested that the dietary acidification induces a greater intestinal Ca absorption through decreasing intestinal pH and increasing Ca solubility (Ender et al., 1971; Lomba et al., 1978). The response of intestinal Ca absorption to anionic diets only happens when there is a positive Ca balance (Lomba et al., 1978; Block, 1984).

The Ca metabolism, including bone resorption, intestinal Ca absorption, and renal Ca excretion, is regulated by the hormones 1,25-dihydroxylvitamin D_3 [1,25-$(OH)_2D_3$] and parathyroid hormone (PTH). Therefore, the effects of DCAD on Ca regulating hormones are worth investigating. Verdaris and Evans (1976) also suggested that the 1,25-$(OH)_2D_3$ was a predominant cause of increased intestinal Ca absorption. Abu Damir et al. (1994) indicated that dietary acidification increases the plasma concentration of 1,25$(OH)_2D_3$. Parathyroid

hormone, secreted by the parathyroid glands, can stimulate renal 1,25-$(OH)_2D_3$ production. Gaynor et al. (1989) and Goff et al. (1991) suggested that the 1,25-$(OH)_2D_3$ in dairy cows fed anionic diets are more responsive to PTH than those fed cationic diets. Block (1984) and Goff et al. (1991) also proved that feeding anionic diets to prepartum dairy cows increased the bone resorption and utilization of bone Ca at parturition, which was indicated by an increased plasma hydroxyproline concentration (an index of bone Ca resorption activity).

Decreasing DCAD improves the Ca homeostasis. This finding is of importance for dairy cows because the hypocalcemia caused by large secretion of Ca in colostrum may be prevented by supplementation of anionic salts in prepartum diets. Hypocalcemia is also referred to as milk fever and parturient paresis. Some animals may fail to regulate or get adapt to the fast Ca loss due to colostrum production and subsequently have recumbency and paresis (Goff et al., 1991). Prevention of hypocalcemia in dairy cows by supplementing anionic diets before parturition has been well documented (Oetzel et al., 1988; Goff et al., 1991; Horst et al., 1997). Block (1984), Goff and Horst (1998), and Oetzel et al. (1988) reported that serum ionized Ca or plasma total Ca increased with decreased DCAD. Oetzel et al. (1988) suggested that supplementation of anionic salt (NH_4Cl and $[NH_4]_2SO_4$) with a DCAD of -75 mEq/kg DM for 21 d before parturition reduced the incidence of hypocalcemia from 17 to 4%. Furthermore, Oetzel et al. (1991) and Lean et al. (2006) indicated that levels of DCAD have a strong linear relationship with the incidence of hypocalcemia.

Decreasing DCAD may potentially reduce or prevent the incidence of urinary tract infections by inducing an acidic environment in the urinary tract. Lesions in the urinary tract

(about 18%) are considered as one of the major causes of mortality or culling among sows, whereas, the other causes include reproductive disorders (about 27%), and old age (about 19%) (Tillon and Madec, 1984; Engblom et al., 2007; Sanz et al., 2007). Almond and Stevens (1995) reported that 22 to 40% of sows had urinary tract infections in Europe. Urinary tract infections occur when bacteria, predominantly *Escherichia coli* (*E. coli*) proliferates at the opening of the vulva, vagina, and urethra, then spread up to the bladder and even kidney (Carr and Walton, 1993; Ronald, 2002). The population of *E. coli* may potentially be reduced by changing the pH of urinary tract environment, because *E. coli* colonize better in a neutral environment of the urinary tract (Abdul-Raouf et al., 1993; Presser et al., 1997).

Nutrition and Management of Liquid Milk Feeding in Nursery Pigs

The natural weaning of pigs is a gradual process, which can vary from 11 to 17 wk of age (Worobec et al., 1999). In commercial farms, weaning age nowadays increased from 19. 06 to 20.17 d (PigCHAMP, 2006,2014). Compared with natural weaning, reduced weaning age benefits the efficiency of farms by increasing pigs per sow per year and reducing the disease transfer from sows to piglets. However, weaning under commercial farm conditions generally results in the “postweaning lag” or “growth check”, which could last from 7 to 14 d and is characterized by depressed feed intake, slow growth or weight loss, diarrhea, increased mortality and morbidity (Rivera et al., 1978; Pluske et al., 1995). This is because newly weaned pigs are exposed to several environmental, behavioral, immunological, social, and nutritional stressors (Pluske et al., 1997; Williams, 2003). The predominant stress is partially

attributed to the change of diets from sow milk to less digestible and less palatable plant-based diets. Plant-based diets contain several anti-nutritional factors for newly weaned pigs (Heo et al., 2013). In addition to the anti-nutritional factors, the inadequate feed intake is also considered as the major determinant of slow growth, impaired intestinal morphological integrity, and depressed immune systems (Bilko et al., 1994b; McCracken et al., 1995; Spreeuwenberg et al., 2001a). Especially, pigs with low birth weight or without enough milk intake have lower weaning weight than average pigs. It is of great importance to allow the low-weaning weight pigs to catch up with other pigs. Successful achievement of this goal could decrease the mortality and morbidity of nursery pigs. In addition, lack of early postweaning growth can cause a delay in days to reach market weight (Mahan and Lepine, 1991).

The small intestine of pigs undergoes rapid change in size, structure, brush border enzymes, and immune functions during the immediate postweaning period (Hampson, 1986; Pluske et al., 1997; Heo et al., 2013). The epithelial lining of the small intestine's walls has finger-like projections known as villi, which helps to increase the surface area for absorption. The base of villi is surrounded by multiple epithelial invaginations termed crypt, which is known as the place for proliferating epithelial cells (Clevers, 2013). Several studies have demonstrated that there is a reduction in villus height (villus atrophy), and an increase in crypt depth (crypt hyperplasia) during the immediate postweaning period (Cera et al., 1988; Kelly et al., 1991b; Pluske et al., 1997). Hampson (1986) and Miller et al. (1986) reported that the villus height at d 5 postweaning was approximately 50% of that at weaning. The

morphological change of small intestine at weaning can be associated with the nutrient intake. Kelly et al. (1991a) showed that pigs with continuous nutrient intake have greater villus height and crypt depth than those with restricted nutrient intake, suggesting that a reduction in feed intake may be responsible for villus atrophy and crypt hyperplasia. In addition, postweaning pigs have a hypersensitivity response to certain ingredients like soybean meal, as indicated by reduced villus height and slightly greater crypt depth (Li et al., 1990). The weaning transition in pigs is also followed by changes in activities of brush-border enzymes. Overall, the enzyme activities decrease between 3 to 5 d postweaning and increase gradually thereafter (Hampson and Kidder, 1986; Pluske et al., 1997). However, lactase activity keeps dropping after weaning. It has been shown that that unweaned pigs had an age-dependent decrease in lactase activity (Kelly et al., 1991b). Mccracken (1984) and Kelly et al. (1991a) reported that maltase and glucoamylase activities increased after weaning because of the substrate induction effects. This effects could be similar to the function of inducible enzyme systems in bacteria that the substrates induce a *de novo* synthesis of enzyme protein and removal of the substrates ceases the enzyme protein synthesis (Jacob and Monod, 1961). The changes in structure and brush border enzymes are also associated with enterocyte turnover and malnutrition. Furthermore, McCracken et al. (1999) indicated that inadequate feed intake during the immediate postweaning period also contributed to the intestinal inflammation.

A variety of management and feeding strategies have been developed to improve the gut health and growth performance of nursery pigs. Frequent liquid feeding is a rational

approach to promoting feed intake and reducing the weaning stress of nursery pigs (Lecce et al., 1979; Odle and Harrell, 1998). The studies about liquid feeding started in 1940's, continued through late 1970's and 1990's (Lecce et al., 1979; Azain et al., 1996; Zijlstra et al., 1996). Early studies (Lecce and Coalson, 1973, 1976; Lecce et al., 1979) brought up a management approach for reducing postweaning stress. The management procedure involved removing piglets from sows after colostrum feeding, caging piglet individually, and feeding hourly a liquid milk diet that was composed of corn oil, peanut oil, and nonfat milk solids with 20% DM. By doing this, the stress could be minimized when the pigs were transferred to nursery room, because piglets had already been weaned from their dam for a certain period and was growing at a same or higher rate as compared to piglets reared with their dam. Lecce et al. (1979) also described different weaning dietary-managements strategies targeting of minimizing weaning stress, improving postweaning pig performance, and determining the optimal age for shifting from a liquid to a dry diet. To further reduce the stress caused by diet shifting, a smooth diet-shifting (gruel or slurry feeding, 2 or 3 times a day for a week's transition) was adopted rather than an abrupt diet-shifting from a liquid to dry diet. Using this kind of weaning management, the results showed that piglets that shifted at 14 d of age had a temporary and slight decline in the rate of weight gain; and piglets that shifted at 30 d of age showed no depression in the growth rate. However, Armstrong and Clawson (1980) observed that cow's milk supplementation 3 times daily for 4 d postweaning (3 to 4 wk of weaning age) did not affect the pig performance. The possible reason for not observing a positive improvement by cow's milk supplementation could be due to no enhancement in total feed

intake. In summary, the basis of postweaning management strategies related to liquid feeding is to encourage pigs to consume feed and obtain sufficient feed intake.

More recent studies about liquid feeding are focused on the shift from sow milk to liquid diet right after weaning. Odle and Harrell (1998) summarized a review about liquid diets in commercial production and showed that piglets fed liquid diets *ad libitum* had 60% greater growth rate as compared with sow-reared piglets. Azain (1997) reported that early-weaned pigs (weaned at 7- to 10-d of age) which were fed liquid diets (milk specialties formulation) for first 7 d postweaning had twice as much dry matter intake as pigs fed dry pelleted diets. In this study (Azain, 1997), liquid diets were transitioned to dry pelleted diets after d 7 postweaning. The results showed that final BW of pigs fed liquid diets at d 14 postweaning was greater than those fed dry pelleted diets. In addition, Zijlstra et al. (1996) indicated a more significant difference in growth performance among pigs either fed a liquid diet (Milk Specialties formulation; 25% protein; 16% solids), reared by sows, or fed dry starter diets from 18-d of age. The results indicated that pigs fed liquid diets had 140% greater dry matter intake, and 283% greater weight gain than pigs fed dry starter diets from 18 to 25-d of age. Furthermore, ADG and villus height of proximal small intestine of pigs fed liquid diets was 64% greater and 74% longer, respectively, than those of pigs reared by sows from 18 to 25-d of age (Zijlstra et al., 1996). Similarly, in research of Kim et al. (2001), liquid milk replacer (15 to 16% DM) was provided by automated milk machines. The results showed that pigs (weaned at 11 d of age) fed liquid milk replacer had 44% greater gain, 18% greater feed intake, and 22% greater gain/feed as compared with pigs fed dry pelleted diets

for 14 d postweaning. Moreover, the advantage in weight gain of pigs fed liquid diets could be maintained to market weight (Kim et al., 2001; Wolter et al., 2002). Price et al. (2013) confirmed that liquid, milk replacer-fed pigs (weaned at 20 d of age) grew faster than dry-fed pigs by 46% when both liquid and dry diets had the identical composition. Collectively, feeding manufactured liquid diets may have reduced negative effects of weaning on growth performance, increased feed intake, and improved intestinal development.

It can be speculated that liquid milk-based ingredients can encourage the feed intake through rising the organoleptic signals (similar smell and taste to sow milk) (Oostindjer et al., 2010). Whereas some previous studies showed that some of the dietary milk-based ingredients could be replaced. Ebert et al. (2005) reported that a mixture of vegetable protein with an appropriate balance of amino acids could replace whey in liquid milk-replacer diets which were fed to neonatal pigs from 2 to 19 d of age, and even resulted in a 20% greater final BW than whey. Similarly, Mahan (1992) investigated the lactose and lactalbumin components of dried whey in dry starter diets and commented that the lactose in dried whey is the primary cause of improved gain and feed intake in response to the dried whey supplementation. However, the effects of diet physical form may confound the animal responses to different feed composition. Oliver et al. (2002) reported that replacing lactose in manufactured liquid diets with hydrolyzed corn syrup solids did not affect feed intake, growth, and intestinal morphology of neonatal pigs (from 1 to 20-d of age). Moreover, Oliver et al. (2005) and Corl et al. (2008) suggested that changing fat concentration from 2 to 29% in liquid diets did not make significant difference in growth performance of pigs.

However, the costs of milk replacer and practical management for liquid-diet feeding may become constraints for the application of liquid feeding. Under the practical condition, pig producers want to maintain the high productivity of pigs while minimizing the use of expensive ingredients. Non-saleable cow milk can be a potential ingredient to be used by nursery pigs. Cow milk usually contains 3.2% CP and 3.3% fat on wet-basis, and 30.0% CP and 27.8% fat on dry matter-basis (USDA, 2016). Meanwhile, commercial milk replacer powders contain about 25% CP and 10 to 15% fat. Therefore, nutrient concentration of raw cow milk is greater than that of some milk replacers on a dry matter basis. Cow milk could be a potential liquid feeding ingredient, if pig producers have access to available milk sources at a relatively cheap price.

Non-saleable milk has been fed to calves for many years and the effects of feeding non-saleable milk on calves' performance and economic feedback have been well documented (Kesler, 1981; Jamaluddin et al., 1996a; Godden et al., 2005). Non-saleable milk is composed of excess colostrum, transition milk, mastitic milk or non-saleable milk containing antibiotics. The intensively reared dairy calves are conventionally fed milk twice a day for a total of approximately 10% of the calf's BW for 37 to 42 d before weaning (Jasper and Weary, 2002). Non-saleable milk is an available feed source because the dairy farms have the inventory of non-saleable milk and the cost of feeding calves this ingredient is relatively low. There is about 22 to 62 kg milk discarded per cow per year (Blosser, 1979), and there are concerns related to economic loss, disposal cost, and environmental issues for directly disposing of the non-saleable milk. Chardavoyne et al. (1979) and Keys et al. (1980)

demonstrated that weight gain of calves fed fermented non-saleable milk or fermented colostrum was not different or greater as compared to those fed normal fresh milk from birth through 5 to 8 wk of age. Godden et al. (2005) reported that calves fed pasteurized non-saleable milk have 0.12 kg greater ADG, 28.81% smaller mortality, and $0.69 less cost per calf per day than those fed milk replacer (20% CP and 20% fat) from d 1 to 49 after parturition. Although there are advantages of feeding non-saleable milk to preweaned calves, there are still some limitations for using non-saleable milk as a feed ingredient, especially when the quality of non-saleable milk is very poor with concerns about microbial safety and total solids content (Moore et al., 2009). Pasteurization of non-saleable milk is a strategy for reducing the bacterial contamination in milk. One study (Elizondo-Salazar et al., 2010) evaluated the calf milk pasteurization on 6 dairy farms and showed that 96% of the milk samples had SPC < 20,000 cfu/mL, and 92% had coliform counts < 100 cfu/mL after pasteurization. Jamaluddin (1995) and Jamaluddin et al. (1996b) indicated that calves that were fed pasteurized non-saleable milk during preweaning period had a reduced morbidity and an increased weight gain compared with those fed non-pasteurized non-saleable milk.

Scope of the Present Dissertation

The productivity of sows nowadays has undergone a rapid change. Modern sows produce a greater number of piglets born alive than before. The managements need keep pace with the production changes. Whereas, the corresponding management strategies of litter vary among countries. For instance, the lactation length in Europe is about 1 to 2 wk longer than that in the US. The difference in the litter management could be due to multiple factors including pig production methods, government regulations, especially in regards to the usage of antibiotics. As compositional factors of suckling intensity, both litter size and lactation length affect sow body weight change and milk production; which subsequently affect the litter performance. Effects of lactation length on sow reproductive performance in a subsequent parity have been well studied. However, no study has been conducted to evaluate the effects of litter size and lactation length on sow lactational performance and litter growth in a subsequent parity. Therefore, the first study (Chapter 2) aims to evaluate the effects of high suckling intensity on sow and litter performance in a subsequent parity. Milk production is affected by the mammary growth and development. Suckling intensity, especially litter size, affects the suckling condition of mammary glands. The suckling condition of mammary glands is related to the mammary growth; suckled mammary glands undergo substantial growth whereas mammary glands that are not suckled undergo a regression. Mammary growth and involution during the lactation and postweaning periods have been well investigated. However, little is known about how the suckling-induced additional mammary growth benefits the mammary gland productivities in the subsequent lactation, especially

when interacting with different mammary location as mammary glands at varied anatomical locations may have different productivities. Therefore, the second study (Chapter 3) aims to evaluate the effects of suckling history and suckling location on mammary gland productivities in a subsequent parity.

Sows, which do not consume sufficient dietary protein and minerals during late gestation and lactation, may be under a catabolic condition and be physiologically challenged. Effects of dietary cation-anion difference have been well studied in dairy cows, especially on reducing the incidence of hypocalcemia. Sows during lactation also have a quick and massive Ca excretion into colostrum and milk. Limited research has been conducted on evaluating the effects of dietary cation-anion difference on Ca metabolism of sows. Therefore, the third study (Chapter 4) aimed to estimate the effects of dietary cation-anion difference on Ca homeostasis of lactating sows.

Sow performance and health as well as nursery pig performance are vital in the entire pig production system. Pigs immediately after weaning face multiple stressors, do not have sufficient feed intake, and subsequently exhibit lack of growth during the early nursery period. Therefore, nursery diets require highly digestible and palatable ingredients, such as milk products. A great mass of non-saleable milk is available in the food retailers and on dairy farms. Feeding the non-saleable milk directly to nursery pigs is worthy of investigating. Moreover, feeding milk to nursery pigs requires liquid milk delivery system and additional labors for sanitization of feeders and replenishment of milk. The economic returns should be evaluated to confirm the benefits to pork producers. The fourth study (Chapter 5) aimed to

evaluate the effects of milk supplementation for 4 d postweaning on growth performance, gut health, and economic returns of nursery pigs.

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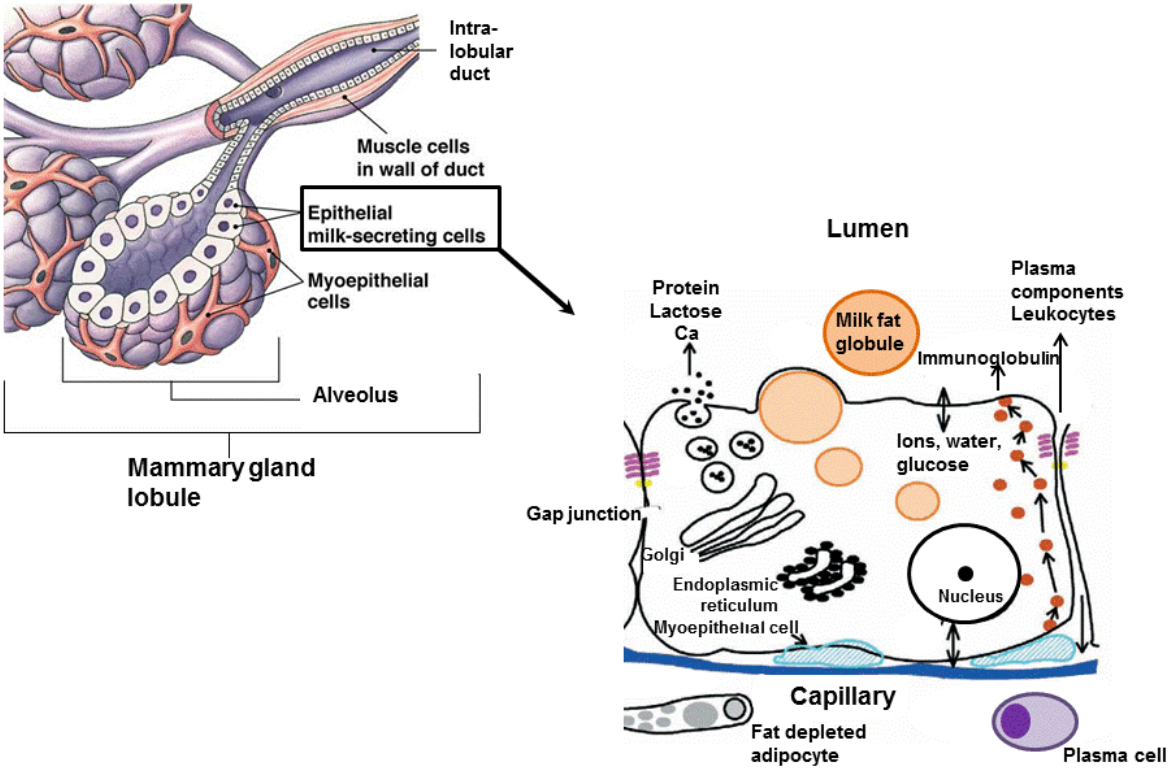

Figure 1. Diagram of mammary alveolus and alveolar epithelial cell for milk secretion (adapted from McManaman and Neville, [2003] and http://www.austincc.edu/apreview/PhysText/Reproductive.html)

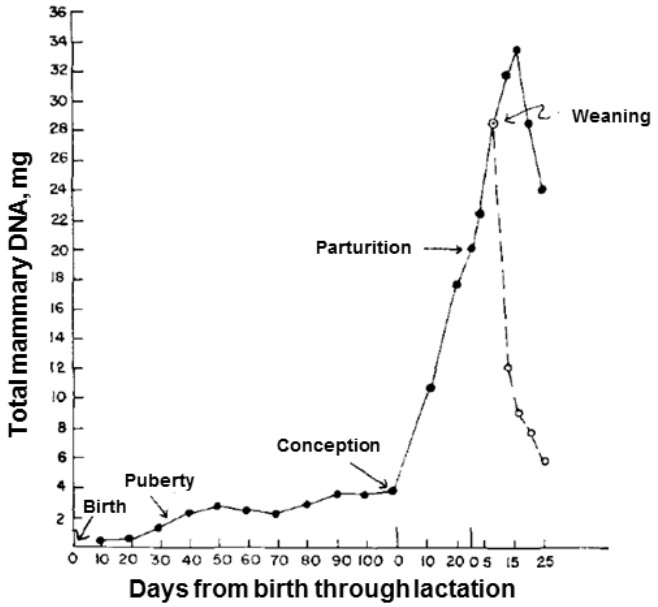

Figure 2. Mammary gland development in rats from birth through lactation (adapted from Tucker, 1969)

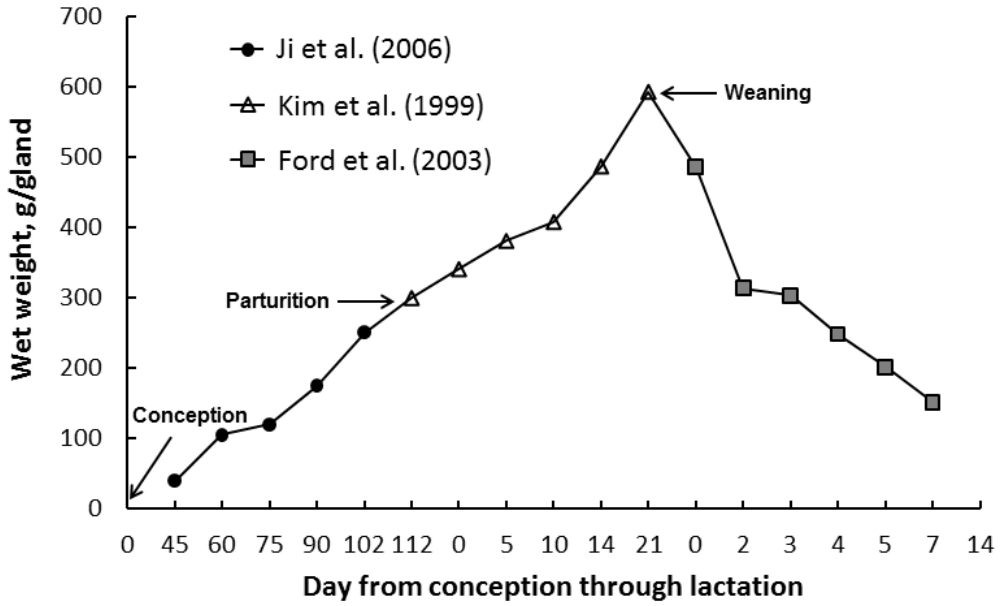

Figure 3. Mammary gland development in sows from birth through lactation (adapted from Kim et al., 1999; Ford et al., 2003; Ji et al., 2006)

CHAPTER 2

EFFECT OF SUCKLING INTENSITY ON SOW PERFORMANCE AND LITTER GROWTH DURING A SUBSEQUENT LACTATION

Abstract: The research was conducted to evaluate the effects of suckling intensity (litter size and lactation length) on sow performance and litter growth in the subsequent lactation. Upon parturition, 115 primiparous sows were initially attributed to 1 of 4 treatments based on a 2 × 2 factorial arrangement with varied litter sizes: 10 and 13 piglets (L10 vs. L13); and varied lactation lengths: 21 and 27 d (D21 vs. D27) in the first parity lactation. In the second parity, sows rebred successfully (n = 66) were continuously used in the experiment. Litter size was set to 10 piglets and lactation length was set to 21 d for all experimental sows in the second lactation. Sows were fed *ad libitum* during both lactations. Feed intake during the lactation, weight loss, backfat loss, and litter weight gain for both lactations were determined. The results showed that litter weight gain in L13 was greater ($P < 0.05$) than that in L10 (54.35 vs. 47.70 kg) in the first lactation. Differences of feed intake of sows among treatments in both lactations were not detected. Sows in L13 tended to have a greater ($P = 0.095$) BW loss than sows in L10 (23.99 vs. 19.70 kg) in the first lactation. Sows in D27 had a smaller ($P < 0.05$) removal rate (ratio of sows culled, aborted, and failed in the first pregnancy check) in parity 2 than those in D21 (28.0 vs. 51.7%). Sows in L13 tended to have a greater ($P = 0.075$) removal rate than those in L10 (47.5 vs. 32.2%). In the second lactation, differences of total number of piglets born and number of piglets born alive among treatments were not detected. Difference of litter weight gain as well as piglet weight gain among treatments were not detected in the second lactation. Sows in D27 had a smaller ($P < 0.05$) BW loss during the second lactation than sows in D21 (3.98 vs. 9.00 kg). Backfat loss in L13 was smaller ($P < 0.05$) than that in L10 during the second lactation (0.5 vs. 2.7 mm). Sows in D27 had a

smaller ($P < 0.05$) concentration of fat in the milk at d 18 of second lactation compared with sows in D21 (7.91 vs. 9.03%). In conclusion, increasing litter size increased the sow removal rate, and increasing duration of lactation decreased the sow removal rate. The sow and litter performance in the second lactation were not negatively affected by increasing the suckling intensity of previous lactation. However, sows with a greater suckling intensity in the first lactation had a smaller amount of body tissues mobilized in the second lactation, implying these sows might maintain a better body condition.

Key words**:** lactation length, litter size, piglet, sow, suckling intensity

Introduction

Milk production is a major limiting factor to support litter growth (Kim and Easter, 2003), and is directly related to sow performance. Suckling intensity is synchronized with capability of mammary gland to produce milk (Marshall et al., 2006). Increasing suckling intensity is expected to increase the milk removal rate, stimulate milk secretion, and subsequently increase litter growth (Hurley, 2001). Suckling intensity comprises several interactions between sows and piglets, including suckling frequency, litter size, lactation length, and size of piglets (King, 2000; Marshall et al., 2006).

Litter size has been improved continuously through genetic selection among these years (Chen, 2003). The number of pigs per litter in United States increased by 2.4 pigs from 1988 to 2012 (NASS, 1994, 2014). Increased number of pigs per litter can affect milk production and mammary growth. Litter size (from 4 to 12 piglets) is linearly related to the milk production (Elsley, 1971; Auldist et al., 1998). Increased litter size (from 6 to 12 piglets) also resulted in an increased postpartum mammary growth, which was indicated by the increased total mass of mammary gland tissue (Kim et al., 1999a). Except for litter size, lactation length is also considered as a factor contributing to suckling intensity. It is a common practice to wean pigs at 21 to 28 d of age when pig weight is larger than 6 kg, particularly in countries other than North American, some farms wean pigs after 21 d (Williams, 2003). Increasing lactation length will enhance milk production and subsequently increase piglet weaning weight, simply because sows can produce more milk during a longer lactation period. Mammary glands continue growing as the lactation processes from d 21 to

28 of lactation (Kim et al., 1999b). In addition, lactation length is also associated with sow reproductive performance, such as weaning-to-estrus intervals, farrowing rates, and subsequent litter sizes (Svajgr et al., 1974; Cole et al., 1975; Xue et al., 1993). However, few studies have examined the effects of increased litter size and lactation length on the productivity and longevity of sows during the subsequent parity.

In addition, primiparous sows have only 75% of the milk production of multiparous sows. The reproduction of primiparous sows is compromised by the active growth and development of body tissues towards maturity, in comparison with that of multiparous sows (Whittemore, 1996). The other concern is that increasing the suckling intensity of primiparous sows may increase the maternal losses and subsequently impair their performance during the later parities. Therefore, this experiment was conducted to evaluate the effects of suckling intensity (litter size and lactation length) on lactational performance of sows and performance of litter in a subsequent lactation.

Materials and Methods

Animals and Experimental Design

A protocol for the use of animals in this study was approved by North Carolina State University Animal Care and Use Committee. The experiment was conducted at a commercial sow farm (Smithfield Foods Inc., Kinston, NC). Gilts were selected for the experiment.

A total of 115 primiparous sows (Smithfield Premium Genetics, Rose Hill, NC, BW of 222.7 ± 20.0 kg) were initially allocated to a 2 × 2 factorial arrangement with varied litter sizes: 10 and 13 piglets (L10 vs. L13); and varied lactation lengths: 21 and 27 d (D21 vs.

D27) in the first lactation. Within 24 h after farrowing, sows were allotted to 2 litter size (the first factor) groups: (1) 61 sows with litters of 9.8 ± 0.5 piglets; (2) 54 sows with litters of 13.4 ± 1.0 piglets (litter size was targeted as 14 piglets but the actual number was rather close to 13 piglets). The litter size was adjusted by cross-fostering using piglets as uniform birth weight as possible (piglets with an average weight among litters). At weaning, sows in either litter size group were further allotted to 1 of 2 sub-groups with lactation length (the second factor) of 21.0 ± 1.4 or 27.0 ± 0.7 d, respectively. The 4 treatments were coded as L10D21, L10D27, L13D21, and L13D27. The number of replications among treatments is different because not all sows successfully farrowed and some sows encountered lactation failure. Numbers of sows or litters during parity 1 were 40, 21, 30, and 24 for L10D21, L10D27, L13D21, and L13D27, respectively. Sows returned to gestation building after weaning and were rebred. At d 42 post breeding, sows were moved from individual stalls (0.61 m × 2.10 m) into the group pens with 6 sows per pen (2.2 m^2 per sow). Pens had quarter stalls and were equipped with a handing nipple. In parity 2, any sows that failed in the first pregnancy test (conception check) were excluded from experiment because the sows allowed for re-breeding after the pregnancy test would have an additional period to grow, which might affect the treatment effects. At d 108 of gestation in parity 2, sows were moved to farrowing crates (1.70 m × 2.30 m) again. Within 24 h after the farrowing, litter size was set to 10.1 ± 1.0 piglets and piglets were weaned at d 20.7 ± 0.6 of lactation in parity 2. After the removal of sows that did not become pregnant or farrow, the number of sows or litters during the second lactation were 20, 18, 14, and 14 for L10D21, L10D27, L13D21, and L13D27,

respectively. All sows were fed a 2-kg common diet daily during the second gestation. Sows were fed *ad libitum* during the entire period of lactation. During both lactations, feed was provided daily at 0630, 1100, and 1630 h. Sows had free access to water.

Sampling

Individual sows and piglets were weighed at farrowing, d 10 of lactation, and weaning in parity 1. At farrowing, d 10 of lactation, and weaning, backfat thickness was measured twice using a digital backfat indicator (Renco lean-meater; Renco Corporation, Minneapolis, MN) with the digital backfat indicator probe placed over the last rib at 6 to 7 cm from dorsal midline; flank-to-flank (in front of hind legs, from bottom of flank on one side over back of sow to the other flank) and heart girth (behind the front legs and in front of the first pairs of mammary glands) were measured with a cloth tape (Cloth Tape Measure, Innovation Frontier Inc., Lakewood, CA). In parity 2, piglet BW, sow BW, and backfat thickness were measured at farrowing and weaning according to the same procedure mentioned previously. In both lactations, total number of piglets born, numbers of piglets born alive, stillborn, and mummified were recorded. In both lactations, feed intake was recorded daily. Colostrum samples (30 mL) were collected within 24 h after parturition in both lactations. Milk samples (30 mL) at 4 d before weaning were collected in both lactations. Colostrum and milk were collected from all functional mammary glands after the injection of 1 mL oxytocin (Oyxtocin 20 IU/mL, Bimeda Inc., Oakbrook Terrace, IL) into ear vein. Colostrum and milk samples were stored at -80ºC for the later analysis of composition including fat, lactose, protein, and solids-not-fat, which were determined by Milkoscan 4000

(Foss Electric, Hillerød, Denmark) and in Virginia Tech United Federation of Dairy Herd Information Association Laboratory (Blacksburg, VA) according to Garst et al. (1999). The instrument for milk composition analysis was regularly calibrated by corresponding chemical analyses, to ensure the concentrations of components of milk within a CV of 3.5% and a standard deviation smaller than 0.04.

Statistical Analysis

Data were analyzed as a completely randomized design. Each individual sow was considered as an experimental unit. Statistical analysis was performed with the MIXED procedure of SAS (SAS Inst. Inc., Cary, NC). Pooled standard error was used in the data set. Data of backfat thickness were analyzed with backfat thickness at farrowing as a covariate at both lactations (Verbeke and Molenberghs, 1997; Littell et al., 2000). Data of piglet weight at weaning, weight gain at weaning, and ADG were analyzed with piglet weight after cross-fostering as a covariate in parity 2. A logistic regression (LOGISTIC procedure of SAS) was used to evaluate the effects of litter size and lactation length on culling rate and sow removal rate with chi-square tests (Peltoniemi et al., 1999; Bates et al., 2003). Mean and SEM of culling rate and sow removal rate were provided by using LSMEANS procedure with ILINK option of SAS. Probability values less than 0.05 were considered statistically significant and between 0.05 and 0.10 as trends.

Results

Performance of Litters in Parity 1

The lactation length in first lactation of D21 and D27 was 21.0 ± 1.4 and 27.0 ± 0.7 ($P < 0.05$), respectively (Table 1). Litter size at the beginning of first lactation of L10 and L13 was 9.8 ± 0.5 and 13.4 ± 1.0 ($P < 0.05$), respectively. There was an interaction ($P < 0.05$) in weaning litter size between litter size and lactation length, showing weaning litter size in L13D27 was greater ($P < 0.05$) than that in L13D21. Sows in L13 had a greater ($P < 0.05$) total number of piglets born than sows in L10, and sows in D27 tended to have a greater ($P = 0.051$) total number of piglets born than sows in D21. An interaction between litter size and lactation length in total number of piglets born tended to exist ($P = 0.062$), indicating sows in L10D21 had a smaller total number of piglets born than sows either in L10D27, L13D21, or L13D27. Sows in L13 had a greater ($P < 0.05$) number of piglets born alive as compared to sows in L10. Sows in D27 tended to have a greater ($P = 0.055$) number of piglets born alive than sows in D21. An interaction in number of piglets born alive between litter size and lactation length tended to exist ($P = 0.075$), specifically showing that the number of piglets born alive in L10D21 was smaller ($P < 0.05$) than that either in L10D27, L13D21, or L13D27. The differences of number of piglets stillborn and mummified were not detected among treatments.

The differences in piglet birth weight among treatments were not detected. At both d 10 of lactation and at weaning, piglet average weight, weight gain, and ADG in L13 were smaller ($P < 0.05$) than those in L10. Average weaning weight, weaning weight gain, and

ADG from birth to weaning in D27 was greater ($P < 0.05$) than those in D21, however, differences in piglet average weight, weight gain, and ADG between D21 and D27 at d 10 of lactation were not detected. There were interaction effects ($P < 0.05$) on average weaning weight, weaning weight gain, and ADG from birth to weaning between litter size and lactation length. Average weaning weight and weaning weight gain of piglets in L13 were smaller ($P < 0.05$) than those in L10 when piglets weaned at 27 d of lactation. Average daily gain from birth to weaning in D27 was greater ($P < 0.05$) than those in D21 when litter size was 10 piglets.

Weight of the litter at birth, at d 10 of lactation, and at weaning in L13 was greater ($P < 0.05$) than those in L10. At both d 10 of lactation and at weaning, litter weight gain in L13 was greater ($P < 0.05$) than in L10. Litter weight and weight gain at d 10 of lactation were not different between D21 and D27; however, litter weight and weight gain at weaning in D27 were greater ($P < 0.05$) than in D21.

Performance of Sows in Parity 1

Weights of sows at farrowing, d 10 of lactation and weaning were not different among treatments (Table 2). Differences in ADFI were not detected among treatments. Body weight loss at d 10 of lactation and weaning in L13 was greater ($P < 0.05$) as compared to those in L10. Differences in body weight loss between D21 and D27 were not detected. Backfat thickness at farrowing was smaller ($P < 0.05$) in L13 than in L10. When assigning backfat thickness at farrowing as a covariate, differences in backfat thickness and backfat loss at both d 10 of lactation and weaning were not detected among treatments. Differences

in flank-to-flank size at farrowing, d 10 of lactation, and weaning were not detected among treatments. Flank-to-flank loss at d 10 of lactation and weaning in L13 was greater ($P < 0.05$) than those in L10. Differences in flank-to-flank loss were not detected between D21 and D27. Differences in heart girth at farrowing and d 10 of lactation were not detected among treatments. Differences in heart girth loss at d 10 of lactation were not detected among treatments. Heart girth at weaning and heart girth loss at weaning in D27 were greater ($P < 0.05$) than those in D21, whereas, difference in heart girth at weaning and heart girth loss at weaning were not detected between L10 and L13.

Culling Rate and Sow Removal Rate

During parity 2, differences in culling rate were not detected among treatments (Table 3). Sows in D27 had a smaller ($P < 0.05$) removal rate in parity 2 than sows in D21. Sows in L13 tended to have a greater ($P = 0.075$) removal rate than sows in L10. Interaction effects of culling rate and removal rate between previous litter size and lactation length were not detected.

Performance of Litters and Sows in Parity 2

Lactation length, litter size at birth, and litter size after cross-fostering in parity 2 were not different among treatments (Table 4). Differences in total number of piglets born, numbers of piglets born alive, stillborn, and mummified in the second lactation among treatments were not detected. In the second lactation, differences in piglet average birth weight among treatments were not detected. Piglet average weight after cross-fostering in D27 tended to be greater ($P = 0.053$) than that in D21 during the second lactation. When

assigning the piglet average weight after cross-fostering as a covariate, differences in piglet weaning weight, weaning weight gain, and ADG at weaning among treatments were not detected. Differences in litter weights at birth, after cross-fostering, and at weaning among treatments were not detected. Differences in litter weight gain among treatments during the second lactation were not detected.

In the second lactation, differences in ADFI among treatments were not detected. In parity 2, differences in weight gain during gestation between L10 and L13 were not detected, whereas, an interaction in weight gain during gestation between previous litter size and lactation length existed ($P < 0.05$), showing that sows in L10D27 tended to have a greater weight gain ($P = 0.074$) during the following gestation than sows in L10D21. Differences in weight of sows at farrowing and at weaning among treatments were not detected. Differences in BW loss between L10 and L13 were not detected, whereas sows in D27 had a smaller BW loss ($P < 0.05$) during the second lactation than sows in D21. An interaction effect on BW loss during the second lactation between the previous litter size and lactation length existed ($P < 0.05$). The BW loss during the second lactation in D27 was smaller ($P < 0.05$) than in D21 when litter size in the previous lactation was 13 piglets. During the second lactation, backfat thickness at farrowing in L13 was smaller ($P < 0.05$) than in L10, whereas differences in backfat thickness at farrowing between D27 and D21 were not detected. When assigning the backfat thickness at farrowing as a covariate, backfat thickness at weaning in L13 was greater ($P < 0.05$) than in L10, and backfat loss at weaning in L13 was smaller ($P <$

0.05) than in L10 during the second lactation. Differences in backfat thickness and backfat loss at weaning between D21 and D27 were not detected.

Composition of Colostrum and Milk in Parity 1 and 2

In the first lactation, differences in colostrum concentrations of fat, protein, and solids-not-fat among treatments were not detected (Table 5). Concentration of lactose in colostrum of sows in D27 was greater ($P < 0.05$) than that in colostrum of sows in D21. The differences in concentration of lactose in colostrum of sows between L13 and L10 were not detected.

In the second lactation, the differences in colostrum concentrations of fat, lactose, protein, and solids-not-fat among treatments were not detected. Sows in D27 had a smaller ($P < 0.05$) concentration of fat in the milk at d 18 of the second lactation than sows in D21. There was an interaction effect ($P < 0.05$) on concentration of fat in milk at d 18 of the second lactation between previous litter size and lactation length. Specifically, concentration of fat in milk of sows in D27 was smaller than in milk of sows in D21 when litter size was 10 piglets in the first lactation. Differences in concentrations of lactose, protein, and solids-non-fat in milk at d 18 of the second lactation among treatments were not detected.

Discussion

Genetic selection and improvement in management have resulted in a gradual increase in total number of piglets born throughout these years. A sow currently gives birth to 10 to 16 piglets per litter and 25 to 30 pigs per year (Kim et al., 2013). More piglets per litter requires greater milk production. Litter size at the beginning of lactation basically determines

the number of functional mammary glands throughout the lactation period. Accordingly, there is a strong positive linear relationship between milk production and litter size (Elsley, 1971; King et al., 1989), although individual growth of piglets decreases in response to increased litter size (Kim et al., 1999a). This study showed the consistent results that litter weight gain increased, whereas individual piglet weight gain decreased with increased litter size. However, increased numbers of functional mammary glands with increased litter size can compensate for the decrease in milk intake of individual piglets (Auldist et al., 1998; King, 2000). Current results showed that increasing litter size from 10 to 13 piglets increased litter weight gain by 19% from d 1 to 10 of lactation and by 12% from d 10 of lactation to weaning, whereas, increasing litter size from 10 to 13 piglets decreased individual piglet weight gain by 9% from d 1 to 10 of lactation and by 24% from d 10 to weaning. The increase in litter weight gain can compensate for the loss in individual piglet weight gain in response to the increased litter size at early lactation (19% in litter increase as compared to 9% in individual decrease) or throughout the lactation (13.9% in litter increase as compared to 13.6% in individual decrease), but not at late lactation (12% litter increase as compared to 24% individual decrease). Therefore, sows seem to have the capability to produce a greater amount of milk during early lactation in response to the increase in litter size (King, 2000).

Correspondingly, increased milk production is associated with increased sow BW loss. Litter size is positively correlated with weight loss of sows during lactation (Rydhmer et al., 1992). This study showed that sows lost 6.6 kg additional weight (3.0% BW) when litter size increased from 10 to 13 piglets. This is simply because sows with a larger litter size

produce a greater amount of milk, which requires more body reserve mobilization to provide the nutrients and energy. Other studies showed that weight loss larger than 5% of BW in parity 1 significantly affects subsequent reproductive performance (Thaker and Bilkei, 2005). In this study, total number of piglets born in parity 2 were not different between sows in 2 litter size groups in this study. A possible reason is that the difference in weight loss induced by altering litter size was not large enough to rise big changes in subsequent reproductive performance.

Increasing lactation length from 21 to 27 d increased the lactating time. Subsequently, either litter weight gain or individual piglet weight gain was increased by their increased milk intake. However, increasing lactation length did not statistically increase weight loss and backfat loss, but did increase the heart girth loss during the first lactation, which partially agrees on what Willis et al. (2003) found that sows weaned at d 24 of lactation had a greater backfat loss and a poorer body condition at weaning than sows early weaned at d 14 of lactation. Sows might get recovered from the lactation BW loss at late lactation. Tantasuparuk et al. (2001) indicated that weight loss during a lactation decreased by 0.6% per day as the lactation length increased from 17 to 34 d. Moreover, an earlier study from Rojkittikhun et al. (1993) showed that primiparous sows could even gain weight from d 28 to 35 of lactation. Collectively, the rate of sow BW loss seems slow down along with lactation period. Increasing lactation length may not directly result in a thinner body condition at weaning.

On the other hand, lots of studies have proven that lactation length is associated with subsequent reproductive performance of sows. Longer lactation length may provide more time for uterine recovery and restoration of the uterine endometrium. Svajgr et al. (1974) indicated that uterine involution was not complete until 30 to 34 d of lactation and incomplete uterine involution may impair subsequent fertility. Therefore, increasing lactation length from 14 to 34 d resulted in a decreased weaning-to-service interval (Xue et al., 1993), an increased farrowing rate (Hays et al., 1978), an increased conception rate (Cole et al., 1975), and subsequently a larger litter size. Surprisingly, this study showed no major improvements in total number of piglets born and number of piglets born alive in the second lactation when the previous lactation length increased from 21 to 27 d, which was consistent with the results from Willis et al. (2003). The possible reason could be due to the management intervention. The other possible reason could be that sows in D21 with lower reproductive performance or in a poorer health condition were removed from experiment, because there was a greater removal rate in D21 in comparison with that in D27. In addition, the subsequent litter size is associated with the weight loss during a previous lactation (Eissen et al., 2003; Thaker and Bilkei, 2005). Weight loss during the first lactation did not differ between sows in 2 lactation length groups in this study, which agrees to the results that subsequent total number of piglets born was also not different between sows in 2 lactation length groups.

Sows with a larger litter size had a greater body weight loss and flank-to-flank loss in the first lactation, whereas farrowing weight in the second lactation was not different

between sows in 2 litter size groups. This is mainly because the weaning weight at the first lactation was not different among treatments. On the other hand, sows with a larger previous litter size had less backfat loss during the second lactation. This is because at both lactations the farrowing backfat thickness in the larger litter size group was lower than in the small litter size groups. Doubtlessly, sows in a thinner body condition have less body reserves to mobilize. As reported by Revell et al. (1998a), leaner sows lost less body fat during the lactation than fatter ones. At the same time, sows with a longer lactation length during the first lactation had less body weight loss during the second lactation than sows that had a shorter lactation length. This could be related to the lighter farrowing BW of sows in D27 during parity 2. When all sows had a litter size of 13 piglets, farrowing weight of sows with a longer first lactation length was 15 kg smaller (not significant) during the second lactation than sows with a shorter first lactation length. The relative lighter farrowing weight is possibly associated with smaller body weight loss during the second lactation. As reported by Mullan and Williams (1989), lighter sows mobilized less body reserves.

However, if we combined these results from both lactations, we can summarize it as follows: 1) sows with a greater suckling intensity mobilized or lost more body reserve during the first lactation; and 2) sows with a greater suckling intensity during the first lactation had less body tissues mobilized (especially for adipose tissue) during the second lactation, as compared with sows subjected to a smaller suckling intensity. Nevertheless, the litter weight gain during the second lactation did not decrease as expected when body tissue mobilization decreased with increased nursery intensity. The responses in litter weight gain reflected the

milk production, whose nutrients are originally from dietary feed intake (exogenous) and body reserves of sows (endogenous) (Revell et al., 1998b). Therefore, our results demonstrated that sows with a greater previous suckling intensity had less body tissue mobilization during the subsequent lactation, but were still capable of maintaining an adequate milk production during the subsequent lactation. One possible reason could be that sows subjected to a greater previous suckling intensity might undergo a subsequent compensatory growth, which is characterized by an accelerated anabolism, a reduced maintenance requirement, or an enhanced efficiency to utilize energy and protein (Wilson and Osbourn, 1960; Hornick et al., 2000). Regardless of feed intake in the second lactation was not different among treatments. Compensatory growth can be induced by a greater previous suckling intensity because both nutrient restrictions, a common cause of compensation growth, and greater suckling intensity during lactation result in a negative nutrient balance. Early studies (MacPherson et al., 1969; O'Grady, 1971; Reese et al., 1982) also demonstrated that primiparous sows having a lower energy or protein intake during lactation could compensate for the greater weight loss during the following gestation. Consistently, current results showed that sows with a longer lactation length in the first lactation had an 8.5-kg greater weight gain during the following gestation than sows with a shorter lactation length, regardless of all sows had the same feed intake during gestation. Moreover, the milk composition, except the concentration of fat, was not affected by increasing nursing intensity of a previous lactation. The reduced concentration of milk fat was consistent with the result of decreased backfat loss. Reduced adipose tissue mobilization

suggested that the available substrates and energy for fatty acid synthesis in mammary glands decreased.

Increasing previous suckling intensity did not negatively affect the litter performance during the subsequent lactation. This result can also be partially explained by the growth and development of mammary glands. Based on known physiologic knowledge of sows, the mammary growth is induced by suckling of piglets (Hurley et al., 2003). Sows with a larger litter size have a greater number of functional mammary glands, subsequently a greater growth in total mass of mammary gland tissues, as compared with sows with a smaller litter size (Kim et al., 1999a). On the other hand, mammary glands may have more development and growth during a longer lactation length simply because sows that are weaned late receive stimulation on mammary glands for a longer period. In the current study, the lactation length was either 21 to 27 d. Kim et al. (1999b) also showed that the amount of protein and dry fat-free tissue in mammary glands continued to increase as the lactation processed from d 21 to 28 of lactation. Therefore, considering the size of mammary glands and number of mammary gland cells is directly associated with milk yield (Tucker, 1966; Knight and Peaker, 1984), it can be speculated that increasing suckling intensity of the first lactation may contribute to a greater total mass of mammary tissues and a greater milk production during the subsequent lactation. Interestingly, an early study from Fraser et al. (1992) demonstrated that suckling mammary glands during the first lactation only resulted in increased productivities during the early stage of the subsequent lactation. However, our study showed that litter weight gain during the second lactation was not statistically different among sows with varied previous

litter sizes and lactation lengths. Apparently, sows with a higher nursing intensity may have greater mammary tissues during the second lactation, however, the potential for greater milk yield could be prevented by limited nutrient intake and body reserve mobilization during the second lactation.

In conclusion, increasing litter size tended to increase the sow removal rate but increasing duration of lactation reduced the sow removal rate. the performance of sows and litters during the second lactation were not negatively affected by increasing suckling intensity of a previous lactation. Interestingly, there was a smaller amount of body tissues mobilized during the second lactation for sows that had a greater suckling intensity during the first lactation, implying these sows maintained better body condition.

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Table 1. Performance of litters in parity 1

	L10		L13			*P*-value[1]		
Item	D21	D27	D21	D27	SEM	L	D	LxD
n, sow	40	21	30	24				
Lactation length, d	21.3	26.6	20.7	26.7	0.3	0.276	<.001	0.195
Litter size at birth, piglet	9.8	10.0	13.3	13.5	0.2	<.001	0.148	0.824
Litter size at weaning, piglet[2]	9.4[c]	8.9[c]	11.8[b]	12.5[a]	0.2	<.001	0.783	0.005
Total born, piglet[2]	11.8[c]	14.1[a]	14.7[a]	14.8[a]	0.5	0.002	0.051	0.062
Born alive, piglet[2]	11.0[b]	13.1[a]	14.0[a]	14.1[a]	0.7	0.001	0.055	0.075
Stillborn, piglet	0.9	1.0	0.7	0.7	0.2	0.265	0.859	0.728
Mummified, piglet	0.7	0.4	0.2	0.2	0.3	0.142	0.610	0.485
Piglet weight								
At birth, kg[3]	1.57	1.53	1.50	1.52	0.04	0.264	0.810	0.455
At d 10, kg[3]	3.50	3.31	3.16	3.22	0.11	0.034	0.532	0.196
At weaning, kg[2,3]	5.98[c]	7.86[a]	5.59[c]	6.71[b]	0.15	<.001	<.001	0.004
Weight gain until d 10, kg	1.93	1.77	1.66	1.70	0.10	0.054	0.546	0.254
Weight gain at weaning, kg[2]	4.41[c]	6.33[a]	4.09[d]	5.19[b]	0.14	<.001	<.001	0.001
ADG from birth to d 10, kg	0.194	0.189	0.178	0.165	0.007	0.013	0.265	0.635
ADG from birth to weaning, kg[2]	0.208[b]	0.238[a]	0.196[bc]	0.195[c]	0.005	<.001	0.006	0.001
Litter weight								
At birth, kg	15.34	15.24	19.95	20.59	0.54	<.001	0.569	0.434
At d 10, kg[2]	33.49[b]	31.04[b]	39.44[a]	41.64[a]	1.26	<.001	0.910	0.038
At weaning, kg	56.36	69.61	65.90	83.33	2.07	<.001	<.001	0.252
Weight gain at d 10, kg[2]	18.15[bc]	15.80[c]	19.49[ab]	21.05[a]	1.13	0.001	0.692	0.051
Weight gain at weaning, kg	41.02	54.37	45.95	62.75	1.95	0.001	<.001	0.319

[1] L stands for litter size; D stands for lactation length.

[2] Within a row, means lacking a common superscript differ ($P < 0.05$).

[3] Average weight of piglet per litter

Table 2. Performance of sows in parity 1

	L10		L14			*P*-value[1]		
Item	D21	D27	D21	D27	SEM	L	D	L x D
n, sow	40	21	30	24				
ADFI, kg/d	4.3	4.5	4.4	4.6	0.1	0.508	0.106	0.728
Sow weight								
At farrowing, kg[2]	220.5	223.7	226.8	219.9	3.7	0.757	0.634	0.202
At d 10, kg	215.9	217.6	218.7	207.5	3.8	0.376	0.251	0.121
At weaning, kg	203.9	205.6	202.4	196.3	4.2	0.238	0.627	0.398
Weight loss at d 10, kg	-4.6	-6.1	-8.1	-12.3	1.6	0.007	0.109	0.445
Weight loss at weaning, kg	-16.6	-18.2	-24.5	-23.6	2.0	0.003	0.879	0.583
Backfat thickness[3]								
At farrowing, mm	28.8	30.0	26.1	23.8	1.9	0.005	0.739	0.265
At d 10, mm	22.5	20.9	21.4	23.3	0.9	0.546	0.867	0.102
At weaning, mm	20.5	18.5	19.0	19.7	1.1	0.912	0.494	0.182
Backfat loss at d 10, mm[4]	-4.7	-6.3	-5.8	-3.9	1.1	0.546	0.867	0.102
Backfat loss, mm[5]	-6.7	-8.7	-8.1	-7.5	1.1	0.912	0.494	0.182
Flank-to-flank								
At farrowing, cm	109.7	109.1	110.1	109.7	1.1	0.572	0.611	0.912
At d 10, cm	110.0	109.5	108.9	107.8	1.1	0.158	0.428	0.722
At weaning, cm	108.1	106.5	106.6	104.5	1.3	0.137	0.117	0.804
Loss at d 10, cm[4]	0.3	0.5	-1.2	-2.0	0.8	0.024	0.729	0.591
Flank-to-flank loss, cm[5]	-1.7	-2.6	-3.6	-5.2	1.1	0.025	0.199	0.693
Heart girth								
At farrowing, cm	111.8	113.0	111.8	112.3	1.4	0.735	0.489	0.774
At d 10, cm	112.1	112.6	111.3	109.5	1.4	0.115	0.623	0.337
At weaning, cm	108.3	106.5	108.0	105.5	1.3	0.550	0.059	0.757
Loss at d 10, cm[4]	0.2	-0.4	-0.5	-2.7	1.4	0.217	0.231	0.504
Heart girth loss, cm[5]	-3.5	-6.5	-3.8	-6.8	1.5	0.845	0.023	0.996

[1] L stands for litter size; D stands for lactation length.

Table 2. Continued

[2] Sows were weighed within 24 h after farrowing.

[3] The sample size in backfat measurement was 23, 20, 16, and 11 for treatment L10D21, L10D27, L14D21, and L14D27, respectively. Data of backfat thickness at d 10 of lactation, at weaning, backfat loss at d 10 of lactation, and backfat loss at weaning were analyzed with backfat at farrowing as a covariate.

[4] Backfat loss at d 10 = backfat thickness at d 10 of lactation – backfat thickness at farrowing. Flank loss and heart girth loss calculations at d 10 of lactation and weaning were similar to backfat loss calculation.

[5] Backfat loss = backfat thickness at weaning – backfat thickness at farrowing; Flank-to-flank loss and heart girth loss calculations were similar to backfat loss calculation.

Table 3. Logistic regression of litter size and lactation length on culling rate and sow removal rate in parity 2

	L10		L14			*P*-value > Chi-square[1]		
Item	D21	D27	D21	D27	SEM	L	D	L x D
Culling rate, %[2]	27.5	14.3	30.0	25.0	7.6	0.257	0.389	0.546
Sow removal rate, %[3]	50.0	14.3	53.3	41.7	7.9	0.075	0.011	0.138

[1] L stands for litter size; D stands for lactation length. Different from the F-test, the null hypothesis was tested in a Chi-square distribution.

[2] Data of culling rate were cataloged as either cull or not cull for each sow. Data of culling rate were analyzed by using the LOGISTIC procedure (SAS Inst. Inc., Cary, NC). Probability modeled was cull = 'YES'. Causing of culling includes death, lameness, low productivity, failing conception and so on.

[3] Causing of sows removal from experiment includes culling, abortion, and failing in first pregnancy check because the sows allowed for re-breeding after the pregnancy test would have additional days to recover and grow from their parity 1, which would affect the treatment effects (suckling intensity).

Table 4. Performance of sows and litters in parity 2

	L10		L14			*P*-value[1]		
Item	D21	D27	D21	D27	SEM	L	D	L x D
n, sow[2]	20	18	14	14				
Lactation length, d	20.6	20.8	20.6	20.6	0.2	0.311	0.415	0.415
Litter size at birth, piglet	13.6	12.1	13.8	13.1	1.0	0.523	0.222	0.666
Litter size after CF, piglet[3]	10.1	10.3	10.4	10.3	0.2	0.489	0.879	0.507
Litter size at weaning, piglet	10.1	10.1	10.2	10.2	0.2	0.619	0.909	0.909
Total born, piglet	13.2	13.6	13.5	13.4	0.7	0.893	0.841	0.774
Born alive, piglet	12.5	12.9	13.0	13.0	0.8	0.668	0.776	0.776
Stillborn, piglet	0.7	0.7	0.5	0.4	0.2	0.317	0.810	0.930
Mummified, piglet	0.2	0.0	0.1	0.0	0.1	0.526	0.183	0.526
Piglet weight[4,5]								
Weight at birth, kg	1.50	1.58	1.53	1.58	0.06	0.798	0.198	0.717
Weight after CF, kg	1.55	1.63	1.53	1.64	0.05	0.840	0.053	0.734
Weight at weaning, kg	6.80	7.18	6.95	6.81	0.15	0.478	0.466	0.108
ADG, kg	0.253	0.269	0.261	0.254	0.007	0.671	0.566	0.155
Litter weight								
Weight at birth, kg	20.06	18.92	20.39	20.05	1.14	0.496	0.487	0.710
Weight after CF, kg	15.58	16.38	15.57	16.76	0.66	0.761	0.111	0.755
Weight at weaning, kg	67.41	72.91	69.52	70.92	2.36	0.977	0.122	0.356
Weight gain, kg	51.83	56.52	53.95	54.16	1.93	0.945	0.178	0.219
Sow lactation ADFI, kg/d[6]	7.1	7.4	7.1	7.1	0.2	0.452	0.567	0.607
Sow weight								
Gestation weight gain, kg[7]	34.6^{B}	55.6^{A}	50.2^{AB}	37.7^{AB}	7.1	0.877	0.581	0.033
At farrowing, kg	235.0	249.9	250.4	235.1	7.8	0.970	0.963	0.033
At weaning, kg	227.8	242.6	239.7	234.3	7.4	0.782	0.474	0.127
Weight loss, kg[8,9]	-7.3^{b}	-7.3^{b}	-10.7^{b}	-0.7^{a}	2.7	0.512	0.038	0.038
Backfat thickness[10]								
At farrowing, mm	19.8	20.6	18.2	17.3	1.2	0.028	0.957	0.429

Table 4. Continued

At weaning, mm	16.3	17.2	18.1	18.6	0.5	0.010	0.238	0.740
Backfat loss, mm	-2.8	-2.1	-1.1	-0.6	0.5	0.010	0.238	0.740

[1] L stands for litter size; D stands for lactation length.
[2] Number of corresponding sows was used in analysis. Some sows were removed from experiment for reasons including abortion, pregnancy failure, lameness, vulva discharge, not conceive, and death.
[3] CF is cross-fostering.
[4] Average weight of piglet per litter
[5] Data of piglet weight at weaning, weight gain at weaning, and ADG were analyzed with the average piglet weight after cross-fostering as a covariate.
[6] Sample size in ADFI of sow was 20, 16, 12, and 13 for L10D21, L10D27, L14D21, and L14D27, respectively.
[7] Gestation weight gain = weight at farrowing in the second lactation – weight at weaning in the first lactation. Within a row, means lacking a common uppercase superscript tend to differ ($0.05 \leq P < 0.10$).
[8] Sample size for sow weight loss was 18, 16, 11, and 13 sows for L10D21, L10D27, L14D21, and L14D27, respectively; sow weight loss = weight at weaning – weight at farrowing.
[9] Within a row, means lacking a common lowercase superscript differ ($P < 0.05$).
[10] Backfat thickness at weaning and backfat loss was analyzed with backfat thickness at farrowing as a covariate. Backfat loss = backfat thickness at weaning – backfat at farrowing.

Table 5. Composition of colostrum and milk in parity 1 and 2 (wet basis)

	L10		L14			*P*-value[1]		
Item	D21	D27	D21	D27	SEM	L	D	L x D
n, sow in parity 1	10	18	15	9				
Colostrum, %								
Fat	10.34	7.54	8.45	9.05	1.20	0.860	0.327	0.133
Protein	9.14	6.16	7.49	8.93	1.13	0.595	0.465	0.041
Solids-not-fat	14.10	13.08	12.62	13.93	1.00	0.733	0.876	0.217
Lactose	3.22	4.23	3.23	3.37	0.29	0.112	0.036	0.104
n, sow in parity 2	20	18	14	14				
Colostrum, %								
Fat	7.92	7.47	7.14	7.95	0.70	0.817	0.788	0.335
Protein	9.86	10.52	10.35	9.69	0.75	0.805	0.997	0.347
Solids-not-fat	14.40	14.84	14.89	14.37	0.62	0.978	0.950	0.414
Lactose	2.93	2.76	2.97	3.08	0.14	0.171	0.790	0.278
Milk, d 18 of lactation, %								
Fat[2]	8.35[b]	8.38[b]	9.71[a]	7.44[b]	0.44	0.609	0.009	0.007
Protein	4.50	4.49	4.68	4.38	0.11	0.815	0.205	0.234
Solids-non-fat	11.64	11.68	11.60	11.52	0.15	0.497	0.907	0.698
Lactose	5.21	5.25	5.00	5.21	0.13	0.299	0.268	0.478

[1] L stands for litter size; D stands for lactation length.

[2] Within a row, means lacking a common superscript differ ($P < 0.05$).

CHAPTER 3

IMPACTS OF SUCKLING LOCATION AND SUCKLING HISTORY ON MAMMARY GLAND PRODUCTIVITY IN TWO CONSECUTIVE PARITIES

Abstract: The research was conducted to evaluate the effects of suckling location and suckling history (mammary glands not suckled or suckled in the previous lactation) of mammary glands on piglet growth during the lactations in 2 parities. In the first experiment, litters of 27 primiparous sows (farrowing BW of 224.2 ± 19.1 kg) were used with litter size of 10.5 ± 1.7 piglets. All sows were rebred in the second parity, with a litter size of 9.9 ± 1.4 piglets in the second lactation. Mammary glands of all sows were allotted into the 3 suckling locations anatomically: anterior location (the first and second pairs of glands), middle location (the third, fourth, and fifth pairs of glands), and posterior location (the sixth, seventh, and eighth pairs of glands). The lactation length in parity 1 and 2 were 22 and 21 d, respectively. In the second experiment, the same 27 primiparous sows were used again. The suckling condition of glands (not suckled or suckled) in the first lactation was considered as the suckling history of glands in the second lactation. In both experiments, individual piglets were weighed at birth and weaning during the lactation. Teat order was observed 3 times from wk 2 to 3 of lactation in both parities. Effects of suckling history on piglet performance during the second lactation were evaluated at each suckling location. Sows were fed *ad libitum* during both lactations. In the first experiment, the results showed that birth weight and weaning weight at varied suckling locations were not different in the first lactation. Average daily gain of piglets which suckled the first 5 pairs of anterior and middle glands was greater ($P < 0.05$) than those which suckled the last 3 pairs of posterior glands in the first lactation (217 vs. 204 g). In the second lactation, birth weight of piglets at varied suckling locations was not different. Average daily gain and weaning weight of piglets that suckled

anterior glands were greater ($P < 0.05$) than those that suckled middle glands (268 vs. 254 g). Average daily gain and weaning weight of piglets that suckled middle glands were greater ($P < 0.05$) than those that suckled posterior glands (254 vs. 220 g). During both lactations, gland usage at the anterior location was greater ($P < 0.05$) than gland usage at the middle location by 12 to 15%, which was greater ($P < 0.05$) than that at the posterior location by 43 to 49%. In the second experiment, suckling history of mammary glands did not affect birth weight, ADG and weaning weight of piglets suckling these glands at either suckling location during the second lactation. Collectively, anterior and middle glands had a greater productivity than other glands during the first lactation. Anterior glands were superior to other glands during the second lactation regarding piglet weight gain. When effects of suckling location were excluded, productivity of a mammary gland during the second lactation was not affected by suckling history (not suckled or suckled) of the first lactation.

Key words: suckling location, suckling history, mammary gland, piglet growth

Introduction

Weaning weight of piglets can affect the management of nursery farms, phase changes, and efficiency of swine production. Weaning weight of piglets within a litter varies in relationship to factors including birth weight and milk intake. Variation of milk production is associated with anatomical locations of mammary glands (Kim et al., 2000). The suckling location of piglets, which is also referred to as the "teat order", was occupied consistently by piglets. Within the first few hours after birth, the teat order is quickly established after birth fighting (MacBride, 1963). Previous studies have showed that piglets that suckled the anterior and middle glands had a greater weight gain than those which suckled posterior glands (Fraster, 1984; Kim et al., 2000). The possible reasons could be that piglets that dominate the anterior or middle glands are born heavier (English et al., 1977; Hartsock and Gravers, 1976) or have a higher milk intake (Fraser and Thompson. 1986; Pluske and Dong, 1998, Skok et al., 2007). Meanwhile, heavier pigs can stimulate a greater milk flow and increase the rate of milk removal (King, 2000; Hurley 2001), and thus increase the milk production of anterior or middle glands during the entire lactation period.

Suckling condition such as not suckled or suckled (involving in suckling length, suckling frequency, litter size, and size of piglets) is associated with mammary growth and development. Glands that are not suckled undergo a process of involution with loss of lactating epithelial cell (Kim et al., 2001; Ford et al., 2003). Glands that are suckled during lactation undergo extensive growth, which was about 100% increase in total gland DNA (Kim et al., 1999a). Even the glands that are suckled will regress after weaning. Ford et al.

(2003) proved that glands that were suckled during lactation contained over twice the amount of DNA than glands that were not suckled at d 7 postweaning. It is still questioned whether the greater tissue retention can contribute to a greater productivity of mammary glands in the subsequent lactation. A recent study in 2 consecutive parities (Farmer et al., 2012) showed that glands suckled in the first lactation contained more parenchymal tissues, more DNA, and more RNA in the second lactation, as compared to mammary glands that are not suckled, however, the weaning weight of piglets that suckled these 2 categories of glands do not differ by the end of the second lactation. Suckling of a gland tends to increase its productivity during the subsequent lactation, whereas the weight gain of piglets is also influenced by the suckling locations of glands (Fraser et al., 1992). However, it is unclear whether suckling condition of mammary glands (not suckled or suckled) affects their productivity during the subsequent lactation without the interference from the suckling location. Therefore, the objective of this study was to characterize the effects of suckling location and suckling history on mammary gland productivity in two consecutive parities.

Materials and Methods

Animals and Experimental Design

The experimental protocol was approved by the North Carolina State University Animal Care and Use Committee. Both experiments were conducted at a commercial sow farm (Smithfield Foods Inc., Kinston, NC).

In the first experiment (Exp. 1), litters of 27 primiparous sows (initial BW at farrowing was 224.2 ± 19.1 kg, Smithfield Premium Genetics, Rose Hill, NC; summer of

2012) were used. During the first lactation, mammary glands of all sows were allotted into the 3 suckling locations anatomically: (1) anterior location (first and second pairs of anterior glands); (2) middle location (the third, fourth, and fifth pairs of middle glands); (3) and posterior location (the sixth, seventh, and eighth posterior glands). Mammary glands were freely picked up by piglets without interference. The first pair of mammary glands was close to the head of sows and far away from the uterus. Sows were fed 2 kg common diet during gestation and allowed *ad libitum* access to lactational common diet during lactation (22 d). Litter size was 10.5 ± 1.7 piglets during lactation. After weaning, all sows returned to gestation building, and they were artificially inseminated after the onset of estrus. At d 42 post-breeding, sows were moved from individual stalls (0.61 m × 2.10 m) into the group pens with 6 sows per pen (2.2 m^2 per sow). Pens had quarter stalls and were equipped with a hanging water nipples. All sows were moved back to farrowing crates (1.70 m x 2.30 m) 1 wk before farrowing. During the second lactation (winter of 2012), mammary glands were assigned into the 3 suckling locations exactly as what was done during the first lactation. Sows were fed 2 kg common diet during gestation and allowed *ad libitum* access to a common lactational diet during the second lactation (21 d). Piglets were cross-fostered to 9.9 ± 1.4 piglets within 48 h after farrowing. Litter management during both lactations including clipping teeth, iron administration, tail docking, treating splay-legged piglets, and castration was conducted within the first 3 d of lactation.

During both lactations, piglets were weighed within 12 h after birth and at weaning. Birth and weaning weights of piglets were also categorized according to the suckling

location. The percentage of mammary gland usage was determined. The percentage of mammary gland usage was calculated as number of suckled (functional) glands divided by total numbers of glands within a certain suckling location. For example, if both 2 glands of the first pair were suckled, then the percentage of mammary gland usage in the first pair of glands was 100%. Another example was: if only 1 gland was suckled at the first 2 pairs of anterior glands, then the percentage of mammary gland usage at the anterior location was 25% (1 suckled gland divided by 4 glands within 2 pairs) for sow A.

In the second experiment (Exp. 2**)**, the same 27 primiparous sows were used again. Mammary glands were freely picked up by piglets without management interference. Glands of all sows in the first lactation were allotted to 1 of 2 suckling conditions: not suckled or suckled. The suckling condition of glands in the first lactation was considered as the suckling history of glands in the second lactation (Fig. 1 as an example). During the second lactation, piglets were weighed at birth and weaning (d 21 of age). Effects of suckling history on birth weight, weaning weight, and ADG of piglets during the second lactation were analyzed at either suckling location (anterior, middle, or posterior location).

Teat Order

For both Exp. 1 and Exp. 2, teat order was observed during wk 2 and 3 of lactation in both parities. The observations were repeated at least 3 consecutive times until piglets and corresponding glands were accurately specified. The observations were done when a stable teat order was established or when the ownership of mammary glands was clearly identified. Some piglets might suckle both teats within one pair of mammary glands. But most of the

piglets could be paired with the specific mammary glands. Data of birth and weaning weights with missing corresponding teat order were removed.

Statistical Analysis

In both experiments, the individual piglet was considered as the experimental unit. Data were analyzed as a completely randomized design by using MIXED procedure of SAS (SAS Inst. Inc., Cary, NC). In Exp. 1**,** the suckling locations of glands (by pair or by sucking location) were regarded as the fixed effect; each sow was regarded as the random effect. Data from parity 1 and parity 2 were analyzed separately. In Exp. 2, the suckling history (suckling condition of parity 1) was regarded as the fixed effect; each sow was regarded as the random effect. In Exp. 2, the fixed effect was evaluated at each suckling location (anterior, middle, or posterior location), separately. Least-squares mean and standard error of the mean were obtained by using LSMEANS procedure with PDIFF option of SAS. Probability values less than 0.05 were considered statistically significant and between 0.05 and 0.10 as trends.

Results

In Exp. 1, average daily gain of piglets at anterior or middle location was greater ($P < 0.05$) than these at posterior location during the first lactation (Table 1). Birth weight and weaning weight of piglets at the varied suckling locations were not different. Individually, average daily gain of piglets that suckled the third pair of glands was greater ($P < 0.05$) than ADG of those that suckled the sixth pair of glands; other than that, piglets that suckled either pair of glands were not different from others in weight gain during the first lactation.

During the second lactation, birth weight of piglets was not different at varied suckling locations. Piglets that suckled anterior glands had greater ($P < 0.05$) ADG and weaning weight than those suckled middle glands, which were greater ($P < 0.05$) than those suckled posterior glands. Individually, piglets that suckled either the first, second, third, or fifth pair of glands had greater ($P < 0.05$) ADG and weaning weight than those suckled either the fourth, sixth, or seventh pair of glands. piglets that suckled the fourth pair of glands had greater ($P < 0.05$) ADG and weaning weight than those suckled the sixth pair of glands. Piglets that suckled the eighth pair of glands had a smaller ($P < 0.05$) weaning weight than those suckled the first pair of glands. Average daily gain and weaning weight of piglets that suckled either 2 pairs among the sixth, seventh, or eighth pair of glands were not different.

During both lactations, the gland usage at the anterior location was greater ($P < 0.05$) than that at middle location, which was greater ($P < 0.05$) than that at posterior location. Individually, the gland usage at either pair among first to fourth pairs location was greater ($P < 0.05$) than those at the fifth to eighth pairs of location. The gland usage at the fifth pair location was greater ($P < 0.05$) than those at the sixth and seventh pairs location, which was further greater ($P < 0.05$) than that at the eighth pair location.

In Exp. 2, at either anterior, middle or posterior location, there was no difference in birth weight, ADG, and weaning weight during the second lactation, between piglets suckling glands with suckling history of not suckled or suckled (Table 2).

Discussion

In general, piglets that suckled the first 5 pairs of anterior and middle glands grew faster than piglets that suckled the last 3 pairs of posterior glands during the first lactation, whereas the birth weight was not different among piglets at the varied suckling locations during the first lactation. This result is consistent with what Kim et al. (2000) found. There is a weak relationship between suckling location and birth weight of piglets (Fraser and Jones, 1975), which explains the insignificant results of birth weight. Weight gain of piglets during lactation highly relies on milk production of mammary glands (Lewis et al., 1978). The results of piglet growth suggest that the first 5 pairs of glands produced greater amount of milk per gland than the last 3 pairs of glands in average. Consistently, a previous study (Skok et al., 2007) showed that anterior and middle glands had a higher milk yield than posterior glands, and consequently piglets that suckled anterior glands had a greater weight gain than those that suckled posterior glands. On the other hand, a larger lactating mammary gland contains more milk secreting cells, so piglet growth is highly correlated to the size of the mammary glands that are suckled (Kim et al., 2000). The different milk production among glands at the varied suckling locations could be due to the difference in mammary growth during the first gestation, or the first lactation, or both. Previous studies have indicated that middle glands grew faster, which was indicated by greater wet weight and content of protein during late gestation (Ji et al., 2006), and greater wet weight, cross-sectional area, contents of protein, and ash in middle glands during lactation, as compared to the posterior glands (Kim et al., 2000).

During the second lactation, piglets that suckled anterior glands had greater weight gain and weaning weight than those that suckled the middle glands or posterior glands. Interestingly, the superiority of gland productivities at varied anatomical locations was different between the first and second lactations. Specifically, the anterior glands become superior to middle glands in regard to piglet weight gain only during the second lactation, but not during the first lactation. This could be explained by that anterior glands might have a higher sensitivity to stimulation of milk let down (Fraser et al., 1992, Hurley, 2001). This is also because the anterior glands might undergo more substantial growth during the second gestation, and because the accumulated growth of anterior glands (number of milk secreting cells) within two lactations were greater than that of other glands. These effects could be accumulated at the second lactation and finally leaded to a bigger size of mammary glands and greater amount of milk yield at the anterior location. In addition, milk produced by anterior glands might have a greater nutrient concentration as compared to milk produced by other glands. A previous study showed that anterior glands produced greater concentrations of immunoglobulins and lactoferrin from colostrum and milk, than posterior glands (Wu et al., 2010), suggesting that piglets that suckled glands at the anterior location had greater passive immunity and intestinal development than others. On the other hand, what is consistent in both lactations is that there was a greater number of piglets that suckled the anterior glands than those that suckled the middle glands. Moreover, there was a greater number of piglets that suckled the middle glands than those that suckled the posterior glands. The preference of gland usage is related to milk flow of glands, specific odor, and the degree

of comfort of suckling location (Gill and Thompson, 1956; Jeppesen, 1982; Fraser, 1984). Even through piglets having a larger BW are more competitive in fighting for the better suckling location, the teat order may be not associated with the birth weight of piglets, because the results showed that the birth weight of piglets that suckled glands at the varied suckling locations was not different during both lactations. However, the teat order was already quickly established within hours after birth (McBride, 1963).

The suckling history results revealed that the suckling history of mammary glands (mammary glands that were not suckled or suckled in the first lactation) did not affect the weight gain of piglets during the second lactation If the suckling location effects were excluded from the variation of piglet weight gain. In the first lactation, mammary glands suckled can grow more tissues whereas mammary glands not suckled regress during the first 7 to 10 d of lactation (Kim et al., 2001). Even the mammary glands that are suckled during the lactation undergo a quick regression once the suckling is terminated or the piglets are weaned. Moreover, mammary glands that were suckled during the lactation still had greater amount of wet weight and content of DNA per glands at d 7 postweaning, than mammary glands that were not suckled during the lactation (Ford et al., 2003). However, Farmer et al. (2012) indicated that, from d 2 to 14 of the second lactation, weight gain of piglets using mammary glands that were suckled during the first lactation was 0.34 kg greater at d 14 of the second lactation, than piglets using mammary glands that were not suckled during the first lactation; even though their BW at weaning were not different. The different results between these 2 studies could be partially due to the differences in experimental designs

related to maternal effects. Farmer et al. (2012) used sows that had the same glands or different glands suckled between the first and second lactations, and therefore arranged these sows into 2 sucking treatments. However, in the current study sow was considered as a random effect with mammary glands of each sow divided into 2 treatments according the suckling history of not sucked or suckled, and subsequently the effects of individual difference of sows were excluded. The reason for excluding the individual difference of sows is that effects of individual difference of sows in 2 parities may interfere the treatment effects. As the dietary energy and protein intake are significant factors influencing both the milk production and mammary growth in lactating sows (King and Dunkin, 1986; Kim et al., 1999b; Hurley, 2001), the significant results from Farm et al. (2012) could also be due to the difference in nutrient intake of sows between treatments.

In addition, Farm et al. (2012) also attributed the greater milk yield of the second lactation produced by glands that were suckled during the first lactation to the greater number of milk secreting cells and greater mammary gene expression. Pitkow et al. (1972) reported that the greater loss of alveolar cells during the first lactation can be compensated by a greater proliferation of alveolar cells during the following lactation in rats. Therefore, it is possibility that mammary glands that were suckled and regressed in the first parity may undergo an additional proliferation. However, the superiority of suckled glands to non-suckled glands in mammary growth decreased along the reproduction life. Kim et al. (1999a, 2001) showed that the amount of DNA of suckled glands was 825% (1489 vs. 160 mg/gland) greater than the amount of DNA of non-suckled glands at d 21 of lactation. The difference in

DNA content was mainly because suckled glands continuously grew whereas non-suckled glands underwent the involution. Until d 7 after weaning, the superiority of suckled glands over non-suckled glands in regard to DNA content still existed with a 146% (278 vs. 113 mg/gland) greater amount of DNA, even though the glands suckled during the first lactation also undergone a regression once suckling or nursing was ceased (Ford et al., 2003). However, until d 17 of the second lactation, glands suckled during the first lactation had only a 21.7% greater amount of DNA at d 17 of the second lactation than glands that were not suckled during the first lactation (Farmer et al., 2012), on the premise that both types of glands were suckled in the second lactation. Therefore, the beneficial effects of the first-lactation suckling on the content of DNA per gland are mitigated along with the time. This could be related to the maturity and growth rate of sows along with their life time.

Suckling or not suckling glands during the first lactation did not affect mammary productivity at either anatomical suckling location, which means that suckling condition of the first lactation did not affect mammary productivity in regard to piglet weight gain during the second lactation, when suckling location effects were excluded. Lacking of the interaction between effects of suckling history and suckling locations suggested that suckling history (not sucked or suckled during a previous lactation) was not a limiting factor influencing the difference in superiority of mammary gland productivity among different anatomical locations. The possible reasons for bringing up the difference in superiority of mammary gland production have been discussed previously.

In conclusion, the first 5 pairs of anterior and middle glands had a greater milk yield per pair of glands during the lactation of parity 1, whereas, anterior glands were superior to the other glands during the lactation of parity 2 in regards to weight gain of piglets. Suckling or not suckling a gland during parity 1 did not affect its productivity during parity 2 at either anatomical suckling location.

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Table 1. Birth weight, ADG, weaning weight, and percentage of gland usage of pigs according to suckling location in parity 1 and 2 of Exp. 1

	n	Birth weight, kg	ADG, g	Weaning weight, kg	Gland usage, %
Suckling location of mammary gland, pair[1]					
Parity 1[2]					
1st	51	1.49 ± 0.04[a]	216.9 ± 7.2[ab]	6.32 ± 0.22[a]	94.4 ± 5.1[a]
2nd	50	1.50 ± 0.04[a]	210.6 ± 7.2[ab]	6.15 ± 0.22[a]	92.6 ± 5.1[a]
3rd	46	1.57 ± 0.04[a]	221.0 ± 7.6[a]	6.46 ± 0.23[a]	87.0 ± 5.1[a]
4th	46	1.55 ± 0.04[a]	218.7 ± 7.6[ab]	6.36 ± 0.23[a]	87.0 ± 5.1[a]
5th	32	1.53 ± 0.05[a]	220.8 ± 9.1[ab]	6.48 ± 0.28[a]	61.1 ± 5.1[b]
6th	33	1.50 ± 0.05[a]	197.5 ± 9.0[b]	5.88 ± 0.27[a]	61.1 ± 5.1[b]
7th	22	1.50 ± 0.06[a]	209.5 ± 11.0[ab]	6.15 ± 0.33[a]	38.9 ± 5.1[b]
8th	4	1.54 ± 0.15[a]	221.0 ± 25.8[ab]	6.54 ± 0.78[a]	7.4 ± 5.1[b]
Anterior glands[3]	101	1.50 ± 0.03[a]	213.3 ±5.1[a]	6.24 ± 0.15[a]	93.5 ± 4.1[a]
Middle glands[3]	124	1.55 ± 0.03[a]	220.0 ± 4.6[a]	6.43 ± 0.14[a]	78.4 ± 3.4[b]
Posterior glands[3]	59	1.50 ± 0.04[a]	203.5 ± 6.7[b]	6.03 ± 0.20[a]	35.8 ± 3.4[c]
Parity 2[2]					
1st	50	1.50 ± 0.04[a]	268.1 ± 7.9[a]	7.01 ± 0.19[ab]	94.4 ± 4.5[a]
2nd	47	1.57 ± 0.04[a]	268.2 ± 8.1[a]	7.07 ± 0.19[a]	89.9 ± 4.5[a]
3rd	49	1.57 ± 0.04[a]	266.9 ± 8.1[a]	7.06 ± 0.19[a]	94.4 ± 4.5[a]
4th	44	1.49 ± 0.05[a]	243.9 ± 8.4[b]	6.50 ± 0.20[b]	85.2 ± 4.5[a]
5th	31	1.50 ± 0.05[a]	246.2 ± 10.0[ac]	6.57 ± 0.24[a]	57.4 ± 4.5[b]
6th	22	1.48 ± 0.07[a]	208.5 ± 11.9[d]	5.78 ± 0.28[c]	42.6 ± 4.5[c]
7th	21	1.40 ± 0.07[a]	226.2 ± 12.2[bcd]	6.06 ± 0.29[c]	40.7 ± 4.5[c]
8th	5	1.54 ± 0.14[a]	239.8 ± 25.0[abcd]	6.45 ± 0.60[c]	7.4 ± 4.5[d]
Anterior glands[3]	97	1.53 ± 0.03[a]	268.2 ± 5.6[a]	7.04 ± 0.14[a]	91.7 ± 3.7[a]
Middle glands[3]	124	1.52 ± 0.03[a]	253.6 ± 5.0[b]	6.74 ± 0.12[b]	79.0 ± 3.0[b]
Posterior glands[3]	48	1.45 ± 0.04[a]	219.5 ± 8.1[c]	5.97 ± 0.19[c]	30.2 ± 3.0[c]

[1]According to the suckling location anatomically, the first and second pairs of glands were defined as "anterior" glands; the third, fourth, and fifth pairs of glands were defined as "middle" glands; the sixth, seventh, and eighth pairs of glands were defined as "posterior" glands.

[2]Within a column, from first to eighth pair, means lacking a common superscript letter differ ($P < 0.05$).

3 With a column, among anterior, middle, and posterior glands, means lacking a common superscript letter differ ($P < 0.05$).

[3] The percentage of mammary gland usage was calculated as number of suckled mammary glands divided by total numbers of mammary glands at a specific suckling location. For example, if only 1 gland was suckled at the anterior glands, then the percentage of mammary glands usage at anterior glands was 25% for sow A.

Table 2. Performance of individual glands at parity 2 according to suckling history (not suckled or suckled in the first lactation) at varied suckling locations in Exp. 2

	Suckling history						*P*-value		
	Anterior[1]		Middle[1]		Posterior[1]		Anterior	Middle	Posterior
	Not suckled[2]	Suckled[2]	Not suckled	Suckled	Not suckled	Suckled	Not suckled vs. Suckled		
Parity 2									
n	6	91	23	101	26	22			
Birth weight, kg	1.67 ± 0.12	1.52 ± 0.03	1.47 ± 0.07	1.54 ± 0.03	1.47 ± 0.06	1.44 ± 0.06	0.254	0.326	0.771
ADG, g	270 ± 21	268 ± 5	256 ± 13	253 ± 6	221 ± 10	217 ± 11	0.923	0.851	0.781
BW gain, kg	5.53 ± 0.32	5.53 ± 0.13	5.16 ± 0.29	5.21 ± 0.11	4.54 ± 0.23	4.49 ± 0.25	0.994	0.886	0.873
Weaning weight, kg	7.20 ± 0.51	7.03 ± 0.13	6.74 ± 0.30	6.74 ± 0.14	6.01 ± 0.25	5.93 ± 0.28	0.749	0.994	0.833

[1] According to the suckling location of sows anatomically, the first and second pairs of mammary gland were defined as “anterior” glands; the third, fourth, and fifth pairs of mammary gland were defined as “middle” glands; the sixth, seventh, and eighth pairs of mammary gland were defined as “posterior” glands.

[2] “Not suckled” means that the mammary glands were not suckled in the first lactation; “Suckled” means that the mammary glands were suckled in the first lactation.

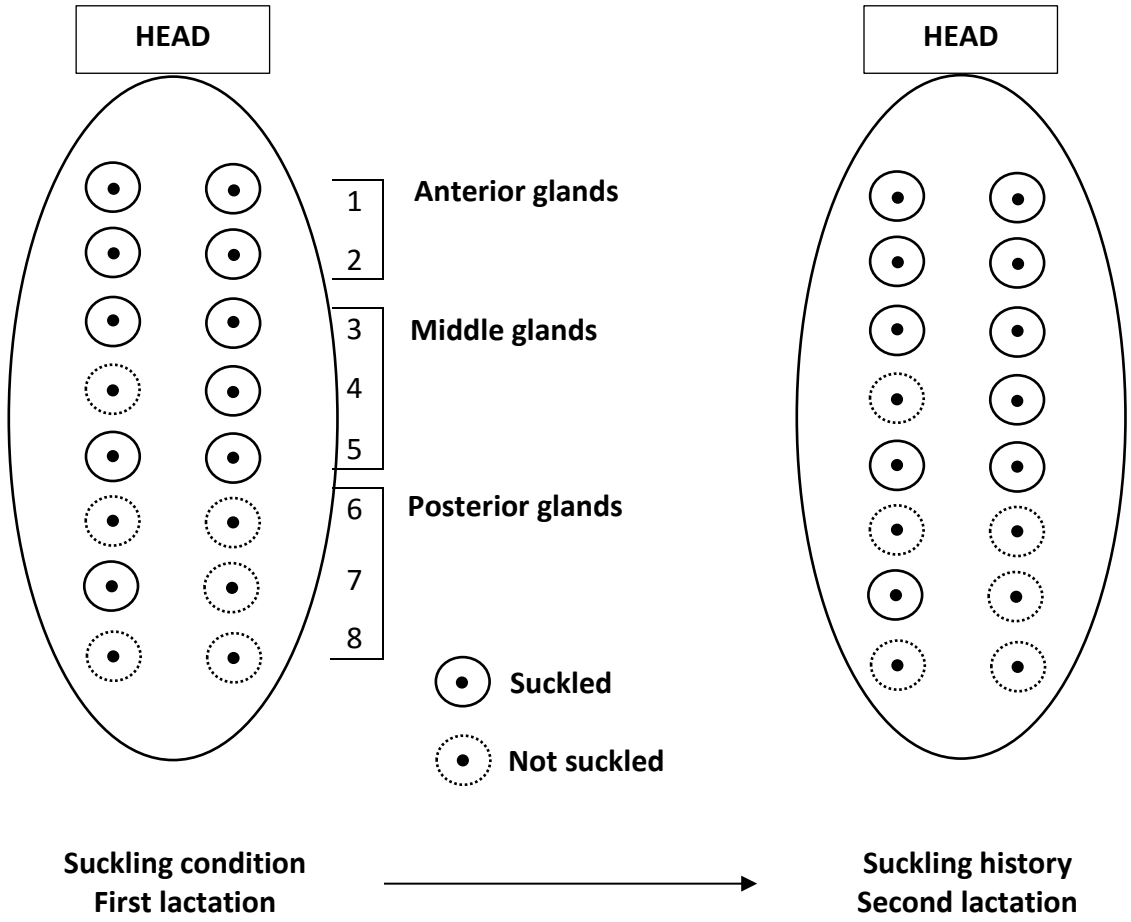

Figure 1. Suckling history of mammary glands. In the second lactation, mammary glands that were not suckled in the first lactation were considered as having a suckling history of 'not suckled'; mammary glands that were suckled in the first lactation were considered as having a suckling history of 'suckled'. The suckling condition (not suckled or suckled) of mammary glands in the first lactation was determined when piglets picked mammary glands with no interference by man.

CHAPTER 4

EFFECT OF DIETARY CATION-ANION DIFFERENCE ON ACID-BASE BALANCE AND CALCIUM HOMEOSTASIS IN MULTIPAROUS SOWS DURING LATE GESTATION AND LACTATION

Abstract: The study was conducted to evaluate the effects of dietary cation-anion difference (DCAD) on acid-base balance and Ca homeostasis in sows during late gestation and lactation. The DCAD was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S])/kg diet. A total of 22 multiparous sows (parity: 4.5 ± 2.9 and BW: 224.1 ± 38.7 kg) were initially allotted to 2 dietary treatments: CON (a control diet with a DCAD of 67 and 78 mEq/kg at late gestation and lactation) or ANI (an anionic diet with a DCAD of -122 and -148 mEq/kg at late gestation and lactation). An anionic salt (Cad-mate, Granco Minerals, Disputanta, VA) was supplemented in the anionic diet. Experimental diets were fed to sows from d 94 of gestation to d 18 of lactation. Sows had a daily access to 2 kg feed during gestation and *ad libitum* access to feed during lactation. Sows in ANI had greater ($P < 0.05$) concentrations of serum Ca at d 1 (9.7 vs. 9.2 mg/dL) and 18 (10.1 vs. 9.7 mg/dL) of lactation than sows in CON. There was a greater ($P < 0.05$) concentration of colostrum Ca in ANI than in CON (79.3 vs. 66.1 mg/dL). There was no difference in concentration of urine Ca between treatments. Sows in ANI had a lower ($P < 0.05$) blood pH at d 1 of lactation (7.37 vs. 7.48) and lower urine pH at d 108 of gestation, d 1, 9, and 18 of lactation as compared to sows in CON (5.23 vs. 6.25, 5.41 vs. 6.42, 5.12 vs. 6.48, and 5.06 vs. 6.26, respectively). At d 1 of lactation, the concentration of serum Cl in ANI was greater ($P < 0.05$) than that in CON (105 vs. 102 mEq/L). Concentrations of parathyroid hormone and 1,25-dihydroxycholecalciferol were not different between treatments at either d 1 or 18 of lactation. Concentrations of serum albumin in ANI were greater ($P < 0.05$) at either d 1 or 18 of lactation than those in CON (3.67 vs. 3.40 g/dL and 3.77 vs. 3.55 g/dL, respectively). Sows in ANI tended to have a

smaller (P = 0.086) concentration of total alkaline phosphatase in serum at d 18 of lactation than sows in CON (43.3 vs. 63.8 U/L). Feed intake, BW loss, and litter performance were not different between treatments. Collectively, feeding an anionic diet with a DCAD of -148 mEq/kg to sows can induce a mild metabolic acidosis at farrowing, reduce the urine pH consistently, and increase serum Ca and colostrum Ca during lactation, possibly due to increased bone resorption without affecting performance of sows.

Key words**:** acid-base equilibrium**,** calcium, dietary cation-anion difference, sow

Introduction

Modern highly prolific sows are producing larger litters with heavier piglets demanding increased milk production, as compared with those in the past (Kim et al., 2013), subsequently requiring increased amounts of minerals. Calcium is required for a series of physiologic functions for sows. The requirement for Ca is greatest during late gestation and lactation when Ca is used to maintain fetal skeleton development and milk production of pigs (Miller et al., 1994; Close and Cole, 2001; NRC, 2012).

Calcium is the predominant mineral in milk of sows. In the interest of maintaining milk Ca concentration at an adequate level, maternal bone resorption commonly increases to compensate for the insufficient Ca intake (Mahan and Vallet, 1997). During pregnancy, parturition, and lactation adjustments in hormones are required to regulate Ca metabolism. Reduction of Ca concentration in extracellular fluid triggers the secretion of parathyroid hormone (PTH) and formation of 1,25 dihydroxycholecalciferol (1,25-$[OH]_2D$), which is able to increase intestinal Ca absorption by activating the active transportation facilitated by Ca binding protein: calbindin. Parathyroid hormone elevates the Ca concentration by increasing Ca reabsorption in kidney and Ca resorption from bone.

Dietary cation-anion difference (DCAD) is used to determine the relationship between strong cations and anions and to predict whether the diets will elicit acidic or alkalinic response when fed to animals (Oetzel, 1991). Diets supplemented with an anionic diet to decrease DCAD are effective in reducing hypocalcemia by increasing bone resorption and inducing a metabolic acidosis in lactating ruminants (Oetzel, 1991; DeGaris and Lean,

2008). Consequently, it is hypothesized that sows fed an anionic diet may mobilize Ca more effective to meet the high Ca demand during lactation. Our objective was to determine the effects of different DCAD during late gestation and lactation on acid-base balance and Ca homeostasis of lactating sows.

Materials and Methods

A protocol for the use of animals in this study was approved by North Carolina State University Animal Care and Use Committee.

Animal and Experimental Diets

At the beginning of the experiment, a total of 30 sows (Yorkshire Landrace cross, 15 sows per treatment) were blocked according to their parity and initial BW. Within blocks, sows were randomly allotted to 1 of 2 dietary treatments: CON (a control diet targeting DCAD of 70 mEq/kg) or ANI (an anionic diet targeting DCAD of -140 mEq/kg) in a randomized complete block design. Four sows in each treatment were removed from the experiment due to pregnancy failure. Therefore, 22 multiparous sows with an average parity of 4.5 ± 2.9 and an average BW of 224.1 ± 38.7 kg with successful farrowing remained in the study. Dietary cation-anion difference was calculated as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram of diet (Tucker et al., 1991; Host et al., 1997). Sows were fed 2 kg experimental diets from d 94 of gestation, 3 kg experimental diets from d 108 of gestation, and allowed *ad libitum* access to feed during lactation (18 d). The control diet in CON was formulated with a DCAD of 67 or 78 mEq/kg during late gestation or lactation, respectively; the anionic diet in ANI was formulated with a DCAD of -122 or -148 mEq/kg during late

gestation and lactation. The anionic diet in ANI was supplemented with 2.0 and 2.2% anionic salt (Cad-mate, Granco Minerals, Disputanta, VA) during gestation and lactation, respectively. Cad-mate contained 12% Cl, 3% Mg, and 3% S at minimum provided by $CaSO_4$, $MgCl_2$, $MgSO_4$, and $(NH_4)_2HPO_4$. The experimental diets (Table 1) were corn-soybean meal based and formulated to meet or exceed the nutrient requirements by NRC (2012). Sows were housed in gestation crates (2.0 m x 0.6 m) at North Carolina State University Swine Educational Unit (Raleigh, NC), and were moved to farrowing crates (2.1 m x 1.5 m) at d 106 of gestation. Temperature in farrowing rooms was maintained at a minimum of 20ºC. Heat lamps were provided to piglets on both sides of farrowing crates.

Litter size and the number of liveborn, stillborn, and mummified piglets were recorded within 24 h of parturition. Cross-fostering was done to adjust the litter size to 9.8 ± 0.2 piglets within 48 h after parturition. Piglets were only cross-fostered within the sow's treatments. Sows were weighed at d 94 and 108 of gestation, at d 1, 9, and 18 of lactation to calculate the BW loss. During lactation, feed was provided twice (0630 and 1300 h daily) after the feed residue was collected and weighed at 0600 h. The feed intake of sows during lactation was calculated. Piglets were weighed at 1, 9, and 18 d of age to calculate the piglet and litter weight gain. Piglet survivability in each litter was calculated at d 18 of lactation.

Sampling and Analysis

Feed samples were collected after feed mixing. During the process of feed bagging, feed sampling was collected from the beginning, middle, and end of the batch. Dry matter, CP, Ca, total P, Mg, Na, K, and S in feed samples were determined by North Carolina

Department of Agriculture Feed and Forage Laboratory (Food and Drug Protection Division, Raleigh, NC). Feed samples were analyzed for moisture (method 930.15; AOAC, 2003) and CP by combustion (method 999.03; AOAC, 2003). Procedure of mineral analysis was adapted from AOAC (Method 985.01, AOAC, 2003). A total of 0.5 g dried, ground feed samples were weighed and pre-digested with 5 mL distilled H_2O, 4 mL HNO_3, and 1mL HCl for 30 min following a microwave digestion (MARS 6, CEM Corp., Matthews, NC). An Inductively Coupled Plasma Optical Emission Spectrometer (ICP-OES, Optima 5300 DV, Perkin Elmer Inc., Waltham, MA) with a Scott double-pass spray chamber were used to analyze feed Ca, total P, Mg, Na, K, and S with 3 replicate readings taken. The relative standard deviation of samples varied from 0.32% to 3.69%. Concentration of Cl was analyzed by a commercial laboratory (Dairy One, Ithaca, NY). In detail, a total of 0.5 g dried, ground feed samples were extracted for 15 min in a 50 mL 0.1 equivalent per liter of HNO_3 solution, followed by the potentiometric titration with $AgNO_3$ using Brinkman Metrohm 716 Titrino Titration Unit (Metrohm Ltd., C-H-9101, Herisau, Switzerland) as described by Cantliffe et al. (1970). The instrument for analyzing Cl was calibrated with 1,000 mg/kg of Cl standard solution with a CV within 5%. Standard samples from Association of American Feed Control Officials were used for calibration. Dietary cation-anion difference of both control and anionic diets were also calculated according to the analyzed compositions of Na, K, Cl, and S (Table 1).

Blood samples from jugular vein were collected, 2 h after feeding, at d 93 and 108 of gestation, d 1, 9, and 18 of lactation (0900 to 1000 h). Blood samples were collected in S-

Monovette tube (Sarstedt, Newton, NC) and centrifuged at 3,000 x g for 15 min at 4°C to obtain serum. Serum samples at d 1 and 18 of lactation were sent to a commercial laboratory (Antech Diagnostics, Cary, NC) for biochemistry assays according to Chaytor et al. (2011). Concentrations of total protein, albumin, globulin, total alkaline phosphatase, creatinine, blood urea N, and minerals including Na, K, Ca, P, and Cl were determined by a chemistry-immuno analyzer (AU640e, Olympus American Inc., Center Valley, PA) using the reagents: total protein, OSR6x32, biuret method; albumin, OSR6x02, modified bromocresol green method; total alkaline phosphatase, OSR6x04, p-nitrophenyl phosphate method; creatinine, OSR6x78, modified Jaffe kinetic method; and blood urea N, OSR6x34, urease glutamate dehydrogenase method. Concentrations of Na, K, P, and Cl were determined by ion selective electrodes method; Ca was determined by arsenazo III method; and P was determined by phosphomolybdate complex method. Controls samples with known concentrations of these parameters were tested per shift (30 samples) on the chemistry-immuno analyzer with a CV within 5%. Concentrations of serum Mg, urine Ca, and milk Ca were determined by a flame atomic absorption spectroscopy (AA-6701F, Shimadzu Scientific Instruments, Kyoto, Japan) as described by Armstrong et al. (2000). To determinate the concentrations of Mg and Ca, samples of serum, urine, and milk were digested with 5% HNO_3 and were deionized with 0.1% $LaCl_3$ and 0.2% KCl solutions. Concentrations of Mg and Ca were analyzed in duplicate. If analyzed concentrations of samples had a CV greater than 5%, the samples were analyzed again. Calibration was done by every 10 samples. Concentrations of PTH and 1,25-$(OH)_2D$ in serum were determined by ELISA kits (Porcine Intact PTH ELISA kit,

Immutopics International LLC, San Clemente, CA; 1,25-dihydroxy vitamin D EIA, IDS Ltd., Tyne and Wear, UK, respectively) according to the manufacturer's instructions. The sensitivity of the porcine intact PTH assay, as determined on 16 duplicate determinations of the 0 pg/mL standard, was 1 pg/mL. The sensitivity of 1,25-$(OH)_2$D assay, defined as the concentration corresponding to the mean minus 2 standard deviations of 20 replicates of the 0 calibrator, was 6 pmol/L.

Urine samples (50 mL) were collected at midstream when sows first urinated in the early morning (0630 to 0800 h). At least 30 mL of colostrum samples were collected within 24 h of parturition. Thirty mL milk samples were collected on d 18 of lactation. Colostrum and milk samples were obtained from all functioned mammary glands by injecting 0.5 mL of oxytocin into ear vein (DeRouchey et al., 2003). Blood and urine pH were measured by a pH meter (Accumet AB15, Fisher Scientific, Dubuque, IA) at room temperature immediately after the samples were obtained according to DeRouchey et al. (2003) and Razzaghi et al. (2012). The pH meter was standardized and calibrated by every 30 samples with standard buffers at pH 4, 7, and 10. The pH glass electrode was placed back in electrode storage buffer immediately after using. Blood pH was adjusted to a common body temperature of 39ºC as described by Patience et al. (1987), using the factor of 0.0147 per ºC (Severinghaus et al., 1956). At the same time, urinary special gravity was measured by a refractometer (Handheld Refractometer, Fisher Scientific, Dubuque, IA) as a parameter for density of urine. Samples of urine, colostrum, and milk were stored in -80ºC until further analysis. Concentrations of fat, lactose, protein, and solids-non-fat in colostrum and milk were analyzed by mid infrared

spectrophotometric technique using Milkoscan 4000 (Foss Electric, Hillerød, Denmark) at Virginia Tech United Federation of Dairy Herd Information Association Laboratory (Blacksburg, VA) as described by Garst et al. (1999). The instrument for milk composition analysis was regularly calibrated by using of samples (composition is known) to ensure the concentrations of components of milk within a CV of 3.5% and a standard deviation smaller than 0.04.

Rebreeding Performance

Sows returned to gestation buildings after weaning. All sows were artificially inseminated 3 times after estrus onset. The pregnancy status of sows was determined at d 30 postbreeding by using an ultrasound scanner (VSS700 EZ Preg Checker; Veterinary Sales and Service Inc., Stuart, FL). During the periods of rebreeding and gestation, sows were fed a common diet without supplementation of the anionic salt. Sows were moved back to farrowing crate at d 106 of gestation. During this period, the number of sows culled, farrowed, or did not farrow were recorded to calculate the culling rate and farrowing rate.

Statistical Analysis

All data except culling rate and farrowing rate were analyzed using the MIXED procedure of SAS (SAS Inst. Inc., Cary, NC) following a randomized complete block design. Initial BW and parity were considered as blocking variables. Initial BW was considered as a random effect; dietary treatments and parity were considered as fixed effects. Data sets of blood and urinary pH at d 108 of gestation, d 1, 9, and 18 of lactation were analyzed with either initial blood or initial urine pH as a covariate (Verbeke and Molenberghs, 1997; Littell

et al., 2000). A logistic regression (LOGISTIC procedure of SAS) was used to evaluate the effects of DCAD on culling rate and farrowing rate with chi-square tests (Peltoniemi et al., 1999; Bates et al., 2003). The individual sow was considered as the experimental unit. Probability values less than 0.05 were considered statistically significant and between 0.05 and 0.10 as trends. During analysis, data greater than (75th Percentile + 1.5 × Interquartile Range) and smaller than (25th Percentile – 1.5 × Interquartile Range) were considered as outlier and excluded from analysis according to LeBlanc (2004).

Results

Serum Ca, Milk Ca, and Urine Ca, Concentrations of PTH, and 1,25-$(OH)_2D$

At d 1 of lactation, concentrations of serum total Ca and colostrum Ca of sows in ANI were greater ($P < 0.05$) as compared to those in CON (Figure 1). At d 18 of lactation, the concentration of serum total Ca was greater ($P < 0.05$) in ANI than in CON. The concentration of milk Ca was not different between treatments. Concentrations of urine Ca did not differ between treatments at either d 1 or 18 of lactation. Concentrations of PTH and 1,25-$(OH)_2D$ were not different between treatments at either d 1 or 18 of lactation.

Serum Macrominerals

At d 1 of lactation, the concentration of serum Mg in ANI tended to be greater ($P = 0.097$) as compared with that in CON (Table 2). Concentrations of Na, K, and total P in serum were not different between treatments at d 1 of lactation, however, the concentration of serum Cl in ANI was greater ($P < 0.05$) than that in CON. At d 18 of lactation, the

concentration of serum Mg in ANI tended to be greater ($P = 0.087$) than that in CON, whereas the concentration of serum Cl was not different between treatments.

Blood pH, Urine pH, and Urinary Special Gravity

At d 94 of gestation, initial blood pH or urine pH was used as covariate for further pH data analysis (Figure 2). Urine pH in ANI were smaller ($P < 0.05$) at d 108 of gestation, d 1, 9, and 18 of lactation than those in CON. Blood pH in ANI was smaller ($P < 0.05$) than that in CON at d 1 of lactation. Blood pH (range from 7.30 to 7.48) was not different between treatments at d 108 of lactation, d 9, and 18 of lactation. Urinary special gravity was not different between treatments throughout the experimental period.

Blood Biochemistry Assays

At d 1 of lactation, the concentration of serum total protein in ANI tended to be greater ($P = 0.085$) than that in CON (Table 3). The concentration of serum albumin in ANI was greater ($P < 0.05$) than that in CON. The concentrations of total alkaline phosphatase and creatinine in serum did not differ between treatments at d 1 of lactation. At d 18 of lactation, concentration of total protein in serum was not different between treatments. The concentration of albumin in serum was greater ($P < 0.05$) in ANI as compared with that in CON at d 18 of lactation. Both concentrations of total alkaline phosphatase and creatinine in serum tended to be smaller ($P = 0.086$ and 0.080, respectively) in ANI that those in CON at d 18 of lactation. Concentrations of serum globulin and blood urea N were not different between treatments at either d 1 or 18 of lactation.

Colostrum and Milk Analysis

The compositions of colostrum and milk at either d 9 or 18 of lactation including fat, lactose, protein, and solids-non-fat were not different between treatments (Table 4).

Sow and Piglet Performance

Initial BW of sows at d 94 of lactation did not differ between treatments (Table 5). Weight gain of sows at d 108 of gestation was not different between treatments. At d 1 of lactation, differences in sow BW, litter weight, and piglet birth weight between treatments were not detected. Differences in numbers of piglets liveborn and stillborn between treatments were not detected. The number of piglets mummified in ANI was greater ($P < 0.05$) than that in CON. During d 0 to 9 of lactation, differences in weight loss and ADFI of sows between treatments were not detected. Differences in litter size, and litter weight gain between treatments were not detected. Differences in piglet weight gain and piglet survivability between treatments at d 9 of lactation were not detected. Differences in weight loss and ADFI from d 1 to 18 of lactation between treatments were not detected as well. Differences in litter weight gain, piglet weight gain, and piglet survivability from d 1 to 18 of lactation were not detected between ANI and CON.

During the subsequent parity, difference between treatments in culling rate and farrowing rate were not detected (Table 6).

Discussion

Calcium requirements during late gestation and lactation increase in proportion to the need of fetal growth and the levels of milk production (Miller et al., 1994; Close and Cole,

2001; NRC, 2012). Sows can mobilize Ca from bone tissues to maintain the milk Ca concentration when dietary Ca is insufficient (Mahan and Vallet, 1997). In this study, an acidifying diet was fed to sows in ANI from late gestation until the end of lactation. Anionic salt supplementation has been commonly used to acidify diet and as an approach to reduce the incidence of milk fever in dairy cows (Darriet et al., 2010). Previous studies (Block, 1984; Fredeen et al., 1988; Goff et al., 1991; Abu Damir et al., 1994) in ruminants have demonstrated that there were increased Ca absorption and bone resorption when an anionic diet was fed.

Dietary acidity or alkalinity is commonly defined by DCAD. Dietary cation-anion difference is commonly expressed as milliequivalents ([Na + K] – Cl) per kilogram diet in majority of publications in swine (Patience et al., 1987; Golz and Crenshaw, 1990; Dove and Haydon, 1994; NRC, 1998) and poultry (Mongin, 1981). However, S can be included in the DCAD equation because S is a negatively fixed ion with high ionic strength, processing 60% of the acidifying strength of Cl (Tucker et al., 1991). Studies (Tucker et al, 1991; Host et al., 1997) on association of DCAD and Ca metabolism in dairy cows most commonly adopt the equation expressed as milliequivalents ([Na + K] – [Cl + S]) per kilogram DM. In this study, DCAD was calculated by using the equation of milliequivalents ([Na + K] – [Cl + S]) per kilogram diet. The DCAD was formulated to 67 and 78 mEq/kg for the control diets and -122 and -148 mEq/kg for the anionic diets during gestation and lactation. The DCAD calculated from analyzed mineral composition was smaller (range difference: from 28 to 49 mEq/kg) compared with that calculated from mineral profiles in formulation (Table 1). The analyzed

composition showed that the diets were more acidic in general than what were calculated. Even small changes in Na or K or S or Cl could impact DCAD calculation. Possibly, there were small differences between mineral profiles and composition of minerals in ingredients used.

Increasing dietary anions such as Cl and S and reducing dietary cations such as Na and K alters the acid-base status by inducing a mild metabolic acidosis (Oetzel, 1991; Abu Damir et al., 1994; Goff and Horst, 1998), which can increase the mobilization of Ca from liver mitochondria (Akerman, 1978; DeGaris and Lean, 2008) and bone (Beck and Webster, 1976; Block, 1984). Measuring urine pH is considered as a sensitive method for assessing the acid-base balance of extracellular fluid (Seifi et al., 2004). This study showed that urine and blood pH was decreased, by feeding an anionic diet to sows. Commonly, sows maintain the urine and blood pH at a normal range from 6 to 7.5 and 7.25 to 7.45, respectively. The urine pH of sows fed an anionic diet was relatively lower in this study as compared to that in other studies (Tucker et al. 1988; Budde and Crenshaw, 2003), and was similar to what DeRouchey et al. (2003) found. The possible reason could be that the urine samples were collected by different methods, which causes the variability in measure environment such as temperature (Rosenthal, 1948). The decreased pH of urine and blood indicates that a mild metabolic acidosis occurred when sows were fed an anionic diet, which had 0.15% more S and 0.30% more Cl than the control diet as fed basis in this study. Excessive amount of strong cation intake can add additional H ion and trigger a decrease in extracellular fluid pH. The mild metabolic acidosis in this study was associated with the increased serum Cl

concentration in ANI sows. The result of increased serum Cl concentration agrees to what Budde and Crenshaw (2003) reported that increasing Cl intake increased Cl retention in postweaning pigs. Secondly, urine pH usually declines initially and subsequently increases when dairy cows were fed an anionic diet (Goff and Horst, 1998; Roche et al., 2007). Similarly, the decrease in urine pH in this study was great initially and subsequently became smaller when an anionic diet was fed to sows continuously. The pattern of change in urine pH could be due to effects of buffering when Ca is released from bone reserves at an acidic environment (Roche et al., 2007). In addition, previous literature (Patience and Chaplin, 1997; DeRouchey et al., 2003; Darriet et al., 2009, 2010) also indicated that the long-term buffering effects on a sow body in response to the dietary anionic load (anionic salt) are characterized by the increased renal excretion of H ions, Cl, SO_4, and NH_4 for regulating blood gas within normal physiological ranges.

In this study, the total Ca in serum was mildly elevated when sows were fed an anionic diet, which agrees to the result of other studies (Goff et al., 1991; Budde and Crenshaw, 2003; Razzaghi et al., 2012). The total Ca includes 3 fractions, which are ionized (44%), complexed (10%), and protein-bound Ca (46%) (Goldstein, 1990). It is speculated that the increased total Ca in serum occurred with the increased 3 fractions of Ca in the same ratio, resulting in a constant proportion of Ca (Goldstein, 1990). Among the protein-bound Ca, about 81% of it binds to albumin and about 19% of it binds to globulin (Moore, 1970). Decreasing blood pH is associated with the increased disassociation of Ca from albumin and an increased ionized Ca concentration (Loken et al., 1960; Agnes et al., 1993). Previous

studies (Oetzel et al., 1988; DeRouchey et al., 2003; Darriet et al., 2009) have demonstrated that ionized Ca increased as DCAD decreased. In this study, serum albumin increased when sows were fed an anionic diet. Because albumin concentration is positively correlated to the total Ca concentration in blood (Agnes et al., 1993), the result of serum albumin is consistent with the results of increased serum total Ca concentration in ANI sows during the lactation. Concentration of urine Ca in this study was numerically different but not statistically different between treatments. However, other studies consistently found that urine Ca concentration and urine total Ca extraction increase with decreased DCAD (Lemann et al., 1967; Goff and Horst, 1998; Budde and Crenshaw, 2003; Razzaghi et al., 2012).

Generally, blood Ca concentration is maintained within a narrow range (2.2 to 2.6 mmol/L or 8.5 to 10.5 mg/dL) (Swenson, 1984; Kim et al., 2009; Nitovski et al., 2011). Hypocalcemia in sows is described with symptoms of muscle weakness and posterior paralysis when there is a great Ca excretion and milk production (Durrell, 1942; Jennings, 1985; NRC, 1998). However, hypocalcemia in sows was not observed in other studies (Roux et al., 2008; Hintz and Billing, 2013). No hypocalcemia sows were observed in this study either, which was indicated by a total Ca concentration in blood, ranging from 8.3 to 10.3 mg/dL in experimental sows. The possible reason is that blood Ca concentration of sows is tightly regulated by calcitonin, PTH, and 1,25-$(OH)_2D$ (Miller et al., 1994). Mahan and Vallet (1997) also indicated that sows could mobilize Ca from bone reserves when dietary Ca and P are inadequate. The other possible reason could be that the lactation of this study was terminated early (d 18 of lactation). Hypocalcemia could be critical especially for sows with

extensive lactation, considering there are increased milk yield and milk Ca concentration at late lactation (Pond and Maner, 1974; Miller et al., 1994).

Calcium excretion in milk increases as the period of lactation processes (Pond and Maner, 1974; Miller et al., 1994). In this study, sows fed an anionic diet had a greater colostrum Ca concentration (increased from 66 to 79 mg/dL) than those fed control diet, whereas Ca concentration in milk at d 18 of lactation (CON vs. ANI: 156 vs. 158 mg/dL) was not different between treatments. The concentration range of milk Ca agrees to what other studies reported that Ca concentration in colostrum is about 87 mg/dL and Ca concentration in milk at the end of lactation increases to about 180 mg/dL (Kent et al., 1998). Even though there was a 20% increase in colostrum Ca of ANI sows, concentrations of nutrients such as protein, lactose, and fat in colostrum and milk at both d 9 and 18 of lactation were not different between treatments, which is the one of the possible reasons for the result that no difference in litter performance was observed between treatments.

Calcium absorption and bone resorption increase in ruminants with high Ca demand when an anionic diet is fed (Fredeen et al., 1988; Abu Damir et al., 1994; Roche et al., 2007). Budde and Crenshaw (2003) reported that postweaning pigs which were fed an anionic diet had a greater Ca retention than those fed a control diet. Even blood total Ca increased with decreased DCAD in this study, the Ca-regulating hormones, such as 1,25-$(OH)_2$D and PTH, did not increase with decreased DCAD as expected. Possible reason could be that metabolic acidosis which was induced by decreasing DCAD augmented the effects of PTH on mobilizing Ca from bone and increasing tubular reabsorption of Ca (Beck and Webster,

1976; Goff et al., 1991; Horst et al., 1997). On the other hand, there was a tendency for decreased serum total alkaline phosphatase in ANI sows. Alkaline phosphatase is negatively associated with bone resorption because alkaline phosphatase hydrolyzes pyrophosphate, which provides inorganic phosphate in bone mineralization (Orimo, 2010). Therefore, decreasing DCAD could have increased bone resorption by reducing the effects of alkaline phosphatase to bone mineralization. Likewise, the increased serum Mg concentration in ANI sows is consistent with our speculation about increased bone resorption.

There was no difference in performance between sows in CON and sows in ANI, which is in agreement to the results of other studies (Dove and Haydon, 1994; DeRouchey et al., 2003) that decreasing DCAD at gestation and lactation from 250 to130 mEq/kg or from 500 to 0 mEq/kg did not affect the performance of sows. The reason for increased mummified piglets from sows which fed decreased DCAD is not clear to understand at this moment. Previous literature had not reported the association between mummification and cation-anion difference either. Patience et al. (1987) reported that growth performance of growing pigs did not change when DCAD ranged from 0 to 341 mEq/kg but decreased at -85 mEq/kg. Other studies (Golz and Crenshaw, 1990; Haydon et al., 1990) subsequently predicted an optimal DCAD for postweaning and growing pigs, which was 250 mEq/kg. However, the DCAD of most these publications in swine (Austic and Calvert, 1981; Patience et al., 1987; Golz and Crenshaw, 1990; Haydon et al., 1990; Dove and Haydon, 1994; DeRouchey et al., 2003) were expressed as milliequivalents ([Na + K] – Cl) per kilogram diet. In this study, the DCAD for the control and anionic diets during lactation were 78 and -

148 mEq/kg, respectively when expressed as milliequivalents ([Na + K] – [Cl + S]) per kilogram diet, 178 and 76 mEq/kg, respectively when expressed as milliequivalents ([Na + K] – Cl) per kilogram diet. There was no difference in performance between treatments, which could be explained by that DCAD of both diets in this study was within the safe margin. In addition, creatinine is an index of muscle catabolism (Schutte et al., 1981; Nelssen et al., 1985) and blood urea N is an index of N excretion (Kohn et al., 2005). Serum creatinine concentration and blood urea N were not different between treatments, which is consistent with the performance results.

Urinary tract infections occur when bacteria, predominantly *Escherichia (E.) coli*, proliferate at the opening of the vulva, vagina, and urethra, then spread up to bladder and even kidney (Carr and Walton, 1993; Ronald, 2002). Lesions in the urinary tract are considered as the primary cause (more than 50%) of sudden death or culling of sows (Tillon and Madec, 1984; Sanz et al., 2007). Almond and Stevens (1995) confirmed that 22 to 40% of sows had urinary tract infections in Europe. The *E. coli* colonizes well in a neutral environment of the urinary tract (Abdul-Raouf et al., 1993; Presser et al., 1997). Decreasing DCAD may potentially reduce or prevent the incidence of urinary tract infections by inducing an acidic environment in the urinary tract. However, in current study, effects of decreasing DCAD on culling rate and farrowing rate of sow herd were not detected. Further studies are required to determine the effects of DCAD on microbial composition of urinary tract, incidence of urinary tract infections, culling rate, and reproductive performance in a larger sow herd.

In conclusion, feeding an anionic diet (DCAD of -122 or -148 mEq/kg) induced a mild metabolic acidosis at farrowing, and reduced the urine pH consistently during the lactation. Deceasing DCAD increased Ca concentrations in serum and colostrum of sows during lactation, implying that there might be increased bone resorption activities. The effects of decreasing DCAD on sow performance were not detected.

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Table 1. Composition of experimental diets (as-fed basis)

Item	Dietary treatment[1]			
	Gestation		Lactation	
	CON	ANI	CON	ANI
Ingredient, %				
Corn, yellow dent	81.30	78.30	70.10	66.40
Soybean meal, dehulled	13.85	13.85	23.50	23.50
Cad-mate[2]	0	2.00	0	2.20
L-Lys HCl	0	0	0.16	0.16
L-Thr	0	0	0.01	0.01
Poultry fat	1.00	2.00	2.08	3.58
Salt	0.50	0.50	0.50	0.50
Vitamin premix[3]	0.04	0.04	0.04	0.04
Mineral premix[4]	0.15	0.15	0.15	0.15
Dicalcium phosphate	2.05	2.05	2.38	2.38
Limestone, ground	1.11	1.11	1.08	1.08
Total	100.00	100.00	100.00	100.00
Calculated composition[5]				
DM, %	89.67	90.02	89.94	90.34
ME, Mcal/kg	3.36	3.34	3.40	3.40
CP, %	13.33	13.08	17.14	16.83
SID Lys, %	0.54	0.54	0.91	0.90
Ca, %	0.94	0.94	1.03	1.03
Total P, %	0.70	0.69	0.80	0.79
Available P, %	0.43	0.43	0.51	0.50
Mg, %	0.14	0.26	0.16	0.29
Na, %	0.27	0.27	0.33	0.33
K, %	0.57	0.56	0.73	0.73
S, %	0.13	0.28	0.16	0.36
Cl, %	0.33	0.66	0.37	0.73
DCAD, mEq/kg[6]	67.26	-121.96	78.18	-148.14
DCAD without S, mEq/kg[7]	148.36	52.71	177.98	76.43
Analyzed composition				
DM, %	88.64	88.77	89.33	89.48
CP, %	12.50	13.38	16.52	16.71

Table 1. Continued

Ca, %	0.97	0.87	1.12	1.10
Total P, %	0.73	0.66	0.93	0.81
Mg, %	0.12	0.22	0.15	0.25
Na, %	0.24	0.21	0.18	0.19
K, %	0.52	0.52	0.74	0.70
S, %	0.16	0.31	0.21	0.37
Cl, %	0.39	0.65	0.38	0.72
DCAD, mEq/kg[8]	27.54	-152.43	29.32	-172.28

[1] Dietary cation-anion difference (DCAD) was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram diet. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation).

[2] Cad-mate (Granco Minerals, Disputanta, VA) is an anionic salt, which contains 12% Cl, 3% Mg, and 3% S at minimum provided by $CaSO_4$, $MgCl_2$, $MgSO_4$, and $NH_4H_2PO_4$.

[3] The vitamin premix provided the following per kilogram of complete diet: 6613.8 IU of vitamin A as vitamin A acetate; 992.0 IU of vitamin D_3; 19.8 IU of vitamin E; 2.64 mg of vitamin K as menadione sodium bisulfate; 0.03 mg of vitamin B_{12}; 4.63 mg of riboflavin; 18.52 mg of D-pantothenic acid as calcium pantothenate; 26.46 mg of niacin; 0.66 mg of biotin.

[4] Mineral premix provided the following per kilogram of complete diet: 39.6 mg of Mn as manganous oxide; 165 mg of Fe as ferrous sulfate; 165 mg of Zn as zinc sulfate; 25.15 mg of Cu as copper sulfate; 0.30 mg of I as ethylenediamine dihydroiodide; and 0.30 mg of Se as sodium selenite.

[5] Calculated composition was based on NRC (2012).

[6] DCAD was based on calculated composition of minerals.

[7] DCAD without S was calculated as mEq ([Na + K] – Cl) per kilogram diet.

[8] DCAD was based on analyzed composition of minerals.

Table 2. Serum macromineral composition

	Dietary treatment[1]			
Item	CON	ANI	SEM	*P*-value
No. of sows	11	11		
Serum, d 1 of lactation				
Na, mEq/L	143.64	145.36	0.90	0.191
K, mEq/L	4.78	4.87	0.14	0.653
Total Ca, mg/dL	9.23	9.71	0.15	0.031
Mg, mg/dL	1.87	1.98	0.04	0.097
P, mg/dL	7.31	7.21	0.26	0.804
Cl, mEq/L	101.82	104.73	0.79	0.017
Na/K ratio	30.18	30.09	0.88	0.943
Serum, d 18 of lactation				
Na, mEq/L	141.64	141.00	0.52	0.401
K, mEq/L	5.42	5.25	0.11	0.300
Total Ca, mg/dL	9.74	10.14	0.10	0.012
Mg, mg/dL	2.21	2.40	0.07	0.087
P, mg/dL	6.45	6.18	0.20	0.361
Cl, mEq/L	103.09	102.82	0.65	0.769
Na/K ratio	26.18	27.00	0.60	0.348

[1] Dietary cation-anion difference (DCAD) was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram diet. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation).

Table 3. Blood biochemistry assays of sows at parturition and weaning

	Dietary treatment[1]			
Item	CON	ANI	SEM	*P*-value
No. of sows	11	11		
Serum, d 1 of lactation				
Total protein, g/dL	6.23	6.52	0.11	0.085
Albumin, g/dL	3.40	3.67	0.06	0.004
Globulin, g/dL	2.83	2.85	0.10	0.898
Total alkaline phosphatase, U/L	46.66	44.73	3.54	0.703
Creatinine, mg/dL	2.16	2.01	0.10	0.293
Blood urea N, mg/dL	9.91	9.00	0.26	0.264
Serum, d 18 of lactation				
Total protein, g/dL	7.17	7.31	0.18	0.591
Albumin, g/dL	3.55	3.77	0.07	0.042
Globulin, g/dL	3.63	3.54	0.20	0.753
Total alkaline phosphatase, U/L	63.80	43.27	8.20	0.086
Creatinine, mg/dL	1.60	1.43	0.07	0.080
Blood urea N, mg/dL	14.45	12.73	0.92	0.198

[1] Dietary cation-anion difference (DCAD) was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram diet. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation).

Table 4. Colostrum and milk compositions in lactating sows[1]

	Dietary treatment[2]			
Item	CON	ANI	SEM	*P*-value
No. of sows	11	11		
Colostrum				
Fat, %	7.28	7.09	0.60	0.837
Protein, %	8.70	8.15	0.79	0.631
Solids-non-fat, %	15.33	15.07	0.71	0.807
Lactose, %	4.91	5.04	0.16	0.556
Ca, mg/dL	66.12	79.34	4.12	0.047
Milk, d 9 of lactation				
Fat, %	7.05	6.92	0.42	0.827
Protein, %	4.90	4.80	0.11	0.512
Solids-non-fat, %	13.28	13.11	0.33	0.736
Lactose, %	6.50	6.44	0.15	0.782
Milk, d 18 of lactation				
Fat, %	6.85	6.18	0.32	0.157
Protein, %	4.61	4.77	0.12	0.323
Solids-non-fat, %	12.96	13.07	0.11	0.505
Lactose, %	6.63	6.57	0.05	0.473
Ca, mg/dL	156.26	158.05	4.42	0.783

[1] Wet weight basis

[2] Dietary cation-anion difference (DCAD) was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram diet. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation).

Table 5. Effects of dietary cation-anion difference (DCAD) on performance of sows and litters

	Dietary treatment[1]			
Item	CON	ANI	SEM	*P*-value
No. of sows	11	11		
Sow BW (d 94 of gestation), kg	224.4	223.8	12.0	0.973
Parity	4.0	3.7	0.9	0.835
d 108 of gestation				
BW gain, kg	5.8	7.4	1.1	0.355
d 1 of lactation				
BW, kg	222.7	222.4	11.0	0.986
Litter weight, kg	14.9	15.9	0.4	0.345
Piglet birth weight, kg	1.46	1.52	0.04	0.346
Litter size after CF, piglet	9.8	9.7	0.2	0.697
Liveborn	10.6	9.2	0.7	0.147
Stillborn	0.8	0.6	0.4	0.761
Mummified	0	0.7	0.2	0.027
d 9 of lactation				
BW loss, kg	12.0	6.5	2.7	0.168
Litter size, piglet	9.5	9.3	0.2	0.502
ADFI, kg	7.4	7.0	0.7	0.256
Litter weight gain, kg	15.5	14.7	0.5	0.526
Piglet weight gain, kg	1.61	1.59	0.08	0.845
Piglet survivability, %	93.0	89.9	2.7	0.333
d 18 of lactation				
BW loss during lactation, kg	14.3	9.1	3.3	0.284
ADFI during lactation, kg	7.7	7.4	0.5	0.543
Litter size, piglet	9.5	9.3	0.2	0.435
Litter weight gain, kg	39.5	36.9	1.7	0.284
Piglet weight gain, kg	4.11	3.98	0.17	0.618
Piglet survivability, %	93.0	89.9	2.7	0.450

[1] Dietary cation-anion difference was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram diet. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation).

Table 6. Effects of dietary cation-anion difference (DCAD) on culling rate and farrowing rate of sows during a subsequent parity[1]

	Dietary treatment[1]			
Item	CON	ANI	SEM	*P*-value > Chi-square
Culling rate, %[2]	18.2	9.1	9.8	0.541
Farrowing rate, %[3]	100.0	90.0	9.5	0.961

[1] Dietary cation-anion difference was expressed as milliequivalents (mEq) ([Na + K] – [Cl + S]) per kilogram diet. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation). Numbers of sows for CON and ANI were 11 and 11, respectively.

[2] Data of culling rate were cataloged as either cull or not cull for each sow. Data of culling rate were analyzed by using the LOGISTIC procedure (SAS Inst. Inc., Cary, NC). Causing of culling includes death, lameness, low productivity, failing conception and so on. Mean and SEM of culling rate were provided by using LSMEANS procedure with ILINK option (SAS Inst. Inc., Cary, NC).

[3] Data of farrowing rate were cataloged as either farrowed or not farrowed for each sow. Data of farrow rate were analyzed by using the LOGISTIC procedure (SAS Inst. Inc., Cary, NC).

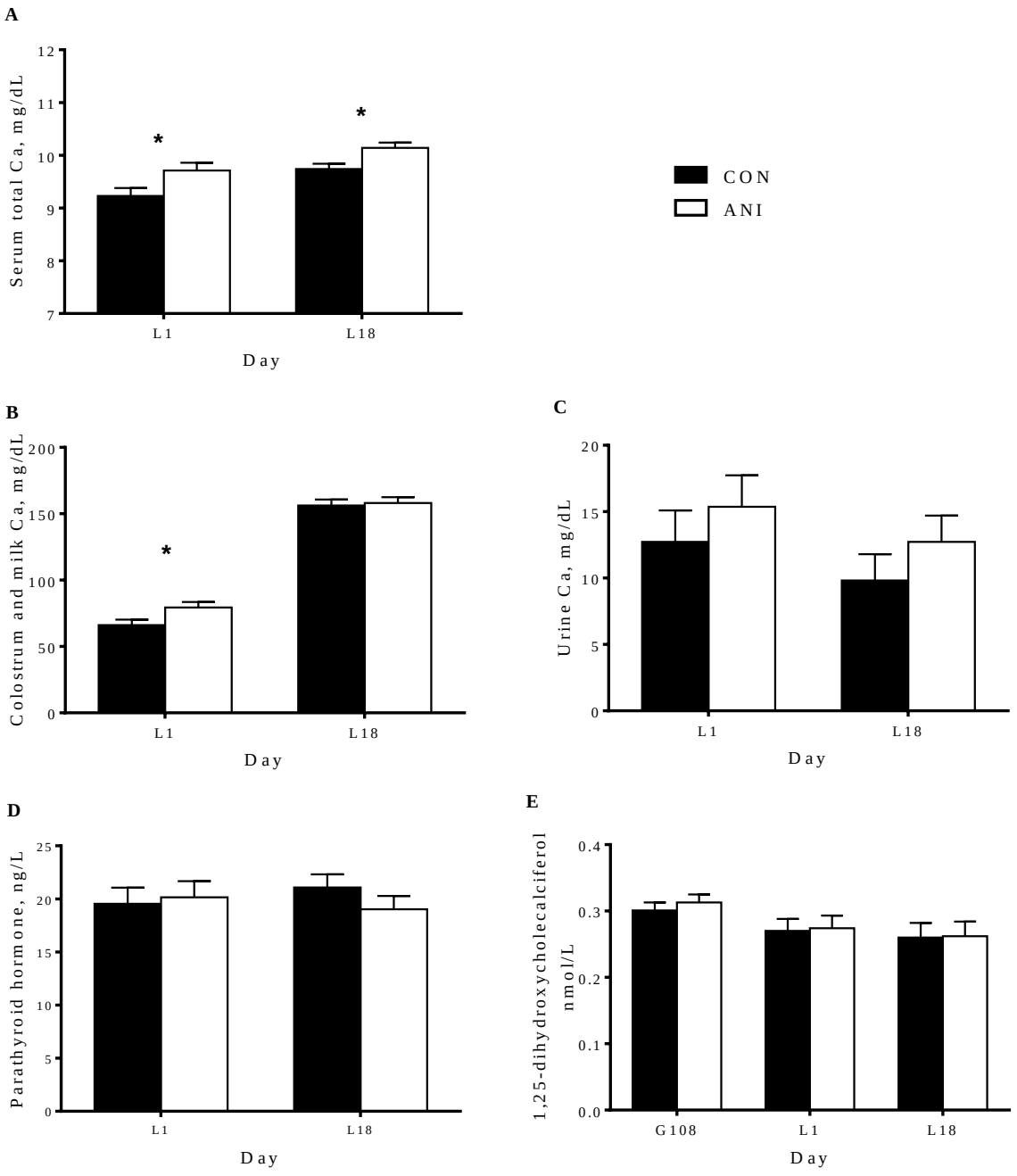
A
Serum total Ca, mg/dL
12
11
10
9
8
7
*
*
L1
L18
Day
CON
ANI
B
Colostrum and milk Ca, mg/dL
200
150
100
50
0
*
L1
L18
Day
C
Urine Ca, mg/dL
20
15
10
5
0
L1
L18
Day
D
Parathyroid hormone, ng/L
25
20
15
10
5
0
L1
L18
Day
E
1,25-dihydroxycholecalciferol
nmol/L
0.4
0.3
0.2
0.1
0.0
G108
L1
L18
Day

Figure 1. Effect of dietary cation-anion difference (DCAD, [Na + K] – [Cl + S]) on serum Ca, milk Ca, and urine Ca concentrations, parathyroid hormone, and 1,25-dihydroxycholecalciferol concentrations in gestating and lactating sows. Values were provided as least square mean and standard error, n = 11. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation). * Standing for difference ($P < 0.05$) between CON and ANI.

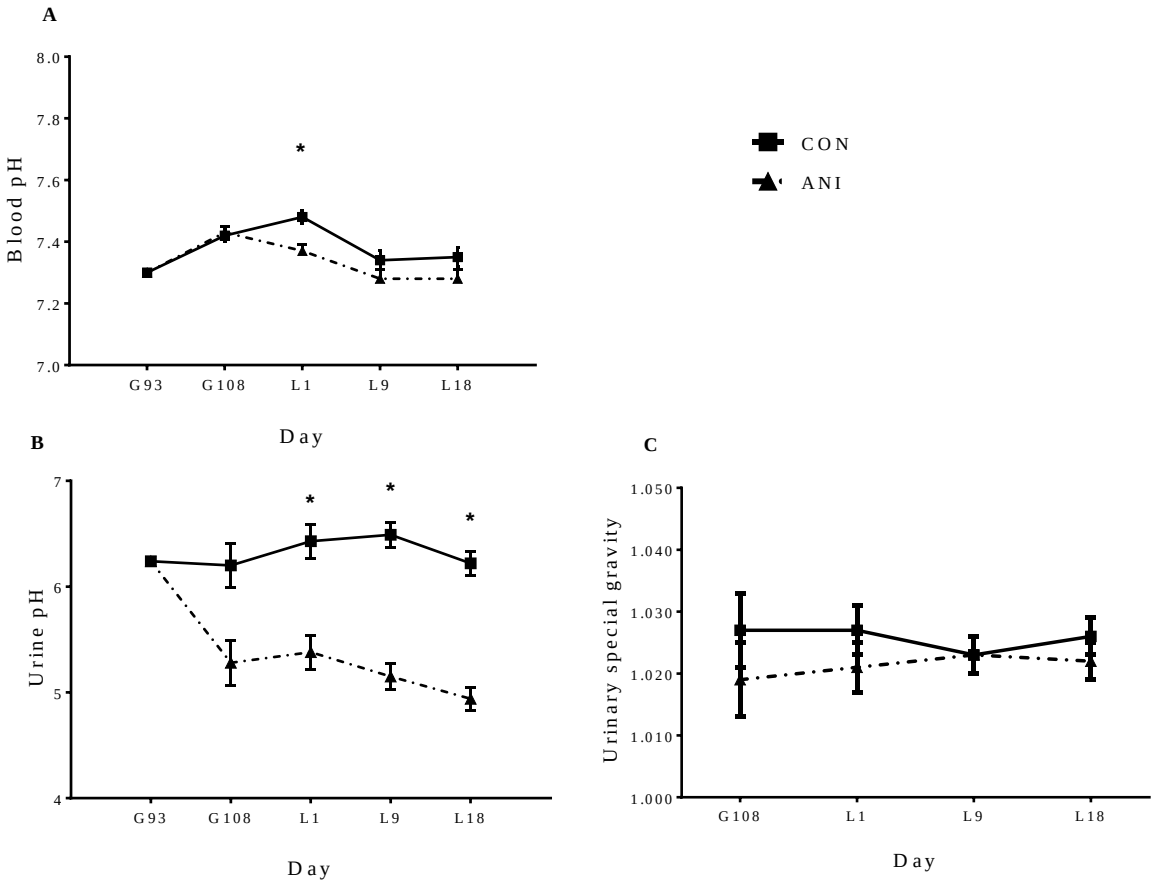

Figure 2. Effect of dietary cation-anion difference (DCAD, [Na + K] – [Cl + S]) on blood pH, urine pH, and urinary special gravity in gestating and lactating sows. Values were provided as least square mean and standard error, n=11. Two dietary treatments were: CON (a control diet with a DCAD of 67 or 78 mEq/kg at gestation or lactation, respectively); and ANI (an anionic diet with a DCAD of -122 or -148 mEq/kg at gestation or lactation). * Standing for difference ($P < 0.05$) between CON and ANI.

CHAPTER 5

EFFECT OF SUPPLEMENTAL RETAIL NON-SALEABLE MILK DURING FIRST FOUR DAYS POSTWEANING ON GROWTH PERFORMANCE, NUTRIENT DIGESTIBILITY, INTESTINAL INTEGRITY, DIARRHEA, MORTALITY, AND ECONOMIC RETURNS OF NURSERY PIGS

Abstract: The experiment was conducted to evaluate the effects of supplemental milk (6.5, 8.7, 10.9, and 10.9 g DM milk/pig/d; 10% of estimated nursery feed intake) during the first 4 d postweaning on growth performance, nutrient digestibility, diarrhea, mortality, and economic returns of nursery pigs. A total of 644 crossbred pigs, weaned at 3 wk of age (6.4 ± 1.2 kg of BW), were randomly assigned to 2 dietary treatments (12 pens/treatment, 27 pigs/pen) in a randomized complete block design with sex and initial BW as blocking variables. Pigs were fed pelleted feed either with or without milk supplementation (milk was mixed with pelleted feed to form a gruel) from d 1 to 4 postweaning (4 times daily, 2-h interval) in a liquid feeding trough. All pigs had free access to another nursery metal feeder filled with pelleted feed during the entire nursery period in 3 phases (phase I: 10 d; phase II: 14 d; and phase III: 25 d). Fecal scores were evaluated daily according to the observation of fresh feces in pens from d 1 to 4 postweaning, following a scale of 1 to 3 (3 = liquid diarrheal feces). Apparent ileal digestibility (AID) of DM, CP, ether extract, and GE was determined at the end of phase I. Jejunal tissues from 1 pig per pen were collected at d 4 and 10 postweaning. Feed cost, revenue, and gross margin/pig were calculated. The results showed that milk supplementation did not affect the ADG and ADFI during the entire nursery period. However, milk supplementation tended to increase ($P = 0.073$) G:F ratio (0.708 to 0.748) from d 4 to 24 postweaning. The AID of DM, CP, ether extract, and GE at d 10 of postweaning did not differ between treatments. Milk supplementation did not affect the jejunal morphology at both d 4 and 10 postweaning. Milk supplementation tended to reduce ($P = 0.065$) the fecal scores (2.04 to 1.84) and decreased ($P < 0.05$) mortality (4.35 to 1.55%)

during the entire nursery period. Feed cost, revenue, and gross margin per pig or per pen did not differ between treatments. Collectively, supplemental milk 4 times daily for the first 4 d postweaning reduced the severity of diarrhea during phase I, increased feed efficiency during phase I and II, decreased mortality but did not enhance the weight gain, intestinal integrity, and economic returns of nursery pigs during the entire nursery period.
Key words: growth performance, gut health, milk, nursery pigs

Introduction

In the United States, 10% of available food supply was lost at the retail-level and 21% was lost at the consumer-level; among these, 32% (about 7.7 billion kg) of available fluid milk supply was wasted with a total loss of 6.4 billion dollars in 2010 (Buzby et al., 2014). The discarded milk was about 850 kg per milk cow per year (NASS, 2010; Buzby et al., 2014). Dairy milk is a valuable and integral ingredient for young pig diets. Milk products such as casein, lactose, skim milk powder, whey permeate, whey powder, and whey protein concentrate are highly palatable ingredients for nursery pigs to provide readily digestible nutrients.

Nursery pigs may undergo postweaning growth lag, which is a phenomenon that nursery pigs display slow growth, depressed feed intake, scouring, frequent diarrhea, and increased mortality when they are exposed to nutritional, environmental, and social stressors after weaned at 3 to 4 wk of age. Weaning is generally characterized by the reduced integrity of intestinal structure (Kelly et al., 1991), decreased brush border enzyme activity (Miller et al., 1986), depressed immunological response, and increased intestinal inflammation (McCracken et al., 1999). During the suckling-weaning transition, the dietary components are transitioned from lactose and animal protein in a liquid form (ie., milk) into the plant sources of polysaccharides and protein in a dry meal or pelleted form. The negative performance or postweaning lag is mainly attributed to the inadequate feed intake (Armstrong and Clawson, 1980; McCracken et al., 1995), and limited nutrient digestibility (Williams, 2003). Previous studies showed that liquid milk feeding or milk replacer feeding

can improve the DM intake and growth rate of pigs during the first to second wk postweaning (Pluske et al., 1996; Odle and Harrell, 1998; Price et al., 2013). Moreover, the enhanced growth performance of pigs fed liquid milk based diets could be maintained to market weight (Kim et al., 2001; Wolter et al., 2002). However, the increased costs for equipment, maintenance, and labor become the major constraints for widespread application of milk products in a liquid feed system.

Pasteurized non-saleable milk has been fed to calves for many years with high economical and nutritional efficiencies, to replace the usage of more expensive milk replacer (Kesler, 1981; Jamaluddin et al., 1996; Godden et al., 2005). Considering that the milk could be a highly palatable and digestible ingredient for nursery pigs, a major question has been raised, can the non-saleable milk from retailers be supplemented with pelleted feed and benefit nursery pigs by improving their growth and economic returns? Therefore, the objective of this study was to evaluate the effect of non-saleable milk supplementation on growth performance, nutrient digestibility, intestinal integrity, diarrhea, mortality, and economic returns of nursery pigs.

Materials and Methods

Animals and Experimental Design

The experimental protocol was approved by the North Carolina State University Animal Care and Use Committee.

The experiment was conducted in a commercial farm (N.G. Purvis Farms, Inc., Robbins, NC). A total of 644 crossbred pigs (3 wk of weaning age, 6.4 ± 1.2 kg of BW),

were randomly assigned to 2 dietary treatments (12 pens/treatment, 27 pigs/pen) in a randomized complete block design with sex and initial BW as blocks. Pigs were fed pelleted feed either with or without milk supplementation (MILK or CON) from d 1 to 4 postweaning (4 times daily) in polyvinyl chloride (PVC) troughs that were placed in the pens for 2 h at each feeding (0630, 0830, 1030, and 1230 h). The pelleted feed and milk supplementations in PVC troughs at d 1 postweaning were offered on a different schedule (0830, 1030, 1230, and 1430 h) due to the delay caused by transfer pigs from farrowing facilities to nursery facilities and allotment. The PVC troughs were used in both MILK and CON pens from d 1 to 4 postweaning. In MILK pens, milk was mixed with pelleted feed evenly into gruel (pelleted feed was added firstly and milk was added secondly in the PVC troughs) at the ratios of 3 to 1, 2 to 1, 1 to 1, and 1 to 1 (or 340 to 113, 454 to 227, 567 to 283, and 567 to 567 g, as-fed basis) at d 1, 2, 3, and 4 postweaning, respectively. An equal amount of pelleted feed (equal to the sum of milk [estimated as 13% DM] and pelleted feed on a DM basis in MILK) was also provided in PVC troughs of CON. After each feeding, feed residue in PVC troughs was collected, weighed, and oven-dried for later DM intake calculation. The PVC troughs were cleaned and sanitized before each feeding. In addition to the feed supplementation in PVC troughs, all pigs were fed *ad libitum* the same pelleted feed, which was placed in another nursery metal feeder, during the entire nursery period in 3 phases (phase I: 10 d; phase II: 14 d; and phase III: 25 d). The PVC troughs provided extra feeder space (feeder area: 0.77 m^2 per pen; linear feeder space: 2.79 m per pen) in addition to the nursery metal feeders (linear

feeder space: 0.91 m per pen). The space allocation of nursery room was 6.07 m^2 per pen or 0.23 m^2 per pig.

Milk was purchased from the recycle company, which collected the non-saleable milk from the retailers. In the experiment, milk was obtained from a pipeline connected to the milk tank during each feeding. Before obtaining the milk, workers would smell the milk and test the milk temperature by hand. Milk would not be used for nursery pigs if it smelled "off" and felt warm. If milk was acceptable, milk in an amount required for each feeding was filled a plastic milk tank and delivered to the nursery rooms immediately. The milk contained 9.71% total solid, 2.00% fat, 7.61% solids nonfat, 2.09% protein, and 4.66% lactose. Titanium dioxide (0.3%) was added in the phase I pelleted feed as an indigestible marker to measure apparent ileal digestibility (AID) of DM, CP, ether extract and GE.

Growth Performance, Body Weight Loss of Pigs, and Mortality

The ADFI, ADG, and G:F ratio were obtained at phase I, II, III, and combined phases of nursery period. The ADFI of liquid, pellet, and total feed from d 0 to 4 postweaning was calculated on a DM basis. The G:F ratio from d 0 to 4 postweaning was provided on a DM basis. Regarding of the pigs which lost weight from d 0 to 4 postweaning, the BW loss and the percentage of pigs which lost weight were calculated. Among all these performance calculation, pen was considered as the experimental unit. The mortality during the entire nursery period was calculated.

Fecal Scores

Fecal scores were recorded at d 2, 3, 4, and 5 postweaning (0630 and 1600 h), based on the observation of fresh feces in the pens. Fecal scores were based on the following scale: 1 = normal, solid feces; 2 = soft, looser than normal feces; 3 = liquid diarrheal feces. Scale system of fecal scores was adopted from the literature (Gomez et al., 1998; Jensen et al., 2006). During the observation, certain type of feces appeared more than 50% within pens were used to determine the fecal scores.

Sample Collection

A total of 48 pigs (1 from each pen at d 4 and 10 postweaning, respectively; 12 pens per treatment) was euthanized with whole intestine tract below the diaphragm removed. The whole small intestine was dissected out and separated carefully without pulling or stretching. About 10 cm of the middle segment of jejunum toward the proximal end was cleaned with distilled water with all digesta gently removed. The jejunal mucosa was scraped with a glass slide and stored in -80ºC for further analysis. About 20 cm of middle segments of jejunum toward the distal end was carefully cleaned with distilled water, and fixed in 10% formalin for morphology measurement. The weight and length of the previous 2 segments of jejunum were measured before the mucosa and morphological samples collections. The rest segments of small intestine were measured for length, and weighed with intestinal digesta washed free, as described by Kelly et al. (1991). As described by Shen et al. (2012), jejunal mucosa (0.5 g) was weighed and homogenized with a handheld homogenizer (Tissuemiser, Thermo Fisher Scientific Inc., Waltham, MA) on ice for 60 s with 1 mL PBS buffer included. The

homogenate was centrifuged at 15,000 × g at 4ºC for 30 min. The supernatant was stored in -80ºC for determining concentrations of protein and tumor necrosis factor alpha (TNFα) later. At d 10 postweaning, ileal digesta was collected in 50 mL sterilized tube and stored in -80ºC for the analysis of nutrient AID.

Chemical Analysis

Feed residue per day was pooled, mixed, and oven-dried (method 934.01; AOAC, 2006) to obtain an accurate DM intake. The procedure of TiO_2 analysis was adapted from Myers et al. (2004). In details, Phase-I feed and ileal samples at d 10 postweaning were digested in concentrated H_2SO_4 for 2.5 h, followed by addition of 30% H_2O_2, and the absorbance was measured at 410 nm. Moisture content was measured with feed samples oven-dried in a forced-air oven (Isotemp Forced Air Convection Lab Oven, Thermo Fisher Scientific Inc., Waltham, MA) at 95ºC (method 934.01; AOAC, 2006). Frozen ileal digesta samples were freeze-dried (24D x 48, Virtis, Gardiner, NY) for chemical analysis. Gross energy was analyzed by using the bomb calorimeter (C2000, IKA-Works Inc., Wilmington, NC). The ether extract was analyzed by the indirect method (method 920.39; AOAC, 2006). Nitrogen was analyzed by using the combustion method (FP528, Leco, St Joseph, MI) to calculate CP (method 992.15; AOAC 2006). Apparent ileal digestibility was calculated as AID (%) = 100 – ([ND/NF] × [TiF/TiD] × 100), where ND is the nutrient concentration in ileal digesta, NF is the nutrient concentration in diets, TiF is the TiO_2 concentration in diets, and TiD is the TiO_2 concentration in digesta.

Jejunal Morphology

Two segments of middle sections of jejunum were fixed in 10% formalin, embedded in paraffin, and stained with hematoxylin and eosin at North Carolina State University Histopathology Laboratory (College of Veterinary Medicine, Raleigh, NC) before the examination by light microscopy. The slides were examined under a digital charge-coupled device camera (INFINITY2-2, Lumenera Corp., Ottawa, ON, Canada), which is attached by a camera adapter (U-CMAD3, Olympus Corp., Tokyo, Japan) to a biological microscope (CX31, Olympus Corp., Tokyo, Japan). A total of 15 well-oriented villi and their associated crypts per pig were measured for villus height (from the tip of the villi to the villus-crypt junction), villus width (measured in the middle of villi), and crypt depth (from villus-crypt junction to the base of crypt) according to Shen et al. (2009). Apparent villus surface area was calculated using the trigonometric relationship between villus height, apical villus width, and basal villus width according to Cummins et al. (1990). Apical and basal villus width ratio was calculated as dividing the apical villus width by its associated basal villus width. The apical and basal villus width ratio was used to determine the degree of shrinkage of villus tip (as suggested by the committee member Dr. Peter Ferket, the villus tip shrinks because of the transition of diets or weaning stresses). Villus height and crypt depth ratio was calculated by dividing the villus height by its associated crypt depth.

Tumor Necrosis Factor α Measurement

The concentration of protein in homogenization supernatant of jejunal mucosa was analyzed with a commercial kit (Pierce BCA Protein Assay Kit, Thermo Fisher Scientific

Inc., Rockford, IL) as described by Smith et al. (1985). Samples were tested in duplicate. The intra-assay CV was 3.6%.

As described by Chaytor et al. (2011), the concentration of TNFα for homogenization supernatant of jejunal mucosa was analyzed with an ELISA kit (Porcine TNF-alpha Quantikine ELISA Kit, R&D Systems, Inc., Minneapolis, MN) according to the instructions of the manufacturer. Samples were tested in duplicate. The intra-assay CV was 2.6%. Jejunal TNFα per unit of mucosa protein was calculated as the concentration of TNFα divided by the concentration of protein for homogenization supernatant of the jejunal mucosa.

Economic Analysis

Milk, labor, and pelleted feed costs were provided in Table 1. Feed cost/pig was calculated as (total milk intake × milk cost + pelleted feed intake × pelleted feed cost + additional labor cost)/pig. Feed cost/kg gain was calculated as feed cost × feed/gain. Revenue per pig was calculated as $65.34 + (BW of pig – 18.18 kg) × $0.30/kg, as described by USDA - Iowa Department of Agriculture Market News (2016). Gross margin/pig was determined by revenue – feed cost/pig. With regard to the mortality effects on economic returns, gross margin/pen was calculated as gross margin/pig × number of pig/pen at the end of nursery period.

Statistical Analysis

Data were analyzed as a randomized complete block design. Pen was the experimental unit. Data were analyzed using MIXED procedure of SAS (SAS Inst. Inc., Cary, NC). Milk supplementation and sex were considered as fixed effects and initial BW was

considered as a random effect. Mortality during the entire nursery period and BW loss during first 4 d postweaning were analyzed with LOGISTIC procedure of SAS. Mean and SEM of mortality and BW loss were provided by using LSMEANS procedure with ILINK option (SAS Inst. Inc., Cary, NC). For morphological parameters, the mean of 15 measurements was used in statistical analysis. Data of fecal scores were analyzed by MIXED procedure with repeat measurement. Time was considered as a random effect. Pooled standard error was used in the data set. Statistical differences between treatment means were considered significant with $P < 0.05$ and considered as trends with $0.05 \leq P < 0.10$.

Results

Growth Performance

Body weight of pigs during the overall nursery period was not different between treatments (Table 2). During phase I, ADG, ADFI of pelleted feed on a DM basis, total ADFI (sum of ADFI of milk and pelleted feed on a DM basis), and G:F were not different between treatments from d 0 to 4 postweaning; ADG, feed intake, and G:F from d 4 to 10 postweaning did not differ between treatments, either. During phase II or phase III, ADG, feed intake, and G:F were not different between treatments. During the combined period of phase I and II (d 4 to 24 postweaning, excluding 4-d milk supplementation), ADG and ADFI did not differ between treatments, whereas The G:F ratio tended to be increased ($P = 0.073$) by milk supplementation. During the entire nursery period (d 4 to 49, excluding 4-d milk supplementation), ADG, ADFI, and G:F were not different between treatments.

Fecal Scores, Body Weight Loss, and Mortality

Milk supplementation decreased ($P < 0.05$) fecal scores from d 2 to 5 postweaning (Table 3). During d 0 to 4 postweaning, percentage of pens with BW loss during d 0 to 4 postweaning did not differ between treatments. The average BW loss of pigs (only including the pigs which lost weight during d 0 to 4 postweaning) was not different between treatments. Milk supplementation during the first 4 d postweaning reduced ($P < 0.05$) the mortality rate from d 0 to 49 postweaning.

Small Intestine Weight and Length

The weight of small intestine in a percentage of BW of pigs did not differ between treatments at either d 4 or 10 postweaning (Table 4). The length of small intestine was not different between treatments. Weight per unit length of small intestine did no differ between treatments at either d 4 or 10 postweaning.

Apparent Ileal Digestibility of DM, CP, Ether Extract, and GE

Apparent ileal digestibility of DM, CP, ether extract and GE did not differ between treatments at d 10 postweaning (Table 5).

Morphology of Villi and Crypts

At d 4 and 10 postweaning, the jejunal villus height, apical villus width, basal villus width, and crypt depth were not different between treatments (Table 6). Villus surface area, Apical villus width:basal villus width ratio, and villus height:crypt depth ratio did not differ between treatments.

Jejunal TNFα

Jejunal TNFα at either d 4 or 10 postweaning did not differ between treatments (Table 7).

Economic Returns

The milk cost and additional labor cost for milk supplementation were $0.004 and $0.045 per pig, respectively, for 4-d milk feeding. Feed cost per pig, which included pelleted feed cost, milk cost, and additional labor cost, was not different between treatments (Table 8). Feed cost per kg weight gain, revenue per pig, gross margin per pig, and gross margin per pen (26 to 27 pigs per pen, the same total number of pigs per treatment initially) were not different between treatments.

Discussion

In this study, pigs in both treatments had relatively low feed intake and slow growth rate during the first 4 d postweaning. After weaning, pigs typically have depressed feed consumption (Leibbrandt et al., 1975). The poor performance observed in the phase I of the nursery period reflected the postweaning check, which is normally observed during the early postweaning period. One of the major cause of the poor performance of pigs was the low feed intake. In this study, the pig weight gain was not enhanced by milk supplementation during the first 4 d postweaning. Lacking of growth could be also due to the low milk intake, which was only 9 g DM per pig per day during the first 4 d postweaning. In addition, the feed intake of pellet feed during phase I (d 4 to 10 postweaning, 145 g) was also not increased by the previous milk supplementation. Like our results, Armstrong and Clawson (1980) also

reported that milk supplementation did not improve the growth performance of pigs during the early nursery period. On the contrary, Zijlstra et al. (1996) reported that pigs (weaned at 18 d of age) fed milk replacer (25.0% protein and 4.2 Mcal/kg of DM) through the nipple feeding system had a 139% greater ADFI, and subsequently a 283% greater ADG from d 18 to 25 postweaning, as compared with nursery pigs fed dry starter diet (20.0% protein and 3.9 Mcal/kg of DM) only. Moreover, McCracken et al. (1995) also indicated that pigs (weaned at 21 d of age) which were fed milk replacer had a higher feed intake than pigs which were only fed corn-soybean meal based diets (0.50 kg vs. 0.13 kg). Pluske et al. (1996) also reported the similar result that pigs (weaned at 29 d of age) fed fresh cow milk had a 39.9% greater ADFI and a 78.5% greater ADG at d 5 postweaning, as compared to nursery pigs fed pelleted starter diets only. Collectively, the different growth performance in regards to the milk feeding could be largely due to the feed intake and milk intake. The milk intake was limited in this study, because the non-saleable milk, which was equal to about 10% of estimated nursery feed intake, was supplemented when pelleted feed was fed *ad libitum* at the same time. Whereas, other publications adopted the *ad libitum* milk or milk replacer feeding method. In addition, different weaning age may also result in the varied effects of milk feeding on growth performance of pigs through influencing the maturity of intestinal system, enzyme activities, and feed intake. Moreover, the feed intake of this study was calculated according to the feed disappearance. The gruel feed (supplemental cow milk added with pellet feed) in PVC troughs of MILK group is more thick and greasy than the pelleted feed in PVC troughs of CON group. Therefore, it appeared that the feed spillage in PVC troughs of

CON was greater than that in MILK group. Because of the condition of the commercial farm, the exact amount of feed spillage was not measured in this study.

The feed efficiency from d 4 to 24 postweaning was enhanced by liquid milk supplementation. The improvement in postweaning feed efficiency could be due to the liquid feeding and additional nutrients from milk. Similarly, Efird et al. (1982) reported that pigs fed diets containing 24% of milk protein in a liquid form had a greater feed efficiency than those fed the same diets in a dry form. The liquid feeding could benefit the postweaning growth performance by promoting feed and water intake, especially for newly weaned pigs, that may have difficulty in identifying nipple drinkers, competing for solid feed in a feeder with limited space, and avoiding being dehydrated and starved (Brook et al., 2001; Kim et al., 2001). Moreover, milk supplementation provided additional protein and energy sources. Previous studies have proved that increasing energy concentration of nursery diets improved the performance and feed efficiency of nursery pigs during nursery periods (van Heugten et al., 1996; Beaulieu et al., 2006). Lactose, whey, and casein in milk is considered as more digestible and palatable carbohydrate or protein sources, as compared with other plant-formed feed ingredients for newly weaned pigs (Wilson and Leibholz, 1981; Lecce et al., 1985). Feeding liquid supplemental milk or fermented milk to postweaning pigs increases diet palatability and acidification, and subsequently may promote growth performance by encouraging the feed intake and reducing enterotoxigenic bacterial proliferation (Dunshea et al., 1999, 2000). In this study, the results showed that the nutrients apparent ileal digestibility were not different between treatments. However, liquid milk supplementation increased the

nutrient digestibility numerically (increased CP digestibility by 6.6%, increased ether extract digestibility by 18.0%, and increased GE digestibility by 14.4%). The lack of sufficient feed intake during the early nursery period may diminish the potential benefit effects of milk supplementation on dietary nutrient concentration and nutrient digestibility. Armstrong and Clawson (1980) also reported increasing nutrients such as energy and protein concentration in dry diet did not improve pig performance immediately after weaning without observing enhancement in feed intake.

Pigs after weaning normally have impaired intestinal integrity and reduced nutrient digestibility as compared with pigs before weaning (Miller et al., 1986; Cera et al., 1988; Pluske et al., 1997). One of the major cause of these negative effects was transition from liquid sow milk to solid complex diets. The lack of energy intake of enterocytes results in reduced villus height or villus atrophy (Kelly et al., 1991; Pluske et al., 1996; Sun et al., 2006). On the contrary, improvement in feed intake after weaning is significantly correlated to the enhancement in small intestinal morphology such as villus height and crypt depth (Pluske et al., 1996). Increased villus height can suggest a greater surface area and an increased capability of nutrient absorption (Caspary, 1992). Crypt depth is positively associated with enterocyte proliferation (Hampson, 1986). Together, the villus height:crypt depth ratio is described as a characteristic of the changes in intestinal morphology and changes in epithelial cell turnover (Fan et al., 1997; Van Der Hulst et al., 1998). Higher villi and shallower crypts can promote nutrient absorption and accelerate growth performance (Xu et al., 2003). Zijlstra et al. (1996) reported that pigs fed milk replacer had 74% longer villi of

proximal small intestine as compared to pigs fed dry starter diets. It is possible that because feed intake and nutrient digestibility was not affected by milk supplementation in the current study, the nutrient intake of enterocytes was also not enhanced by milk supplementation. Therefore, we failed to detect any significant difference in villus height, apical villus width, crypt depth, and villus surface area between treatments. Interestingly, by comparing the gut morphology at d 4 with that at d 10, the villus height and villus surface area of pigs, which were fed diets with supplemental milk. increased by 20.7 and 88.2% from d 4 to 10 postweaning; whereas, the villus height and villus surface area of pigs, which were fed a dry pelleted feed, only increased by 5.4 and 52.6% from d 4 to 10 postweaning, respectively. This suggested that milk supplementation may benefit the gut morphology by enhancing the recovery and growth rate of villi after the postweaning stress. This could be due to an additional supply of lactose in milk. Because lactose can act as a key nutrient for maintaining epithelial integrity and controlling paracellular transport (Spreeuwenberg et al., 2001). Small intestine weight, and weight per unit of length at both d 4 and 10 postweaning were not affected by dietary supplementation of milk. This result is consistent with the growth performance and the gut morphology results. However, Efird et al. (1982) reported that pigs fed diets with milk protein in liquid form had greater intestinal weight and intestinal length per kilogram body weight than pigs fed diets with soy protein in dry form. Above all, feed intake or energy intake is still a predominant factor influencing the mucosal integrity during the first 4 d postweaning. Interestingly, the apical:basal villus width ratio, as a parameter of shrinkage of villus tip, decreased from d 4 to 10 postweaning in both treatments. This

suggested that the shrinkage of villus tip caused by the transition of diets or weaning stresses was slowly reduced by d 10 postweaning.

The intestine becomes susceptible to enteropathogens at weaning (Lecce, 1983). The postweaning morphology and function changes of the intestine at weaning could result in malabsorption of nutrients and diarrhea (Nabuurs, 1998). In this study, pigs after weaning had a relatively high incidence of diarrhea, which was indicated by the results that the fecal scores ranged from 1.84 to 2.04 (3 = liquid diarrheal feces) from d 2 to 5 postweaning. The diarrhea severity was mitigated by liquid milk supplementation. Postweaning diarrhea is associated with the disrupted intestinal barrier function, reduced intestinal secretory activity, and increased intestinal permeability (Boudry et al., 2004; Moeser et al., 2007; Smith et al., 2010). The reduced fecal scores suggest that milk supplementation may mitigate postweaning diarrhea and improve the intestinal barrier function during the early nursery period. The reduced severity of diarrhea could be also possibly because of the effects of milk supplementation on intestinal secretion of fluid and electrolyte and on intestinal permeability (Wijtten et al., 2011; Vente-Spreeuwenberg et al., 2003). Moreover, milk fermentation produces lactic acid, which may further reduce stomach pH and subsequently reduced the population of coliform bacteria (Rateliffe et al., 1986). Another study (Maswaure and Mandisodza, 1995) reported that pigs fed diets with liquid fresh whey had a greater incidence of diarrhea as compared to pigs fed diets without milk products. The possible reason for having a severer diarrhea was associated with the contamination of liquid feeding system by enterotoxigenic bacteria (Lecce, 1986; Dunshea et al., 2000).

Newly weaned pigs need to obtain amino acids and energy from diets to support the morphology and the functions of the small intestinal mucosa. On the other hand, dietary antigens could induce the immune response (Miller et al., 1984). The reduced intestinal integrity is associated with altered T-lymphocyte subsets and increased intestinal inflammation (Spreeuwenberg et al., 2001; Pié et al., 2004). In details, the intestinal permeability and integrity are influenced by the pro-inflammatory cytokines including IL-1β, IL6, and TNF-α (McKay and Baird, 1999). Pié et al. (2004) reported that genes of these pro-inflammatory cytokines were up-expressed during the first wk postweaning. In current study, the TNF-α in jejunum reduced from d 4 to 10 postweaning. However, milk supplementation for 4 d postweaning did not reduce the TNF-α concentration. Because the inadequate feed intake during the immediate postweaning period is considered as one of the major limiting factors contributing to intestinal inflammation (McCracken et al., 1999). The insignificant effects of liquid milk feeding on intestinal inflammation in this study may be associated with the results of feed intake as well. However, some literature (McCracken et al., 1995; Spurlock, 1997) also reported that local acute pro-inflammation imposed by weaning stressors may not be significantly attenuated by the ingredient changes.

Supplemental milk for first 4 d postweaning reduced the mortality of nursery pigs by 2.8%. Survivability of nursery pigs is associated with nutrition and management factors related to health and growth of pigs. Light-weight pigs with low feed intake are difficult to maintain the energy and nutrient intake and subsequent have a higher incidence of mortality (Dewey et al., 2006). As discussed previously, the feed intake was not different between

treatments. Nabuurs (1998) reported that the mortality, caused by diarrhea, is highly connected with villus shortening and crypt deepening. In this study, the light-weight pigs and dead pigs were not selected for gut morphology measurement. But the reduced mortality could be explained by the improvement in light-weight pigs' growth, gut integrity, and health in milk supplementation group. Dewey et al. (2006) also reported that not using high digestible ingredients in start diets such as spray-dried plasma is associated with increased nursery-barn mortality rates.

Because of the simple design of milk feeding system and the relative less expensive cost of milk and labor, the total feed cost including the additional labor cost per pig was not increased by milk supplementation. However, because there was no significant difference in weight gain of pigs, the revenue and gross margin per pig or per pen were not different between treatments.

Collectively, milk supplementation for the first 4 d postweaning did not improve the gut integrity but decreased the severity of diarrhea. Milk supplementation tended to enhance the feed efficiency and decreased the mortality during the phase I and II of nursery period, but did not enhance the pig weight gain and economic returns during the entire nursery period.

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Table 1. Variable input costs

Variable	Cost, $/unit[1]
Milk, kg of DM	0.15
Labor, wage/h	7.25
Pelleted feed	
Phase I, kg	0.66
Phase II, kg	0.42
Phase III, kg	0.28

[1]The variable input costs were provided by the commercial farm, where the experiment was conducted in North Carolina.

Table 2. Growth performance of pigs from d 0 to 49 postweaning

Item	CON[1]	MILK[1]	SEM	*P*-value
Body weight, kg				
d 0	6.43	6.45	0.14	0.910
d 4	6.46	6.52	0.10	0.696
d 10	7.06	7.16	0.12	0.598
d 24	11.55	11.86	0.32	0.507
d 49	26.21	26.33	0.54	0.400
Phase I, d 0 to 10				
d 0 to 4				
ADG, g	9	17	11	0.629
ADFI-liquid, g[2]	--	9	--	--
ADFI-pellet, g[3]	88	81	4	0.268
ADFI-total, g[4]	88	90	4	0.722
G:F	0.032	0.141	0.122	0.544
d 4 to 10				
ADG, g	89	94	8	0.689
ADFI, g	153	145	4	0.181
G:F	0.551	0.638	0.046	0.209
Phase II, d 10 to 24				
ADG, g	315	334	16	0.405
ADFI, g	427	436	18	0.710
G:F	0.736	0.767	0.017	0.236
Phase III, d 24 to 49				
ADG, g	586	579	10	0.535
ADFI, g	838	846	16	0.749
G:F	0.703	0.687	0.008	0.182
Phase I and II, d 4 to 24				
ADG, g	240	254	12	0.402
ADFI, g	337	340	13	0.889
G:F	0.708	0.748	0.014	0.073
Total period, d 4 to 49				
ADG, g	429	431	10	0.902
ADFI, g	610	615	14	0.790
G:F	0.704	0.703	0.006	0.838

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form. In MILK, gruel was provided by mixing milk with a pelleted feed into gruel.

[2] Average daily feed intake of milk on a DM basis from d 0 to 4 postweaning.

[3] Average daily feed intake of pelleted feed on a DM basis from d 0 to 4 postweaning.

Table 2. Continued
[4] Total average daily feed intake including both ADFI of milk and pelleted feed on a DM basis from d 0 to 4 postweaning.

Table 3. Fecal scores, weight loss in first 4 days postweaning, and mortality of pigs

Item	CON[1]	MILK[1]	SEM	*P*-value
Fecal scores[2]	2.04	1.84	0.10	0.065
BW loss, d 0 to 4				
Average BW loss in pens with BW loss, g[3]	27	15	6	0.366
Percentage of pens with BW loss, %[4]	59.0	40.8	14.2	0.395
Mortality, d 0 to 49, %[4]	4.35	1.55	0.69	0.045

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form.

[2] Fecal scores were based on the following scale:

1 = normal, solid feces

2 = soft, looser than normal stools

3 = liquid diarrheal feces

Fecal scores were measured at d 2, 3, 4, and 5 postweaning. Time was considered as a fixed effect. Data were analyzed with Proc MIXED with repeat measurement.

[3] Body weight changes of pigs were calculated only in the pens in which pigs lost weight in average.

[4] Mortality and BW loss was analyzed with LOGISTIC procedure of SAS (SAS Inst. Inc., Cary, NC). Mean and SEM of mortality and BW loss were provided by using LSMEANS procedure with ILINK of SAS.

Table 4. Small intestine weight and length of pigs

Item	CON[1]	MILK[1]	SEM	*P*-value
Total small intestine				
d 4				
Weight, g	216.5	220.6	18.3	0.873
Length, m	8.93	8.90	0.37	0.959
d 10				
Weight, g	351.0	337.4	15.5	0.549
Length, m	10.43	10.29	0.33	0.760
Total small intestine/BW				
d 4				
Weight, g/kg BW	30.77	32.22	2.38	0.664
Length/kg, m/kg BW	1.28	1.31	0.07	0.772
Weight/length, g/m	24.33	24.62	1.31	0.876
d 10				
weight/kg BW	47.58	45.67	2.72	0.624
Length/kg BW	1.43	1.40	0.08	0.799
Weight/length, g/m	33.88	32.82	1.81	0.684

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form.

Table 5. Apparent ileal digestibility of DM, CP, ether extract, and GE of pigs at d 10 postweaning

Item	CON[1]	MILK[1]	SEM	*P*-value
AID				
DM, %	91.67	91.84	0.83	0.869
CP, %	41.58	44.32	4.07	0.673
Ether extract, %	65.86	77.73	6.46	0.322
GE, %	42.76	48.93	4.19	0.375

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form.

Table 6. Morphology of villi and crypts in the jejunum of pigs

Item	CON[1]	MILK[1]	SEM	*P*-value
Jejunum, d 4 postweaning				
Villus height, µm	317	294	27	0.541
Apical villus width, µm	82	88	3	0.170
Basal villus width, µm	94	101	4	0.235
Crypt depth, µm	163	149	7	0.182
Villus surface area, mm^2	0.019	0.017	0.003	0.702
Villus height:crypt depth ratio	2.08	2.12	0.15	0.593
Apical:basal villus width ratio	0.92	0.91	0.03	0.979
Jejunum, d 10 postweaning				
Villus height, µm	334	355	23	0.540
Apical villus width, µm	43	43	2	0.999
Basal villus width, µm	120	121	4	0.831
Crypt depth, µm	177	177	6	0.944
Villus surface area, mm^2	0.029	0.032	0.003	0.444
Villus height:crypt depth ratio	2.04	2.14	0.14	0.627
Apical:basal villus width ratio	0.77	0.77	0.02	0.851

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form.

Table 7. Jejunal tumor necrosis factor α (TNFα) of pigs

Item	CON[1]	MILK[1]	SEM	*P*-value
Jejunal TNFα, ng/g protein				
d 4	2.07	2.24	0.25	0.641
d 10	1.18	1.38	0.12	0.255

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form.

Table 8. Economic returns of pigs

Item	CON[1]	MILK[1]	SEM	*P*-value
Feed cost/pig, $	9.34	9.43	0.224	0.789
Milk cost/pig, $	--	0.004	--	--
Pelleted feed cost/pig, $[2]	9.34	9.38	0.22	0.906
Additional labor cost/pig, $	--	0.045	--	--
Feed cost/kg gain, $[3]	0.47	0.47	0.004	0.803
Revenue/pig, $[4]	70.64	70.72	0.36	0.882
Gross margin/pig, $[5]	61.30	61.29	0.15	0.967
Gross margin/pen, $[6]	1,462.38	1,496.26	21.77	0.297

[1] From d 0 to 4 postweaning, pigs in CON were fed pelleted feed; pigs in MILK were fed the pelleted feed with milk supplementation in a gruel form.
[2] Feed cost/pig = milk cost/pig + pelleted feed cost/pig + additional labor cost/pig
[3] Feed cost/kg gain = (feed cost/pig)/weight gain.
[4] Revenue per pig = $65.34 + (BW of pig – 18.18 kg) × $0.30/kg, as described by USDA-Iowa Department of Agriculture Market News (2016).
[5] Gross margin per pig = revenue/pig –feed cost/pig.
[6] Each pen had 26 or 27 pigs in total. The Control and Treatment has the same number of pigs (n = 322).

CHAPTER 6

GENERAL CONCLUSIONS

Increasing litter size may increase the milk production and body weight loss of sows during lactation. Elongating lactation length may or may not affect the body weight loss of sows during lactation. The first study concluded that increasing litter size and elongating lactation length in parity 1 did not affect the milk production in parity 2. In addition, sows with increased suckling intensity had decreased body tissue mobilization during the lactation of parity 2. The results of the first study also imply that even though increasing suckling intensity in parity 1 increased sow body weight loss, these sows could maintain the body condition and milk production with a slower breakdown of body tissue during the lactation of parity 2.

In addition, the milk production of individual mammary glands varied according to anatomical location. Piglets, which suckled mammary glands at different anatomical locations, had different weight gain. The second study showed that the first 5 pairs of anterior and middle glands were superior to the last 3 pairs of posterior glands in milk production in parity 1. However, the first 2 pairs of anterior glands were superior to other glands in milk production in parity 2. Consistently, anterior or middle glands were suckled in a greater percentage as compared to posterior glands during both parities. However, suckling or not suckling of individual mammary glands in parity 1 did not affect the mammary gland productivities in parity 2. The conclusion of the second study suggested that the suckling history of individual mammary glands might not be the limiting factor to piglet weight gain in a subsequent lactation.

Lactating sows may be under a catabolic condition for maintaining milk production. The sow health is important, in regards to sow longevity, culling rate, and animal welfare. Similar to other lactating animals, lactating sows have a massive and quick Ca excretion into colostrum and milk. The third study concluded that decreasing DCAD during late gestation and lactation can improve the Ca homeostasis by increasing the serum and colostrum Ca concentration. In addition, decreasing DCAD induced a mild metabolic acidosis with a significantly reduced urine pH. Therefore, the effects of DCAD on reducing urinary tract infections of sows require further investigation. The third study implies that use of anionic salt supplementation with reduced the cation-anion difference of late gestation and lactation diets can effectively improve the capability of mobilizing Ca during lactation.

The performance of baby pigs relies on milk intake, whereas the performance of pigs after weaning is largely affected by feed intake during this period and how well the nursery pigs adapt to the dry corn-soybean based diets. The fourth study showed that milk supplementation for 4 d postweaning reduced severity of postweaning diarrhea during the early nursery period. Milk supplementation did not affect the feed intake, growth performance, and economic returns per pig, although mortality was reduced during the entire nursery period. The conclusion of the fourth study implies that even short-time milk supplementation did not enhance the performance but reduced the mortality of nursery pigs, when the nutrient intake was not enhanced. In addition, liquid feeding in pig farms may raise sanitation and economic concerns. Proper hygiene of milk feeding systems and milk is crucial to avoid contamination and to reduce the incidence of diarrhea caused by

enterotoxigenic bacteria. The effects of milk pasteurization on growth performance and economic returns of nursery pigs need further investigation. Access to cheaper, local non-salable milk and usage of simpler designed milk feeding trough is possible to diminish the feed and labor costs.

Overall, litter management through increasing litter size and lactation length of the first-parity sows did not negatively affect the litter and sow performance in the lactation of parity 2. Nutritional management through reducing DCAD during late gestation and lactation could improve the health of lactating sows, which was indicated by an increased ability to mobilize Ca. For nursery pigs, nutritional management through supplementing milk for a short-time could increase feed efficiency, and reduce mortality, although it could not increase the growth performance of nursery pigs.