EFFECTS OF SOW GUT MODIFYING FEED ADDITIVES ON REPRODUCTIVE CHARACTERISTICS AND PROGENY GROWTH PERFORMANCE

by

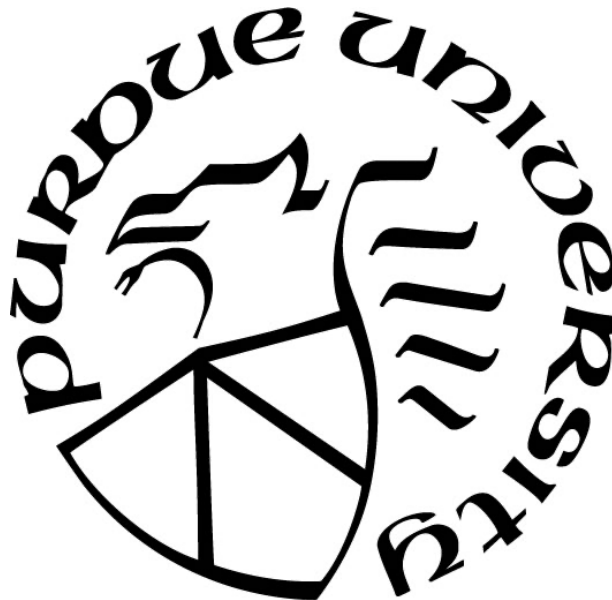**Morgan Taylor Thayer**

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To my husband, Ryan, for your never-ending love and support, and to my parents for always encouraging me to work hard and dream big.

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ABSTRACT

Providing wholesome pork products to consumers involves raising healthy pigs to grow well and be feed efficient from birth to market. Raising these pigs starts with ensuring the sow is healthy and provided good nutrition in gestation and lactation. Therefore, this dissertation primarily focuses on research of gut modifying feed additives fed to sows in gestation and lactation (and to their progeny in Chapter 3) to enhance reproductive performance and litter growth to weaning (and in the nursery). In Chapter 2, a total of 606 sows and their progeny were used to determine if feeding gestating and lactating sows a proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast product (WCY; CitriStim, ADM Animal Nutrition, Quincy, IL) improves sow and litter performance in a commercial production system. Sows were fed a control (CON) diet or control diet fortified with 0.15% of the WCY from d 35 of gestation through lactation. Sows supplemented with WCY in gestation and lactation had a greater number of total born piglets by 0.45 pigs ($P < 0.04$), piglets born alive ($P < 0.04$), heavier born alive litter weights ($P < 0.001$), and greater post cross-foster litter size ($P < 0.001$) compared to CON fed sows. Litter size at weaning was increased by 0.54 pigs when sows were fed WCY compared to CON ($P < 0.001$). However, litter weaning weights and 21-day adjusted litter weaning weights were similar ($P > 0.158$) with the 21-day adjusted litter weaning weights being numerically greater for the WCY sows. The average piglet weaning weights from CON fed sows were heavier by 0.35 kg compared to WCY ($P < 0.001$). This increase in body weight of piglets from CON fed sows is partially explained by their 0.93 days longer lactation ($P < 0.001$) and may also be due to the smaller litter size nursed throughout lactation. The percent of litters treated for scours decreased from 38.3% to

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14.2% when sows were fed WCY ($P < 0.001$). The distribution of birth and weaning weights was not different ($P > 0.2461$) between treatments.

Chapter 3 encompasses a sow experiment where progeny were followed onto the nursery for a 28-d study. Forty-seven sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or in combination improves sow lactation feed and water intake, litter growth, and subsequent reproductive performance. At weaning, offspring were fed a positive control diet (PC), negative control diet (NC), or a diet representative of their dam's treatment to determine if there is an additive benefit to also feeding DFM and/or OA to nursery pigs in addition to their dams. On approximately d 80 of gestation, sows were fed one of four diets in a 2×2 factorial design: 1) gestation control (CON), 2) CON with DFM (1.6×10^9 CFU/kg of complete feed), 3) CON with 0.4% OA, 4) CON with both DFM and OA. Dietary treatments were also fed throughout lactation. Sows fed the OA diets had fewer mummies per litter ($P < 0.010$) compared to diets not containing OA. Sows fed diets with the DFM gave birth to lighter pigs born alive (1.5 vs. 1.7 kg; $P < 0.003$) compared to non-DFM fed sows, and a tendency for an interaction ($P < 0.092$) existed where feeding DFM+OA lessened the decrease in born alive BW. There was a tendency ($P < 0.093$) for pigs from DFM fed sows to also be lighter at weaning (5.8 vs. 6.2 kg) compared to pigs from sows not fed DFM, with no differences in litter sizes at weaning ($P < 0.815$). There was a tendency ($P < 0.079$) for the DFM to decrease the amount of sow BW loss in lactation compared to sows not fed the DFM (approximately 6 vs. 8% BW loss, respectively). The maintained BW in lactation was likely related to DFM sows numerically ($P < 0.124$) consuming 8.4% more feed during d 7-14 of lactation and 6.4% more feed ($P < 0.234$) from d 1 of lactation

to weaning. The interaction was approaching a trend ($P < 0.133$) where sows fed DFM returned to estrus 1.0 day sooner than CON, but only 0.4 days sooner when sows were fed the DFM+OA diet.

Progeny weaned from these sows ($n = 384$, Initial BW = 6.15 kg) were blocked by initial BW and sex and allotted (6 pigs/pen, 8 pens/treatment) to one of 8 nursery treatments. Pigs from CON sows were fed a negative (NC; no antibiotics, no pharmacological Zn or Cu) or positive (PC; neomycin-oxytetracycline in phases 1 and 2 (827 and 551 ppm) and carbadox in phases 3 and 4 (55 ppm)) control diet. Pigs from sows fed DFM, OA, or DFM+OA were fed the NC diet or a diet representative of their dam's treatment. Diets with DFM contained 1.6×10^9 CFU/kg of complete feed and diets with OA contained 0.5, 0.4, 0.3, and 0.0% OA in phases 1-4, respectively. Weaning weight was used as a covariate for nursery performance due to the DFM offspring being significantly lighter at weaning. For all phases and overall, PC fed pigs had greater ADG ($P < 0.003$) and ADFI ($P < 0.059$) than NC pigs. PC fed pigs had greater G:F ($P < 0.010$) than NC pigs for all phases and overall except d 21-28 ($P < 0.532$). Feeding DFM or OA in sow diets improved (interaction; $P < 0.049$) nursery pig G:F, but DFM+OA offspring had similar G:F compared to NC pigs from CON fed sows for d 7-14, 0-14, 0-21, and 0-28. Feeding DFM or OA to sows and their progeny decreased ADFI (interaction; $P < 0.042$) but improved G:F (interaction; $P < 0.028$) for d 7-14 and 0-14 with DFM+OA having similar performance to NC. For d 14-21 and 0-21, feeding DFM or OA to sows and their progeny decreased ADFI whereas DFM+OA increased ADFI above NC (interaction; $P < 0.019$). Overall, d 0-28, feeding DFM or OA to sows and their progeny improved G:F (interaction; $P < 0.001$) with DFM+OA having poorer G:F compared to NC. When the DFM was fed to sows and nursery pigs, progeny harvested on d 6 post-weaning had a decreased ratio of villus height to crypt depth ($P < 0.035$) compared to sows and pigs not consuming the DFM (average 1.34 vs. 1.67). Comparing pigs fed PC vs. NC from CON fed sows, expression of

interleukin 10 (IL-10) was greater (0.51-fold increase; $P < 0.046$) for NC pigs than PC pigs. Expression of occludin (OCLN) was lower ($P < 0.010$) when OA was fed to the sows and pigs compared to when OA was not fed to the sows and pigs (0.78 vs. 1.00, respectively).

Chapter 4 is the only chapter that does not include maternal nutrition. In this chapter, maternal line gilts (Topigs Norsvin TN70) were bred with frozen semen from Duroc boars born from 2000 to 2017 divided into two genetic groups: semen from boars born in 2000 to 2005 and 2011 to 2017. These genetic groups had vastly different terminal sire indexes (TSI) of 88.2 and 112.0 for 2000 to 2005 and 2011 to 2017, respectively. A total of 155 pigs were weaned into 44 pens in a wean-to-finish facility to determine if genetics from two decades of sires and sex of the progeny impact progeny growth performance and carcass characteristics. The expected large growth performance differences indicated by the TSI's of the two genetic groups were not observed. However, barrows had greater feed intake ($P < 0.031$) and fatter carcasses ($P < 0.004$) than the more feed efficient ($P < 0.006$) and leaner ($P < 0.015$) gilts in this study. Modern swine genetics have been selected to be leaner and results from this study agree, although the differences in live scan and carcass measurements were not as large as expected. The lack of differences between genetic groups could possibly be due to environmental differences including nutrition and rearing conditions from when these sires were alive compared to what was experienced by these progeny.

In conclusion, feeding gestating and lactating sows a proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast product increased the number of piglets born and weaned as well as decreased the prevalence of scours during lactation. Feeding a *Bacillus licheniformis* DFM to sows may decrease pig born alive weight and subsequent weaning weight but reduce sow BW loss through 6.4% more lactation feed intake, quickening the return to estrus.

Other than decreasing the number of mummies per litter, feeding the OA alone or in combination did not improve sow reproductive or litter growth performance in this study. Feeding DFM or OA to sows or their offspring may improve nursery feed efficiency but did not result in a difference in ADG or final BW in this study. Feeding the combination diet (DFM+OA) to the sow and nursery pigs tended to increase ADFI. Feeding antibiotics post-weaning continued to improve pig growth performance resulting in 2.7 kg heavier pigs at the end of the 28-d nursery period. Lastly, the expected large growth performance differences indicated by the TSI's of two genetic groups created by using frozen semen from boars born in 2000 to 2005 and 2011 to 2017 were not observed.

CHAPTER 1.   LITERATURE REVIEW

In recent decades, genetic selection, advanced nutrition, and improved reproductive technologies have successfully increased litter sizes of the modern sow to 12-15+ piglets born alive. Although more pigs are being born, the increased litter sizes have resulted in increased pre-weaning mortality rates of 10-20% (PigCHAMP, 2020). The swine industry today is also seeking to optimize antibiotic use in order to meet changing regulatory standards and address concerns from the general public. Effective January 2017, antibiotics used for growth promotion in swine has been eliminated in the U.S. (GFI #213; U.S. Food and Drug Administration, 2020). Therefore, researchers are motivated to find other feed additives that promote not only growth, but additionally gut health and immunity. This dissertation highlights nutritional strategies to address these grand challenges through yeast, direct fed microbial, and organic acid technologies fed to the sow and/or her piglets.

1.1   Porcine Fetal Development

1.1.1   Fertilization to Day 35 of Gestation

A mature gilt or sow mates with a boar during her 24 to 72-hour period of estrus and estrus in the female pig occurs every 21 days. Ovulation, the release of ova from follicles of the ovary, is characterized by a pre-ovulatory surge of luteinizing hormone (LH) and occurs about 44 hours after the onset of estrus (Bazer and Johnson, 2014). Normal ova development by the female, normal sperm development by the male, and proper timing of insemination are all necessary for successful fertilization of the ova, which is thought to occur at greater than 90% in pigs (Anderson, 2009). Once successful fertilization occurs, the maintenance of a functional corpus luteum (CL) and its production of progesterone is required for establishment and maintenance of pregnancy

(Bazer and First, 1983). Progesterone is required to activate proper secretory functions of the endometrial uterine glands that provide a multitude of proteins essential for embryonic development, implantation, and placentation (Bazer et al., 2010).

The successfully fertilized ova spend a short time near the ampullary-isthmic junction (AIJ) of the oviduct where they begin mitotically dividing and are classified as an embryo. The embryos will migrate from the AIJ toward the uterus and enter the uterus 48 to 56 hours after ovulation (Bazer and First, 1983). When the embryo migrates into the uterus it is about at the four-cell stage of embryo development. It reaches the blastocyst stage by about d 5 and it sheds the zona pellucida, called hatching, between d 6 and 7 of gestation (Bazer and First, 1983; Bazer et al., 2009; Figure 1.1). The pig embryo transforms from spherical to tubular and filamentous form by elongating from a size of 9 to 10 mm long to 100 to 200 mm long by d 12 of gestation (Bazer and First, 1983; Bazer et al., 2010). At this time, the embryo produces estrogen, which serves as the maternal recognition of pregnancy for the sow to maintain pregnancy and progesterone production (Bazer et al., 2010). The filamentous conceptus continues to grow to about 800 to 1000 mm long and is coiled up like a ball of thread by d 16 of pregnancy (Bazer and Johnson, 2014).
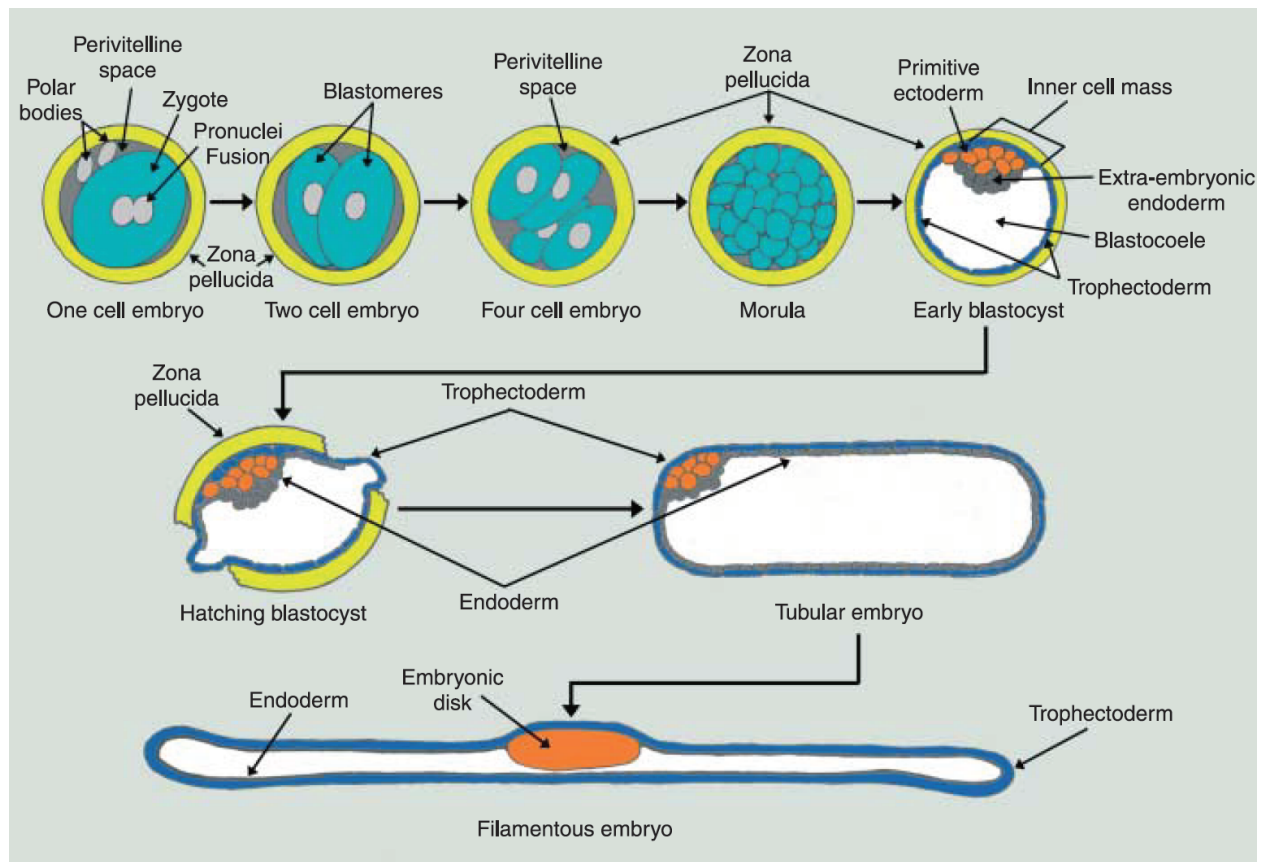
Figure 1.1 Early pregnancy events (Bazer et al., 2009).

The developing conceptus (the embryo and its associated extra-embryonic membranes) migrates throughout the uterine lumen to become evenly spaced within the two uterine horns of the sow at d 11 to 12 and conceptus attachment and the initial wave of uterine angiogenesis occurs at about d 13 to 19 of gestation (Keys et al., 1986; Dyck and Ruvinsky, 2011; Ziecik et al., 2011). The non-invasive "central-type" implantation involves the attachment of the conceptus trophectoderm, the most outer layer of embryonic cells, to the sow's uterine luminal epithelium (Bazer et al., 2010; Bazer and Johnson, 2014). Furthermore, the chorion is derived from the conceptus trophectoderm and it is the chorion that interacts with the maternal uterus which gives rise to the epitheliochorial placental structure in the pig (Bazer et al., 2010). Bazer and Johnson

(2014) estimate that 30 to 40% of conceptuses die before successful implantation, potentially due to elongation failure or inadequate contact of trophectoderm with the luminal epithelium.

The placenta serves as the exchange point for gasses, nutrients, hormones, and other regulatory molecules to be transported from the uterine luminal epithelium to the conceptus. Non-invasive implantation initiates placentation in the pig which is characterized by extensive remodeling of cells to reduce the diffusion distance between maternal and fetal blood (Bazer and Johnson, 2014). Because the uterine luminal epithelium and conceptus trophectoderm do not physically fuse together, many pockets between them form known as the areolae (Friess et al., 1980). The lumen of an areolae is filled with histotroph from uterine glands that is then transported into placental capillaries leading to fetal-placental tissues (Bazer and Johnson, 2014). These areolae form by d 30 of pregnancy (Friess et al., 1980).

Sexual determination of the conceptus begins at conception when the embryo inherited either XX or XY chromosomes. Although the primordial germ cells that migrate and colonize the gonadal genital ridge are neither male or female at that stage, female (XX) primordial germ cells are predetermined to develop into oogonia (germ cells that transform into oocytes; Sarraj and Drummond, 2012). This predetermination is due to the absence of the sex-determining region on the Y chromosome known as the *SRY* gene that would initiate development of the testes rather than ovaries from the bipotential fetal gonad (Koopman et al., 1991). By d 35 of gestation, the bipotential fetal gonad is differentiated to an ovary and is accompanied by the appearance of egg nests (Anderson, 2009).

1.1.2   Day 35 to Day 80 of Gestation

Due primarily to genetic selection, ovulation rates have increased from approximately 13 ova in 1984 to upwards of 20 ova in 2011 of parity 1 females (Kemp et al., 2018). Patterson et al.

(2008) observed an ovulation rate of 23 to 24 ova in multiparous sows. Interestingly, many of these embryos are surviving the pre-implantation period (to approximately d 18). Uterine capacity on d 25 to 30 is exceeded by the number of embryos surviving. As a result, prenatal loss is greater during the post-implantation period (Foxcroft et al., 2006). The overcrowding of the sow's uterus by these increased numbers of embryos may impair placental development and therefore embryo and fetal development of surviving offspring (Kemp et al., 2018). As the next few days progress, an embryo becomes a fetus at about d 35 of gestation, marked by when organs are at their earliest recognizable stage of development (McGeady et al., 2006).

The placenta continues to grow and develop at a rapid rate from approximately d 20 until d 60 to 70 of gestation when placental growth rate slows thereafter (Dyck and Ruvinsky, 2011; Bazer and Johnson, 2014). The number of areolae formed in the placenta are maximized by d 70 of pregnancy when the placenta of each piglet has approximately 2500 areolae associated with it (Knight et al., 1977).

The piglet's primary muscle fibers form from d 25 to 50 of gestation and secondary muscle fibers form from d 50 to 90 of gestation (Zhang et al., 2019). The esophagus, stomach and intestine form at about d 18 of gestation and the mass of the pig GI tract increases more than 170-fold from d 45 to 110 of gestation (McPherson et al., 2004). While the ovaries are differentiated at d 28, the clitoris is formed by d 44 and oviducts, uterus, and anterior vagina are formed from the mesonephros ducts by d 51 of gestation (Pond et al., 1991). The humerus, femur, and ribs of the fetal pig are ossified by d 35 of gestation (Pond et al., 1991).

1.1.3   Day 80 of Gestation to Parturition

Although the rate of placental growth decreases after d 70 of gestation, fetal growth occurs rapidly between d 70 of gestation and parturition (Bazer and Johnson, 2014). Wilson et al. (1998)

observed that conceptuses continued to still increase placental size in attempts to keep up with increasing nutrient demands of the fetus between d 90 of gestation and parturition. During the last 2 to 4 weeks of gestation, the fetus begins retaining glycogen in skeletal muscle and liver in preparation for thermoregulation after parturition and energy needed to suckle and consume colostrum (Theil, 2015).

Parturition (d 114 to 117 of gestation) is initiated by the fetus and the maturation of the fetal hypothalamic-pituitary-adrenal axis. Fetal adrenocorticotropic hormone (ACTH) is released in response to uterine crowding and ACTH stimulates secretion of cortisol. Cortisol promotes synthesis of enzymes which convert progesterone to estradiol and aids in the removal of the pregnancy "progesterone block". Progesterone decreases and estradiol increases promoting the start of parturition (Bazer and First, 1983; Senger, 2012; Bazer and Johnson, 2014).

At birth, the gilt's ovaries are developed, and the non-renewable pool of oocytes remain in the diplotene phase of meiosis until the gilt reaches puberty (Borum, 1967; Anderson, 2009). The gilt's rudimentary uterus is present at birth; however, significant postnatal development occurs in the uterus. The uterine wall isn't considered functionally mature with essential endometrial glands until the gilt is about 120 days of age (Bartol et al., 1993; Cooke et al., 2013). Therefore, reproductive tract development of gilts begins during their fetal development, when the mother's uterine environment can have major impacts on the developing fetuses and is completed in the early neonatal periods of life (Bazer, 1975; Bartol et al., 1993).

1.2 Factors Influencing Number Born Alive

The maximum number of pigs born alive is determined by the female's ovulation rate, the capacity of the uterus to support developing fetuses, and survival of piglets through the farrowing process. The ovulation rate is the number of follicles that ovulated and released ova for the chance

to be fertilized, leaving the respective number of corpora lutea on the ovaries (Bazer and First, 1983). Most data have shown that fertilization rates are approximately 97% in pigs, however, fewer pigs are born than the number of fertilized oocytes due to embryonic and fetal mortality (Bazer and First, 1983). Ford et al. (2002) attribute embryonic and fetal mortality to limitations of uterine capacity that begin to impact conceptus survival after d 30 of gestation. These authors address the surface area of placental exchange of nutrients and waste being a limitation and cause of conceptus loss. Also, the number of uterine glands is determined during early adenogenesis which impacts the amount of histotroph secreted and therefore the number of fetuses a uterus can support (Cooke et al., 2013). Lastly, during the farrowing process piglets can die due to asphyxiation from dystocia and are called stillborn piglets.

An increased number of stillborn piglets results in a decreased proportion of piglets born alive in the litter. Naturally, as litter size increases, one would think it takes more time for the sow to farrow all of her piglets. However, Theil (2015) did not observe an association between farrowing duration and the number of total piglets born. A relationship exists between farrowing duration and the number of stillborn piglets, as the time between the first and last pig increases, the number of stillborn pigs increases (1.5 stillborns when longer than 300 minutes vs. 0.4 stillborns when less than 300 minutes; Oliviero et al., 2010). However, it is unclear if the longer farrowing duration increases the number of stillborn pigs or if the stillborn piglets are causing the increased time to farrow the litter (Theil, 2015). Oliviero et al. (2010) also observed a significant correlation between backfat and farrowing duration where sows with more than 17 mm of backfat prior to farrowing had a longer farrowing duration than sows with less than 17 mm of backfat (230 vs. 385 minutes).

Litter size in swine production is an important component in evaluating reproductive efficiency. Litter size can be defined as the total number of pigs born or the number of pigs born alive. Specifically, the number of pigs born alive is the total pigs born minus the stillborn piglets. Stillborn piglets are those that are alive at the start of farrowing but die intrapartum (Koketsu et al., 2017). Mummies are a separate category because they did not have the opportunity to be born alive due to dying before the pregnancy term was complete. To determine approximately when the fetus died during development, the crown-rump length of the mummified fetus can be measured. Jang et al. (2014) measured crown-rump length of fetuses located in the middle of the uterine horn. Fetuses measured 6.11, 18.50, 23.19, and 30.61 cm long on d 43, 73, 91, and 108 of gestation, respectively.

Although genetic selection for increased ovulation rate has been a successful strategy to increase litter size, it is important to recognize that there are also many challenges with larger litters. These challenges include a longer farrowing duration, increased variation in birth weights, increased percentage of light birth weight pigs, and increased competition for colostrum, resulting in negative impacts on survival of these piglets to weaning as well as growth and survival to market (Schinckel et al., 2010; Islas-Fabila et al., 2018; Kemp et al., 2018; Oliviero et al., 2019; Vandelannoote et al., 2020).

1.2.1   Parity Effects

1.2.1.1   Gilt

Raising females to be productive and healthy for many parities starts with the management of replacement gilts. An optimal nutrition program should target gilts to be mated for the first time when they reach 135 kg at about 200 days of age (Williams et al., 2005; NSNG, 2010). Ovulation

rates of gilts increase during the second and third estrous cycle compared to their first cycle. Therefore, gilts should not be mated on their pubertal estrus. When van Wettere et al. (2006) compared mating gilts during their 1st or 2nd estrus cycles, ovulation rate increased from 14.9 to 15.5, the number of embryos increased from 11.3 to 12.3 and embryo survival increased from 76.5 to 79.8% at 20 days post-mating.

Also, the strategy of flushing is an option where feeding gilts a high energy diet or higher feed intake of the same diet 11 to 14 days prior to breeding increases their ovulation rate (NSNG, 2010). Mallmann et al. (2020) investigated the effects of flush feeding on modern replacement gilts and found that gilts fed 3.6 kg/d before the second estrus had 1.6 more medium to large sized follicles compared to gilts fed 2.1 kg/d (14.3 vs. 15.9 follicles). These feeding levels represented 1.7 and 2.8 times the maintenance energy requirements of these gilts at the start of the experiment. Mallmann et al. (2020) sacrificed gilts at approximately d 30 of gestation and therefore were unable to correlate differences in the number of follicles to litter sizes at birth.

After conception, gilts must continue to grow and prioritize nutrients toward their own growth and development (e.g. mammary growth) as well as fetal growth (Theil et al., 2012). Gilts must enter the farrowing house at an optimal body condition (e.g. BCS 3) in order to maximize feed intake and milk yield for their growing litter (NSNG, 2010). Williams et al. (2005) suggest that gilts mated at 135 kg body weight should achieve proper body mass by weighing greater than 180 kg at their first farrowing.

1.2.1.2   Sow – Parity 1 to 5

After gilts give birth to their first litter, they are considered parity 1 sows. First parity sows tend to have smaller litters of lighter birth weights than older parity sows. Hoving et al. (2010) observed that parity 1 sows had 10.3 pigs born alive compared to 11.2 pigs born alive for parity 2

sows. This may be due to parity 1 sows having lower ovulation rates as well as a smaller uterine capacity which limits the number of developing fetuses (Sell-Kubiak et al., 2019). Interestingly, a parity 1 sow with a large number of pigs born alive will very likely produce a large number of pigs born alive throughout all subsequent parities (Koketsu et al., 2017).

It is important to note that some sows undergo what is called the "second litter syndrome" (Schenkel et al., 2010). This is where first parity sows are unable to consume enough feed to meet the energy requirements for lactation and have to mobilize a large amount of fat and protein mass in order to provide enough milk to the piglets. Consequently, the extreme nutrient mobilization leads to a reduced ovulation rate and lower embryo survival rate leading to lower pregnancy rates and reduced litter sizes of sows having their second litter (Kemp et al., 2018).

With approximately 11.5 vs. 10.5 pigs born alive, sows in parities 2 through 5 had larger litter sizes than parity 1 and parity 8 sows (Koketsu and Dial, 1997). Although litter sizes have increased with genetic selection, this general trend of parity 3 to 5 sows being the most productive (approximately 14.2 total born piglets) is still true today (Sell-Kubiak et al., 2019).

1.2.1.3 Geriatric – Parity 6+

Sows that are parity 6 and older typically have smaller litter sizes than parities 3 to 5 (Koketsu and Dial, 1997). Sell-Kubiak et al. (2019) estimated parity 3 to 5 sows to have about 14.2 total born piglets and parity 6, 7, 8, 9 and 10 sows to have 14.0, 13.7, 13.3, 12.9, and 12.7 total born piglets, respectively. Once a geriatric sow produces smaller litter sizes than parity 1 sows within the same production system, they should be culled and replaced. This lower reproductive performance of older parity sows can be attributed to decreased ovulation and fertilization rates in aged sows and they tend to have a higher rate of embryonic mortality due to slower responses to fetal demands for uterine space (Koketsu et al., 2017). Geriatric sows also have more stillborn

piglets due to slower responses to stimuli during the parturition process (Koketsu et al., 2017). Parity 5 or higher females also have a higher risk of abortion (Iida et al., 2016).

In addition to decreased litter sizes past parity 5, sow body weight increases as they age and have more litters. In practice, geriatric sows who have farrowed 6+ litters get fed the same amount of feed in gestation as a parity 3 sow once body condition has been restored after lactation (Boyd et al., 2008). These authors believe this constant feeding procedure is acceptable for energy and protein needs of the sow, but the geriatric sows that also weigh more require increased micronutrients (equivalent on a per kg body weight basis) to help support tissue metabolism and combat the age-related decline in litter size. Boyd et al. (2008) fed geriatric control sows the standard 0.15% vitamins and trace minerals (VTM) and fed geriatric test sows additional VTM, choline, and chromium in order for a parity 6 sow to achieve a similar grams VTM/kg body weight to a parity 3 sow. The additional VTM in the diet resulted in an increase of 0.60 pigs weaned per litter and 1.44 more pigs weaned per sow per year for the geriatric sow (Boyd et al., 2008). This indicates that the decline in older sows is also related to improper nutrition and not only reproductive system functionality.

1.2.2   Nutrition

After females have achieved enough growth and development for optimal productivity and longevity, it is important to then consider how individual nutritional modifications may impact litter size. As noted previously, the practice of flushing may increase ovulation rate of gilts (NSNG, 2010). Also, immediately after mating, it may be necessary to reduce feed intake back down to normal gestation levels to prevent a higher incidence of embryonic mortality due to higher energy intake and decreased plasma progesterone (Jindal et al., 1997). Interestingly, Leal et al. (2019) conducted a systematic review of immediate post-mating feed allowance on embryonic survival,

and the authors concluded that feed restriction immediately post-mating is no longer relevant with modern prolific dam lines used today.

Sows typically are unable to consume enough feed in lactation to meet their litter's milk demand. Once the sow becomes catabolic, she uses her own body reserves to put fat, protein, and minerals into her milk (Clowes et al., 2003; Schenkel et al., 2010). In primiparous sows with "second litter syndrome", lower feed intake and mobilization of body stores in lactation results in a reduced ovulation rate and lower embryo survival leading to lower pregnancy rates and reduced litter sizes of sows having their second litter (Schenkel et al., 2010; Kemp et al., 2018). The lower embryo survival rate may be due to reduced follicle size and developmental capacity of the oocytes (Kemp et al., 2018).

Interestingly, during a 21-day lactation period where primiparous sows were subjected to restricted feed intake during the last week of lactation, they lost 13% of protein and 17% of fat mass (d 0-21; predicted from equations including body weight and backfat depth). However, wean-to-estrus interval and ovulation rate of the subsequent cycle for feed restricted sows were not different compared to ad libitum fed sows. Embryonic survival of the feed restricted sows was reduced on d 30 of gestation (Vinsky et al., 2006).

Primiparous sows with low dietary protein intake in the entire lactation period lost 16% of their body protein mass (d 0-23; predicted from equations including body weight and backfat depth). By d 20 of lactation, milk protein concentration was also decreased resulting in poorer piglet growth rate. At weaning (d 23), sows that lost 16% of their body protein mass had fewer medium size follicles with less follicular fluid contained in the follicles (Clowes et al., 2003). Therefore, restricted feed intake early in lactation may reduce embryonic survival and increase

protein mass loss of sows which may have adverse effects on follicular development creating potential negative impacts on the subsequent reproductive performance.

Feeding sows dietary fiber prior to farrowing may alleviate constipation and decrease farrowing duration (Oliviero et al., 2010). Dietary fiber may also prolong digestion and absorption of energy compared to easier digestible feedstuffs (e.g. starch) providing a more consistent glucose status for sows at the time of farrowing (Serena et al., 2009). Gourley et al. (2020) attempted to feed sows multiple small meals before farrowing to provide energy closer to parturition however, authors observed no difference in farrowing duration or stillborn rate. In contrast, Feyera et al. (2018) observed a strong positive correlation ($r = 0.76$; $P < 0.001$) between farrowing duration and time since last meal until the onset of farrowing when analyzing a dataset retrospectively. These authors conclude that many sows suffer from a low-energy status at the time of farrowing and this had negative impacts on the farrowing process including longer farrowing duration and more stillborns.

1.2.3 Genetics

Johnson et al. (1999) conducted genetic selection for 14 generations to increase litter size using ovulation rate and embryonal survival in their selection index. Their estimate of heritability for number born alive per litter was 0.17. They observed responses at generation 11 including 7.4 more ova, 3.8 more fetuses present at d 50 of gestation, 2.3 more fully formed pigs, and 1.1 more live pigs at birth due to genetic selection. However, the embryonal survival rate decreased with increased ovulation rate. Also, they observed that ovulation rate and number of fetuses had a positive genetic correlation with the number of stillborns per litter. Therefore, Johnson et al. (1999) suggested that genetic improvement programs should select on pigs born alive because they

observed undesirable genetic relationships between ovulation rate and the numbers of stillborn and mummified pigs.

Holm et al. (2005) analyzed data on a nucleus population of Norwegian Landrace pigs from 1990 to 2000 from the Norwegian national recording scheme. Authors calculated the average heritability to be 0.12 for the number of pigs born alive in the female's first litter and 0.14 for the number of pigs born alive in the females second litter. They also estimated the highest genetic correlation to be between these two traits ($r_g = 0.95$). Results from Holm et al. (2005) illustrated that selection for increased number born alive will slightly increase the female's age at first service and decrease the probability of a return to estrus after weaning.

Chen et al. (2003) observed annual genetic gains of 0.018 piglets per litter per year when analyzing multiple breeds of pigs including U.S. Yorkshire, Duroc, Hampshire, and Landrace pigs from 1984 to 1999. More recently, Camargo et al. (2020) analyzed data on Landrace pigs from 2009 to 2016 raised in southern Brazil. These authors estimated the heritability of the number of pigs born alive to be 0.09 ± 0.04 using a multitrait model for litter traits at birth. Although the heritability estimate was lower than others, this is due to it being a multitrait model. The genetic trend from 2009 to 2016 was positive with annual genetic gains of 0.006 piglets per litter per year (Camargo et al., 2020).

1.2.4   Disease Pressure and Herd Health

In order for sows to achieve optimal reproductive performance and a large litter size, a healthy breeding herd is essential. In the event of an immune challenge, energy, protein, and other nutrients are used to mount an immune response and fight infection. Resources partitioned toward an immune response are therefore not used for reproductive cycles or fetal growth and development (Spurlock, 1997).

Many infectious diseases cause reproductive failure, a few of which include porcine reproductive and respiratory syndrome virus (PRRSV), porcine circovirus 2 (PCV2), porcine parvovirus (PPV), leptospirosis, and influenza A virus (IAV; Althouse et al., 2019; Arent and Ellis, 2019). The PRRSV causes reproductive failure in sows including abortions, early farrowing, increased stillborns and mummies, and more pigs born that are weak (Zimmerman et al., 1997). The low viability pigs at birth are a consequence of in utero viremia and these pigs are more susceptible to secondary diseases (Lewis et al., 2009). Porcine circovirus 2 results in weak pigs at birth, stillborns, and fetal mummifications (Althouse et al., 2019). Porcine parvovirus causes embryonic death and resorption, therefore small litter sizes, and fetal mummification (Althouse et al., 2019). Influenza A virus causes infertility, decreased litter size, abortions, and stillborns (Althouse et al., 2019).

Routine vaccination can prevent or reduce the prevalence of infection for PCV2, PPV1, leptospirosis, and IAV. However, PRRSV prevention relies on strict biosecurity, sanitation, and in swine-dense regions, air filtration. Once a herd is infected with PRRSV, there are several methods to eliminate the virus including total depopulation and repopulation, partial depopulation, test and removal, and the most common method is herd closure (Zimmerman et al., 2019).

1.3   Piglet Environment in the Farrowing House

Briefly, small pigs weighing less than 800 g have a low chance of survival to weaning (Theil et al., 2012). These small pigs loose body heat more rapidly, have smaller glycogen pools, and smaller stomachs which limit colostrum consumption especially when trying to compete within a large litter (Theil et al., 2012). In addition to a piglet's birth weight, the environment in farrowing crates determine a piglet's pre-weaning growth, health, and survival. This environment includes

available nutrients, e.g. colostrum and milk, the presence of infectious pathogens, and a microenvironment focused on the higher temperature needs of the piglet.

1.3.1 Colostrum and Milk

Sow performance can be measured by litter size, total born and born alive, and the number of pigs weaned. Furthermore, the quality of the pig born and weaned is partially determined by their body weight and health status. One important environmental factor that is responsible for piglet survival and growth pre-weaning is colostrum and milk quality and quantity. Sow feed intake of a well formulated diet is essential because there is a very high demand for nutrients from the sow's diet to support milk synthesis (Theil et al., 2012).

The first stage of lactation, lactogenesis 1, begins approximately five weeks prior to farrowing (d 80 of gestation). This is the stage at which colostrum production takes place (Theil et al., 2012). However, immunoglobulin G transfer from sow plasma to lacteal secretions begins about 10 days before parturition (Huang et al., 1992).

Colostrum is the first secretion from the mammary gland after parturition and it is essential for piglets to consume colostrum in order to survive the first few days of life. For approximately 16 hours after birth, piglets use their own glycogen depots as a source of energy (Theil et al., 2011). Thereafter, colostrum consumption as a source of energy is critical to survival. Colostrum is also a source of immunity (Rooke and Bland, 2002). Piglets are born without a developed immune system and therefore, they rely on passive humoral immunity from their mothers in the form of immunoglobulins, IgG, from colostrum (Oliviero et al., 2019).

The ability of piglets to consume IgG and to transfer IgG from the gut to the bloodstream is diminished after the first 24 to 36 hours of life due to gut closure. Thereafter, the dominant immunoglobulin in the milk is IgA and this immunoglobulin functions to provide protection at the

mucosal surfaces of the intestine (Rooke and Bland, 2002). Piglets are able to produce their own immunoglobulins at about 3 to 4 weeks of age but development of the intestinal lymphoid tissue isn't complete until about 7 to 9 weeks of age and can be delayed further when weaned early at 3 to 4 weeks of age (Rooke and Bland, 2002; Thomson et al., 2019).

After colostrum, the sow's milk transitions into whole milk by d 3 of lactation. The amount of milk produced by the sow is dependent on both endocrine factors as well as nursing stimuli. Prolactin is the most important hormone responsible for milk production in the sow (Farmer, 2016). In addition to circulating hormones, milk synthesis in the sow is stimulated by milk removal from the mammary gland and milk is synthesized at the highest rate during the first 30-35 minutes after suckling (Theil et al., 2012) with piglets nursing on average every 30 to 50 minutes (Auldist et al., 2000; Moreira et al., 2020). Prolactin and piglet suckling are important for the maintenance of milk synthesis.

Milk composition is largely influenced by the sow's feed intake and dietary composition, with fat being the macronutrient most sensitive to changes in the sow's diet. A high feed intake in lactation is essential to meet the sow's energy demands and decrease her catabolism due to the demand for milk production. When the dietary intake of nutrients and energy is not adequate, sows will mobilize their own body stores to provide protein and fat in their milk placing themselves in a negative energy balance (Whittemore, 1996). Milk is mainly composed of lactose, protein, minerals, and fat. Of these components, the most variable is fat. Lauridsen and Danielsen (2004) fed 8% supplemental fat of varying sources and observed an increased daily output of fat and/or energy in sow milk. Supplementation of fat in the sow diet has been used to improve litter performance by influencing the milk fat component and quantity of milk produced (Lauridsen and Danielsen, 2004; Theil et al., 2012).

Fat is made up of triglycerides and more specifically, fatty acids. Milk fatty acid composition is highly influenced by the sow's dietary fatty acid composition (Lauridsen and Danielsen, 2004). Sow's milk typically contains little to no medium chain fatty acids (Decuypere and Dierick, 2003; Lauridsen and Danielsen, 2004). The fatty acid profile of sow's milk is commonly comprised of long-chain fatty acids, but it may be possible to increase the presence of medium chain fatty acids by feeding sows fat sources high in medium-chain fatty acids, e.g coconut and palm kernel oils (Lauridsen, 2020). Increased concentrations of medium-chain fatty acids in sow milk may increase pre-weaning litter weight gain and increase survival rates to d 3 of life (Jean and Chiang, 1999; Lauridsen and Danielsen, 2004).

1.3.2   Shedding of Pathogens in Sow Feces to Piglets

Piglet diarrhea, also called scours, in the farrowing house is a common contributor to pre-weaning mortality (Saif et al., 2019). Some common causes of farrowing house scours include *Escherichia coli*, *Clostridium perfringens* type C, *Cystoisospora suis* (coccidiosis), and porcine epidemic diarrhea virus (PEDV; McCormick et al., 2017; Lindsay et al., 2019; Saif et al., 2019; Uzal and Songer, 2019; Posthaus et al., 2020). Piglet scours result in inefficient use of dietary nutrients, dehydration, weakness, and often mortality.

Assuming the farrowing crates are effectively sanitized between sow groups, the other concerning route of pathogenic transmission to piglets is fecal-oral transfer (Mackie et al., 1999). Reduction of pathogen shedding from the sow's gastrointestinal tract to the piglet environment in the farrowing crate can beneficially alter the developing gastrointestinal microbiota of the neonatal piglet, e.g. DFMs reducing *Clostridium* populations responsible for scours (Baker et al., 2013). Feed additives that can reduce pathogenic bacteria in the gut of the pig include antibiotics, organic acids, zinc and copper, prebiotics, DFMs and yeast, and plant extracts (Liu et al., 2018).

1.3.3 Piglet Microenvironment

In the United States, sows and piglets are typically kept in farrowing crates while in the farrowing barn. These farrowing crates are designed to keep the sow in the center of the crate while also providing space on either side for the piglets to lay. Other than to prevent the sow from crushing the piglets, these piglet areas of the crates are equipped with heat lamps and/or heating pads. This additional heat creates a warmer thermal environment for the piglets than what the sow is comfortable in. The lactating sow prefers 18 to 22°C whereas, the piglets prefer 32°C with a lower critical temperature (LCT) of 25°C (Quiniou and Noblet, 1999; Tucker et al., 2020).

It is important for newborn piglets to have a warmer environment in order to decrease the negative impacts of cold stress. Due to the absence of brown adipose tissue in the piglet, their main mechanism for thermoregulation when they are too cold is shivering (Berthon et al., 1994). Shivering induces an increased utilization of the piglet's stored energy in the form of lipids and glycogen (Berthon et al., 1996) and when newborn piglets are too cold, their intake of colostrum also decreases leading to a greater likelihood of starvation (Le Dividich and Noblet, 1981). A pig experiencing hypothermia and limited nutrition will become weak and disoriented which increases the chances for that piglet to be crushed by the sow.

Proper ventilation provides fresh, clean air to the animals in the farrowing barn without the presence of drafts which could chill the piglets (Tucker et al., 2020). Ventilation goals are also dependent on the season (e.g. summer vs. winter). For fully slatted facilities, Brumm (2019) recommends a minimum ventilation rate of 34, 85, and 1100 m^3/h when in cold, mild, and hot weather, respectively. These minimum ventilation rates are designed to target the desired humidity level of 60-80% in cold weather and higher ventilation rates are necessary to decrease humidity in response to warming outside temperatures (Brumm, 2019). In addition to ventilation, sows are

provided cooling strategies like a water drip to allow for evaporative cooling. Two consequences to a water drip for the sows include a higher risk of chilling the piglets that often also become wet and the water from the drip increases the humidity in the farrowing room.

1.4   Leaky Gut – Sow and Weaned Pig

1.4.1   The Importance of Gut Barrier Function

The main function of the gastrointestinal tract (GIT) is to digest and absorb dietary nutrients while excluding potential pathogens. In order for the GIT to remain healthy and functional, the gut barrier needs to be maintained. There are many elements involved in maintaining the gut barrier including the mucosal immune system, mucus secretion, bicarbonate, immunoglobulin A, antimicrobial peptides, epithelial cells, and the intracellular tight junction proteins between epithelial cells (Johnson et al., 2012). Disfunction of any of these elements may result in a leaky gut due to a compromised barrier which allows unwanted macromolecules and bacteria to enter the pig through the digestive tract (Johnson et al., 2012).

Within the pig's GIT, there is a protective layer of mucus between the intestinal lumen and the epithelial absorptive cells. Goblet cells within the intestine are a key component of the gut barrier because they serve to produce mucus (Pluske et al., 2002). Many pathogenic bacteria produce mucolytic enzymes which degrade the mucin layer compromising the epithelial gut barrier and leading to bacterial translocation into the lamina propria layer of the intestine (Pluske et al., 2002).

Tight junction proteins serve as a paracellular barrier between adjacent intestinal epithelial cells. These proteins are located near the apical brush boarder and within the apical junction complex (Johnson et al., 2012). A few of the tight junction proteins commonly researched in swine

include claudins, zonula occludens-1 (ZO-1; also known as TJP1 or tight junction protein-1), and occludin (Johnson et al., 2012; Hu et al., 2013; Wang et al., 2015).

The degradation of the protective layer of mucus or tight junction proteins could lead to gastrointestinal disfunction if pathogenic bacteria are present. Gastrointestinal disfunction commonly involves diarrhea, impaired nutrient absorption, and reduced growth rates in pigs. Post-weaning colibacillosis (PWC) is one very common disease of the small intestine due to the colonization of pathogenic *E. coli* in the first 3 to 10 days post-weaning. Enterotoxigenic *E. coli* release enterotoxins which cause pigs to have hypersecretory diarrhea and dehydration (Evans and Evans, 1996). A major contributor to mortality and morbidity in post-weaned pigs is PWC (Pluske et al., 2002).

In order to avoid sacrificing sows for dissection research of gastrointestinal disfunction, there are ways to indirectly monitor gut health and gut barrier function in sows and other ages of pigs. D-lactate concentrations in the plasma of pigs has been associated with a change in intestinal permeability and bacterial translocation (Celi et al., 2019). Specifically, high D-lactate levels are indicative of increased intestinal permeability (Xun et al., 2015). While mammalian cells form D-lactate in nanomolar concentrations through the methylglyoxal pathway, the exogenous source of D-lactate comes from microbial fermentation in the colon (Ewaschuk et al., 2005) which when the intestinal wall is compromised, leaks into the blood. Another non-invasive method to measure intestinal barrier permeability is feeding lactulose and mannitol which are non-digestible oligosaccharides that should just pass through the pig's gastrointestinal tract. A high lactulose:mannitol ratio detected in urine is an indication that there is damage to the intestinal barrier tight junctions (Celi et al., 2019). Lactoferrin and myeloperoxidase (MPO) measured in feces are additional biomarkers used to quantify intestinal inflammation (Celi et al., 2019). In

bowel samples collected from the jejunum, MPO activity was greater for newborn piglets provided total parenteral nutrition compared to enterally fed piglets (Kansagra et al., 2003). These authors also observed a correlation between MPO activity measured in the jejunum and intestinal permeability measured by lactulose ($R^2 = 0.32$) and polyethylene glycol 4000 ($R^2 = 0.38$) recovery in urine after oral gavage.

1.4.2   GIT Immune System

Intestinal penetration of luminal antigens through a leaky gut leads to activation of an immune response and intestinal inflammation (Johnson et al., 2012). The first line of defense against invading pathogens is the rapid and non-specific innate immune system involving neutrophils, macrophages, dendritic cells, and natural killer cells (Uematsu and Fujimoto, 2010). These cells are responsible for presenting the detected antigen to lymphocytes (B and T cells), phagocytosis of the pathogen, free radical production, and cytokine production (Volman et al., 2008).

Pathogen associated molecular patterns (PAMP) of invading pathogens are recognized by pattern recognition receptors (PRR) including toll-like receptors (TLR) and dectin-1 (Volman et al., 2008). These PRRs are located on the basolateral surface of intestinal epithelial cells and they are also highly expressed on antigen presenting cells such as macrophages and dendritic cells (Uematsu and Fujimoto, 2010). Activated TLRs can initiate many different responses including inflammatory responses, activation of the adaptive immune system, and even tissue repair (Prendergast and Jaffee, 2013).

Additionally, Peyer's patches and their specialized microfold cells (M cells) are a large part of the mucosa-associated lymphoid tissue, where M cells serve to transport antigens into the Peyer's patches from within the intestinal lumen (Volman et al., 2008). Peyer's patches house

dendritic cells, B cells, and T cells. The PRRs on dendritic cells recognize the antigen, phagocytose, process, and present the antigen to T cells (Volman et al., 2008). Detection of antigens result in increased cytokine production and B cell activation (Volman et al., 2008; Johnson et al., 2012).

Inflammation and associated damage are driven by many pro-inflammatory cytokines, some of which include TNF-α, IFN-γ, IL-1β, IL-6, IL-8, IL-12, and IL-18. There are also anti-inflammatory cytokines which may act through immunosuppressive mechanisms or inhibition of pro-inflammatory cytokine production including IL-4, IL-10, IL-11, IL-13, and TGF-β (Pié et al., 2004; Johnson et al., 2012; Dembic, 2015; Andrews et al., 2018). In the gut, inflammatory cytokines such as TNF-α increase tight junction permeability and anti-inflammatory cytokines such as TGF-β have protective effects on the intestinal epithelial barrier (Xiao et al., 2017). In addition to regulating intestinal epithelial barrier integrity, cytokines also regulate intestinal epithelial cell proliferation and apoptosis (Andrews et al., 2018).

Although these two groups of pro- and anti-inflammatory cytokines seem clear cut, Andrews et al. (2018) stress that literature contains evidence of some cytokines exhibiting pro-inflammatory and also anti-inflammatory effects depending on the location and specific situation throughout the body. For example, TNF-α and IL-6 are known to contribute to gut inflammation, however these two cytokines also help promote epithelial proliferation of new cells to support gut homeostasis (Andrews et al., 2018). Also, IL-17 induces pro- and anti-inflammatory effects in the mucosa (Neurath, 2014). Specificlly, IL-17 pro-inflammatory functions include upregulation of TNF, IL-1β, IL-6, IL-8, and recruitment of neutrophils potentially leading to tissue destruction in inflammatory bowel disease (IBD). Research has also shown IL-17 to have anti-inflammatory effects such as promoting tissue homeostasis (Neurath, 2014).

In addition to the initial and quick innate immune response and inflammatory response, as previously stated, antigen presenting cells such as macrophages and dendritic cells serve as the critical bridge to activate the adaptive immune system via presentation to B and T lymphocytes (Prendergast and Jaffee, 2013). The adaptive immune system has defining characteristics including specificity, inducibility, diversity, memory, and non-responsiveness to self (Prendergast and Jaffee, 2013). The two types of adaptive immune responses include the humoral-B cell and cell-mediated-T cell responses.

From the humoral response, intestinal immunoglobulin A (IgA) is secreted from IgA producing plasma cells (activated B cells) residing in the lamina propria layer of the intestine (Johnson et al., 2012). IgA helps protect against antigens such as enterotoxigenic *E coli* (ETEC; Evans and Evans, 1996). From the cellular response, T cells can produce cytokines and they can directly kill target cells via specialized T cells or natural killer cells (Prendergast and Jaffee, 2013).

Tissue inflammation is one part of the systemic immune response. Two potential ways to indirectly evaluate intestinal inflammation in sows include lactoferrin and myeloperoxidase measured in feces. Lactoferrin (LF) is a glycoprotein in mature neutrophilic granulocytes and myeloperoxidase is an enzyme in young neutrophils, monocytes, and macrophages (Prata et al., 2016). These fractions of neutrophils are a valuable target because neutrophils infiltrate intestinal mucosa during inflammation (Prata et al., 2016).

1.5   Gut Modifiers

Antibiotics used for growth promotion in swine has been eliminated in the U.S. effective January 2017 (GFI #213; U.S. Food and Drug Administration, 2020). Therefore, researchers are motivated to find other feed additives that promote not only growth, but additionally gut health which has impacts on feed efficiency, overall animal health, and high-quality pork products

(Metzler et al., 2005). Organic acids, including medium and short chain fatty acids, direct fed microbials, and yeast feed additives are a few classes of feed additives with gut modification features discussed in this review.

1.5.1 Medium Chain Fatty Acids

Nutritionists have been adding organic acids (OA) to pig diets for many years (Metzler et al., 2005). A few specific organic acids include citric acid, lactic acid, fumaric acid, sorbic acid, benzoic acid, short chain fatty acids (SCFA), and medium chain fatty acids (MCFA). Specifically, MCFA are saturated fatty acids made up of 8 to 12 carbon atoms, although some also classify C6 as an additional MCFA (Hanczakowska, 2017). In general, they are lipids with antimicrobial properties against bacteria, fungi, protozoa, and viruses (Yoon et al., 2018). These MCFA may be an effective alternative to in-feed antibiotics because they reduce the colonization of pathogenic bacteria in the gastrointestinal tract of the pig (Decuypere and Dierick, 2003).

The mode of action for MCFA involves disruption of the phospholipid membrane around bacteria and lipid bilayer-enveloped viruses (Jackman et al., 2020). The most important disruptions caused by MCFA include increased membrane permeability, cell lysis, disruption of the electron transport chain, uncoupling of oxidative phosphorylation, inhibition of membrane enzyme activities, and inhibited nutrient uptake (Yoon et al., 2018). Medium chain fatty acids, especially lauric acid (C12), more strongly inhibit gram-positive bacteria than gram-negative bacteria because of the more simplistic lipid bilayer cell membrane of gram-positive bacteria (Jackman et al., 2020). However, some *in vitro* studies have reported that caprylic acid (C8) has a high antibacterial activity against gram-negative *E. coli* and *Salmonella* bacterial species (Yoon et al., 2018).

Medium chain fatty acids also serve as a readily available energy source to promote pig growth and gut development and integrity (Zentek et al., 2011). They are a source of quick energy because they are absorbed into the enterocyte where they can be utilized directly or transported through portal blood to the liver avoiding the time-consuming mechanisms of chylomicron formation or re-esterification (Velázquez et al., 1996; Zentek et al., 2011). Additionally, MCFA are rapidly metabolized in the mitochondria to CO_2 and ketone bodies which are used to stimulate lipogenesis (Azain, 1993). Neonatal energy stores may be improved and therefore increase pre-weaning survival by feeding MCFA to sows prior to parturition (Azain, 1993).

Some literature describes feeding medium chain triglycerides (MCT) in addition to or separately from MCFA (Azain, 1993; Dierick et al., 2002). Medium chain triglycerides contain three MCFA bound to a glycerol molecule. In contrast to free MCFA, MCT do not have strong odors which avoids the possible feed intake deterrent MCFA may have (Jackman et al., 2020). Endogenous, or exogenous, lipases facilitate the release of MCFA from MCT within the stomach and duodenum. As discussed below, feeding pure forms of MCFA and monoglycerides have an additional benefit of inhibiting viral and bacterial pathogen survival in feed and therefore reduce risk of disease transmission to pigs (Jackman et al., 2020).

In addition to many effective biosecurity protocols, MCFA may be added to swine diets to mitigate feed pathogens, especially viruses (Dee et al., 2020). Viruses including PEDV, PRRSV, senecavirus A (SVA), and African swine fever virus (ASFV) can survive in feed or on feed ingredients and be transmitted to swine consuming the infected feed (Dee et al., 2014; Niederwerder et al., 2019). The addition of a 1% MCFA blend containing 1:1:1 caproic (C6), caprylic (C8), and capric acids (C10) or these FA individually (0.66%) in addition to lauric acid (C12) has been added to PEDV infected feed and fed to 10-day-old pigs (Cochrane et al., 2020).

Pigs fed the MCFA blend or C6, C8, or C10 alone in the PEDV infected feed did not show evidence of PEDV infectivity analyzed by qRT-PCR of rectal swabs and tissues collected 7 days post inoculation. When the 0.66% C12 and 0.3% dry lauric acid product were added to infected feed, pigs were considered infected on d 4, 6, and 7 post inoculation which authors speculate could be due to the longer MCFA being too lipophilic to approach the PEDV cellular membrane (Cochrane et al., 2020). Therefore, some MCFA feed additives function as protective measures in the feed even before they are consumed by the animal.

1.5.1.1 Combinations of Medium Chain Fatty Acids

Hansen et al. (2012) concluded that feeding MCFAs, C8 or C12 sources, to sows during the transition period beginning one week before parturition may beneficially impact colostrum synthesis as observed by increased concentrations of MCFAs in the sow's plasma around parturition and an increased piglet live weight gain from 0 to 24 h of life. Hansen et al. (2012) did not observe a difference in 0 to 24 h piglet mortality between control and C12 coconut oil diets (4.8 vs. 3.6%, respectively), but the C8 supplementation diet reduced mortality (0%).

Jean and Chiang (1999) also reported low birth weight pigs (weighing less than 1,100 g) born to sows fed 10% coconut oil (C12 source) or 10% MCFA had greater survival rates to d 3 of life (80 and 98%, respectively) compared to pigs from control sows fed 10% soybean oil (48%). The MCFA diet fed from d 84 of gestation until farrowing contained 920 g/kg C8, 20 g/kg C10 and 60 g/kg C12 (Jean and Chiang, 1999). In agreement with Azain (1993), milk from sows fed MCFA contained decreased fat content compared to control fed sows (79 vs. 93 g/kg; Jean and Chiang, 1999). The improved survival of low BW pigs appears to be related more to the observed increased hepatic glycogen levels at 4 h after birth because milk from MCFA fed sows had a lower fat and energy content.

Nursery pigs were fed MCFA supplemented diets for 35 days (Gebhardt et al., 2020). A 1:1:1 blend of caproic (C6), caprylic (C8), and capric acids (C10) were fed in a dose dependent manner in 5 diets from 0 to 1.5% of the diet. Additionally, diets with 0.5% of each acid alone were also fed. Average daily gain, feed intake, and G:F linearly and dose dependently improved with increasing 1:1:1 blend of MCFA. From d 0-14 and d 0-35, pigs fed the 0.5% C8 diet had greater ADG, G:F, and body weights compared to control. Pigs fed 0.5% C6 or C10 also had improved overall G:F compared to control fed pigs, with no differences in ADG or ADFI (Gebhardt et al., 2020).

In a similar study, Thomas et al. (2020) fed a MCFA feed additive (C8 and C10) to nursery pigs for 34 days in a dose dependent manner of 0%, 0.5%, 1.0%, and 2.0% inclusion. Additionally, authors fed a diet containing 1.0% MCFA 1:1:1 blend of caproic (C6), caprylic (C8), and capric acids (C10). Overall (d 0-34), ADG and ADFI increased linearly with increasing MCFA feed additive. Feed efficiency also increased up to 1.0% of the diet. Pigs on d 34 were 1.8 kg heavier when fed 2.0% MCFA additive compared to 0.0% (23.6 vs. 21.8 kg). There was also no difference in growth performance between pigs fed the 1.0% MCFA feed additive and 1.0% 1:1:1 (C6:C8:C10) blend.

In a bacterial challenge model with Enterotoxigenic β-hemolytic *Escherichia coli* (ETEC: serotype O149:K91: K88), Cochrane et al. (2018) investigated feeding MCFAs as a chlortetracycline (CTC) antibiotic alternative to nursery pigs. Dietary treatments were fed for 14 days post-challenge including: 1) control with no CTC or MCFA; 2) with a therapeutic dose of 0.44 g/kg CTC; 3) a MCFA blend of 3.5, 3.6, and 3.6 g/kg; 4) a MCFA blend of 1.2, 4.8, and 4.0 g/kg ; or 5) a MCFA blend of 0.4, 5.6, and 4.0 g/kg of caproic (C6), caprylic (C8), and capric (C10) acids, respectively for all treatments. For the 14-day period, pigs challenged with *E. coli* and

fed any of the 3 MCFA diets had similar G:F compared to the CTC fed pigs with no difference in ADG or ADFI. Pigs fed CTC and the 0.4, 5.6, and 4.0 g/kg blend had increased G:F compared to the control fed pigs (Cochrane et al., 2018).

1.5.1.2   Caprylic Acid (C8) and Capric Acid (C10)

Azain (1993) did not observe a difference in d 1 survival but authors did observe 10% medium chain triglycerides (MCT; C8 and C10) fed to sows improved pre-weaning survival (d 0-21) and additionally increased MCFA in milk collected on d 7 of lactation. Sow's milk typically does not contain any MCFA (Decuypere and Dierick, 2003). Therefore, Azain (1993) explained that the increased MCFA in milk only accounted for less than 5% of the milk fatty acids which likely did not cause the improvement in survival rate. Interestingly, milk from the MCT fed sows contained 76.2 mg/mL of total lipid which was significantly less than control fed sows (87.8 mg/mL; Azain, 1993).

Piglets fed 0.2% caprylic acid (C8) and/or capric acid (C10) were provided diets starting at 7 days of age, weaned on d 35, and concluded the experiment at 84 days of age (Hanczakowska et al., 2011). Although impractical, these authors restricted feed intake of piglets from weaning until d 84 increasing intake by about 200 g every 7 days. At weaning on d 35, pigs fed C8, C10, or the combination diet had a heavier body weight and greater ADG compared with control fed pigs. During this time, pigs fed C8 had the greatest body weight, ADG, and best feed conversion ratio. Subsequently on d 84, pigs fed the MCFAs alone or in combination had greater ending body weights and overall experimental ADG and feed conversion ratios compared to control diets without MCFAs or antibiotics (Hanczakowska et al., 2011).

Hanczakowska et al. (2011) also observed an improvement in protein and fiber digestibility when pigs were fed the MCFAs alone or in combination compared to the controls. Microbial

counts of *Clostridium perfringens* in ileal digesta were reduced when C8 and C10 were fed alone with no difference between the combination fed pigs and the controls. There was also no difference in the microbial counts of *E. coli* in the ileum digesta. Pigs fed C10 also had increased ileal villus height and increased crypt depth compared to control fed pigs (Hanczakowska et al., 2011).

A later experiment by many of the same authors was conducted where piglets fed 0.3% caprylic (C8) or 0.3% capric acids (C10) from 7 days of life until 70 days of life were weaned at 28 days of life (Hanczakowska et al., 2016). For the entire experimental period, pigs fed C10 had a greater average daily gain (323 vs. 298 g/d) and greater ending body weight (24.08 vs. 22.37 kg) on d 70 compared to control fed pigs, with pigs fed C8 intermediate and not different than either group. Jejunal villus height, villus width, crypt depth, and villus height/crypt depth ratio were not different between control, C8, or C10 fed pigs. Microbial counts of *E. coli* in the jejunum and cecum digesta of pigs fed C8 were decreased in addition to a reduction of *Clostridium perfringens* counts in the cecum digesta compared to control with pigs fed C10 intermediate and not different than either group (Hanczakowska et al., 2016).

Dierick et al. (2002) fed 2.5% medium chain triglyceride oil (MCT; C8 and C10) to pigs with and without 0.1% lipase 1 week before weaning at 21 days of age until 3 weeks post-weaning. Authors reported that pigs fed MCT with or without lipase had increased ADG during the first 2 weeks post-weaning compared to control (164 and 165 vs. 127 g/d from d 0-7; 160 and 161 vs. 127 g/d from d 7-14). There were no differences in growth during the week before weaning, the third week post-weaning, or the overall post-weaning period. Feed intake and feed conversion ratio was also not different between treatments at any point of the experiment. On post-weaning d 18, a subset of 5 barrows per treatment group were harvested. *Streptococci* and *E. coli* counts from

stomach contents were reduced when MCT was fed without the addition of lipase and these statistical reductions did not occur in the duodenal contents (Dierick et al., 2002).

1.5.1.3 Lauric Acid (C12) and Glycerol Monolaurate (GML)

Lauric acid (C12:0) is a saturated MCFA and is the primary fatty acid of coconut oil (Dayrit, 2015). Lauric acid is also the most active saturated fatty acid against gram-positive bacteria (Kabara et al., 1972). Glycerol monolaurate, also known as monolaurin, is a monoglyceride meaning it is a monoester of glycerol and one free fatty acid, lauric acid (Schlievert et al., 2019).

Monoglycerides form micelles, an active state, at lower concentrations than free MCFA and therefore may explain why they are more biologically potent, e.g. antibacterial properties of membrane disruption (Jackman et al., 2020). Compounds with longer chain lengths also exhibit more potent inhibitory activity than shorter chain lengths. Glycerol monolaurin has a lower critical micelle concentration value and greater potency than free lauric acid and glycerol monocaprin (Yoon et al., 2017; Valle-González et al., 2018).

Lauric acid also has a lower critical micelle concentration at lower pH values indicating it causes greater phase separation in lipid bilayers in acidic conditions where the proton is not dissociated from lauric acid's carboxylic acid group (Valle-González et al., 2018). Therefore, the more potent inhibitory activity of GML throughout the entire gastrointestinal tract may be because GML will not dissociate within the more neutral environments of the intestine due to its pK_a value of 14 (Jackman et al., 2020). In broth and biofilm cultures, GML has more than 200 times the bactericidal activity of lauric acid alone and both components have antimicrobial properties that act on nearly all Gram-positive bacterial species (Schlievert and Peterson, 2012).

Lauridsen and Danielsen (2004) fed 8% supplemental fat of varying sources including coconut oil (C12) to sows 1 week prior to farrowing and throughout a 28-d lactation. Litter weight gain from sows fed 8% coconut oil tended to be greater than sows fed 8% fish oil (66.3 vs. 59.3 kg in 28 days). Coconut oil fed sows also had a greater daily output of fat in milk compared to fish oil and sunflower oil fed sows. The concentration of C10 and C12 in milk of sows was greatest in sows fed coconut oil and palm oil also provided more C12 compared to other fat sources.

In a Pig Progress article, Dansen (2016) reported a case study of C12 fed to sows on a PRRSV infected farm in southern Europe in 2014. Sow diets were supplemented with 15 g/d of a C12 supplement containing GML for 1 week pre-farrowing until the next service. With 28 sows/litters per treatment, the number of pigs weaned per sow increased 4.3% (11.75 vs. 12.25), pre-weaning mortality decreased 30.8% (5.2% vs. 3.6%), and the days until next service decreased 36.8% (6.52 vs. 4.12) when sows were fed the C12 supplement.

Pigs were fed blends of MCFA, lactic acid, and GML for 35 days in the nursery (Thomas et al., 2020). Diets consisted of a control diet without MCFA, the control with 1.0% MCFA 1:1:1 blend of caproic (C6), caprylic (C8), and capric acids (C10), and the control diet with 1.0% of 4 different blends of 50% C6, 20% lactic acid, and increasing amounts of GML (0%, 10%, 20%, and 30%) at the expense of C12 (30%, 20%, 10%, 0%). Overall (d 0-35), pigs fed the 1.0% MCFA 1:1:1 blend had greater ADG and ADFI compared to control fed pigs with a 0.9 kg heavier ending weight (21.6 vs. 22.5 kg). However, the pigs fed the 4 different blends of MCFA, lactic acid, and GML did not have a linear or quadratic growth response. When calculating the mean of the 4 blends and comparing it to control, the MCFA fed pigs had better feed efficiency from d 0-14 but not overall. The mean of the 4 blends for d 0-35 ADG was 435 g/d compared to 422 g/d for control fed pigs but the p-value was only approaching a trend ($P = 0.149$) and the mean final body weight

was 22.0 kg vs. 21.6 kg for blends and control fed pigs, respectively ($P = 0.134$; Thomas et al., 2020).

1.5.2 Short Chain Fatty Acids and Benzoic Acid

Short-chain fatty acids (SCFA) are fatty acids made up of 1 to 6 carbon atoms in the acyl chain including: formate (C1), acetate (C2), propionate (C3), butyrate (C4), valerate (C5), and caproate (C6; Mills et al., 2009). Other organic acids commonly fed to swine include citric acid, lactic acid, fumaric acid, sorbic acid, and benzoic acid (Radcliffe et al., 1998; Kim et al., 2005; Suiryanrayna and Ramana, 2015).

In general, SCFA are used for acidification of the gut, pH reduction, in order to decrease the growth of pathogenic bacteria in the gut (Gabert and Sauer, 1994). Uncharged weak acids are able to diffuse into the cytoplasm of microbes, decreasing their cytoplasmic pH, and dissociating into protons and anions (Hirshfield et al., 2003). The anions have effects on osmolarity of the cytoplasm causing increased membrane permeability and anions also disrupt microbial enzyme activity causing metabolism malfunctions (Hirshfield et al., 2003; Heo et al., 2013; Suiryanrayna and Ramana, 2015). Acidification also improves nutrient digestibility via increased pepsin activity and stimulated gut development and integrity in pigs (Gabert and Sauer, 1994; Suiryanrayna and Ramana, 2015). Reducing pathogenic overgrowth via pH reduction is one way to control pig post-weaning diarrhea (Lauridsen, 2020).

Supplementation with 0.1% or 0.2% protected organic acids were fed to sows from d 95 of gestation to weaning (21 days; Mohana Devi et al., 2016). The protected organic acids were formulated to be released slowly throughout the gastrointestinal tract and contained 17% fumaric acid, 13% citric acid, 10% malic acid, and 1.2% MCFA consisting mainly of capric and caprylic acids. In summary, 0.2% protected organic acid fed to sows increased digestibility of DM, N, and

energy in lactation, decreased fecal *E. coli* concentration, and increased *Lactobacillus* concentration throughout lactation compared to the control fed sows. Piglets born to sows fed 0.2% protected organic acids had greater white blood cell counts and IgG at weaning compared to pigs from control fed sows but there was no difference in piglet survival, ADG, or fecal scores (Mohana Devi et al., 2016).

Benzoic acid is a cyclic SCFA with a double bond in the benzene ring classified as an organic acid (Menegat et al., 2019a). Knarreborg et al. (2002) observed, *in vitro*, benzoic acid was superior to 5 other organic acids in exhibiting bactericidal effects on coliform and lactic acid bacteria in the stomach as well as the small intestine of piglets. Benzoic acid has also been fed *in vivo* to sows (Kluge et al., 2010; Benthem de Grave et al., 2016), weaned pigs (Kluge et al., 2006; Guggenbuhl et al., 2007; Chen et al., 2017), and weaned pigs challenged with *E. coli*, specifically to reduce the incidence of diarrhea (Silveira et al., 2018).

Kluge et al. (2010) fed lactating sows 0, 0.5, 1.0, or 2.0% benzoic acid. When included at 2.0% of the diet, digestibility of organic matter, crude protein, crude fat, and crude fiber increased compared to controls with a 0.87 kg/d reduction in feed intake although it was not statistically significant (6.24 kg/d vs. 5.37 kg/d). Sows fed benzoic acid also had decreasing urinary pH values with the greatest reduction in the sows fed 2.0% (7.20 control vs. 5.39 at 2.0%) which may help prevent urinary tract infections and/or endometritis (Kluge et al., 2010).

In a study evaluating the possible effects of feeding benzoic acid to gestating and lactating sows, Benthem de Grave et al. (2016) found no difference in the number of pigs born alive, weaned, or growth rate of litters when sows were fed 0, 0.5, 1.0, or 2.0% benzoic acid from insemination to weaning. Although there was no statistical difference in feed intake in gestation and lactation, sows fed the 2.0% inclusion of benzoic acid decreased body weight and backfat measured at

weaning compared to control fed sows. There was also no difference in bone mineral composition or prevalence and severity of stomach ulcers among treatments. Therefore, authors concluded that long term administration of benzoic acid fed to sows at a dose lower than 2.0% did not affect sow or piglet welfare or performance (Benthem de Grave et al., 2016).

Kluge et al. (2006) observed benzoic acid improved body weight gain from 338 g/d in control pigs to 387 g/d in 1% benzoic acid fed pigs during a 35-d nursery experiment. Although the 1% benzoic acid fed pigs outperformed the control fed pigs by 9% in feed intake and 6% in feed conversion ratio, these improvements were not statistically significant. Benzoic acid did improve nitrogen retention from 15.6 g/d in control pigs to 16.5 g/d in 1% benzoic acid fed pigs but there were no differences in nutrient digestibility. Authors attributed the improved growth rate may be due to the antimicrobial effects of benzoic acid decreasing the number of total bacteria in the stomach, gram-negative bacteria in the duodenum, and aerobic bacteria in the ileum (Kluge et al., 2006).

Guggenbuhl et al. (2007) reported that 0.5% benzoic acid fed to weaned pigs improved daily weight gain and feed conversion ratio during the 32-day nursery study. At the end of the study, pigs were harvested and stomach and cecal contents were collected to be plated for bacterial counts. When pigs were fed 0.5% benzoic acid, total lactic acid bacteria in the stomach increased and *E. coli* in the cecum decreased. During a second experiment with 60-d-old ileal canulated pigs, ileal apparent digestibility of total energy and nitrogen were increased when pigs were fed 0.5% benzoic acid compared to control fed pigs (Guggenbuhl et al. 2007).

A group of 21-d-old weaned pigs were fed a control diet, control plus 0.2% benzoic acid, or control plus 0.5% benzoic acid for 42 days in the nursery (Chen et al., 2017). Pigs fed the 0.2% benzoic acid diet had heavier body weight and greater ADG at d 14 (d 0-14) and 42 (d 0-42) of the

experiment compared to controls and 0.5% benzoic acid supplemented pigs were heavier with a greater ADG on d 42 (d 0-42). Pigs fed benzoic acid diets were also more feed efficient from d 0-42 of the study, where only 0.2% benzoic acid fed pigs had a greater overall ADFI. Jejunal pH values were lower on d 14 when pigs were fed benzoic acid but not on d 42. Stomach pH was also reduced when pigs were fed 0.5% benzoic acid on d 14 and 42. Jejunal villus height:crypt depth was also increased in pigs fed either benzoic acid diet compared to controls on d 14 and 42. *Lactobacillus* in ileal digesta from pigs fed 0.2% benzoic acid, and not 0.5%, was increased when sampled on d 14 and 42. In 42-d ileal digesta, *Bifidobacterium* was increased in 0.2% benzoic acid fed pigs and *E. coli* was reduced in pigs fed both benzoic acid diets. Higher mRNA expression on d 14 was observed for ZO-1 and occludin in the jejunum when pigs were fed 0.2% and only ZO-1 when pigs were fed 0.5% benzoic acid compared to controls. Occludin and ZO-1 expression was also upregulated on d 42 for both benzoic acid supplemented diets (Chen et al., 2017).

1.5.3   *Bacillus* Direct-Fed Microbials

A fetus has a sterile gut until microbial inoculation occurs in the birth canal during parturition (Mackie et al., 1999). After birth, piglets ingest environmental and cutaneous microbes from the sow and the microbiota of the piglet are established within the first few days after birth. A balanced gut is composed of beneficial bacteria thriving to support a healthy micro-ecosystem and restricting the growth of harmful bacteria (Liao and Nyachoti, 2017). Direct fed microbials (DFM), also known as probiotics, are live microorganisms added to the diet to improve microbial balance in the gut (Fuller, 1989; Menegat et al., 2019a). A healthy micro-ecosystem and symbiosis between the microorganisms and the pig host helps prevent diarrhea, improve nutrient utilization, growth performance, and health status of the pig (Liao and Nyachoti, 2017).

Strains of *Bifidobacterium*, *Enterococcus*, *Lactobacillus*, *Bacillus*, *Pediococcus*, and *Streptococcus* are most commonly used probiotics in animal nutrition (Yirga, 2015). Specifically, *Bacillus*-based DFMs are spore forming bacteria and these spores are stable under heat and acidic pH making them resistant to harmful impacts of feed processing and breakdown in the stomach of the pig before reaching the intestine and germinating to become vegetative cells (Menegat et al., 2019a). *Bacillus subtilis* and *Bacillus licheniformis* have little outgrowth in the GIT and therefore can't permanently colonize the gut which means continued intake of these DFMs are necessary when included in pig diets (Leser et al., 2008). It is understood that vegetative cells of *Bacillus* species produce enzymes that may enhance nutrient digestibility. However, due to little outgrowth of vegetative cells within the GIT, Leser et al. (2008) concluded that a significant number of growing vegetative cells of *B. subtilis* or *B. licheniformis* is not a prerequisite for the possible mode of action of *Bacillus* based DFMs.

Direct fed microbials fed to sows can provide production and health promoting benefits to sows and litters (Alexopoulos et al., 2004a; Baker et al., 2013). These benefits may include increased lactation feed intake, reduced sow weight loss in lactation, decreased piglet diarrhea scores, decreased pre-weaning mortality, and increased piglet body weight at weaning when Alexopoulos et al. (2004a) fed a combination of *Bacillus subtilis* and *Bacillus licheniformis* spores to gilts 14 days prior to parturition until weaning. Also, DFMs can reduce pathogen shedding from the sow feces to the piglet environment in the farrowing crate which can beneficially alter the developing gastrointestinal microbiota of the neonatal piglet, e.g. reducing *Clostridium* populations responsible for scours (Baker et al., 2013).

Sows and/or piglets were fed a diet supplemented with *Bacillus subtilis* C-3120 (Menegat et al., 2019b). Sows were fed a control diet in gestation and lactation or diets containing 5.0×10^8

cfu/kg of gestation feed and 1.0×10^9 cfu/kg of lactation feed. Progeny of these sows were followed into the nursery for a 42-d study where pigs were fed a control diet or diet containing 5.0×10^8 cfu/kg of nursery feed. Lactation feed intake tended to increase when sows were fed the DFM however, there was no difference between treatments in sow reproductive performance, piglet growth in the farrowing house, or pre-weaning mortality. There was also no interaction or significant main effects in overall nursery performance (d 0-42). Interestingly, from d 21-42 of the nursery, pigs from sows fed the control diet in gestation and lactation actually had a greater ADG and ADFI compared to pigs from sows fed the DFM. There was no difference between treatments in fecal consistency in the farrowing house or nursery when fecal score evaluation was conducted.

In contrast with Menegat et al. (2019b), in feed *Bacillus subtilis* and *Bacillus licheniformis* spores fed to weaned pigs decreased morbidity and mortality associated with post-weaning *E. coli* diarrhea and had a greater ADG and better feed conversion compared to the control fed pigs, with no difference in ADFI (Alexopoulos et al., 2004b). Authors attributed these benefits to a decrease in GIT pathogenic bacteria proliferation via the addition of the DFM but the exact mechanism is still unclear. Other mechanisms might include competition for receptors on the gut mucosa, nutritional competition between bacteria species, antibacterial properties, or stimulation of immunity (Alexopoulos et al., 2004b).

Kritas and Morrison (2005) fed a *Bacillus subtilis* and *Bacillus licheniformis* DFM to nursery pigs and determined these pigs had very similar ADG, ADFI, and G:F compared to pigs fed low doses of antibiotics. Kritas and Morrison (2005) concluded that in a high health farm, DFMs may be a viable substitute to in-feed antibiotics. It is important to note however, that Kritas and Morrison (2005) did not have a negative control treatment group (no antibiotics or probiotics added to the feed) and therefore could not determine the value of in-feed antibiotics in this system

to then compare to the DFM. Positive nursery growth performance benefits of DFM studies are not consistent across the literature which may be due to species of bacteria, dietary inclusion, age of the pig, environmental conditions, etc. (Buntyn et al., 2016). Liu et al. (2018) also comment in their review that DFM efficacy in the past has been inconsistent but they believe there has been improvements in technology and strains used in some commercial DFMs today.

Regarding gut barrier function, a low dose of *Bacillus subtilis* and *Bacillus licheniformis* spores (3.9×10^8 CFU/day) provided to weaned pigs via oral liquid solution challenged with *E. coli* lead to an increase in ZO-1 jejunal mucosa gene expression compared to the *E. coli* infected pigs with no DFM administration (Yang et al., 2016). However, authors did not observe this difference in occludin gene expression. An increase in ZO-1 gene expression suggests a stronger barrier function with this DFM administration.

1.5.4 Mannan Oligosaccharides

Dietary yeast products fed to swine have been identified as potential growth promoting feed additives, which may also have beneficial effects on the pig's immune system and health (Davis et al., 2002; Shen et al., 2011). There are benefits of feeding a whole cell yeast product because the cell wall component of the yeast is a source of mannan oligosaccharides (MOS) which are short chains of mannose sugars (Cromwell, 2012). Metzler et al. (2005) consider MOS to be a prebiotic because it gives beneficial bacteria a better opportunity to attach to the epithelium and colonize the GIT.

Mannan oligosaccharides positively impact gut health by beneficially shifting bacterial populations in the gut via the mannose binding of pathogenic bacterial glycoproteins (lectins) in the intestine and preventing pathogenic bacterial adhesion to host epithelium (Kogan and Kocher, 2007; Peisker et al., 2017). As a result of preventing pathogenic bacteria from adhering to the

intestinal lining, beneficial microorganisms have a greater opportunity to attach and colonize. This overall process is known as competitive exclusion (Metzler et al., 2005; Cromwell, 2012).

Yeast cell wall derived MOS has been investigated for their immune modulation properties when fed to sows which some authors speculate may lead to the observed improvements in piglet pre-weaning growth performance (Newman and Newman, 2001; O'Quinn et al., 2001). Feeding sows 5 g of MOS per day from 2 weeks pre-farrowing until weaning increased colostrum IgM (440 vs. 316 mg/dL) and numerically increased IgG (4,215 vs. 3,565 mg/dL) compared to control fed sows (Newman and Newman, 2001). Twenty-four hours after farrowing, the IgM (227 vs. 184 mg/dL) and IgG (1,572 vs. 1,130 mg/dL) concentrations in colostrum were still numerically greater in sows fed MOS. Pigs born to MOS sows had a heavier body weight on d 7, 14, and 21 of lactation compared to the controls. Authors observed no differences in colostrum IgA, pre-weaning mortality, or sow fecal bacterial concentrations (Newman and Newman, 2001).

In agreement, O'Quinn et al. (2001) fed sows 0.20% of the Bio-Mos Alltech, Inc. MOS product 3 weeks prior to farrowing and through 21 days of lactation where they fed a reduced inclusion rate of 0.10%. Authors observed no differences in sow weight loss, number born alive, stillborns, or mummies but they did observe an increase in litter birth and weaning weights and a decrease in pre-weaning mortality when sows were fed MOS. They also observed an increase in IgG (5,853 vs. 4,842 mg/dL), IgM (273 vs. 241 mg/dL), and a tendency for an increase in IgA (1,178 vs. 1,097 mg/dL) in pre-nursing colostrum samples (O'Quinn et al., 2001).

In a more recent study, MOS was supplemented to sows and progeny using a 2×2 factorial treatment arrangement (Duan et al., 2019). Sows were fed 400 mg/kg MOS from d 86 of gestation through 20 days of lactation and piglets were fed 800 mg/kg MOS starting at 7 days of age through 35 days of age. Sows were removed from farrowing crates when the piglets reached 20 days of

age. Secretory IgA in jejunal mucosa at 35 days of age was greater in piglets from sows fed MOS (Duan et al., 2019).

When MOS was in the piglet diet, the number of *Lactobacillus* in the ileum digesta was greater and also tended to be greater in the jejunum digesta whereas the number of *Escherichia coli* in the jejunum and cecum digesta tended to decrease compared to the piglet diets not containing MOS (Duan et al., 2019). When MOS was fed to the sow and piglet, a greater amount of *Lactobacillus* and lesser amount of *Escherichia coli* in the jejunum digesta was present compared to control fed piglets from control fed sows (Duan et al., 2019).

Piglets from sows fed MOS had decreased serum pro-inflammatory cytokines interleukin (IL)-2 and IL-4 compared to control at 35 days of age (Duan et al., 2019). When the piglets themselves consumed MOS, pro-inflammatory IL-2, IL-4, and interferon (IFN)-γ were reduced and anti-inflammatory IL-10 was increased in piglet serum compared to control fed piglets. Duan et al. (2019) concluded that feeding sows and piglets MOS could improve intestinal microbiota and suppress systemic inflammation in the piglet.

White et al. (2002) fed 21-day-old weaned pigs a 3% *Saccharomyces cerevisiae* diet (yeast contained 5.2% MOS equaling 0.156% dietary MOS) for 4 weeks in the nursery. Growth performance was not improved with the addition of MOS from the yeast. At the termination of the study, *Lactobacillus* count from fecal swabs of yeast fed pigs were greater than control fed pigs. Serum IgG on d 28 tended to be 24% greater for yeast fed pigs than control fed pigs with no difference in IgA or IgM (White et al., 2002).

In a second experiment, authors placed piglets in isolation units at 11 days of age for a 29-day acclimation period followed by a 10-day challenge period with *E. coli* K88. Authors fed pigs a 3% *Saccharomyces cerevisiae* yeast supplemented diet. When harvested at 50 days of age, yeast

fed pigs had reduced colonization of total coliforms in the jejunum and cecum compared to the control. Pigs fed yeast also tended to have higher IgG concentration in the serum compared to control fed pigs (White et al., 2002).

Zhao et al. (2012) also reported increased ADG and ADFI when nursery pigs were fed 0.1% MOS for 28 days compared to the negative control fed pigs. Pigs fed MOS also had greater apparent total tract digestibility of dry matter and nitrogen on d 14 of the experiment compared to negative controls. Although there were no differences in IgG, red and white blood cells, and lymphocyte counts, Zhao et al. (2012) did observe a decreased diarrhea score when pigs were fed MOS compared to the negative control fed pigs.

In a meta-analysis of 54 comparisons from 29 experiments, MOS from *Saccharomyces cerevisiae* yeast increased growth performance by 4.1% in the nursery compared to pigs not fed MOS (Miguel et al., 2004). Yeast fed pigs also had a 2.1% increase in ADFI and a 2.3% improvement in feed efficiency. Authors explained that slower-growing pigs the first 1 to 2 weeks post-weaning responded to MOS better than the average or accelerated growth rate pigs. Miguel et al. (2004) speculated that not only may the MOS eliminate pathogenic bacteria from the gut to enhance health and growth of the nursery pig but another mode of action may be a reduced immune system activation and therefore more nutrients available to be utilized for growth.

1.5.5 Beta Glucans

In addition to mannan oligosaccharides, the cell wall component of yeast is also a source of β-glucans. Distinguishable from the bacterial, fungal, and cereal grain β-glucans, the molecular structure of yeast β-glucans consists of 1,3 β-linked glycopyranosyl residues (glucose polymers) with few long 1,6 β-linked branches (Volman et al., 2008). Yeast β-glucans are recognized by the

immune system's pattern recognition receptors (PRRs) as pathogen associated molecular patterns (PAMPs; Volman et al., 2008). When orally ingested, β-glucans bind membrane PRRs of innate immune cells, located in the small intestinal mucosal lining, which signals the immune system to prepare for potential pathogen threat (Batbayar et al., 2012; Baert et al., 2015).

Specifically, within the small intestinal mucosal lining, there are intestinal intraepithelial lymphocytes as well as Peyer's patches and their specialized microfold cells (M cells) that are involved in regulating the immune response (Volman et al., 2008). When M cells aid in absorbing β-glucans from the intestinal lumen into Peyer's patches, β-glucans come in contact with dendritic cells, B cells, and T cells housed there (Volman et al., 2008). Dectin-1 is a receptor highly expressed on dendritic cells, neutrophils, macrophages, and some B- and T cells that is reported to aid in the immune response to feeding yeast β-glucans (Volman et al., 2008; Baert et al., 2015). Binding of β-glucans to dectin-1 results in NF-kB activation which leads to induction of gene transcription and therefore cytokine production, phagocytosis, and respiratory burst (Volman et al., 2008).

From the accumulation of *in vitro* and *in vivo* studies, Volman et al. (2008) believes β-glucans initiate a type 1 helper T-lymphocyte (Th1) response. In this scenario, IL-12 from activated macrophages for example stimulates naïve helper T-lymphocytes to develop into Th1 cells and Th1 cells produce IL-1β, interferon (IFN)-γ, IL-2, and tumor necrosis factor (TNF)-α to aid in cell-mediated immunity (Volman et al., 2008). Th1 responses may include activation of cytotoxic T cells and macrophages functioning to eliminate and clear intracellular pathogens (Xiao et al., 2004).

Literature on sows fed yeast β-glucans is limited. Chau et al. (2009) fed 5 mg β-glucans per kg of body weight to a small sample of sows ($n = 5$) 5 weeks prior to farrowing until 2 weeks

post-farrowing. Another treatment group was fed the β-glucan diet and also vaccinated for *Actinobacillus pleuropneumoniae* 5 and 2 weeks pre-farrowing ($n = 7$). There was no difference in number born alive, piglet birth weights, and weaning weights when sows were fed β-glucans and/or vaccinated. Sows fed β-glucans alone did not have increased immunoglobulins in colostrum or milk and therefore did not reflect enhanced passive immunity in piglet serum. However, when sows were fed β-glucans and vaccinated, the β-glucans acted as an oral adjuvant to increase IgM in milk after vaccination but an increase in piglet serum IgM was not observed (Chau et al., 2009).

Nursery pigs were fed diets with 0, 0.01, 0.02, 0.03, or 0.04% β-glucans from *Saccharomyces cerevisiae* for 5 weeks (Hahn et al., 2006). Overall, there was a tendency for a linear increase in ADG as the concentration of β-glucans in the diet increased. There was also a linear increase in the digestibility of dry matter, gross energy, crude protein, ether extract, Ca, and P as dietary inclusion of β-glucans increased (Hahn et al., 2006). These authors conducted a second experiment feeding a diet of 0.02% β-glucans to nursery pigs for 8 weeks and evaluated multiple immune parameters. Blood collected at the end of week 4 from pigs fed β-glucans contained a greater percentage of MHC-II lymphocytes than control fed pigs. At the end of week 8, β-glucan fed pigs had greater $CD4^+$ and $CD8^+$ T cells than control fed pigs with no differences in B cell populations (Hahn et al., 2006).

In contrast with Hahn et al. (2006), Zhou et al. (2013) did not observe growth performance improvements when feeding 0.1 g β-glucan/kg diet from *Saccharomyces cerevisiae*. Zhou et al. (2013) did observe that pigs fed β-glucans had decreased fecal *E. coli* counts on d 28 compared to control fed pigs. These authors conducted a second experiment feeding the same diet to nursery pigs for 42 days and infected pigs with *E. coli* lipopolysaccharide (LPS). In agreement with Hahn et al. (2006), Zhou et al. (2013) observed increased $CD4^+$ and $CD8^+$ T cells in β-glucan fed pigs

after LPS challenge which authors claim indicates the stimulation of the immune system via β-glucan inclusion in the diet. Similarly, Stuyven et al. (2009) reported that weaned pigs fed 0.5 g β-glucan/kg diet for 2 weeks were less susceptible to F4+ enterotoxigenic *E. coli* infection which may be a result of the β-glucans preventing adhesion and colonization of *E. coli* in the small intestine as suggested by reduced bacterial excretion and diarrhea. Stimulation of the immune system may also be involved, but Stuyven et al. (2009) did not measure $CD4^+$ and $CD8^+$ T cells in their experiment.

Some studies have fed a yeast fermentation product to sows without specifically distinguishing between MOS or β-glucan components of the yeast cell walls (Kim et al. 2008; Shen et al., 2011; Hasan et al., 2018). A *Saccharomyces cerevisiae* yeast fermentation product fed to sows during the entire gestation and lactation periods resulted in no difference in reproductive performance but did tend to increase total litter weight at weaning and litter body weight gain compared to litters from sows not fed the yeast product (Shen et al., 2011). IgG was also not different between treatment groups when measured in colostrum, milk, and plasma of piglets. Authors conclude the yeast fermentation product may improve the maternal health status because they observed a decreased neutrophil cell count in the blood of sows on d 110 of gestation and d 17 of lactation (Shen et al., 2011).

When sows were fed 0.20% of the Bio-Mos Alltech, Inc. MOS product, O'Quinn et al. (2001) observed an increase in IgG, IgM, and a tendency for an increase in IgA in pre-nursing colostrum samples. However, Hasan et al. (2018) reported no effects of 2 g of yeast derivatives fed to multiparous sows on colostrum immunoglobulin concentration but did observe an increase in fat content by 21% and colostrum yield by 24%. Additionally, Hasan et al. (2018) observed that sows fed the yeast derivatives had more beneficial bacteria (*Roseburia*, *Paraprevotells*, and

Eubacterium) and suppressed opportunistic pathogenic bacteria (*Proteobacteria*, specifically the genera *Disulfovibrio*, *Escherichia/Shigella* and *Helicobacter*) in their feces. Piglets from these sows at one week of age also had more beneficial bacterial populations and fewer opportunistic pathogenic bacteria in their feces (Hasan et al. 2018).

There was no difference in litter size at birth or litter birth weight when Kim et al. (2008) supplemented sow diets with a *Saccharomyces cerevisiae* yeast culture at 12 to 15 grams per sow per day starting on d 35 of gestation, however authors did observe an increased litter wean weight due to dietary treatment. Although milk samples were not collected, the authors speculated the increased litter wean weight could have been the result of sows being fed the yeast culture product producing more milk and/or of higher quality (Kim et al. 2008).

In nursery pigs, a feed additive containing yeast cell walls improved ADG, ADFI, and final BW at post-weaning d 28 compared to controls (Gerritsen et al., 2012). Improved ADG, digestibility of dry matter, crude protein, and gross energy, increased jejunal villus height, and increased villus height:crypt depth ratio was observed when diets supplemented with 5 g/kg of yeast culture was fed to weaned pigs for 21 days (Shen et al., 2009). Shen et al. (2009) also observed an increase in the Th1 cytokine IFN-γ present in the jejunum measured by ELISA compared to control fed pigs when sacrificed on d 21 of the study.

1.6   Economic Evaluation of Feed Technologies

Pigs raised without antibiotics in feed typically grow slower and may also encounter more frequent health challenges. Therefore, due to elimination of in feed antibiotics in the U.S. for growth promotion, the swine industry has committed to the development of new feed technologies to alleviate the decrease in growth performance without antibiotics. Research and development teams are tasked to conceptualize new ideas, design technologies, and test the effectiveness of new

products. Additionally, companies designing new products need to determine their cost of production and if it is profitable to offer the product for sale at all. The customer offered the product must conduct their own economic evaluation to justify investing in the new feed additive. Both parties must consider the volatility of many biological and economic factors included in their evaluations.

Each feed technology or product on the market claims to provide some form of benefit to swine producers when fed to the animal. These benefits may include a greater number of pigs born per litter, a more feed efficient pig that takes less feed to achieve the same weight, a faster growing pig that takes less time to get to market, a healthier pig that requires fewer injectable therapies or a herd with a greater survivability rate. Although each of these benefits are considered production improvements, they aren't necessarily proven to be economically advantageous and each of the products aren't guaranteed to perform the same in every production system.

Each feed technology comes with direct costs of adding a new ingredient to the diet. The new ingredient may also impact productivity and profitability indirectly as well for example, in the form of storage, increased labor to handle the ingredient, and the learning curve of how to incorporate the ingredient most efficiently. On the surface, the economic gain of the production improvement may be greater than the direct cost of the added feed ingredient, but there are many other considerations that need to be reviewed before determining if feeding the new product produces an economic return.

Costs of production highlighted by Widmar et al. (2011) include feed costs, veterinary services, barn space and utilities, labor, mortality, and marketing. The interest on the value of pigs on feed, grow-finish barn and equipment costs, and grow-finish labor are also indirect market hog costs. In production, there is a common motivation of spreading these costs over increasing

numbers of reproductive units. However, Widmar et al. (2011) evaluated the economic implications of increased litter sizes and found that these larger litter sizes also come with challenges of lighter birth and weaning weights, therefore slower growth rates, and potentially pigs with a lower immune status and greater chance of mortality. Considering all of these fluid components, calculating cost on a per pig marketed basis aids in on-farm decision making surrounding the addition of a new feed technology or not.

Boland et al. (1999) optimized feed formulation with phase feeding to determine what nutrient intake maximizes economic returns for producers rather than maximizing animal growth. These authors constantly considered the marginal return of feeding the animal more nutrients or the opportunity cost of selling the animals at the current weight and bringing in a new set of younger animals to take the cost of facility space. Boland et al. (1999) also determined the addition of protein or lysine had diminishing marginal returns and that there is a huge economic incentive for producers to adopt a multiple phase feeding program.

In regard to marketing, producers are incentivized for having a high proportion of a load of pigs hit the packer's target marketing weight or come very close to it. Producers are also monetarily penalized when they send pigs that are very heavy or very light in comparison to the target market weight. Additionally, carcass lean quantity is evaluated by packers. The percent lean of each carcass is calculated based on loin depth and fat depth at a given carcass weight. Carcasses below the percent lean cut off are discounted and carcasses above the cut off are assigned a carcass grade premium which is added to the base meat price. Typically, as the percent lean of the carcass increases, the carcass grade premium increases. Therefore, if a feed technology reduces the variation in market weights or increases carcass leanness at heavier weights, one must consider the

reduction in penalties that come from more homogenous pigs at marketing and the potential for greater carcass grade premiums.

Additional considerations include any local, state, or federal regulations that may be in place. For example, Boland et al. (1998) developed an optimization model to evaluate alternative feed ingredients, phytase and synthetic amino acids, to decrease excreted nitrogen and phosphorus in swine manure. These authors highlight that state regulations of nitrogen and phosphorus application on crop land indirectly apply a maximum on animal inventory for a producer when land is fixed. Boland et al. (1998) discussed that if a producer is constrained by land, it is economical to use a combination of technologies including phytase and synthetic amino acids to avoid an additional cost of more manure storage, even when the unit cost of the technologies is greater than the ingredients that they are replacing, e.g. corn or soybean meal.

As discussed, there is volatility in biological and economic factors associated with decision making under risk in swine production. An economic evaluation deeper than simply comparing economic gain to the cost of the added feed ingredient must be conducted for a technology developer to craft a plausible and convincing marketing strategy and for a producer to justify investing in a new feed additive technology.

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CHAPTER 2. FEEDING A WHOLE-CELL INACTIVATED *PICHIA GUILLIERMONDI* YEAST TO GESTATING AND LACTATING SOWS IN A COMMERCIAL PRODUCTION SYSTEM

2.1 Abstract

A total of 606 sows (PIC 1050) and their progeny (PIC 1050 × 280) were used to determine if feeding gestating and lactating sows a proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast product (WCY; CitriStim, ADM Animal Nutrition, Quincy, IL) improves sow and litter performance in a commercial production system. Once confirmed pregnant at d 35 post-breeding pregnancy check, sows were fed either a basal gestation control (CON) diet (0.55% SID Lysine) or the control diet fortified with 0.15% of the WCY replacing corn in the CON diet. Dietary treatments were also fed in lactation (1.05% SID Lysine) once sows were moved into farrowing crates on approximately d 112 of gestation until weaning. Sows supplemented with WCY in gestation and lactation had a greater number of total born piglets by 0.45 pigs ($P < 0.04$), piglets born alive (13.85 vs. 14.27; $P < 0.04$), heavier born alive litter weights ($P < 0.001$), and greater post cross-foster litter size ($P < 0.001$) compared to CON fed sows. Litter size at weaning was increased by 0.54 pigs when sows were fed WCY compared to CON ($P < 0.001$). However, litter weaning weights and 21-day adjusted litter weaning weights were similar ($P > 0.158$) with litter weights being numerically greater for the WCY sows. The average piglet weaning weights from CON fed sows were heavier by 0.35 kg compared to WCY ($P < 0.001$). This increase in body weight of piglets from CON fed sows is partially explained by their 0.93 days longer lactation ($P < 0.001$) and may also be due to the smaller litter size nursed throughout lactation. The percent of litters treated for scours decreased from 38.3% to 14.2% when sows were fed WCY ($P < 0.001$). The distribution of birth and weaning weights by body weight range was not different ($P > 0.2461$)

between treatments. In conclusion, feeding gestating and lactating sows a proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast product increased the number of piglets born and number weaned as well as decreased the prevalence of scours during lactation.

2.2 Introduction

Dietary yeast products fed to swine have been identified as potential growth promoting feed additives, which may also have beneficial effects on the pig's immune system and health (Shen et al., 2011). *Saccharomyces cerevisiae* is the common yeast species that has been studied for some time, however this yeast has a larger cell surface area and weaker hydrophobic properties compared to *Pichia guilliermondi* (Peisker et al., 2017). These physical differences may contribute to *Pichia guilliermondi* having a stronger ability to inhibit pathogenic bacteria from binding to small intestinal epithelium of swine and broiler chickens (Peisker et al., 2017). A proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast (WCY) is a co-product of citric acid fermentation and was fed in this experiment.

The yeast cell wall fraction of many yeast species represents approximately 20 to 30% of the dry weight of the yeast cell and consists of about 85 to 90% polysaccharide and 10 to 15% protein (Nguyen et al., 1998; Lesage and Bussy, 2006). Specifically, yeast cell walls are a source of mannan-oligosaccharides and β-glucans. Mannan-oligosaccharides positively impact gut health by beneficially shifting bacterial populations in the gut via the binding of bacterial pathogens in the intestine and preventing their adhesion to host epithelium (Kogan and Kocher, 2007). Beta glucans bind membrane receptors of innate immune cells, located in the small intestinal mucosal lining, which signals the immune system to prepare for potential pathogenic threats (Baert et al., 2015; Batbayar et al., 2012). The overall reduction in pathogenic stressors to the sow, the potential

shift in gut microbiota, and immune system activation may improve sow reproductive performance and her progeny's preweaning performance.

The objective of this study was to determine if feeding gestating and lactating sows a proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast product (CitriStim, ADM Animal Nutrition, Quincy, IL) improves sow and litter performance in a commercial production system. Performance parameters included litter characteristics at birth, birth weights individually and/or whole litter weights, individual or whole litter weaning weights, survival rate, and percent of litters treated for scours.

2.3   Materials and Methods

2.3.1   General

The experimental procedures used in this study were approved by the Purdue Animal Care and Use Committee (PACUC # 1909001949)**.** This study was conducted at Martin Family Farms (Williamsport, IN) breed-to-wean facility. Feed samples were analyzed by ADM Animal Nutrition (Quincy, IL).

2.3.2   Animals and Diets

A total of 606 sows (PIC 1050) and their progeny (PIC 1050 × 280) were used in this study. Treatments were gestation and lactation diets as a basal control (CON) diet or the control diet fortified with 0.15% CitriStim, a whole-cell inactivated yeast product (WCY; ADM Animal Nutrition, Quincy, IL), at the expense of corn in the CON diet. Sows were bred in stalls, group housed on d 35 of gestation post-pregnancy check, and fed the dietary treatments beginning at the time of group gestational housing. Group pens used electronic sow feeders (AP Schauer ESF Stations, AGCO, Duluth, GA) that delivered approximately 2.27 kg of feed per sow per day,

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varying based on the individual sow's body condition. Industry standard diet formulations were targeted to meet or exceed the swine nutrient requirements (NRC, 2012) for gestating and lactating sows (Table 2.1). The monthly average high temperatures when these sows were gestating were 27.3, 27.6, 28.0, 27.7, and 26.7°C from May to September of 2018, respectively.

Sows were moved into the farrowing crates on approximately d 112 of gestation and were allowed *ad libitum* access to lactation feed. Sows and litters for the piglet weight data were selected for the experiment in the order they farrowed as long as the number born alive was 8 or more piglets and no sow was above parity 7. Parity was equalized across treatments. When approaching the targeted number of 300 sows on test per treatment, sows were selected so that the parity distribution of the experiment resembled the farm's parity distribution from their electronic records program (PigKnows). The number of parity 2 sows for data collection were limited near the end of the data collection period. Therefore, we selected a few more parity 1, 3, and 4 sows to replace the unavailable parity 2 sows. The farm's parity distribution over the previous 6 months before the study started was: 20% parity 1, 18% parity 2, 12% parity 3, 12% parity 4, 11% parity 5, 11 % parity 6, 8% parity 7, and 8% parities 8 to 11.

Whole litter weights were collected on the first day of life (d 0) and on the day before weaning from about 200 litters per treatment. An additional 100 litters per treatment were tagged and weighed on an individual piglet bases on d 0 and the day before weaning. Weights were collected using Avery Weigh-Tronix digital scales (Fairmont, MN). Individual pigs were weighed with a 45.72 x 45.72 cm scale (model BS-1818-50-NO). Whole litters were predominantly weighed with a 60.96 x 60.96 cm scale (model BS-2424-200-NO). Two check weights (5 and 22.7 kg) were used to check the scales for accuracy before the start of each weigh day.

Day 0 care included an oral dose of Marquis (15% w/w ponazuril, Merial, Inc., Duluth, GA), 0.2 mL injectable Excede (ceftiofur crystalline free acid, Zoetis, Parsippany, NJ), and 2.0 mL injectable iron (100 mg/mL). Cross-fostering within treatments occurred after d 0 care and weights to equalize litter size within treatment. If litters were scouring, SpectoGard (spectinomycin, Bimeda, Dublin, Ireland) was administered orally between days 1 to 3 of life depending on the severity of the diarrhea (2 mL given once daily). Castration and tail docking occurred between d 3 and 7 depending on labor availability.

2.4 Chemical Analysis

All diets were formulated by United Animal Health (Sheridan, IN). Gestation and lactation diets were sampled at the sow farm and subsampled at Purdue University before being shipped to ADM Animal Nutrition (Quincy, IL) for analysis. Feed samples were analyzed for crude protein (AOAC 990.03, 2006), crude fat (AOAC 920.39, 2006), crude fiber (AOCS Ba6a-05, 2006), ash (AOAC 942.05, 2006), and moisture content (Shreve et al., 2006; Table 2.2).

2.5 Statistical Analysis

Data were analyzed as a completely randomized design using the MIXED procedure in SAS 9.4 (SAS Institute, Inc., Cary, NC) with sow or piglet as the experimental unit. For sow, litter, and individual piglet performance, dietary treatment of the sow and parity (1 to 7) were fixed effects. Stillborn and mummy data were square root-transformed to meet assumptions of normality. When modeling individual birth weights, dietary treatment and parity group (parity 1, 2, and 3+) were included as fixed effects with total born used as a covariate. When modeling adjusted 21 day weaning weights of individual piglets, dietary treatment, parity group, birth weight within treatment, post cross-foster litter size, and if the piglet was cross-fostered or not were all included.

The GLM procedure in SAS 9.4 was used to simply model adjusted 21 day weaning weights of individual piglets, including birth weight and dietary treatment as independent variables. The FREQ procedure in SAS 9.4 was used to generate a frequency plot and chi-square analysis of the distribution of birth and weaning weights separated by dietary treatment. To assess differences among parities the PDIFF option of the LS Means function was used for the multiple comparisons among parities. Differences were considered significant at $P \leq 0.05$ and trends at $0.05 < P \leq 0.10$.

2.6 Results

2.6.1 Dietary Treatment Effects

There were no dietary treatment × parity interactions for sow, litter, and individual piglet performance ($P > 0.05$; Table 2.3 and 2.4), therefore the interaction was removed from the model. Results presented in Table 2.3 include all 606 sows and litters, about 2/3 of which piglets were weighed on a whole litter bases and 1/3 of the litters were weighed on an individual piglet basis. Sows supplemented with WCY in gestation and lactation had a greater number of total piglets born (+0.45 pigs; $P < 0.04$), a greater number of pigs born alive (0.42 pigs; $P < 0.04$), and a 1.02 kg heavier born alive litter weight compared to CON fed sows ($P < 0.001$; Table 2.3). At 12 hours after birth, there was no difference ($P > 0.05$) in survival rate between treatments, and therefore sows fed WCY had a larger litter size by 0.44 pigs ($P < 0.027$) and 0.99 kg heavier litter weight ($P < 0.001$) compared to CON. Post cross-foster litter size was also greater ($P < 0.001$) for WCY fed sows by 0.68 pigs. Litter size at weaning was increased by 0.54 pigs when sows were fed WCY compared to CON ($P < 0.001$).

Piglet body weights were calculated by dividing the whole litter weight by the number of piglets in the litter at each time point. Average piglet born alive weight and weight at 12 hours was not different due to treatment ($P > 0.12$; Table 2.3). The average piglet weaning weights from

CON fed sows were heavier by 0.35 kg compared to WCY ($P < 0.001$). This increase in body weight of piglets from CON fed sows may be due to the smaller litter size throughout lactation of the CON sows. However, WCY litters were 0.93 days younger than CON ($P < 0.001$), therefore average weaning weights of litters from both treatments were adjusted to 21 days using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 (National Swine Improvement Federation, 1987). After this adjustment, the average 21-day adjusted pig weaning weight was still greater for CON litters compared to WCY by 0.18 kg ($P < 0.012$), however the whole litter 21-day adjusted weaning weights were similar ($P > 0.158$) but numerically greater for the WCY sows. The percent of litters treated for scours decreased from 38.3% to 14.2% when sows were fed WCY ($P < 0.001$).

A subset of about 100 litters per dietary treatment were weighed on an individual piglet basis (Table 2.4). Piglets were weaned 0.89 days earlier from WCY sows compared to CON ($P < 0.001$) and the percent of litters treated for scours decreased from 45.55% to 12.24% when sows were fed WCY ($P < 0.001$). There was a trend for increased born alive litter weight ($P < 0.063$) and 12-hour litter weight ($P < 0.109$) but decreased individual piglet weaning weight ($P < 0.071$) when sows were fed WCY compared to the control diet. However, when weaning weights were adjusted to 21 days, individual pig weaning weights were nearly identical between treatments. This 100 sows/treatment subset had a similar, though non-significant, 0.40 piglet increase in total born ($P < 0.320$; Table 2.4) whereas the total dataset with about 300 sows/treatment had a 0.45 piglet increase in total born ($P < 0.04$; Table 2.3).

As an added reference to the sow and litter performance during this study, all sows that were fed the WCY and CON diets during gestation and lactation during the same dates as our data was collected and extracted from the farm's record program (PigKnows) and is presented in Table

2.8. This data, while not statistically analyzed, reports a similar 0.47 total pigs born alive and 0.54 pigs weaned increase when fed the WCY product compared to the CON fed sows.

2.6.2   Parity Effects

There were no parity by treatment interactions, therefore the parity effects are being provided as additional information gleamed from the data in this experiment and had no effect on the dietary treatment results presented above. For the whole 600+ sow data set, there was an effect of parity ($P < 0.049$) for all performance parameters except for the number of mummies in a litter ($P < 0.244$) and weaning age tended to be affected by parity ($P < 0.092$; Table 2.5). Parity 1 sows had fewer total born piglets compared to all other parities ($P < 0.008$) and parity 6 sows had fewer piglets total born compared to parity 5 sows ($P < 0.033$). Parity 1 sows had fewer born alive piglets and a lighter born alive litter weight compared to parities 2, 3, 4, and 5 ($P < 0.01$). Parity 6 and 7 sows had fewer born alive piglets compared to parities 3 and 5 ($P < 0.033$). Parity 7 sows had a smaller percent born alive compared to all other parities ($P < 0.043$), except parity 6 ($P > 0.05$). Parity 5 and 6 sows had a smaller percent born alive compared to parity 1 sows ($P < 0.017$). Parity 6 and 7 sows had a smaller percent born alive compared to parity 1 and 2 sows ($P < 0.013$). Parity 1 sows had a fewer number and percent stillborn piglets compared to all other parities ($P < 0.045$), except parity 2 ($P > 0.05$). Parity 7 sows had more stillborn pigs compared to parities 1 and 2 ($P < 0.007$). Parity 7 sows had a larger percent stillborn compared to parities 1, 2, and 3 ($P < 0.039$). Parity 5 sows had a lighter born alive litter weight compared to parities 2 and 3 ($P < 0.003$). Parity 1 and 6 sows had a lighter born alive litter weight compared to parities 2, 3, 4, and 5 ($P < 0.028$). Parity 7 sows had a lighter born alive litter weight compared to parities 2, 3, and 4 ($P < 0.001$).

Parity 1 sows had a higher percent survival at 12 hours before cross-foster compared to parities 4, 5, and 6 ($P < 0.028$) and parity 2 sows were greater percent survival than parity 6 ($P <$

0.045). Parity 1 sows had a lighter litter weight at 12 hours compared to parities 2, 3, 4, and 5 (P < 0.001). Parity 2 and 3 sows had a heavier litter weight at 12 hours compared to parities 5, 6, and 7 (P < 0.003). Parity 4 sows had a heavier litter weight at 12 hours compared to parities 6 and 7 (P < 0.003) and parity 5 greater than parity 6 (P < 0.013). Parity 1 and 6 sows had fewer pigs in the litter at 12 hours compared to parities 2, 3, and 5 (P < 0.006). Parity 4 sows had fewer pigs in the litter at 12 hours compared to parity 3 (P < 0.029). Parities 2 and 3 sows had more pigs in the litter post cross-foster compared to parities 4, 6, and 7 (P < 0.040).

Parities 1 and 2 sows had a higher percent survival from cross-foster to weaning compared to parities 3 and 7 (P < 0.026). Parity 1 sows had a lighter litter weaning weight and 21-day adjusted litter weaning weight compared to all other parities (P < 0.001), except parity 7 (P < 0.284). Parity 2 sows had a heavier litter weaning weight compared to parities 5, 6, and 7 (P < 0.002). Parities 3 and 4 had heavier litter weaning weights compared to parities 6 and 7 (P < 0.010), and parity 5 greater than parity 7 (P < 0.003). Parities 2 and 4 sows had a heavier 21-day adjusted litter weaning weight compared to parities 5, 6, and 7 (P < 0.034). Parity 3 sows had a heavier 21-day adjusted litter weaning weight compared to parities 6 and 7 (P < 0.006) and parity 5 sows were greater than parity 7 (P < 0.006). Parity 2 sows weaned the most pigs and had a greater number of pigs weaned compared to parities 4, 5, 6, and 7 (P < 0.009). Parity 1 and 3 sows had a greater number of pigs weaned compared to parities 6 and 7 (P < 0.007). Parity 4 and 5 sows had a greater number of pigs weaned compared to parity 7 sows (P < 0.017).

Parity 1, 5, and 7 sows had a lighter average pig born alive weight and average pig weight at 12 hours compared to parities 2, 3, and 4 (P < 0.003). Parities 2 and 4 sows had a heavier average pig born alive weight and average pig weight at 12 hours compared to parities 5, 6, and 7 (P < 0.010). Parity 3 sows had a heavier average pig weight at 12 hours compared to parities 5, 6, and

7 ($P < 0.047$). Parity 1 sows had a lighter average pig weaning weight and lighter 21-d adjusted average pig weaning weight compared to all other parities ($P < 0.001$). Parity 4 sows had a heavier average pig weaning weight compared to parities 3, 5, 6, and 7 ($P < 0.025$). Parity 4 sows had a heavier 21-d adjusted average pig weaning weight compared to all other parities ($P < 0.031$). Parity 1 sows had a greater percent of litters treated for scours compared to all other parities ($P < 0.001$). Parity 2 sows had a greater percent of litters treated for scours compared to parity 6 sows ($P < 0.030$).

In the individual piglet subset of about 100 litters per treatment, there was a main effect of parity ($P < 0.044$) for all performance parameters except for percent born alive and stillborn, number stillborns and mummies, percent survival before and after cross-foster, post cross-foster and weaning litter size, and weaning age ($P > 0.05$; Table 2.6). Parity 1 sows had fewer total born piglets compared to parities 2, 3, 4, and 5 ($P < 0.012$). Parity 3 sows had more total born piglets compared to parity 6 ($P < 0.015$). Parity 1 sows had fewer piglets born alive compared to parities 2, 3, and 5 ($P < 0.04$) and parity 6 had fewer than parity 3 ($P < 0.006$). Parity 1 sows had a lighter born alive litter weight and lighter litter weight at 12 hours compared to parities 2, 3, and 4 ($P < 0.003$). Parities 2, 3, and 4 sows had a heavier born alive litter weight and heavier litter weight at 12 hours compared to parities 6 and 7 ($P < 0.029$) and litters of parity 3 sows were heavier than parity 5 sows for both weights ($P < 0.026$). Parity 1 sows had fewer pigs in the litter at 12 hours compared to parities 3 and 5 ($P < 0.030$). Parity 3 sows had more pigs in the litter at 12 hours compared to parity 6 sows ($P < 0.010$).

Parity 1 sows had a lighter litter weight post cross-foster compared to parity 2, 3, and 7 sows ($P < 0.040$). Parities 2 and 3 sows had heavier litter weights post cross-foster compared to parity 5, 6, and 7 sows ($P < 0.017$) and parity 4 sows heavier than parity 7 sows ($P < 0.012$). Parity

1 sows had a lighter litter weaning weight and 21-d adjusted litter weaning weight compared to all other parities ($P < 0.013$), except parity 7 ($P < 0.272$). Parity 2 sows had a heavier litter weaning weight compared to parities 5, 6, and 7 ($P < 0.025$) and parity 4 sows heavier litter weaning weight than parity 7 sows ($P < 0.013$). Parity 2 sows had a heavier 21-d adjusted litter weaning weight compared to parities 6 and 7 ($P < 0.017$).

Parity 1 sows had a lighter piglet born alive weight, lighter piglet weight at 12 hours, and lighter pig weight after cross-foster compared to parity 2 sows ($P < 0.034$). Parities 2, 3, and 4 sows had a heavier piglet born alive weight compared to parity 5 and 7 sows ($P < 0.038$). Parities 2 and 4 sows had a heavier piglet weight at 12 hours and heavier pig weight after cross-foster compared to parity 5 and 7 sows ($P < 0.042$) and piglet weights at 12 hours from parity 3 sows were heavier than parity 7 sows ($P < 0.032$). Piglets from parity 3 sows were heavier after cross-foster compared to parity 5 sows ($P < 0.028$). Parity 1 sows had a lighter pig weaning weight and lighter 21-d adjusted weaning weight compared to all other parities ($P < 0.006$). Parity 4 sows had a heavier pig weaning weight and heavier 21-d adjusted weaning weight compared to parity 5 and 7 sows ($P < 0.028$). Parity 1 sows had a greater percent of litters treated for scours compared to all other parities ($P < 0.001$).

2.6.3 Modeling Individual Pig Data

The total piglets with individual birth weights was 2960 piglets of which 2507 survived to weaning (84.7%). Individual birth weight means were adjusted for parity group (1, 2, and 3+) as a fixed effect, and total born was used as a covariate. Although in Tables 2.3 and 2.4 there was no difference in piglet born alive birth weights between treatments ($P > 0.12$), when adjusting for parity group and total born, piglets from sows fed WCY were heavier compared to CON (1.43 kg vs. 1.39 kg; $P < 0.001$). For every 1 more pig born, birth weight per pig decreased 0.037 kg ($P <$

0.001). Piglets born to parity 1 sows were 0.092 kg lighter compared to parity 2 and 3+ sows ($P <$ 0.001).

To visualize the distribution of birth weights, individual piglet birth weights were divided into ranges in 0.09 kg increments. The distribution of birth weights by weight range was not different ($P < 0.2461$; Figure 2.1) between sow dietary treatments. Individual piglet weaning weights were divided into ranges separated at 0.50 kg increments. The distribution of weaning weights by weight range was not different ($P < 0.3551$; Figure 2.2) between sow dietary treatments. Although not statistically analyzed, the percent of pigs weaned less than 3.60 kg of body weight was 5.06% for control fed sows and 6.34% for WCY fed sows.

To visualize the relationship between birth weight and mortality, raw data was used to group birth weights into 0.09 kg weight ranges and the proportion of pigs that died within each weight range was plotted (Figure 2.3). Although not statistically analyzed, visually the percent mortality decreased as birth weight increased. Pigs weighing less than 0.61 kg at birth had a less than 40% survival rate.

As noted previously, due to a difference in wean age between treatments, weaning weights were adjusted to a standard 21 days of age. Weaning weights were also adjusted for dietary treatment, parity group (1, 2, and 3+), birth weight within treatment, post cross-foster litter size, and if the piglet was cross-fostered or not. There was no treatment effect ($P > 0.05$) on weaning weights, however there were effects due to all other factors listed above ($P < 0.001$). Pigs born to parity 1 sows weighed 0.837 kg lighter at weaning compared to parities 2 and 3+ ($P < 0.001$). For every 0.1 kg increase in birth weight, pigs weighed 0.441 kg heavier at weaning ($P < 0.001$). For every 1 pig increase in litter size post cross-foster, pigs weighed 0.12 kg lighter at weaning ($P <$ 0.001). Lastly, if pigs were cross-fostered to another litter, they were 0.58 kg lighter at weaning (P

< 0.001). This decrease in weaning weight due to cross-fostering held true for pigs cross-fostered up to 2.02 kg of birth weight (Table 2.7).

To visualize the relationship between 21-day adjusted weaning weight and birth weight, birth weights were grouped into 0.09 kg weight ranges and the average adjusted weaning weight within each birth weight range was plotted for each dietary treatment (Figure 2.4). When modeling 21-day adjusted weaning weight using a general linear model with birth weight and dietary treatments as independent variables, the overall model was significant ($P < 0.001$) where weaning weight increased as birth weight increased for both dietary treatments. Within the model, birth weight was significant ($P < 0.001$), however treatment ($P < 0.26$) and the interaction of birth weight and treatment ($P < 0.16$) were not significant. Birth weight and dietary treatment explained 39.6% of the variation in 21-day adjusted weaning weight ($R^2 = 0.396$; Figure 2.5).

2.7   Discussion

Research investigating *Pichia guilliermondi* products fed to sows is fairly limited when compared to the common yeast, *Saccharomyces cerevisiae*, derived products. Veum et al. (1995) fed sows *Saccharomyces cerevisiae* yeast culture from d 60 of gestation through d 21 of lactation at 0, 0.5, 1.0, or 2.0% of complete feed and observed no differences in sow reproductive performance. When sows were fed a *Saccharomyces cerevisiae* fermentation product 5 d before breeding through lactation (12 g/d in gestation and 15 g/d in lactation), Shen et al. (2011) observed no differences in reproductive performance of the sow. However, sows fed *Saccharomyces cerevisiae* did tend to increase total litter weight at weaning and litter body weight gain compared to litters from sows not fed the yeast product which could be due to yeast fed sows weaning numerically more pigs (10.3 vs. 9.2; Shen et al., 2011).

Bass et al. (2019) fed sows an inactivated, whole yeast cell *Pichia guilliermondi* product at 0, 0.1, or 0.2% of complete feed within 24 hours of breeding through the end of the 21-day lactation period. Sows were housed in individual gestation stalls and individual farrowing crates. Inclusion of *Pichia guilliermondi* in the sow diet linearly increased the number of piglets born alive per litter as the dietary inclusion increased (12.49, 13.33, and 13.43 for 0, 0.1, or 0.2%, respectively; Bass et al., 2019).

The differences in reproductive performance when feeding yeast products may be due to differing physical properties between the yeast species. *Saccharomyces cerevisiae* has a larger cell surface area and weaker hydrophobic properties compared to *Pichia guilliermondi* (Peisker et al., 2017). These physical differences may contribute to *Pichia guilliermondi* having a stronger ability to inhibit pathogenic bacteria from binding to small intestinal epithelial cells of swine and broiler chickens via the yeast cell wall mannan-oligosaccharide component (Kogan and Kocher, 2007; Peisker et al., 2017). Beta glucans, the other major yeast cell wall component, bind membrane receptors of innate immune cells. These cells, located in the small intestinal mucosal lining, signal the immune system to prepare for potential pathogenic threats (Baert et al., 2015; Batbayar et al., 2012). The overall reduction in pathogenic stressors to the sow, the potential shift in gut microbiota, and immune system activation may improve reproductive performance of sows fed *Pichia guilliermondi*.

Data from the current study supports findings of Bass et al. (2019). The addition of the inactivated, whole yeast cell *Pichia guilliermondi* product at 0.15% of the sow diet in this study resulted in a greater number of piglets born alive (13.85 vs. 14.27 for 0 or 0.15%, respectively). The experiment by Bass et al. (2019) was comprised of about 30 sows per treatment in a university research setting using gestation stalls whereas the current study collected data on about 300 sows

per treatment in a commercial swine production system where sows were housed in gestation pens. It is interesting to note that an increase in the number of pigs born alive was consistent across the two very different studies when an inactivated, whole yeast cell *Pichia guilliermondi* product was fed.

Sows in the current study were housed in gestation stalls without yeast supplementation for 35 d before being confirmed pregnant. Once confirmed pregnant, they were moved into gestation pens with electronic sow feeders and fed the control diet or the diet supplemented with 0.15% *Pichia guilliermondi* whole cell yeast (WCY). Sows fed the WCY had an increased number of pigs total born and born alive compared to CON fed sows. Because sows were fed the same diets until d 35 of gestation, it is logical to assume the two groups of sows had similar ovulation rates and conceptus implantation success because both of these events occur before d 35 of gestation (Ziecik et al., 2011).

According to Ford et al. (2002), 30-50% of fertilized ova do not make it to term in U.S. pig breeds and 75% of these losses occur before d 30 of gestation. There are two additional periods of significant conceptus loss in sows which occur from d 30-40 and d 90-114 of gestation when uterine capacity, including space and nutrients, becomes a critical limiting factor due to competition among litter mates (Ford et al., 2002). These two time periods are defined by abrupt increases in surface area between the placenta and uterine luminal surface where microscopic interdigitations develop known as primary and secondary rugae (Bjørkman and Dantzer, 1987). The exact mechanism of how the mannan-oligosaccharide and β-glucan cell wall components of the WCY may impact conceptus survival due to uterine capacity at these two time periods is unclear. It is also possible that this dietary yeast product, a by-product of citric acid fermentation,

contains other undiscovered active *Pichia guilliermondi* culture components that may be residual from the citric acid fermentation process.

Bass et al. (2019) observed an increase of 0.94 pigs born alive when sows were fed 0.2% *Pichia guilliermondi* starting at breeding. The current study fed 0.15% *Pichia guilliermondi* WCY to sows starting on d 35 of gestation and an increase of 0.42 pigs born alive was observed. The response observed by Bass et al. (2019) was two times the response observed in the current study which could be due to the longer duration of feeding the *Pichia guilliermondi* WCY and feeding it during a critical time frame of embryo loss before d 30 of gestation.

As previously stated, it is logical to assume the two groups of sows in the current experiment had similar ovulation rates and conceptus implantation success because both of these events occur before d 35 of gestation (Ziecik et al., 2011). Therefore, one might expect to observe an increased number of mummies in litters of the CON sows because they had fewer total born piglets. In this study, there was no difference in the number of mummies per litter due to dietary treatment. According to Flowers (2019), calcification of fetal bones begins at about d 38-45 of gestation and fetal losses after this point will result in formation of a mummified fetus due to the failure of the uterus to reabsorb bone. It is possible that the CON fed sows had more conceptus loss from d 35-45 in which those losses did not result in formation of mummies. It is also possible that mummies formed a short time period after d 45 may not have been large enough to be detected in a commercial production system where employees are more concerned with assisting the next sow than looking for small mummies.

In the current study, sows fed *Pichia guilliermondi* gave birth to more pigs resulting in a heavier born alive litter weight compared to CON fed sows. There was no difference in litter size at birth or litter birth weight when Kim et al. (2008) supplemented sow diets with a *Saccharomyces*

cerevisiae yeast culture starting on d 35 of gestation (12 g/d) until d 21 of lactation (15 g/d), however authors did observe an increased litter wean weight due to supplemental *Saccharomyces cerevisiae* yeast culture. Although milk samples were not collected, Kim et al. (2008) speculated the increased litter wean weight could have been due to an increased production of milk and/or of higher quality milk when sows were fed the yeast culture product. In contrast, this experience observed no difference in 21-day adjusted litter weaning weights due to dietary treatment.

In this experiment, pre-cross-fostering survival was not different between dietary treatments and cross-fostering within treatments occurred after d 0 care and weights to equalize litter size. Due to WCY fed sows having larger litter sizes at birth and no difference in survival before cross-fostering, CON fed sows had 0.68 fewer pigs per litter to nurse during lactation. Sows fed the WCY diet weaned 0.54 more pigs per litter compared to CON fed sows. The smaller number of pigs on the CON sows were potentially consuming more milk nutrients per pig which could be one reason why individual CON pigs were heavier at weaning.

Additionally, 38% of CON litters ($n = 300$) were treated for *E. coli* scours whereas only 14% of WCY litters ($n = 306$) were treated. Litters with diarrhea between days 1 to 3 of life received at least one dose of spectinomycin. Although diarrhea likely had negative impacts on growth performance up to the first week of life, the antibiotic treatment could have helped eliminate other subclinical challenges in the farrowing house allowing pigs from CON fed sows to grow better thereafter to weaning. There was no difference in pre-weaning mortality between dietary treatments. However, if pigs did not receive spectinomycin on this farm, a decreased survival to weaning likely would have been observed for CON litters compared to WCY litters. Although not specific to *E. coli* scours, a Swine 2012 USDA report found that 10% of all preweaning mortality is from scours (USDA, 2015).

In recent decades, genetic selection, advanced nutrition, and improved reproductive technologies have successfully increased litter sizes of the modern sow to 12-15+ piglets born alive. The increased litter sizes have resulted in a greater variation of within litter birth weights which is also accompanied by a greater proportion of light weight pigs (less than 1 kg BW; Quesnel et al., 2008). Although previous studies have reported a decrease in pigs born weighing less than 0.91 kg when an inactivated, whole yeast cell *Pichia guilliermondi* product was fed (Bass et al., 2019), data from this study suggested no difference in the distribution of birth weights.

Lighter birth weight pigs have a greater chance of pre-weaning mortality. These small pigs loose body heat more rapidly, have smaller glycogen pools, and smaller stomachs which limit colostrum consumption especially when trying to compete within a large litter (Theil et al., 2012). In this study, pigs weighing less than 0.61 kg at birth had a less than 40% survival rate. In agreement, Feldpausch et al. (2019) evaluated data including 4,068 piglets from 394 litters on four commercial farms. Authors observed that 15.2% of pigs weighed less than or equal to 1.11 kg at birth and these pigs had a 34.4% mortality rate representing 43% of all pre-weaning mortalities.

Differences in reproductive and litter performance were detected due to dietary treatment as well as differences due to parity of the dams. Parity 1 sows had the fewest pigs total born per litter (13.7) compared to all other parities (14.7 to 15.7) and fewer pigs born alive per litter (13.2) than parities 2 through 5 (14.0 to 14.7). In agreement, with approximately 11.5 vs. 10.5 pigs born alive, sows in parities 2 through 5 had larger litter sizes than parity 1 and parity 8 sows (Koketsu and Dial, 1997). Although litter sizes have increased with genetic selection, this general trend of parity 3 to 5 sows being the most productive is still true today (Sell-Kubiak et al., 2019).

Litter sizes may be smaller due to parity 1 sows having a smaller uterine capacity which limits the number of developing fetuses (Sell-Kubiak et al., 2019). In geriatric sows that are parity

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6 and older, lower reproductive performance can be attributed to decreased ovulation and fertilization rates in aged sows and they tend to have a higher rate of embryonic mortality due to slower responses to fetal demands for uterine space (Koketsu et al., 2017). Geriatric sows also have more stillborn piglets due to slower responses to stimuli during the parturition process (Koketsu et al., 2017). This was also observed in the present study where parity 7 sows had more stillborn pigs per litter (1.4) compared to parities 1 and 2 (0.55 and 0.70, respectively).

Kim et al. (2008) observed a difference in litter birth and weaning weights due to parity, where the primiparous sows produced litters with a lighter birth weight compared to multiparous sows which contained parities 2 through 12. Also, litters from primiparous sows were heavier at weaning than multiparous sows, but the heavier weaning weight could be due to about a half of a pig increase in litter size at weaning for litters from primiparous sows. In the current study when all 606 sows were included, there was a parity main effect for born alive litter weight and 21-day adjusted litter weaning weight. In agreement with Kim et al. (2008), parity 1 sows had a lighter born alive litter weight compared to parities 2, 3, 4, and 5, however there was no difference in born alive litter weight between parities 1, 6, and 7 in this experiment. In contrast to Kim et al. (2008), parity 1 sows in this experiment had a lighter 21-day adjusted litter weaning weight compared to all other parities, except parity 7. Interestingly, litter size at weaning was greater for parity 1 sows compared to parities 6 and 7, therefore parity 1 sows simply weaned smaller pigs than all other parities.

The observation of parity 1 sows having smaller pigs at birth and at weaning is common (Craig et al., 2017). After conception, gilts must continue to grow and prioritize nutrients toward their own growth and development (e.g. mammary growth) as well as fetal growth (Theil et al., 2012). Therefore, if litter sizes were similar, it is logical that multiparous sows that prioritize more

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nutrients toward fetal growth would have larger pigs at birth and larger framed, later parity, sows would have the capacity to carry heavier litters. Considering weaning weight, a parity 1 sow compared to a multiparous sow will be physically smaller and therefore will not have as large of a stomach capacity for feed intake in lactation to support milk production. Milk yield of multiparous sows is typically greater than milk yield of primiparous sows which results in multiparous sows raising heavier pigs to weaning (Quesnel et al., 2015). Additionally, multiparous sows have more and better developed mammary glands that produce more milk compared to primiparous sows (Farmer, 2018).

There was also an effect of parity on the percentage of litters treated for scours between d 1 and 3 of life. Parity 1 litters were treated more often for scours than any other parity which may be due to parity 1 sows providing less passive immunity to their litters. Quesnel (2011) measured colostrum concentrations of immunoglobulin G (IgG) at 24 hours after birth and found that IgG concentrations were greater in older sows than primiparous sows.

2.8   Conclusion

In conclusion, the addition of WCY to sows in gestation and lactation increased total number born by about one-half of a pig which increased the born alive litter weight compared to CON fed sows. Litter size at weaning was increased by about one-half of a pig when sows were fed WCY compared to CON. However, pigs born to WCY sows had lighter weaning weights compared to CON which may be due to their increased litter size and potential limits of this sow's genetics or nutrition fed to meet the increased milk demand of the increased litter size. The percent of litters treated for scours decreased dramatically due to feeding WCY, adding to the economic, labor savings, and piglet welfare potential benefits of feeding WCY.

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Table 2.1 Sow diet composition (as-fed basis)[1]

Ingredient, %	Control (CON)		CitriStim (WCY)	
	Gestation[2]	Lactation[3]	Gestation[2]	Lactation[3]
Corn	67.03	56.93	66.88	56.78
Soybean meal, 47% CP	4.56	26.89	4.56	26.89
DDGS, 7% Fat	22.50	10.00	22.50	10.00
Choice white grease	------	2.50	------	2.50
Soybean Hulls	2.50	------	2.50	------
Limestone	1.52	1.35	1.52	1.35
Monocalcium Phos. (21% P)	0.77	0.86	0.77	0.86
Salt	0.05	0.11	0.05	0.11
L-Lysine-HCl	0.28	0.28	0.28	0.28
L-Threonine	0.03	0.07	0.03	0.07
L-Tryptophan	0.01	------	0.01	------
Superior Sow 12 Vitamin and Mineral Premix[4]	0.60	0.60	0.60	0.60
Epson Plus Pak[5]	------	0.25	------	0.25
Choline Chloride (60%)	0.10	0.10	0.10	0.10
Visano Sow[6]	0.05	0.05	0.05	0.05
NAT-P-E 2500[7]	------	0.01	------	0.01
CitriStim[8]	------	------	0.15	0.15
Total	100.00	100.00	100.00	100.00
Calculated analysis[9]				
ME, Kcal/kg	3076.8	3238.7	3076.8	3238.7
Crude Protein, %	13.25	19.31	13.25	19.31
Crude Fat, %	3.64	5.65	3.64	5.65
Digestible lysine, %	0.55	1.05	0.55	1.05
Digestible lysine:ME, g/Mcal	1.79	3.24	1.79	3.24
Digestible methionine+cystine:lysine, %	70.00	50.00	70.00	50.00
Digestible threonine:lysine, %	70.00	63.00	70.00	63.00
Digestible tryptophan:lysine, %	18.00	18.50	18.00	18.50
Ca, %	0.85	0.85	0.85	0.85
P, %	0.56	0.59	0.56	0.59
Available P, %.	0.45	0.45	0.45	0.45

[1]Formulations were completed by United Animal Health (Sheridan, IN).

[2]Diets were fed from d 35 post-pregnancy check until they were loaded into farrowing crates on approximately d 112 of gestation.

[3]Diets were fed from d 112 of gestation until weaning.

[4] Provided per kg of diet: vitamin A, 11,161 IU; vitamin D_3, 2545 IU; vitamin E, 66 IU; vitamin K, 1.42 mg; riboflavin, 6.6 mg; pantothenic acid, 23.6 mg; niacin, 44.2 mg; B_{12}, 31μg; biotin, 0.44 mg; folic acid, 1.62 mg; thiamine, 0.25 mg; pyrdoxine-B_6, 0.25 mg; iron, 129 mg; zinc, 125 mg; manganese, 60 mg; copper, 20 mg; iodine, 1.26 mg; selenium, 0.3 mg; cobalt, 0.02 mg; calcium, 0.095%; sodium, 0.08%, chloride, 0.12%; phytase, 371 FTU; and estimated 0.12% phosphorus release from the phytase.

[5]Provided per kg of diet: potassium, 0.057%; magnesium, 0.012%; sodium, 0.012%; sulfur, 0.030%; chloride, 0.039%.

[6]Visano Sow is a direct fed microbial (DFM) for sows from United Animal Health. It is a multi-strain *Bacillus spp.*

[7]Natuphos E phytase enzyme (*Aspergillus niger*; 2500 FTU/g, BASF, Florham Park, NJ) provided 250.7 FTU/kg phytase activity in lactation diets.

[8]CitriStim (ADM Animal Nutrition, Quincy, IL) is a proprietary strain of *Pichia guilliermondi*, a whole-cell inactivated yeast product. Nutrient value was assumed to be equal to corn that it replaced in the control diet.

[9]Calculated nutrients were targeted to meet or exceed the NRC 2012. Nutrient Requirements of Swine. 11th ed. Natl. Acad. Press, Washington, DC.

Table 2.2 Analyzed sow diet composition (as-fed basis)[1]

Item	Control (CON)		CitriStim (WCY)	
	Gestation	Lactation	Gestation	Lactation
Crude Protein, %	12.71	20.60	13.63	19.77
Crude Fat, %	3.70	4.70	3.44	5.10
Crude Fiber, %	3.83	3.07	4.05	3.36
Ash, %	4.50	5.16	4.83	4.98
Moisture, %	13.08	14.68	12.49	12.29

[1] Samples were collected at Martin Family Farms (Williamsport, IN), subsampled at Purdue University, and shipped to ADM Animal Nutrition (Quincy, IL) for analysis.

Table 2.3 Effects of feeding a whole-cell inactivated yeast product (WCY) to gestating and lactating sows on sow and whole litter performance in a commercial production system[1]

	Diet [2]		SEM	Probability, $P <$	
	Control (CON)	CitriStim (WCY)		Treatment	Parity
Sows, *n*	300	306	---	---	---
Parity	3.53	3.57	---	---	---
Litter characteristics					
Total born,[3] n	14.78	15.23	0.158	0.040	< 0.001
Born alive, n	13.85	14.27	0.146	0.039	< 0.001
Born alive, %	94.17	94.04	0.447	0.832	< 0.001
Stillborn,[4] *n*	0.93	0.96	0.076	0.492	0.001
Stillborn,[4] %	5.82	5.96	0.441	0.530	0.002
Mummies,[4] n	0.33	0.33	0.045	0.458	0.244
Born alive litter weight, kg	18.83	19.85	0.192	< 0.001	< 0.001
12-hour measurements[5]					
Survival before cross-foster, %	96.00	96.25	0.387	0.633	0.049
Litter weight, kg	18.53	19.52	0.195	< 0.001	< 0.001
Litter size, n	13.26	13.70	0.142	0.027	< 0.001
Post cross-foster litter size, n	13.14	13.82	0.120	< 0.001	0.005
Weaning measurements[6]					
Sows weaned, n	295	294	---	---	---
Weaning age	19.42	18.49	0.062	< 0.001	0.092
Survival after cross-foster, %	88.44	87.59	0.663	0.354	0.029
Litter weight, kg	65.54	64.41	0.664	0.220	< 0.001
21-day adjusted litter weight,[7] kg	71.47	72.89	0.722	0.158	< 0.001
Litter size, n	11.52	12.06	0.100	< 0.001	< 0.001
Piglet body weight,[8] kg					
Born alive	1.40	1.43	0.012	0.125	< 0.001
12-hour	1.41	1.44	0.012	0.122	< 0.001
Weaning[6]	5.71	5.36	0.045	< 0.001	< 0.001
21-day adjusted weaning	6.24	6.06	0.049	0.012	< 0.001
Litter treated for scours,[9] %	38.33	14.18	2.406	< 0.001	< 0.001

(Table continues)

[1] A total of 606 sows and their progeny (PIC 1050 × 280) were used to determine if feeding a whole-cell inactivated yeast product (WCY; ADM Alliance Nutrition, Inc., Quincy, IL), to sows in gestation and lactation diets influences sow and pre-weaning pig performance in a commercial production system.

[2] Two maternal dietary treatments were fed. In gestation, post pregnancy check on d 35, and in lactation, sows were fed a control basal diet or the basal diet with 0.15% added WCY.

[3] Total born was calculated as born alive plus stillborn piglets and does not include mummies.

[4] Data were square root-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and p-values represent the square root-transformed data.

[5] The 12-hour measurements were collected between 1 and 12 hours of birth from living piglets and before cross-fostering. The 12-hour survival rate does not include stillborn mortality.

[6] Weaning weights were collected 1 day before actual weaning age.

[7] Due to a significant difference in weaning age, weaning litter weights were adjusted for weaning age to a 21-day basis using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 written by the National Swine Improvement Federation in December 1987.

[8] Piglet body weights were calculated by dividing the litter weight by the number of pigs in the litter. Born alive is the litter weight at birth not including stillborn pigs. The 12-hour weight is the litter weight before cross-fostering, which would exclude any crushed or non-viable pigs at that time. Weaning is the litter weight at weaning. The 21-day adjusted weaning is the 21-day adjusted litter weight divided by the number of pigs at weaning.

[9] Percent of litters treated for scours at least 1 time.

Table 2.4 Effects of feeding a whole-cell inactivated yeast product (WCY) to gestating and lactating sows on sow and individual piglet performance in a commercial production system[1]

	Diet [2]			Probability, $P <$	
	Control (CON)	CitriStim (WCY)	SEM	Treatment	Parity
Sows, n	105	106	---	---	---
Parity	3.55	3.58	---	---	---
Litter characteristics					
Total born,[3] n	15.00	15.40	0.284	0.320	0.005
Born alive, n	14.16	14.39	0.256	0.515	0.012
Born alive, %	94.91	93.95	0.820	0.399	0.526
Stillborn,[4] n	0.85	1.01	0.147	0.378	0.207
Stillborn,[4] %	5.09	6.05	0.820	0.375	0.192
Mummies,[4] n	0.34	0.30	0.070	0.887	0.936
Born alive litter weight, kg	19.24	20.10	0.332	0.063	< 0.001
12-hour measurements[5]					
Survival before cross-foster, %	96.01	96.49	0.615	0.572	0.614
Litter weight, kg	18.96	19.70	0.334	0.109	< 0.001
Litter size, n	13.55	13.86	0.248	0.378	0.044
Post cross-foster measurements					
Litter weight, kg	18.84	19.29	0.294	0.266	< 0.001
Litter size, n	13.42	13.72	0.186	0.242	0.077
Weaning measurements[6]					
Sows weaned, n	105	106	---	---	---
Weaning age	19.39	18.50	0.096	< 0.001	0.523
Survival after cross-foster, %	88.86	88.59	1.160	0.863	0.511
Litter weight, kg	65.90	64.86	1.139	0.507	< 0.001
21-day adjusted litter weight,[7] kg	71.99	73.35	1.245	0.429	< 0.001
Litter size, n	11.84	12.04	0.168	0.381	0.125
Piglet body weight,[8] kg					
Born alive	1.40	1.43	0.021	0.281	0.017
12-hour	1.41	1.44	0.021	0.325	0.025
After cross-foster	1.41	1.44	0.021	0.375	0.031
Weaning[6]	5.59	5.40	0.076	0.071	< 0.001
21-day adjusted weaning	6.11	6.10	0.083	0.976	< 0.001
Litter treated for scours,[9] %	45.55	12.24	3.957	< 0.001	< 0.001

(Table continues)

[1] A total of 211 sows and their progeny (PIC 1050 × 280) were used to determine if feeding a whole-cell inactivated yeast product (WCY; ADM Alliance Nutrition, Inc., Quincy, IL), to sows in gestation and lactation diets influences sow and pre-weaning pig performance in a commercial production system.

[2] Two maternal dietary treatments were fed. In gestation, post pregnancy check on d 35, and in lactation, sows were fed a control basal diet or the basal diet with 0.15% added WCY.

[3] Total born was calculated as born alive plus stillborn piglets and does not include mummies.

[4] Data were square root-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and p-values represent the square root-transformed data.

[5] The 12-hour measurements were collected between 1 and 12 hours of birth from living piglets and before cross-fostering. The 12-hour survival rate does not include stillborn mortality.

[6] Weaning weights were collected 1 day before actual weaning age.

[7] Due to a significant difference in weaning age, weaning litter weights were adjusted for weaning age to a 21-day basis using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 written by the National Swine Improvement Federation in December 1987.

[8] Piglet body weights were calculated by dividing the litter weight by the number of pigs in the litter. Born alive is the litter weight at birth not including stillborn pigs. The 12-hour weight is the litter weight before cross-fostering, which would exclude any crushed or non-viable pigs at that time. Weaning is the litter weight at weaning. The 21-day adjusted weaning is the 21-day adjusted litter weight divided by the number of pigs at weaning.

[9] Percent of litters treated for scours at least 1 time.

Table 2.5 Effects of parity when feeding a whole-cell inactivated yeast product (WCY) to gestating and lactating sows on sow and whole litter performance in a commercial production system[1,2] (606 sows; corresponding to Table 2.3 results)

	Parity							SEM	Probability, $P <$
	1	2	3	4	5	6	7		
Sows, n	135	81	96	86	81	72	55	---	---
Litter characteristics									
Total born,[3] n	13.70[d]	14.98[abc]	15.64[ab]	14.97[abc]	15.75[a]	14.74[bc]	15.23[abc]	0.363	< 0.001
Born alive, n	13.15[e]	14.28[abc]	14.77[a]	14.06[abcd]	14.76[ab]	13.54[cde]	13.83[cde]	0.336	< 0.001
Born alive, %	96.35[a]	95.70[ab]	94.78[abc]	94.37[abc]	93.79[bc]	92.63[cd]	91.10[d]	1.026	< 0.001
Stillborn,[4] n	0.55[c]	0.70[bc]	0.87[ab]	0.91[ab]	0.99[ab]	1.21[ab]	1.40[a]	0.175	0.001
Stillborn,[4] %	3.65[d]	4.30[cd]	5.22[bc]	5.59[abc]	6.21[abc]	7.36[abc]	8.88[a]	1.024	0.002
Mummies,[4] n	0.21	0.28	0.38	0.42	0.28	0.49	0.25	0.102	0.244
Born alive litter weight, kg	17.33[f]	20.90[ab]	20.98[a]	20.27[abc]	19.38[cde]	18.21[f]	18.32[def]	0.441	< 0.001
12-hour measurements[5]									
Survival before cross-foster, %	97.49[a]	96.85[abc]	95.96[abcd]	95.49[bcd]	95.41[bcd]	94.70[d]	96.99[ab]	0.889	0.049
Litter weight, kg	17.20[e]	20.64[a]	20.61[ab]	19.83[abc]	19.08[cd]	17.75[e]	18.10[de]	0.448	< 0.001
Litter size, n	12.80[d]	13.81[abc]	14.16[a]	13.38[bcd]	14.06[ab]	12.73[d]	13.41[abcd]	0.326	< 0.001
Post cross-foster litter size, n	13.57[abc]	13.85[ab]	14.08[a]	13.20[c]	13.58[abc]	12.99[c]	13.12[c]	0.274	0.005
Weaning measurements[6]									
Sows weaned, n	131	80	95	85	79	67	52	---	---
Weaning age	18.76	19.21	18.87	18.96	19.06	18.96	18.87	0.096	0.092
Survival after cross-foster, %	89.72[ab]	90.85[a]	86.36[c]	88.98[abc]	87.56[abc]	87.39[abc]	85.25[c]	1.546	0.029
Litter weight, kg	57.63[f]	71.27[a]	68.02[abc]	69.02[ab]	65.68[bcd]	63.41[de]	59.76[ef]	1.549	< 0.001
21-day adjusted litter weight,[7] kg	64.52[e]	78.42[a]	75.82[ab]	76.71[a]	72.68[bc]	70.45[cd]	66.66[de]	1.685	< 0.001
Litter size, n	12.13[ab]	12.54[a]	12.06[abc]	11.67[bcde]	11.84[bcd]	11.33[def]	10.96[f]	0.234	< 0.001
Piglet body weight,[8] kg									
Born alive	1.35[d]	1.50[a]	1.46[abc]	1.49[ab]	1.35[d]	1.40[cd]	1.35[d]	0.028	< 0.001
12-hour	1.36[c]	1.51[a]	1.48[ab]	1.50[ab]	1.36[c]	1.41[c]	1.36[c]	0.028	< 0.001
Weaning[6]	4.79[c]	5.72[ab]	5.69[b]	5.95[a]	5.55[b]	5.63[b]	5.46[b]	0.106	< 0.001
21-day adjusted weaning	5.35[c]	6.29[b]	6.34[b]	6.60[a]	6.14[b]	6.25[b]	6.08[b]	0.114	< 0.001
Litter treated for scours,[9] %	59.73[a]	27.31[b]	23.67[bc]	19.49[bc]	22.37[bc]	12.84[c]	18.40[bc]	5.521	< 0.001

(Table continues)

[a,b,c,d,e,f]Means within a row without common superscripts differ ($P < 0.05$).

[1] A total of 606 sows and their progeny (PIC 1050 × 280) were used to determine if feeding a whole-cell inactivated yeast product (WCY; ADM Alliance Nutrition, Inc., Quincy, IL), to sows in gestation and lactation diets influences sow and pre-weaning pig performance in a commercial production system.

[2] Two maternal dietary treatments were fed. In gestation, post pregnancy check on d 35, and in lactation, sows were fed a control basal diet or the basal diet with 0.15% added WCY.

[3] Total born was calculated as born alive plus stillborn piglets and does not include mummies.

[4] Data were square root-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and p-values represent the square root-transformed data.

[5] The 12-hour measurements were collected between 1 and 12 hours of birth from living piglets and before cross-fostering. The 12-hour survival rate does not include stillborn mortality.

[6] Weaning weights were collected 1 day before actual weaning age.

[7] Due to a significant difference in weaning age, weaning litter weights were adjusted for weaning age to a 21-day basis using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 written by the National Swine Improvement Federation in December 1987.

[8] Piglet body weights were calculated by dividing the litter weight by the number of pigs in the litter. Born alive is the litter weight at birth not including stillborn pigs. The 12-hour weight is the litter weight before cross-fostering, which would exclude any crushed or non-viable pigs at that time. Weaning is the litter weight at weaning. The 21-day adjusted weaning is the 21-day adjusted litter weight divided by the number of pigs at weaning.

[9] Percent of litters treated for scours at least 1 time.

Table 2.6 Effects of parity when feeding a whole-cell inactivated yeast product to gestating and lactating sows on sow and individual piglet performance in a commercial production system[1,2] (211 sows; corresponding to Table 2.4 results)

	Parity							SEM	Probability, $P <$
	1	2	3	4	5	6	7		
Sows, n	38	36	38	28	26	29	16	---	---
Litter characteristics									
Total born,[3] n	13.71[c]	15.39[ab]	16.34[a]	15.57[ab]	15.77[ab]	14.61[bc]	15.00[abc]	0.714	0.005
Born alive, n	13.24[c]	14.50[ab]	15.37[a]	14.46[abc]	14.81[ab]	13.58[bc]	13.94[abc]	0.644	0.012
Born alive, %	97.23	94.55	94.44	93.57	94.20	94.08	92.93	2.057	0.526
Stillborn,[4] n	0.47	0.89	0.97	1.11	0.96	1.03	1.06	0.368	0.207
Stillborn,[4] %	2.78	5.45	5.56	6.43	5.80	5.92	7.07	2.057	0.192
Mummies,[4] n	0.29	0.25	0.32	0.43	0.27	0.48	0.19	0.178	0.936
Born alive litter weight, kg	18.01[d]	21.03[ab]	21.60[a]	20.78[abc]	19.59[bcd]	18.69[d]	18.01[d]	0.832	< 0.001
12-hour measurements[5]									
Survival before cross-foster, %	97.71	96.67	95.25	95.07	96.72	96.17	96.19	1.543	0.614
Litter weight, kg	17.79[d]	20.69[ab]	21.18[a]	20.31[abc]	19.27[bcd]	18.36[d]	17.69[d]	0.837	< 0.001
Litter size, n	12.92[c]	13.97[abc]	14.61[a]	13.75[abc]	14.31[ab]	12.99[bc]	13.38[abc]	0.624	0.044
Post cross-foster measurements									
Litter weight, kg	18.90[cd]	20.61[a]	20.38[ab]	19.43[abc]	18.39[cde]	18.64[cde]	17.08[e]	0.737	< 0.001
Litter size, n	13.97	13.89	14.18	13.18	13.77	13.20	12.81	0.467	0.077
Weaning measurements[6]									
Sows weaned, n	38	36	38	28	26	29	16	---	---
Weaning age	19.00	19.14	18.95	19.00	18.77	18.67	19.06	0.240	0.523
Survival after cross-foster, %	87.67	91.07	85.76	90.75	88.67	87.81	89.35	2.911	0.511
Litter weight, kg	57.34[c]	72.17[a]	66.99[ab]	70.09[ab]	65.53[b]	64.38[b]	61.14[c]	2.859	< 0.001
21-day adjusted litter weight,[7] kg	63.62[c]	79.73[a]	74.41[ab]	77.63[ab]	73.39[ab]	72.19[b]	67.72[bc]	3.125	< 0.001
Litter size, n	12.13	12.61	11.97	11.89	12.12	11.48	11.38	0.422	0.125
Piglet body weight,[8] kg									
Born alive	1.39[bcd]	1.50[a]	1.46[abc]	1.48[ab]	1.34[d]	1.41[abcd]	1.33[d]	0.052	0.017
12-hour	1.40[bcd]	1.50[a]	1.47[abc]	1.49[ab]	1.36[cd]	1.42[abcd]	1.34[d]	0.052	0.025
After cross-foster	1.39[bcd]	1.50[a]	1.47[abc]	1.48[ab]	1.35[d]	1.42[abcd]	1.34[d]	0.053	0.031
Weaning[6]	4.74[c]	5.77[ab]	5.63[ab]	5.91[a]	5.40[b]	5.62[ab]	5.37[b]	0.190	< 0.001
21-day adjusted weaning	5.26[c]	6.36[ab]	6.25[ab]	6.55[a]	6.05[b]	6.31[ab]	5.95[b]	0.207	< 0.001
Litter treated for scours,[9] %	68.42[a]	27.78[b]	26.32[b]	25.00[b]	15.38[b]	14.37[b]	25.00[b]	9.931	< 0.001

(Table continues)

[a,b,c,d,e]Means within a row without common superscripts differ ($P < 0.05$).

[1] A total of 211 sows and their progeny (PIC 1050 × 280) were used to determine if feeding a whole-cell inactivated yeast product (WCY; ADM Alliance Nutrition, Inc., Quincy, IL), to sows in gestation and lactation diets influences sow and pre-weaning pig performance in a commercial production system.

[2] Two maternal dietary treatments were fed. In gestation, post pregnancy check on d 35, and in lactation, sows were fed a control basal diet or the basal diet with 0.15% added WCY.

[3] Total born was calculated as born alive plus stillborn piglets and does not include mummies.

[4] Data were square root-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and p-values represent the square root-transformed data.

[5] The 12-hour measurements were collected between 1 and 12 hours of birth from living piglets and before cross-fostering. The 12-hour survival rate does not include stillborn mortality.

[6] Weaning weights were collected 1 day before actual weaning age.

[7] Due to a significant difference in weaning age, weaning litter weights were adjusted for weaning age to a 21-day basis using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 written by the National Swine Improvement Federation in December 1987.

[8] Piglet body weights were calculated by dividing the litter weight by the number of pigs in the litter. Born alive is the litter weight at birth not including stillborn pigs. The 12-hour weight is the litter weight before cross-fostering, which would exclude any crushed or non-viable pigs at that time. Weaning is the litter weight at weaning. The 21-day adjusted weaning is the 21-day adjusted litter weight divided by the number of pigs at weaning.

[9] Percent of litters treated for scours at least 1 time.

Table 2.7 Effects of cross-fostering on weaning weight[1]

	Average Wean Weight (*n*)		
	Cross-fostered	Not Cross-fostered	Difference, kg
Birth weight range, kg			
0.61 – 1.12	4.01 (9)	4.65 (389)	0.64
1.13 – 1.58	5.15 (32)	5.88 (1189)	0.73
1.59 – 2.03	5.97 (27)	6.93 (753)	0.96
2.04 – 2.38	9.52 (1)	8.08 (97)	- 1.43

[1] Modeling adjusted 21-day weaning weights of individual piglets that survived to weaning. Dietary treatment, parity group, birth weight within treatment, post cross-foster litter size, and if the piglet was cross-fostered or not were all included. Average weaning weights within each birth weight range was calculated and number of pigs in each category noted.

Table 2.8 Compiled PigKnows Data[1]

	Diet[2]	
	Control (CON)	CitriStim (WCY)
Sows, *n*	905	647
Parity	3.99	3.97
Birth Litter characteristics		
Total born,[3] *n*	14.00	14.47
Born alive, *n*	13.21	13.46
Born alive, %	94.63	93.49
Still born, *n*	0.79	1.01
Stillborn, %	5.26	6.51
Mummies, *n*	0.25	0.29
Weaning measurements		
Sows weaned, *n*	858	602
Litter size, *n*	10.81	11.35
Weaning age	19.46	18.50

[1] A total of 1552 sows and their progeny (PIC 1050 × 280) were used to determine if feeding CitriStim (ADM Alliance Nutrition, Inc., Quincy, IL) to sows in gestation and lactation diets influences litter characteristics at birth and at weaning. Sows of all parities and litter sizes were included. Sows with less than 10 days or greater than 24 days of lactation were excluded. Sows were weaned 3 days per week in this facility.

[2] Two maternal dietary treatments were fed. In gestation, post pregnancy check on d 35, and in lactation, sows were fed a control basal diet or the basal diet with 0.15% added CitriStim.

[3] Total born was calculated as born alive plus stillborn piglets and does not include mummies.
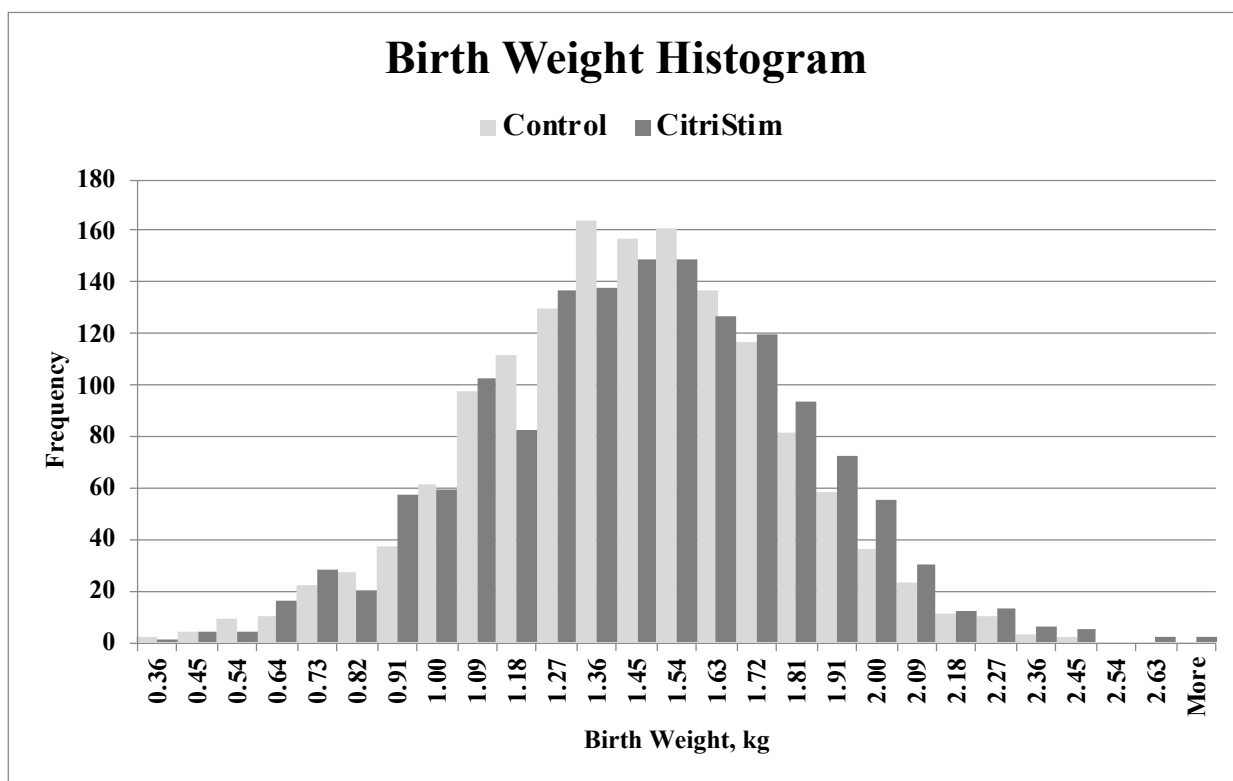
Figure 2.1 Combined histogram of distribution of birth weight ranges. Sows were fed a control basal diet or the basal diet with 0.15% added whole cell yeast (WCY) in gestation starting on d 35 post pregnancy check through lactation. Individual piglet birth weights were divided into ranges separated at 0.09 kg increments. The distribution of birth weights by birth weight range was not different ($P < 0.2461$) between sow dietary treatments.
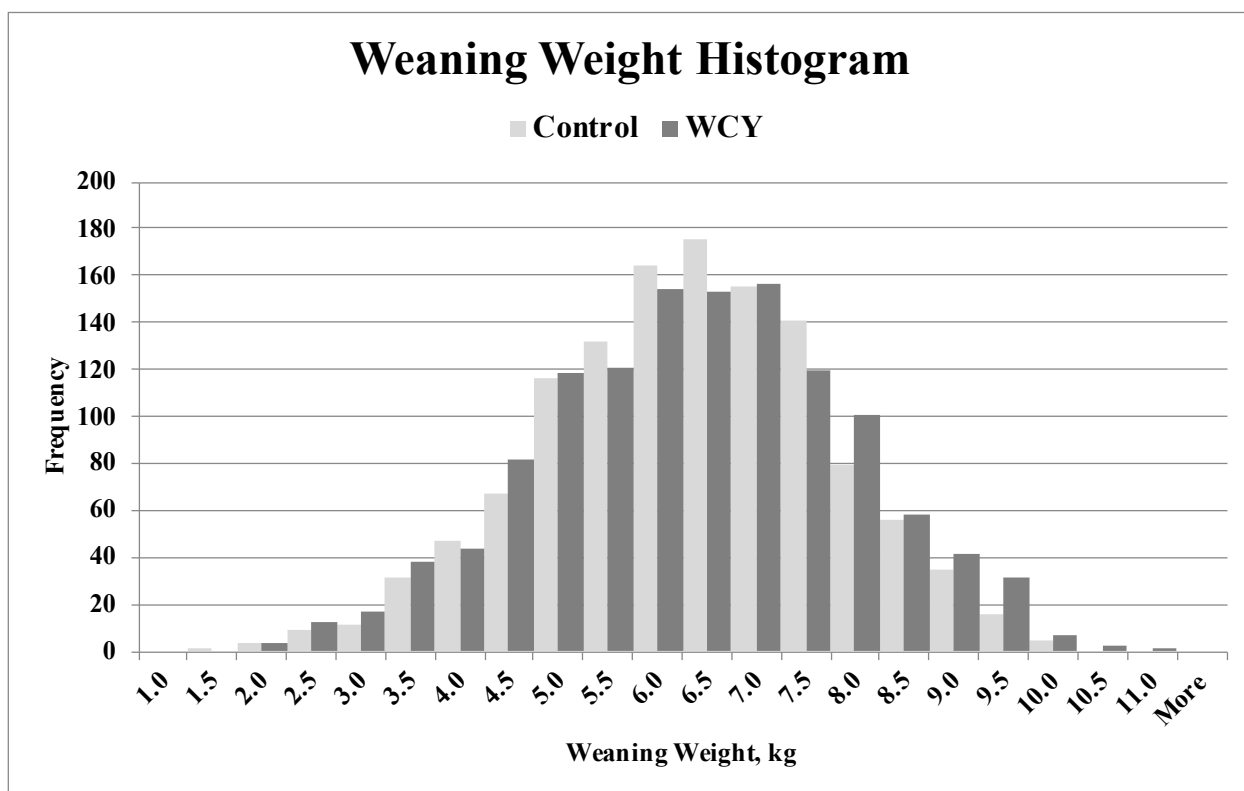
Figure 2.2 Combined histogram of distribution of weaning weight ranges. Sows were fed a control basal diet or the basal diet with 0.15% added whole cell yeast (WCY) in gestation starting on d 35 post pregnancy check through lactation. Individual piglet weaning weights were divided into ranges separated at 0.50 kg increments. The distribution of weaning weights by weaning weight range was not different ($P < 0.3551$) between sow dietary treatments.
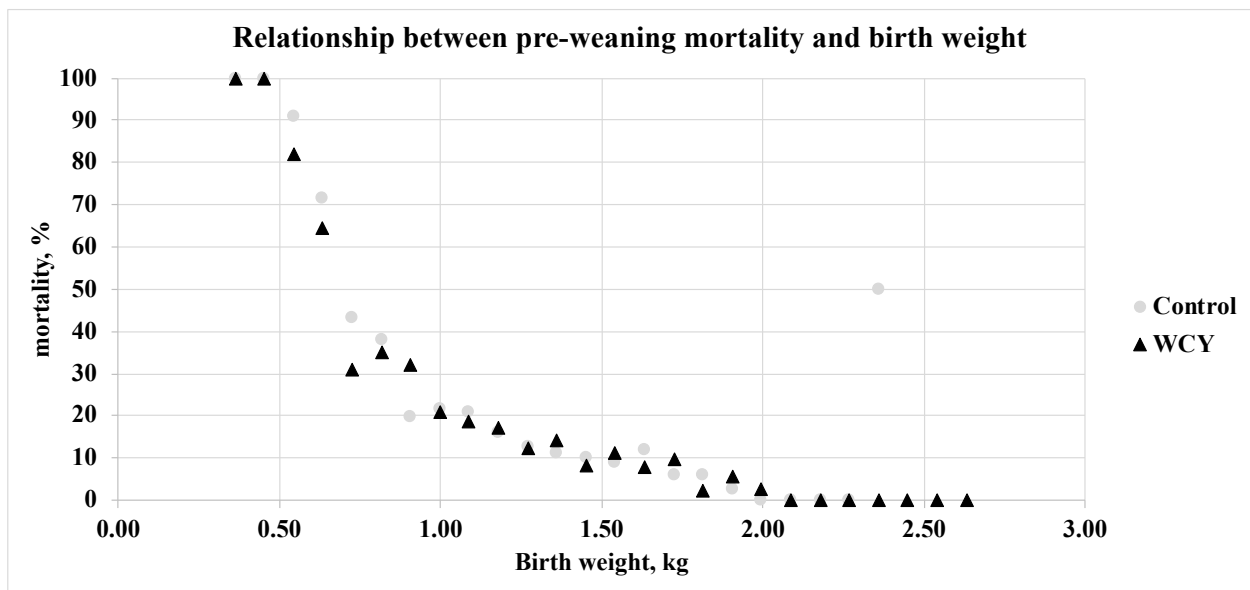
Figure 2.3 Relationship between pre-weaning mortality and birth weight. Sows were fed a control basal diet or the basal diet with 0.15% added whole cell yeast (WCY) in gestation starting on d 35 post pregnancy check through lactation. Individual piglet birth weights were divided into weight ranges separated at 0.09 kg increments. Within each birth weight range, the proportion of piglets that did not survive was calculated and presented as percent mortality. The 2.36 kg birth weight range for the control treatment was comprised of only 2 pigs in which one piglet did not survive to weaning.
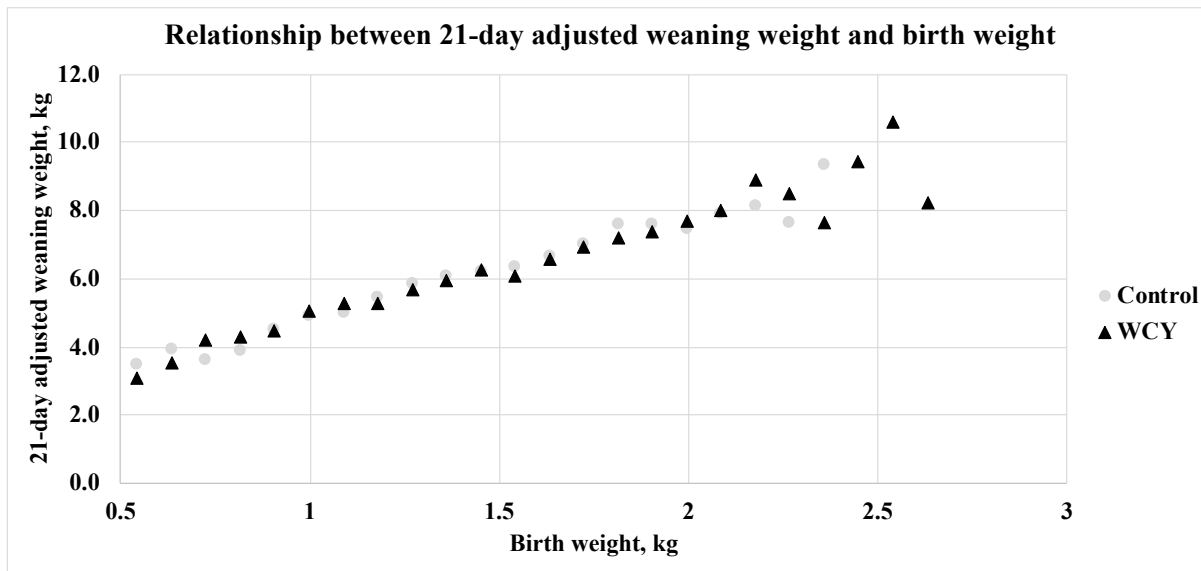
Figure 2.4 Relationship between 21-day adjusted weaning weight and birth weight. Sows were fed a control basal diet or the basal diet with 0.15% added whole cell yeast (WCY) in gestation starting on d 35 post pregnancy check through lactation. Individual piglet birth weights were divided into weight ranges separated at 0.09 kg increments. Within each birth weight range, the average adjusted weaning weight was calculated. Due to a significant difference in weaning age between treatments, individual weaning weights were adjusted for weaning age to a 21-day basis using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 written by the National Swine Improvement Federation in December 1987. The generalized regression reveals for every 0.1 kg increase in birth weight, piglet weaning weight increased by 0.441 kg ($P < 0.001$).
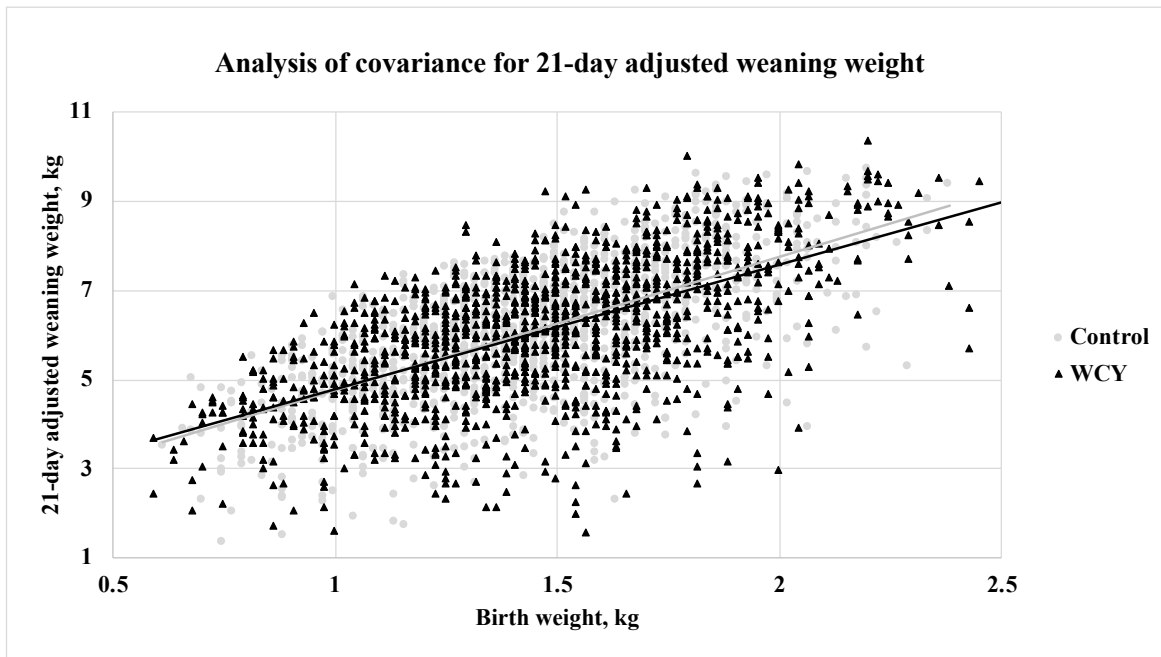
Figure 2.5 Relationship between 21-day adjusted weaning weight and birth weight. Sows were fed a control basal diet or the basal diet with 0.15% added whole cell yeast (WCY) in gestation starting on d 35 post pregnancy check through lactation. Due to a significant difference in weaning age between treatments, individual weaning weights were adjusted for weaning age to a 21-day basis using adjustment factors from the Guidelines for Uniform Swine Improvement Programs 10.2.1 written by the National Swine Improvement Federation in December 1987. In a general linear model, birth weight and sow dietary treatment explained 39.6% of the variation in 21-day adjusted weaning weight ($R^2 = 0.396$). Birth weight was significant in the model ($P < 0.001$), however sow dietary treatment ($P < 0.26$) and the interaction of birth weight and treatment ($P < 0.16$) were not significant.

CHAPTER 3.   EVALUATION OF A DIRECT-FED MICROBIAL AND ORGANIC ACID BLEND, ALONE OR IN COMBINATION, ON SOW REPRODUCTIVE AND PROGENY GROWTH PERFORMANCE

3.1   Abstract

Forty-seven sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or in combination improves sow lactation feed and water intake, litter growth, and subsequent reproductive performance. On approximately d 80 of gestation, sows were fed one of four diets in a 2×2 factorial design: 1) gestation control (CON; 0.55% SID Lysine), 2) CON with DFM (1.6×10^9 CFU/kg of complete feed), 3) CON with 0.4% OA, 4) CON with both DFM and OA. Dietary treatments were also fed throughout lactation (1.00% SID Lysine) starting on approximately d 112 of gestation when sows entered farrowing facility. Sows fed the OA diets had fewer mummies per litter ($P < 0.010$) compared to diets not containing OA. Sows fed diets with the DFM gave birth to lighter pigs born alive (1.5 vs. 1.7 kg; $P < 0.003$) compared to non-DFM fed sows, and a tendency for an interaction ($P < 0.092$) existed where feeding DFM+OA lessened the decrease in born alive BW. There was a tendency ($P < 0.093$) for pigs from DFM fed sows to also be lighter at weaning (5.8 vs. 6.2 kg) compared to pigs from sows not fed DFM, with no differences in litter sizes at weaning ($P < 0.815$). There was a tendency ($P < 0.079$) for the DFM to decrease the amount of sow BW loss in lactation compared to sows not fed the DFM (approximately 6 vs. 8% BW loss, respectively). The maintained BW in lactation was likely related to DFM sows numerically ($P < 0.124$) consuming 8.4% more feed during d 7-14 of lactation and and 6.4% more feed ($P < 0.234$) from d 1 of lactation to weaning. The

interaction was approaching a trend ($P < 0.133$) where sows fed DFM returned to estrus 1.0 day sooner than CON, but only 0.4 days sooner when sows were fed the DFM+OA diet.

Progeny from these sows were followed into the nursery for a 28-d study where pigs were fed a positive control diet (PC), negative control diet (NC), or a diet representative of their dam's treatment to determine if there is an additive benefit to also feeding DFM and/or OA to nursery pigs in addition to their dams. Weaned pigs from 47 dams ($n = 384$, Initial BW = 6.15 kg) were blocked by initial BW and sex and allotted (6 pigs/pen, 8 pens/treatment) to one of 8 nursery treatments. Pigs from CON sows were fed a negative (NC; no antibiotics, no pharmacological Zn or Cu) or positive (PC; neomycin-oxytetracycline in phases 1 and 2 (827 and 551 ppm) and carbadox in phases 3 and 4 (55 ppm)) control diet. Pigs from sows fed DFM, OA, or DFM+OA were fed the NC diet or a diet representative of their dam treatment. Diets with DFM contained 1.6×10^9 CFU/kg of complete feed and diets with OA contained 0.5, 0.4, 0.3, and 0.0% OA in phases 1-4, respectively. Weaning weight was used as a covariate for nursery performance due to the DFM offspring being significantly lighter at weaning. For all phases and overall, PC fed pigs had greater ADG ($P < 0.003$) and ADFI ($P < 0.059$) than NC pigs. PC fed pigs had greater G:F ($P < 0.010$) than NC pigs for all phases and overall, except d 21-28 ($P < 0.532$). Feeding DFM or OA in sow diets improved (interaction; $P < 0.049$) nursery pig G:F, but DFM+OA offspring had similar G:F compared to NC pigs from CON fed sows for d 7-14, 0-14, 0-21, and 0-28. Feeding DFM or OA to sows and their progeny decreased ADFI (interaction; $P < 0.042$) but improved G:F (interaction; $P < 0.028$) for d 7-14 and 0-14 with DFM+OA having similar performance to NC. For d 14-21 and 0-21, feeding DFM or OA to sows and their progeny decreased ADFI whereas DFM+OA increased ADFI above NC (interaction; $P < 0.019$). Overall, d 0-28, feeding DFM or OA to sows and their progeny improved G:F (interaction; $P < 0.001$) with DFM+OA having poorer

G:F compared to NC. When the DFM was fed to sows and nursery pigs, progeny harvested on d 6 post-weaning had a decreased ratio of villus height to crypt depth ($P < 0.035$) compared to sows and pigs not consuming the DFM (average 1.34 vs. 1.67). Comparing pigs fed PC vs. NC from CON fed sows, expression of interleukin 10 (IL-10) was greater (0.51-fold increase; $P < 0.046$) for NC pigs than PC pigs. Expression of occludin (OCLN) was lower ($P < 0.010$) when OA was fed to the sows and pigs compared to when OA was not fed to the sows and pigs (0.78 vs. 1.00, respectively).

In conclusion, feeding a *Bacillus licheniformis* DFM to sows may decrease pig born alive weight and subsequent weaning weight but reduce sow BW loss through 6.4% more lactation feed intake, quickening the return to estrus. Other than decreasing the number of mummies per litter, feeding the OA alone or in combination did not improve sow reproductive or litter growth performance in this study. Further benefits of feeding the OA alone or in combination may require a larger sample size to be detected. In the nursery, pigs fed the PC diet containing antibiotics had better growth performance than pigs fed the NC diet with no antibiotics nor pharmacological levels of zinc or copper. Feeding DFM or OA to sows or their offspring may improve nursery feed efficiency but did not result in a difference in ADG or final BW in this study. Feeding the combination diet (DFM+OA) to the sow and nursery pigs tended to increase ADFI. The intestinal histology and gene expression of the jejunum in progeny harvested 6 days post-weaning did not provide compelling supportive evidence to feed the DFM or OA to nursery pigs to effect gut health post-weaning.

3.2 Introduction

Direct fed microbials (DFM) fed to sows can provide production and health promoting benefits to sows and litters (Alexopoulos et al., 2004; Baker et al., 2013). These benefits may

include increased sow feed intake for the first 14 days after farrowing, reduced sow weight loss in lactation, decreased piglet diarrhea scores, decreased pre-weaning mortality, and increased piglet body weight at weaning when Alexopoulos et al. (2004) fed a combination of *Bacillus subtilis* and *Bacillus licheniformis* spores to gilts. Also, DFMs can reduce pathogen shedding from the sow feces to the piglet environment in the farrowing crate which can beneficially alter the developing gastrointestinal microbiota of the neonatal piglet, e.g. reducing *Clostridium* populations responsible for scours (Baker et al., 2013).

Bacillus-based DFMs fed to weaned pigs have been shown to provide health and growth benefits (Alexopoulos et al., 2004; Kritas and Morrison, 2005). Alexopoulos et al. (2004) observed decreased morbidity and mortality associated with post-weaning *E. coli* diarrhea and greater ADG and feed conversion when diets were supplemented with *Bacillus subtilis* and *Bacillus licheniformis* spores in the nursery. Kritas and Morrison (2005) also fed a *Bacillus subtilis* and *Bacillus licheniformis* DFM to nursery pigs and determined these pigs had very similar ADG, ADFI, and G:F compared to pigs fed low doses of antibiotics.

Nutritionists have been adding organic acids (OA) to pig diets for many years (Metzler et al., 2005). In general, OA are antimicrobial agents that may be an effective alternative to in-feed antibiotics (Decuypere and Dierick, 2003). The OA blend fed in this experiment contains short and medium-chain fatty acids (MCFA) as well as a cyclic short-chain fatty acid (SCFA), benzoic acid. In general, SCFA are used for acidification of the gut, pH reduction, in order to decrease the growth of pathogenic bacteria in the gut (Gabert and Sauer, 1994). Acidification also improves nutrient digestibility via increased pepsin activity and stimulated gut development and integrity in pigs (Gabert and Sauer, 1994; Suiryanrayna and Ramana, 2015).

Research has been conducted looking at different forms of MCFAs as well as individual or combinations of free MCFA in pig diets with varying success (Lauridsen and Danielsen, 2004; Hansen et al., 2012; Gebhardt et al., 2020). One specific MCFA included in the diets of this study is lauric acid. Lauric acid (C12:0) is a saturated MCFA and is the primary fatty acid of coconut oil (Dayrit, 2015). Lauric acid is also the most active saturated fatty acid against gram-positive bacteria (Kabara et al., 1972). Another advantage of adding free MCFA to swine diets is that they are a source of quick energy because they are absorbed into the enterocyte and mainly transported through the blood to the liver avoiding the time-consuming mechanisms of chylomicron formation or re-esterification (Velázquez et al., 1996).

Feeding a coconut oil source of C12 to sows 1 week prior to farrowing and through lactation may increase litter weight gain (Lauridsen and Danielsen, 2004). Hansen et al. (2012) concluded that feeding MCFAs, C8 or C12 sources, to sows during the transition period beginning one week before parturition may beneficially impact colostrum synthesis as observed by increased concentrations of MCFAs in the sow's plasma around parturition and an increased piglet live weight gain from 0 to 24 h of life.

A 1:1:1 blend of caproic (C6), caprylic (C8), and capric acids (C10) were fed to nursery pigs for 35 days at an inclusion of 0 to 1.5% of the diet or 0.5% of each MCFA alone (Gebhardt et al., 2020). Average daily gain, ADFI, and G:F linearly improved with increasing 1:1:1 blend of MCFA. Overall, pigs fed the 0.5% C8 diet had greater ADG, G:F, and body weights compared to control and pigs fed 0.5% C6 or C10 also had improved overall G:F compared to control fed pigs, with no differences in ADG or ADFI (Gebhardt et al., 2020).

The objective of the sow study was to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-

chain fatty acids (OA; DaaFit Plus, ADM Animal Nutrition, Quincy, IL), or in combination improves sow late gestation and lactation BW and BCS, lactation feed and water intake, litter growth, therapies administered in the farrowing house, and subsequent sow reproductive performance. Litter growth performance parameters included litter characteristics at birth, individual piglet weights collected one day after birth and one day before weaning, post cross-foster ADG, and survival rate.

The objective of the nursery study was to determine if feeding gestating and lactating sows and/or their progeny a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA; DaaFit Plus, ADM Animal Nutrition, Quincy, IL), or in combination improves nursery growth performance, administration rate of therapeutic antibiotic injections, intestinal histology, and gene expression of cytokines and tight junction proteins within the jejunal mucosa.

3.3 Materials and Methods

3.3.1 General

The experimental procedures for this experiment were approved by the Purdue University Animal Care and Use Committee (Sow PACUC # 1909001949; Nursery PACUC # 1303000841). This study was conducted at the Purdue University Animal Sciences Research and Education Center (ASREC) where sows were individually housed in late gestation and piglets were born in farrowing facilities then group housed in nursery facilities post-weaning.

3.3.2 Animals and Diets: Sow Experiment

A total of 54 sows were bred, pregnancy checked on approximately d 30 of gestation, and allotted to dietary treatments on approximately d 80 of gestation. Parity of the sows was equalized

across dietary treatments. Sows were fed one of four diets in a 2×2 factorial design: 1) gestation control (CON; 0.55% SID Lysine), 2) CON with DFM (1.6×10^9 CFU/kg of complete feed), 3) CON with 0.4% OA, 4) CON with both DFM and OA. Sows received 2.27 kg/day of gestation feed delivered once daily. Dietary treatments were also fed *ad libitum* throughout lactation (1.00% SID Lysine) starting on approximately d 112 of gestation when sows entered the farrowing facility. Diets were formulated to meet or exceed the swine nutrient requirements (NRC, 2012) for gestating and lactating sows (Table 3.1). All sows were given a pre-farrowing combination vaccination for rotavirus, *C. perfringens*, and *E. coli* on approximately d 94 of gestation (ProSystem RCE, Merck Animal Health, Madison, NJ).

Due to 6 females being no longer pregnant when entering the farrowing facility (2 CON, 3 DFM, and 1 DFM+OA), only 48 sows farrowed. Three of those 6 non-pregnant females were noted as not very piggy when initially put on test (1 CON, 1 DFM, and 1 DFM+OA). One sow from the OA treatment was removed from the data set because she had a retained pig, was treated many times, and did not eat for a long period after farrowing. All of the pigs in one DFM+OA litter were treated for greasy pig and therefore the weaning, survival, and ADG litter performance data from that litter was removed from the data set.

Sow body weights (BW) were collected on d 80 and 112 of gestation as well as day 1 of lactation and at weaning. Sows were also assigned a body condition score (BCS) and ultrasonic measurements of their last rib backfat and loin depth were recorded on d 80 and 112 of gestation and at weaning. An Aloka SSD 500V Ultrasound (Aloka Co., Ltd., Tokyo, Japan) was used to image and measure last rib backfat and loin depth of the sows. The probe was placed at the sow's last rib (last to approximately 10th rib) and aligned approximately10 cm off midline, and parallel

to the sow's spine. Measurements were collected from the animal's right and left side and the average was calculated.

Individual piglet weights were collected at processing on the day after birth (d 1) and the day before weaning. Piglet processing included ear notching, clipping of needle teeth, tail docking, castration, and a dose of 1.0 mL injectable iron (200 mg/mL; Uniferon 200, Pharmacosmos, Inc, Watchung, NJ). Cross-fostering occurred after processing and within 72 hours of birth. In the attempt to even out litter size, cross-fostering mainly occurred within sow dietary treatment. However, due to timing of litters born, variation in litter sizes, and crushed pigs before processing, a total of 8 piglets from DFM+OA sows were cross-fostered onto DFM sows.

Each sow was fed from her individual 38-liter tub located in front of her farrowing crate. Lactation feed was retrieved from the sow's feeder and weighed on d 1, 3, 7, 14, and at weaning. The remaining feed weight was added to the weight of the feed in the tub on each day. Feed additions to the tub and spoiled feed removed from the feeder was weighed and recorded to calculate average daily feed intake (ADFI). Daily sow water intake was measured in the farrowing house using individual water meters (Assured Automation, Clark, NJ) mounted on 40 farrowing crates (10 per treatment). As stated above, one sow from the OA treatment was removed from the data set, leaving 39 remaining sows with water intake data.

Colostrum was collected within 8 hours of the first pig born from 43 sows. The Brix refractometer (MISCO, Cleveland, OH) was used to indirectly measure immunoglobulin G (IgG) concentration. The meter was calibrated to produce a reading of zero using distilled water prior to each use. Measurements were collected in triplicate from the same sample and the values were averaged. Data from one sow fed the OA treatment was removed because of the Brix value being an outlier (greater than 3 standard deviations from the mean). All but one CON sow (retained as a

lactation nurse sow) was weaned into gestation crates or a cull pen for determination of return to estrus data. Sows were heat checked with a boar twice daily and first signs of estrus were recorded to calculate returning to estrus and the wean to estrus interval.

All piglet therapies in the farrowing house were recorded and divided into four categories: lameness, enteric, unthrifty, and other. The other category was comprised of therapies for greasy pig, *Streptococcus suis* infection, and respiratory. Pigs treated at least once was calculated as a percent of the post cross-foster litter size and the total therapies given in the litter was calculated, which includes pigs treated multiple times.

3.3.3 Animals and Diets: Nursery Experiment

Progeny from sows fed diets containing a *Bacillus licheniformis* direct fed microbial (DFM), an organic acid blend of medium and short chain fatty acids (OA), DFM+OA, or a control (CON) diet from d 80 of gestation until weaning were used in this study. Weaned pigs from 47 dams ($n = 384$, Initial BW = 6.15 kg) were blocked by initial BW and sex and allotted (6 pigs/pen, 8 pens/treatment) to one of 8 nursery treatments. All pigs received a vaccination for circovirus and *Mycoplasma hyopneumoniae* (Fostera Gold PCV MH, Zoetis, Parsippany, NJ) and an iron booster (200 mg) one day prior to weaning.

Pigs from CON sows were fed a negative (NC; no antibiotics, no pharmacological Zn or Cu) or positive (PC; neomycin-oxytetracycline in phases 1 and 2 (827 and 551 ppm) and carbadox in phase 3 and 4 (55 ppm)) control diet. Pigs from sows fed DFM, OA, or DFM+OA were fed the NC diet or a diet representative of their dam's treatment. Diets with DFM contained 1.6×10^9 CFU/kg DFM and diets with OA contained 0.5, 0.4, 0.3, and 0.0% OA in phases 1-4, respectively. Each of the 4 nursery phases were fed for 7 days. Dietary treatments were fed *ad libitum* and

formulated to meet or exceed the swine nutrient requirements (NRC, 2012) for nursery pigs (Table 3.2).

Pigs and feeders were weighed the day before weaning, d 7, 14, 21, and 28 post-weaning to calculate pen ADG, ADFI, and G:F. Each week, feeders were dumped and scraped before adding the next phase diet to the feeder. All therapeutic injectable antibiotic treatments were recorded and divided into four categories: enteric, lameness, unthrifty, and other. The other category was comprised of therapies for *Streptococcus suis* infection, respiratory, and an infected snout. Pigs treated at least once within a period was calculated as a percent of the pigs in the pen at that time.

3.3.4 Jejunal Tissue Collection

One gilt per pen was harvested 6 days post-weaning. Pigs were weighed and then euthanized by CO_2 gas asphyxiation immediately followed by exsanguination as an adjunctive method to ensure death (AVMA, 2013). Anterior from the cecum, 30 cm of intestinal tissue was discarded to eliminate the ileum. The jejunal sections were then cut measuring 100, 20, and 5 cm to collect jejunum intestinal contents, mucosa scrapings, and undisturbed tissue for histology, respectively.

Intestinal contents were collected by stripping the jejunum into a 50 mL conical tube and freezing at -20°C for future microbiome analysis. The sections for mucosa scrapings and histology were first rinsed carefully with phosphate buffered saline (PBS) to remove digesta. Mucosa scrapings were collected by cutting the tissue down the mesenteric line, flattening the tissue on a cutting board, and scraping the tissue with a microscope slide. A pencil eraser size of mucosa tissue, approximately 1 g (5 mm in diameter), was placed in 1 mL of Trizol reagent (Ambion,

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Carlsbad, CA), flash frozen in liquid nitrogen, and stored at -80°C until further processing for gene expression analysis. The section of tissue for histology was cut down the mesenteric line and placed in Tissue-Tek Uni-Cassettes (Sakura Finetek USA, Inc., Torrance, CA) and stored in 10% neutral buffered formalin until further processing for villus height and crypt depth analysis.

3.3.5 Histology

The jejunum sections were processed by the Purdue Histology Research Laboratory in the Purdue University Veterinary School. The 10% neutral buffered formalin fixed tissue was embedded in paraffin, a 5 μm thick section was sliced, and stained with hematoxylin and eosin (H&E). Images were obtained using an AmScope Microscope Digital Camera (MU1000, AmScope, Irvine, CA) at 40X magnification. ImageJ software (NIH, Bethesda, MD) was used to measure villi height and crypt depth of a minimum of 8 well oriented villi and crypts for each tissue sample.

3.3.6 RNA Extraction, cDNA Synthesis, and Quantitative PCR

Total RNA from jejunal mucosa was isolated using a commercially available extraction kit (PureLink™ RNA Mini Kit, Invitrogen, Carlsbad, CA) from samples stored in Trizol reagent. Quantification of RNA was conducted using a NanoDrop spectrophotometer (ND-100, NanoDrop Technologies, Rockland, DE). The quantity of RNA was measured at an absorbance of 260 nm and purity was evaluated by determining the ratio of the absorbances at 260 nm and 280 nm. All samples were measured in duplicate and had an average A260/280 ratio of 1.97 to 2.14. The integrity of total RNA was further checked on a 1.2% denaturing agarose gel electrophoresis with visualization of a clear 28S and 18S ribosomal RNA banding pattern.

After RNA quantification, each sample was diluted to 1 µg of RNA per 4.75 µL of nuclease free water. The complementary DNA (cDNA) was then synthesized by reverse transcription using a cocktail of the following reagents: 2 µL 5X First Strand Buffer (Invitrogen, Carlsbad, CA), 1 µL Bovine Serum Albumin (1 mg/mL; Invitrogen, Carlsbad, CA), 0.5 µL dNTP Mix (10 mM; Promega Corporation, Madison, WI), 0.25 µL Rnasin® Plus Ribonuclease Inhibitor (40 Units/µL; Promega Corporation, Madison, WI), 1 µL Oligo(dT)$_{12-18}$ Primer (0.5 µg/µL ; Invitrogen, Carlsbad, CA), and 0.5 µL M-MLV Reverse Transcriptase (200 Units/µL; Invitrogen, Carlsbad, CA). The total volume of 10 µL containing 1000 ng RNA and cocktail were incubated in a Thermo Hybaid PCR Express thermal cycler (Midwest Scientific, St. Louis, MO) at 25°C for 10 min, followed by 48°C for 30 minutes, and 95°C for 5 minutes. Samples were diluted with 90 µL nuclease free water and stored at -20°C until amplified.

Quantitative polymerase chain reaction (qPCR) for gene expression was conducted in duplicate using iQ™ SYBR® Green Supermix (Bio-Rad Laboratories, Inc., USA) on a Bio-Rad PCR machine (CFX Connect, Real-Time System). Each reaction was performed in a volume of 15 µL containing 7.5 µL iQ™ SYBR® Green Supermix, 0.5 µL forward primer, 0.5 µL reverse primer, 4.5 µL of nuclease free water, and 2 µL of cDNA (20 ng equivalent cDNA).

In accordance with Derveaux et al. (2010), the specificity of the amplification was confirmed by the melting curve. The PCR efficiency for each set of primer probes was evaluated using a serial dilution and found to be greater than 90% for all targets. The primer sequences, annealing temperatures, and amplicon product lengths are listed in Table 3.20. The data was normalized to the geometric mean of three stable housekeeping genes (GAPDH, ACTB, and STX5). Data are presented as the fold change ($2^{-\Delta\Delta Ct}$) relative to the NC group. Due to block being significant in the model, the first 2 blocks of the NC group were averaged and used to calculate 2^-

$^{\Delta\Delta Ct}$ for blocks 1 and 2. The next 2 blocks, 3 and 4, were averaged and used to calculate $2^{-\Delta\Delta Ct}$ for

blocks 3 and 4 and so on for all 8 blocks.

3.3.7 Feed and Therapeutic Drug Cost Analysis

The basal diet cost was $1.043/kg, $0.770/kg, $0.489/kg, and $0.328/kg of complete feed

for phases 1-4, respectively. The antibiotic PC diet contained neomycin-oxytetracycline

($14.330/kg) in phases 1 and 2 and carbadox ($13.669/kg) in phases 3 and 4. The *Bacillus*

licheniformis DFM cost $3.638/kg and OA blend cost $3.748/kg.

Therapeutic drugs administered in the nursery as individual therapies included

enrofloxacin ($0.72/mL), ceftiofur crystalline free acid ($1.34/mL), lincomycin hydrochloride

($0.52/mL), isoflupredone acetate ($0.649/mL), flunixin meglumine ($0.30/mL), penicillin

($0.129/mL), ampicillin ($0.305/mL), and dexamethasone ($0.095/mL).

3.3.8 Chemical Analysis

Feed samples were collected at the Purdue University ASREC, subsampled in the lab at

Purdue University (West Lafayette, IN), and shipped to University of Missouri (Columbia, MO)

for analysis (Tables 3.3, 3.4, 3.5, and 3.6). University of Missouri analyzed for fatty acid profile

(AOAC 920.39; AOAC 996.06; Knittelfelder and Kohlwein, 2017). Feed samples were also

analyzed at Purdue University for gross energy (Parr 6200 Bomb Calorimeter; Parr Instrument

Company, Moline, IL) and crude protein (LECO; combustion method AOAC 990.03) as an

internal check (Tables 3.7 and 3.8).

Feed samples were also shipped to Eurofins Nutrition Analysis Center (ENAC; Des

Moines, IA) to be analyzed for crude protein (AOAC 990.03), crude fat (AOAC 920.39), crude

fiber (AOAC 962.09), acid detergent fiber (ANKOM ADF for A2000 mod), neutral detergent fiber

(ANKOM NDF for A2000 mod), ash (AOAC 942.05), Ca (AOAC 927.02), P (AOAC 984.27), and amino acid profile (AOAC 982.30 mod). Results are pending.

3.3.9 Statistical Analysis

Sow data were analyzed as a completely randomized design with a 2×2 factorial treatment structure using the MIXED procedure in SAS 9.4 (SAS Institute, Inc., Cary, NC). The sow or litter was the experimental unit. For sow BW, BCS, ultrasound scans, litter performance, feed and water intake, Brix value, rebreeding, and piglet therapy data, dietary treatment of the sow was the fixed effect and parity group (1, 2+) was used as a covariate. The litter performance parameters of litter wean weight, pig wean weight, post cross-foster pig gain, and post cross-foster ADG were analyzed with wean age as a covariate in addition to the parity group covariate. Stillborn and mummy data were square root-transformed and the piglet therapy data was log-transformed to meet assumptions of normality. The FREQ procedure in SAS 9.4 was used to conduct a chi-square analysis of the percentage of weaned sows that returned to estrus. Differences were considered significant at $P \leq 0.05$ and trends at $0.05 < P \leq 0.10$.

Nursery data were analyzed as a randomized complete block design using the MIXED procedure in SAS 9.4 (SAS Institute, Inc., Cary, NC). Pigs were blocked by initial BW and sex and pen was the experimental unit for growth performance and injectable therapies. The pig was the experimental unit for intestinal histology and gene expression. Dietary nursery treatment was the fixed effect and initial BW was used as a covariate for growth performance data due to piglets from DFM fed sows being significantly lighter at weaning. Nursery injectable therapy data was log-transformed to meet assumptions of normality. Statistical analysis of the gene expression data was performed on the ΔCt values, and results are presented as fold changes relative to the NC.

Nursery data were analyzed using 7 pre-planned orthogonal contrasts. Pigs from CON sows were fed an antibiotic positive control diet (PC; treatment A) or a negative control diet (NC; treatment B). Pigs from sows fed DFM, OA, or DFM+OA were fed the NC diet (treatments C, D, and E, respectively) or a diet representative of their dam's treatment (treatments F, G, and H, respectively). The first three contrasts were used to investigate the main effects of the sow's diet when fed the DFM, OA, as well as their interaction when the sows were fed dietary treatments and the nursery pigs were fed NC diets. Nursery treatments compared in these first three contrasts included treatment B, C, D, and E. For example, the main effect of the DFM in the sow diet alone would compare the average of treatment C and E vs. the average of treatment B and D. The next three contrasts, 4 to 6, were used to investigate the main effects of the DFM and OA as well as their interaction when the sows and nursery pigs were fed dietary treatments. Nursery treatments compared included treatment B, F, G, and H. For example, the main effect of the DFM in the sow and nursery diet would compare the average of treatment F and H vs. the average of treatment B and G. The seventh contrast was used to investigate the difference between the PC (treatment A) and NC (treatment B) both from CON fed sows. Differences were considered significant at $P \leq$ 0.05 and trends at $0.05 < P \leq 0.10$.

3.4 Results

3.4.1 Sow Experiment

The sow experiment investigated the effect of feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination.

3.4.1.1 Sow BW, BCS, and Ultrasound Scans

There was a DFM × OA interaction ($P < 0.05$; Table 3.9) for loin depth change in gestation from d 80 to 112 measured by ultrasound. Sows fed the DFM maintained loin muscle depth but DFM+OA fed sows lost 5.3% of their loin depth when the OA was added to the DFM, which was comparable to the CON and OA fed sows. Similarly, the interaction ($P < 0.134$) for BCS changes in gestation was approaching a trend where sows fed the DFM increased their BCS by 0.24, OA sows increased by 0.12, however, BCS for DFM+OA sows was similar to CON with little to no change in gestation BCS.

When ultrasonically measuring backfat thickness, there was no DFM × OA interaction at any time point or change calculated ($P > 0.05$). There was a main effect of DFM ($P < 0.001$) and OA tendency ($P < 0.063$) for backfat thickness change in gestation where DFM and DFM+OA sows maintained backfat thickness compared to sows not fed the DFM and OA fed sows tended to lose backfat. In contrast, while the OA fed sows had generally decreased their loin depth and lost backfat in gestation, sows fed OA were approaching a trend ($P < 0.148$) for greater BW gain compared to sows not fed OA (25.5 vs. 22.2 kg).

Lactation body weight loss tended ($P < 0.079$) to be not as severe, approximately 6 vs. 8% loss, and BCS at weaning tended ($P < 0.108$) to be greater when sows were fed DFM compared to sows not fed DFM. The less severe body weight loss and improvement in BCS is likely attributed to the DFM sows numerically ($P < 0.124$; Table 3.11) consuming 8.4% more feed during d 7-14 of lactation. Although not significant in this small study, DFM sows consumed 6.4% more feed in lactation ($P < 0.234$; Day 1 to weaning) compared to sows not fed DFM. There were no other interactions or main effects of dietary treatment on lactation feed or water intake (Tables 3.11 and 3.12).

3.4.1.2 Reproductive and Litter Performance

Sows fed the OA diets had fewer mummies per litter ($P < 0.010$; Table 3.10) compared to diets not containing OA. The number of mummies per litter was approaching a trend for a DFM × OA interaction ($P < 0.114$) where sows fed the DFM alone had 0.41 more mummies than CON, however sows fed the DFM+OA had the fewest number of mummies (0.71 fewer than CON). The percent of total born pigs being born alive was also approaching a trend for an interaction ($P < 0.119$). The percentage being born alive dropped by 6.55%, 2.34%, and 1.89% below the CON sows when sows were fed DFM, OA, and DFM+OA diets, respectively.

The litter weights, of pigs born alive and after cross-fostering, from sows fed the DFM diets were lighter ($P < 0.032$) compared to sows not fed DFM. In agreement, sows fed diets with the DFM gave birth to lighter pigs born alive on an individual basis (1.5 vs. 1.7 kg; $P < 0.003$) compared to non-DFM fed sows, and a tendency for an interaction ($P < 0.092$) existed where feeding DFM+OA lessened the decrease in individual born alive BW. The individual pig BW post cross-foster was also less ($P < 0.008$) when sows were fed DFM or DFM+OA compared to the other two diets.

There was a tendency ($P < 0.093$) for the litter and individual weaning weights of pigs fed DFM to be lighter than litters from non-DFM fed sows (individual, 5.84 vs. 6.20 kg) with no difference in litter sizes at weaning ($P < 0.815$). There was also a tendency for a DFM × OA interaction ($P < 0.074$) where litter weaning weight was less for DFM fed sows compared to the control but feeding OA or DFM+OA lessened the decrease in litter weaning weight.

3.4.1.3 Colostrum Brix Values and Rebreeding Rate

A digital Brix refractometer was used in this study to indirectly measure colostrum IgG concentration. There was no difference ($P > 0.05$; Table 3.13) in average colostrum Brix value

(approximately 25%) due to dietary treatment. For return to estrus post-weaning, the interaction was approaching a trend ($P < 0.133$; Table 3.14) where sows fed the DFM returned to estrus 1.0 day sooner than CON, but only 0.4 days sooner when sows were fed the DFM+OA diet.

3.4.1.4 Injectable Therapies

There was no difference in the total therapies administered ($P > 0.05$; Table 3.15) when calculated as a percentage of the post cross-foster litter size. However, there was a DFM × OA interaction ($P < 0.039$) for therapies in the lameness category where litters from sows fed the DFM and OA alone increased lameness treatments but litters from sows fed the DFM+OA diet had a reduced treatment rate more similar to CON.

The percent of the litter treated at least once for being unthrifty tended to be reduced ($P < 0.086$) when OA was included in the sow diet, average 0.665% vs. 2.64%. There was also a numerical trend ($P < 0.105$) for OA litters to be treated less for being unthrifty when the total number of therapies were counted and calculated as a percent of the total therapies given for that litter. The increased percent of the litter treated at least once for the other category was approaching a trend ($P < 0.110$) when the DFM was included in the sow diet, average 0.165% vs. 4.4%.

3.4.2 Nursery Experiment

The nursery experiment investigated the effect of feeding gestating and lactating sows and/or their progeny a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short chain fatty acids (OA), or the products in combination. One pig fed the NC diet from an OA fed sow was pulled off test on d 7. Two pigs fed the NC diet from DFM+OA fed sows were pulled off test, one on d 14 and one on d 21.

3.4.2.1 Growth Performance

For most of the phases and overall, the antibiotic PC fed pigs had greater ADG, ADFI, G:F, and BW ($P < 0.048$; Table 3.16) compared to NC fed pigs from CON fed sows. The two exceptions include a tendency ($P < 0.059$) for d 0-7 ADFI where PC was numerically greater, and no difference ($P < 0.532$) for G:F for d 21-28. Other than PC pigs being heavier than NC from CON sows throughout the study ($P < 0.008$), there were no weekly or overall differences in nursery pig BW due to any other dietary treatment of the sow or nursery pigs. The only exception was pigs from DFM sows were lighter on d 0 ($P < 0.006$) than all other treatments, requiring d 0 BW to be used as a covariate for all growth performance data.

The following growth performance results encompass contrasts where the DFM and/or OA were added to the sow diet only and progeny were fed the NC diet in the nursery. Feeding DFM or OA in sow diets improved (interaction; $P < 0.049$) nursery pig G:F, but DFM+OA offspring had similar G:F compared to NC pigs from CON fed sows for d 7-14, 0-14, 0-21, and 0-28. Pigs from DFM fed sows had decreased ADFI and pigs from sows fed OA had greater ADG compared to the other treatment groups resulting in improved G:F for d 7-14, 0-14, 0-21, and 0-28. There was a tendency during d 0-7 for the DFM in the sow diet ($P < 0.077$) to decrease nursery pig G:F compared to progeny from sows not consuming DFM. The increased ADG ($P < 0.113$) and ADFI ($P < 0.106$) for d 7-14, and ADFI ($P < 0.105$) for d 21-28 was approaching a trend for pigs fed NC from sows fed OA compared to pigs fed NC from sow not fed OA.

The following growth performance results encompass contrasts where the DFM and/or OA was added to the sow and nursery pig diets. There was a tendency during d 0-7 for the DFM in the sow and nursery diet ($P < 0.096$) to decrease nursery pig G:F compared to progeny from sows and pigs not consuming DFM in the diet (average 0.75 vs. 0.82, respectively). This tendency was

driven by decreased ADG of DFM fed sows and pigs and increased ADFI of DFM+OA fed sows and pigs.

Feeding DFM or OA to sows and their progeny decreased ADFI (interaction; $P < 0.042$) and therefore improved G:F (interaction; $P < 0.028$) for d 7-14 and 0-14 with DFM+OA having similar feed intake and efficiency to NC fed pigs from CON sows. For d 14-21 and 0-21, feeding DFM or OA to sows and their progeny decreased ADFI whereas DFM+OA increased ADFI above NC fed pigs from CON sows (interaction; $P < 0.019$). There was a tendency during d 14-21 for the DFM in the sow and nursery diet ($P < 0.081$) to increase nursery pig ADFI compared to progeny from sows and pigs not consuming DFM in the diet (average 412 vs. 378 g/d, respectively). Feeding DFM or OA to sows and their progeny tended to reduce nursery pig ADG (interaction; $P < 0.059$) during d 14-21 compared with DFM+OA having increased ADG above NC fed pigs from CON sows. The increased nursery pig G:F (interaction; $P < 0.118$) for d 0-21 was approaching a trend for pigs and sows fed the DFM or OA compared with DFM+OA having similar feed efficiency to NC fed pigs from CON sows.

Feeding DFM or OA to sows and their progeny during d 21-28 increased G:F (interaction; $P < 0.001$) due to the increased ADG (interaction; $P < 0.008$) with DFM+OA having decreased G:F and ADG below NC fed pigs from CON sows. There was a tendency during d 21-28 for the DFM in the sow and nursery diet ($P < 0.089$) to decrease nursery pig ADG compared to progeny from sows and pigs not consuming DFM in the diet (average 451 vs. 477 g/d, respectively). This decreased ADG lead to a tendency for DFM in the sow and nursery diet ($P < 0.067$) to decrease nursery pig G:F in the d 21-28 period compared to progeny from sows and pigs not consuming DFM in the diet (average 0.66 vs. 0.70, respectively). When OA was fed to sows and nursery pigs during d 21-28, nursery pigs had a decreased ($P < 0.022$) G:F compared to progeny from sows and

pigs not consuming OA in the diet (average 0.655 vs. 0.705, respectively). The decreased G:F was due to decreased ADG of pigs from sows and pigs fed DFM+OA and slightly increased ADFI of pigs from sows and pigs fed OA and DFM+OA.

Overall, d 0-28, feeding DFM or OA to sows and their progeny improved G:F (interaction; $P < 0.001$) with DFM+OA having poorer G:F compared to NC fed pigs from CON sows. Day 0-28 G:F interaction was due to a tendency (interaction; $P < 0.067$) for pigs from sows and nursery pigs fed DFM or OA consuming less feed and pigs from sows and nursery pigs fed DFM+OA consuming more feed per day than pigs fed NC from CON sows. Additionally, there was a tendency during d 0-28 for the DFM in the sow and nursery diet ($P < 0.053$) to decrease nursery pig G:F compared to progeny from sows and pigs not consuming DFM in the diet (average 0.654 vs. 0.683, respectively). This tendency was driven by pigs having increased ADFI when sows and pigs were fed DFM+OA.

3.4.2.2   Injectable Therapies

All therapeutic injectable antibiotic treatments were recorded and divided into four categories: enteric, lameness, unthrifty, and other (Table 3.17). The other category was comprised of therapies for *Streptococcus suis* infection, respiratory, and an infected snout. Pigs treated at least once within a period was calculated as a percent of the pigs remaining in the pen. Comparing pigs fed PC vs. NC from CON fed sows, there was no difference ($P > 0.05$) in treatment rate for any reason d 0-7, 7-14, or 14-21. The percent of pigs in a pen treated for any reason from d 21-28 was greater ($P < 0.009$) when pigs were fed the NC diet compared to PC (12.5 vs. 0%).

Overall from d 0-28, the percent of pigs treated at least once for any reason during the whole study, as a percent of pigs in the pen, and the total therapies given per pen was greater for NC fed pigs than PC from CON fed sows ($P < 0.050$). When the total therapies were calculated to

achieve a per pig basis, there was a tendency ($P < 0.070$) for NC pigs from CON sows to have a greater average treatment rate from d 0-28 compared to PC (0.23 vs. 0.02 therapies per pig on average). In other words, approximately 1 out of 4 NC pigs from CON fed sows were treated during this 28-day study compared to 1 out of 50 for the PC pigs.

The following injectable therapy results encompass contrasts where the DFM and/or OA was added to the sow diet only and progeny were fed the NC diet in the nursery. From d 0-7, 4.2% of pigs in pens fed NC from OA fed sows were treated for enteric challenges and pigs fed NC from CON, DFM, and DFM+OA sows had a therapy rate of 0%. This resulted in pigs from sows fed DFM having a decreased treatment rate compared to sows not fed the DFM ($P < 0.036$), pigs from sows fed OA having an increased treatment rate compared to sows not fed OA ($P < 0.036$), and the interaction ($P < 0.036$). There was also a tendency ($P < 0.051$) for NC fed pigs from sows fed OA to be treated more for other reasons compared to pigs from sows not fed OA during d 0-7 (average 2.1 vs. 0.0%, respectively).

From d 7-14, 5.0% of pigs in pens fed NC from DFM fed sows were treated for enteric challenges and pigs fed NC from CON, OA, and DFM+OA sows had a therapy rate of 0%. This resulted in pigs from sows fed DFM having an increased treatment rate compared to sows not fed the DFM ($P < 0.036$), pigs from sows fed OA having a decreased treatment rate compared to sows not fed OA ($P < 0.036$), and the interaction ($P < 0.036$). From d 7-14, there was also a tendency ($P < 0.056$) for pigs from sows fed DFM to be treated less for lameness compared to pigs from sows not fed DFM (average 0.0 vs. 3.75%, respectively).

Feeding DFM or OA to sows only (progeny were fed the NC diet in the nursery) increased the treatment rate for enteric challenges (interaction; $P < 0.032$) during d 14-21 where 5.0% of pigs in pens from DFM sows and 2.5% of pigs in pens from OA sows were treated and 0.0% of

pigs in pens from CON sows and DFM+OA sows were treated. During d 21-28, pigs fed NC from sows fed OA were treated less frequently for any reason ($P < 0.026$) compared to pigs fed NC from sows not fed OA (average 3.75 vs. 11.25%). Pigs fed NC from sows fed OA tended to be treated less for lameness ($P < 0.051$) compared to pigs from sows not fed OA (average 0.0 vs. 2.5%). Overall from d 0-28, feeding DFM or OA to sows only tended to increase the nursery pig treatment rate for any reason (interaction; $P < 0.101$) compared with pigs fed NC from sows fed DFM+OA having similar treatment rate to NC fed pigs from CON sows. The percent of pigs treated for being unthrifty tended to increase ($P < 0.099$) for NC fed pigs from DFM fed sows and tended to decrease ($P < 0.099$) for NC fed pigs from OA sows compared to NC pigs from sows not fed the DFM or OA, respectively. The percent of pigs treated for other reasons from d 0-28 was greater when DFM was fed to sows only ($P < 0.041$) compared to when sows were not fed the DFM.

The following injectable therapy results encompass contrasts where the DFM and/or OA was added to the sow and nursery pig diets. When OA was fed to the sow and nursery pigs during d 21-28, a lesser percent of pigs were treated for any reason ($P < 0.042$) and specifically for being unthrifty ($P < 0.008$) compared to when the sow and pigs did not consume OA (any reason; average 6.25 vs. 15%, respectively). Overall from d 0-28, the percent of pigs treated for being unthrifty ($P < 0.082$) and the total therapies given per pen ($P < 0.068$) tended to decrease for pigs and sows fed the OA compared to pigs and sows not fed the OA (total therapies average 0.57 vs. 1.63). When the total therapies were divided by 6 pigs per pen to achieve a per pig basis, sows and nursery pigs fed OA had a reduced average treatment rate ($P < 0.045$) compared to sows and nursery pigs not fed OA (0.095 vs. 0.270 therapies per pig on average).

3.4.2.3   Intestinal Histology

Jejunal villus height and crypt depth were measured and ratio calculated (VH:CD) from samples collected on d 6 post-weaning from one pig per pen. Comparing pigs fed PC vs. NC from CON fed sows, there was a tendency ($P < 0.085$; Table 3.18) for pigs fed PC to have deeper crypts (251 vs. 220 μm) and a tendency for lower VH:CD ratio ($P < 0.106$).

When DFM was fed to sows and nursery pigs were fed NC, there was a tendency ($P < 0.058$) for harvested progeny to have a decreased VH:CD compared to progeny from sows not fed the DFM (average 1.34 vs. 1.59). This reduction in the ratio was mainly due to progeny from DFM and DFM+OA sows having numerically shorter villi. Feeding DFM or OA to sows and NC diet to nursery pigs tended (interaction; $P < 0.057$) to increase progeny crypt depth to approximately 250 μm where progeny from DFM+OA fed sows had a similar crypt depth to the NC fed pigs from CON sows of approximately 225 μm.

When the DFM was fed to sows and nursery pigs, progeny harvested on d 6 post-weaning had a decreased VH:CD ($P < 0.035$) compared to sows and pigs not consuming the DFM (average 1.34 vs. 1.67). This reduction in the ratio was due to a tendency ($P < 0.076$) for reduced villus height when sows and pigs were fed the DFM compared to when sows and pigs were not fed the DFM (average 309 vs. 351 μm).

3.4.2.4   Quantitative Polymerase Chain Reaction (qPCR)

Gene expression was evaluated using qPCR from jejunal tissue samples collected on d 6 post-weaning from one gilt per pen. There was no differences or tendencies detected ($P > 0.10$; Table 3.19) for gene expression of glutathione peroxidase 1 (GPx1), interferon gamma (IFN-γ), transforming growth factor beta 1 (TGF-β1), or tumor necrosis factor alpha (TNF-α). Comparing

pigs fed PC vs. NC from CON fed sows, expression of interleukin 10 (IL-10) was greater (0.51-fold increase; $P < 0.046$) for NC pigs from CON fed sows than PC fed pigs.

Feeding DFM to the sows and NC to the weaned piglets tended to result in a lower expression of occludin (OCLN; $P < 0.104$) compared to offspring from sows not fed the DFM. Feeding DFM or OA in sow diets tended to result in offspring having lower expression (interaction; $P < 0.086$) of claudin 7 (CLDN7), however when sows were fed DFM+OA and pigs fed NC, similar CLDN7 expression was observed compared to NC fed pigs from CON sows. There was also a tendency for expression to be lower for CLDN7 ($P < 0.104$), OCLN ($P < 0.094$), and tight junction protein 1 (TJP1; $P < 0.072$) when sows were fed OA and nursery pigs were fed NC compared to sows not fed OA and the pigs fed NC.

When sows and pigs were fed the OA, pigs tended to have lower expression of CLDN1 (interaction; $P < 0.089$) and interferon alpha (IFN-α; interaction; $P < 0.089$), however when sows and pigs were fed DFM+OA, the reduction in gene expression was attenuated to a level similar to when sows and pigs were fed the DFM. Feeding OA to the sows and nursery pigs resulted in a lower expression of OCLN ($P < 0.010$) and tended to result in a lower expression of CLDN7 ($P < 0.057$) and TJP1 ($P < 0.087$) compared to sows and pigs not fed the OA.

3.4.2.5   Econimic Evaluation

Due to the addition of antibiotics in the PC nursery diet, PC fed pigs had a greater feed cost per pig for all weeks and overall compared to NC pigs from CON fed sows ($P < 0.020$; Table 3.21). The PC fed pigs gained more weight resulting in PC fed pigs having a lower feed cost per kg of BW gain for d 7-14 and d 14-21 compared to NC pigs from CON fed sows ($P < 0.023$). However, the PC fed pigs had a greater feed cost per kg of BW gain for d 21-28 compared to NC pigs from CON fed sows ($P < 0.005$). The therapeutic drug cost per pig and drug cost per pig per day was

greater for NC pigs vs. PC pigs from control fed sows during d 21-28 ($P < 0.005$) and tended to be greater overall for NC fed pigs compared to PC fed pigs (d 0-28; $P < 0.073$).

The following economic evaluation results encompass contrasts where the DFM and/or OA were added to the sow diet only and progeny were fed the NC diet in the nursery. From d 21-28, feeding the OA in the sow diet decreased the therapeutic drug cost on a per pig basis ($P < 0.022$) and per pig per day basis ($P < 0.022$) compared to pigs from sows not fed the OA. Feed cost per kg of BW gain during d 14-21 tended to be greater for pigs from OA fed sows compared to pigs from sows not fed the OA ($P < 0.094$). Feeding DFM or OA in sow diets decreased (interaction; $P < 0.036$) feed cost per kg of BW gain from d 0-28, but for DFM+OA offspring, the feed cost increased above the NC pigs from CON fed sows. There was a tendency for an interaction ($P < 0.078$) for feed cost per pig during d 7-14 where pigs from the DFM fed sows had a decreased feed cost whereas offspring from the DFM+OA fed sows had the same feed cost per pig as the NC pigs from CON sows.

The following economic evaluation results encompass contrasts where the DFM and/or OA was added to the sow and nursery pig diets. Feeding DFM or OA to sows and their progeny decreased (interaction; $P < 0.012$) feed cost per pig during d 7-14, 14-21, and 0-28 with DFM+OA having similar or greater feed cost per pig compared to the NC pigs from CON sows. On a feed cost per kg of BW gain basis, feeding DFM or OA to sows and their progeny decreased (interaction; $P < 0.039$) cost during d 7-14, 21-28, and 0-28 with DFM+OA having similar or greater feed cost per kg of BW gain compared to the NC pigs from CON sows. Feed cost per kg of BW gain was greater when sows and pigs were fed the DFM from d 21-28 ($P < 0.019$) and tended to be greater from d 0-7 ($P < 0.076$) and d 0-28 ($P < 0.083$) compared to pigs and sows not fed the DFM. When sows and pigs were fed the OA, the feed cost per kg of BW gain increased, while therapeutic drug

cost per pig and therapeutic drug cost per pig per day decreased from d 21-28 ($P < 0.016$) and tended to decrease from d 0-28 ($P < 0.103$) compared to sows and pigs not fed the OA. There was a tendency during d 7-14 for the DFM or OA fed to sows and their progeny to increase (interaction; $P < 0.095$) the therapeutic drug cost per pig and drug cost per pig per day whereas the DFM+OA fed sows and pigs were not treated at all resulting in no drug costs during this period.

3.5   Discussion

3.5.1   Sow Experiment

Sows fed the OA in this experiment tended to gain more BW from d 80 to 112 of gestation compared to sows not consuming the OA. This observation was unexpected as all sows were fed a consistent 2.27 kg/day of gestation feed delivered once daily. One possible mode of action to explain the tendency in BW gain is that OA have been shown to increase nutrient digestibility in growing pigs and sows (Guggenbuhl et al., 2007; Mohana Devi et al., 2016), however we did not measure nutrient digestibility in this study.

Supplementation with 0.1% or 0.2% protected organic acids were fed to sows from d 95 of gestation to weaning (21 days; Mohana Devi et al., 2016). The protected organic acids were formulated to be released slowly throughout the gastrointestinal tract and contained 17% fumaric acid, 13% citric acid, 10% malic acid, and 1.2% MCFA consisting mainly of capric and caprylic acids. The 0.1 and 0.2% inclusion of protected organic acids fed to sows linearly increased digestibility of dry matter, N, and energy in lactation and there was no difference in lactation feed intake among treatments. We fed 0.4% of a MCFA blend that analyzed as adding about 0.1% MCFA, similar to the level added by Mohana Devi et al. (2016) giving plausibility to improving nutrient digestibility in our study as a possible mode of action.

The DFM fed sows tended to have less BW loss in lactation which is likely due to 8.4% numerically greater feed consumption and 16.4% greater water intake of DFM sows during d 7-14 of lactation compared to sows not fed the DFM. In agreement, Alexopoulos et al. (2004a) fed a combination of *Bacillus subtilis* and *Bacillus licheniformis* spores to gilts and observed an increased feed intake for the first 14 days after farrowing which reduced sow BW loss in lactation.

Sows fed the OA diets had fewer mummies per litter. Considering that sows started consuming experimental diets on d 80 of gestation, the OA only had the potential to have an effect on mummies from d 80 of gestation to parturition. There was no obvious disease challenge recorded during gestation, however the herd is positive for porcine reproductive and respiratory syndrome virus (PRRSV) and could have had subclinical infections at the time. This OA blend has been reported by Dee et al. (2020) to reduce disease causing organisms in the feed including porcine epidemic diarrhea virus (PEDV), PRRSV, and senecavirus A (SVA). Therefore, the possible subclinical infections may have been reduced when sows were fed OA resulting in fewer mummies.

Additionally, if mummies were formed after d 80 while on the experimental diets, it could be due to uterine crowding and insufficient space and nutrients for all the pigs in the litter. According to Ford et al. (2002), 5-10% of remining conceptuses are lost between d 90 to 114 of gestation due to uterine crowding which would be visualized by the presence of mummies. The mechanism in which the OA may have an impact on uterine environment in late gestation to aid in the survival of at-risk pigs is unclear. However, as discussed above, if nutrient digestibility was improbed, there would be more nutrients available to sustain fetuses. We also did not measure the crown-rump length of mummies to determine if they were formed before or after d 80. It is possible

that the differences in mummies was not related to dietary treatments if mummies were formed before diets were fed.

Born alive litter weights and individual pigs born alive to DFM fed sows were lighter compared to sows not fed the DFM with no statistical difference in litter sizes at birth. However, DFM fed sows did have the greatest total born at 0.91 more pigs than CON and interestingly all treatments had a greater number of total born pigs than CON fed sows. Therefore, the increased number of total born pigs for DFM sows might partially explain their lighter born alive piglet weights. More research with a robust number of sows should be conducted to investigate this potential response of DFM fed sows. In contrast, Baker et al. (2013) observed an increased number of pigs total born and born alive per litter that were also heavier at birth when sows were fed a DFM containing 2 strains of *Bacillus subtilis* spores (3.75×10^8 CFU/kg of complete feed) starting at approximately d 72 of gestation and throughout lactation. Baker et al. (2013) discussed that possibility the increased litter size at birth may be a reflection of conception rate that occurred before feeding the experimental diets on d 72 of gestation, which is similar to the current experiment starting dietary treatments on d 80 of gestation.

Baker et al. (2013) followed piglets to weaning and observed improvements in litter weaning weight, ADG, and decreased pre-weaning mortality when litters were from DFM fed sows compared to control fed sows. It could be the heavier pigs at birth from DFM fed sows may have simply continued to grow faster and were weaned heavier than control pigs simply due to being heavier at birth. However, authors attributed these improvements to a decrease in pathogen shedding from the sow and therefore a decreased challenge from the farrowing crate environment which was supported by reduced *Clostridium* populations in piglets nursing DFM supplemented sows.

In the current experiment, DFM fed sows tended to wean lighter pigs compared to pigs from sows not fed DFM with no differences in litter sizes at weaning. The pigs that were born lighter in this experiment tended to remain lighter at weaning with no difference in pre-weaning mortality. The performance differences between Baker et al. (2013) and the current experiment may be due to the different strains of DFM fed to the sows (*Bacillus subtilis* vs. *Bacillus licheniformis*, respectively). However, Alexopoulos et al. (2004a) fed a combination of *Bacillus subtilis* and *Bacillus licheniformis* spores to gilts ($1.28x10^9$ CFU/kg of complete feed) starting 14 d before farrowing and observed a reduction in scours, reduced pre-weaning mortality, increased number weaned and heavier weaning weights compared to litters from control fed sows with no difference in litter sizes at birth. These inconsistent results with a similar species of DFM, *Bacillus licheniformis*, indicates potentially different efficacies of DFMs fed to swine today (Liu et al., 2018).

The OA blend fed in this experiment contained short and medium-chain fatty acids as well as a cyclic short chain fatty acid, benzoic acid. Azain (1993) observed that 10% medium-chain triglycerides (MCT; C8 and C10) fed to sows improved pre-weaning survival (d 0-21) and additionally increased MCFA in milk collected on d 7 of lactation. In the current experiment, there was no difference in pre-weaning survival when OA was fed to the sow or not. Additionally, Lauridsen and Danielsen (2004) fed 8% supplemental fat of varying sources including coconut oil (C12) to sows 1 week prior to farrowing and throughout a 28-d lactation. Litter weight gain from sows fed 8% coconut oil tended to be greater than sows fed 8% fish oil (66.3 vs. 59.3 kg in 28 days). Coconut oil fed sows also had a greater daily output of fat in milk compared to fish oil and sunflower oil fed sows. In the current experiment, there was no difference in litter weaning weight

due to OA and this may be related to feeding only 0.4% OA in the current study vs. 8-10% in the other studies.

A digital Brix refractometer was used in this study to indirectly measure colostrum IgG concentration. The Brix percentage positively correlates with sow colostrum IgG concentration when measured by a commercially available radial immune diffusion kit (r = 0.56; Balzani et al., 2016) and when measured by an ELISA kit (r = 0.63; Hasan et al., 2016). Hasan et al. (2016) categorized brix readings of 25% to 29% as having an adequate colostrum IgG content (50.7 ± 2.1 mg/mL). Therefore, although there was no difference in average Brix value due to dietary treatment, the colostrum from these sows overall with a Brix value around 25% is considered adequate.

Baker et al. (2013) and Alexopoulos et al. (2004a) observed no difference in wean-to estrus interval. In this study, an interaction was approaching a trend where sows fed DFM returned to estrus 1.0 day sooner than CON, but only 0.6 and 0.4 days sooner when sows were fed the OA and DFM+OA diets, respectively. The quicker return to estrus is likely related to the sows fed the DFM tending to have less BW loss in lactation and consuming numerically 6.4% more feed from d 1 of lactation to weaning compared to sows not consuming the DFM. It has long been recognized that an increased feed intake of sows in lactation is associated with a shorter wean to estrus interval and subsequently larger litter size (Koketsu and Dial, 1997). When Xue et al. (2012) fed 129 sows lactation diets with an SID Lysine:Metabolizable energy ratio of 2.1 to 3.3 g/Mcal, the wean to estrus interval decreased linearly from 6.44 to 5.55 days. Authors also attributed the shorter wean to estrus interval to an observed increase in plasma luteinizing hormone concentration on d 21 of lactation for those sows (Xue et al., 2012). A recent review by Tokach et al. (2019) recognized

that BW loss and mobilization of body protein and fat reserves of lactating sows is responsible for prolonged wean to estrus intervals, however modern sows may be more resilient.

3.5.2 Nursery Experiment

The nursery experiment was designed to evaluate if the DFM and/or OA could be fed to the sow alone or if the nursery pigs also need to consume the DFM and/or OA in order to observe improved gut integrity, gene expression, and nursery pig growth performance. Again, the OA blend fed in this portion of the experiment contained short-chain fatty acids (SCFA) and medium-chain fatty acids (MCFA) as well as a cyclic SCFA, benzoic acid.

The majority of the 8 nursery treatments were formulated with no antibiotics and no pharmacological levels of zinc or copper due to increasing Food and Drug Administration (FDA) and European Union regulations on the use of these feed technologies in swine feed (ZnO banned in European Union by June 2022; GFI #213; U.S. Food and Drug Administration, 2020). One nursery treatment however, served as a positive control (PC) and contained neomycin-oxytetracycline in phases 1 and 2 (827 and 551 ppm) and carbadox in phase 3 and 4 (55 ppm).

Historically since the 1950's, antibiotics have been used in swine feed to suppress or inhibit the growth of certain microorganisms resulting in pig growth promotion, improvements in feed efficiency, and reduction of morbidity and mortality (Cromwell, 2012). According to Dritz et al., (2002), data involving 3648 nursery pigs highlighted that the addition of antibiotics in nursery diets significantly improved ADG by 5.0% from 436 to 458 g/d in modern production systems with trial durations of 28 to 32 days. When LeMieux et al. (2003) fed oxytetracycline and neomycin for 27 days in the nursery, overall ADG significantly increased by 11.2% from 329 to 366 g/d when the antibiotics were added. LeMieux et al. (2003) also did not add pharmacological

levels of zinc to their diets but did include a flavoring agent technology. In the current study, pigs fed the antibiotic PC grew 342 g/d during the 28-d study compared to 245 g/d when pigs were fed NC from CON sows. This 39.6% improvement in ADG when antibiotics were added to the diet is a larger difference compared to Dritz et al. (2002) and LeMieux et al. (2003). While these other authors formulated the negative control diets to also contain other feed technologies, the current study had a true negative control with no other feed technologies (except plasma protein). Therefore, the smaller levels of improvement observed in other studies may be due to the antibiotics working in conjunction with other feed technologies where this study did not.

Interestingly, except for PC fed pigs, the remaining pens of pigs had a growth rate of approximately 196 g/d from d 7-14 which remained at approximately 214 g/d from d 14-21. This observation may be due to pigs experiencing some sort of subclinical health challenge. Although the statistical analysis in this experiment did not test the PC vs. everyone else, from the nursery therapy records, there were 16 pigs treated on d 21 of the experiment (9 for diarrhea and 7 for unthrifty) compared to only 7 on d 14 (1 diarrhea, 3 unthrifty, and 3 lame). The growth rate of pigs, excluding PC, then increased to approximately 473 g/d from d 21-28.

Mohana Devi and Kim (2014) conducted a similar nursery experiment where they fed MCFA alone or in combination with a DFM only to the nursery pigs without the maternal component present in our study. When pigs were fed 0.2% MCFA and/or 0.01% DFM (10^9 CFU *Enterococcus faecium* DSM 7134/kg of complete feed) for 6 weeks in the nursery, the overall ADG and G:F increased when pigs were fed the DFM and combination diets compared to the control (Mohana Devi and Kim, 2014). The improvement in ADG and G:F for the combination diet is in contrast with the current experiment. Additionally, Mohana Devi and Kim (2014) observed the nutrient digestibility (ATTD) of dry matter, nitrogen, and energy were increased

when pigs were fed the combination diet compared to the other 3 diets and feeding MCFA and DFM individually resulted in a greater digestibility compared to the control.

When feeding DFM without MCFA or OA, Alexopoulos et al. (2004b) observed decreased morbidity and mortality associated with post-weaning *E. coli* diarrhea and greater ADG and G:F when diets were supplemented with *Bacillus subtilis* and *Bacillus licheniformis* spores in the nursery. Kritas and Morrison (2005) also fed a *Bacillus subtilis* and *Bacillus licheniformis* DFM to nursery pigs and determined these pigs had very similar ADG, ADFI, and G:F compared to pigs fed low doses of antibiotics.

In contrast to the current study where there were no differences observed in ADG due to dietary treatment, other than the PC, Dierick et al. (2002) fed 2.5% medium-chain triglyceride oil (MCT; C8 and C10) to pigs with and without 0.1% lipase 1 week before weaning at 21 days of age until 3 weeks post-weaning. Authors reported that pigs fed MCT with or without lipase had increased ADG during the first 2 weeks post-weaning compared to control (164 and 165 vs. 127 g/d from d 0-7; 160 and 161 vs. 127 g/d from d 7-14). There were no differences in growth during the week before weaning, the third week post-weaning, or the overall post-weaning period. Feed intake and feed conversion ratio was not different between treatments at any point of the Dierick et al. (2002) experiment which is in contrast with the current study where feeding DFM or OA to sows and/or their progeny improved G:F for the overall experiment (d 0-28).

A group of 21-d-old weaned pigs were fed a control diet, control plus 0.2% benzoic acid, or control plus 0.5% benzoic acid for 42 days in the nursery (Chen et al., 2017). Pigs fed the 0.2% benzoic acid diet had heavier body weight and greater ADG at d 14 (d 0-14) and 42 (d 0-42) of the experiment compared to controls and 0.5% benzoic acid supplemented pigs were heavier with a greater ADG on d 42 (d 0-42). These results were in contrast with the current study where there

were no differences observed in ADG or BW due to dietary treatment, other than the PC. Pigs fed benzoic acid diets were also more feed efficient from d 0-42 of the study, where only 0.2% benzoic acid fed pigs had a greater overall ADFI (Chen et al., 2017). Similarly, in the current study, feeding DFM or OA to sows and/or their progeny improved G:F for the overall experiment (d 0-28).

The main function of the gastrointestinal tract (GIT) is to digest and absorb dietary nutrients while excluding potential pathogens. In order for the GIT to remain healthy and functional, the absorptive capacity and gut barrier needs to be maintained. However, during weaning, pigs are subject to new social and environmental stressors and they transition from a liquid to solid diet. In addition to overall weaning stress, some pigs are slow to consume solid feed which allows for gut inflammation and increased proliferation of harmful gut bacteria like pathogenic *E. coli* (Fairbrother et al., 2005). All of these factors shortly following weaning work together to cause blunting of villi and impaired gut barrier function (Liu, 2015). One pig per pen was harvested 6 days post-weaning to evaluate the ability of these feed additives, DFM and OA, to alleviate the negative effects of weaning on nursery pigs when fed only to the sow or to the sow and nursery pigs. All sacrificed pigs were consuming feed prior to being harvested based on stomach and intestinal contents being present.

It is common in the swine industry to feed SCFA to piglets during the immediate post-weaning period. In general, SCFA are used for acidification of the gut, pH reduction, in order to decrease the growth of pathogenic bacteria in the gut (Gabert and Sauer, 1994). Acidification also improves nutrient digestibility via increased pepsin activity and stimulated gut development and integrity in pigs (Gabert and Sauer, 1994; Suiryanrayna and Ramana, 2015). Reducing pathogenic overgrowth via pH is one way to control pig post-weaning diarrhea (Lauridsen, 2020). Although we did not evaluate fecal scores in this experiment, we did evaluate injectable therapies

administered each week in the nursery and did not observe a large difference in therapy rate for enteric challenges among dietary treatments. However, feeding the OA blend to sows and/or their progeny may decrease the need for therapeutic antibiotic injections (for any reason) during d 21-28. Interestingly, d 21-28 was the first week OA was removed from the nursery diets, but it may have had a lasting carryover effect if there was a subclinical disease challenge occurring. Another intriguing observation of the therapeutic treatments is that the pigs from sows fed DFM+OA and also DFM+OA in the nursery required no treatments at all through d 21 post-weaning, similar to the antibiotic PC.

Another advantage to feeding the OA blend to nursery pigs is the inclusion of MCFA. Adding MCFA to swine diets serve as a readily available energy source to promote pig growth and gut development and integrity (Zentek et al., 2011). Medium-chain fatty acids are a source of quick energy because they are absorbed into the enterocyte and mainly transported through the blood to the liver avoiding the time-consuming mechanisms of chylomicron formation or re-esterification (Velázquez et al., 1996).

Due to the inclusion of MCFA in the OA diets of sows and nursery pigs, we hypothesized that pigs of treatments containing OA would have longer villi in the jejunum when harvested 6 days post-weaning. There was no difference in villus height, crypt depth, or their ratios in this study when the OA was fed to the sow or the sow and nursery pigs. In agreement, although measured 28 days post-weaning, supplementation of short-chain organic acids or in combination with MCFA did not affect morphometric data (Ferrara et al., 2017). Additionally, Chen et al. (2017) fed benzoic acid to nursery pigs and observed that jejunal villus height:crypt depth increased in pigs fed benzoic acid compared to controls on d 14 and 42 post-weaning. The only significant observation in this study on small intestinal histology was when the DFM was fed to the sows and

nursery pigs, harvested progeny had a decreased ratio of villus height to crypt depth compared to sows and pigs not consuming the DFM.

In addition to the absorptive capacity and gut barrier function, the GIT also houses more than 70% of the body's immune cells (Willing et al., 2012). Activation of the immune system as an inflammatory response results in production of signaling molecules, e.g. pro- and anti-inflammatory cytokines. In the current experiment, there was no difference in the gene expression of the pro-inflammatory cytokines: TNF-α, IFN-α, or IFN-γ. Again, the current experiment harvested pigs 6 days post-weaning (21-d-old pigs). Similarly, according to Pié et al. (2004), gene expression of pro-inflammatory cytokines including TNF-α, IL-1β, IL-6, and IL-8 in the mid-small intestine increased the day after weaning but rapidly returned to pre-weaning values between days 2 and 8 post-weaning for 28-d-old pigs. Additionally, Hu et al. (2013a) measured gene expression in the pre-weaned 21-d-old pig and on days 3 and 7 post-weaning. In contrast to this study, authors observed an increased gene expression for TNF-α and IL-6 on d 3 and d 7 post-weaning compared to the pre-weaned control pigs.

In this study, there was also no difference in gene expression for the anti-inflammatory cytokine, TGF-β, which is in agreement with Hu et al. (2013a) when measured in the jejunum of pigs 7 days post-weaning compared to pre-weaning controls. However, pigs from CON sows in this experiment that were fed the antibiotic PC diet had a 0.51-fold lower expression of the anti-inflammatory cytokine, IL-10, in the jejunum compared to NC fed pigs from CON sows. Helm et al. (2019) fed nursery pigs subtherapeutic chlortetracycline (CTC; 40 ppm) for 35 days in the nursery and measured ileum mRNA gene expression of pigs euthanized on d 35 post-weaning. In agreement with the current study, Helm et al. (2019) observed a tendency for an IL-10 gene

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expression reduction (0.36-fold) in the ileal intestinal tissue of CTC fed pigs compared to control fed pigs.

In the gut, inflammatory cytokines such as TNF-α increase tight junction permeability and anti-inflammatory cytokines such as TGF-β have protective effects on the intestinal epithelial barrier (Xiao et al., 2017). Tight junction proteins, e.g. TJP1, OCLN, CLDN1, and CLDN7, serve as a paracellular barrier between adjacent intestinal epithelial cells. These proteins are located near the apical brush boarder and within the apical junction complex (Johnson et al., 2012). Although we did not observe differences in any pro-inflammatory cytokines, the expression of OCLN in jejunal mucosa of pigs harvested 6 days post-weaning was lower when OA was fed to the sows and pigs compared to when the OA was not fed to the sows and pigs. In contrast, a higher mRNA expression was observed on d 14 for TJP1 (previously known as ZO-1) and OCLN in the jejunum when pigs were fed 0.2% benzoic acid and only TJP1 when pigs were fed 0.5% benzoic acid compared to controls. Occludin and TJP1 expression was also upregulated on d 42 for both benzoic acid supplemented diets (Chen et al., 2017).

Although there was no difference in expression of tight junction proteins due to the addition of the DFM in the sow or sow and nursery pig diet, Yang et al. (2016) challenged nursery pigs with *E. coli* after feeding a DFM for 1 week and did see differences when they were sacrificed 1 week after the *E. coli* challenge (d 15 post-weaning). A low dose of *Bacillus subtilis* and *Bacillus licheniformis* spores (3.9×10^8 CFU/day) provided to weaned pigs via oral liquid solution lead to an increase in TJP1 jejunal mucosa gene expression compared to pigs with no DFM administration after being challenged with *E. coli* for 1 week (Yang et al., 2016). The increased TJP1 suggests the DFM prevented loss of intestinal epithelial barrier integrity however, Yang et al. (2016) did not observe this difference in occludin gene expression.

The nursery feed costs were evaluated on a per pig basis as well as a per kg of BW gain basis. Adding antibiotics to nursery diets did increase the feed cost per pig for each week and overall in this study, however the increased BW gain of those pigs justified adding the antibiotics as seen by the reduced feed cost per kg of BW gain during d 7-14 (-$0.232/kg BW gain) and d 14-21 (-$0.140/kg BW gain). Results indicate that antibiotics were not cost effective on a feed cost per kg of BW gain basis during the last week of the study (d 21-28) during which the feed cost was $0.540 per kg BW gain for PC fed pigs compared to $0.447 per kg BW gain for NC fed pigs both from CON fed sows. Interestingly, also during the last week of the study, the therapeutic drug cost per pig and per pig per day was greater for NC pigs vs. PC pigs which may have helped increase the growth rate of NC fed pigs during this period, decreasing their feed cost per kg BW gain.

For the overall experiment (d 0-28), there was an interaction for feed cost per kg of BW gain when the feed additives were in the sow diet only and when the feed additives were also fed to the nursery pigs. In both contrasts, feed cost per kg of BW gain decreased when adding the DFM or OA to the sow and/or nursery diet but increased the feed cost for the DFM+OA diet fed to the sow and/or nursery pigs similar to or greater than the feed cost for the NC fed pigs from CON sows. Although the feed cost per kg of the diet increased when adding the feed additives, these results were driven by a slight reduction in feed intake when adding the DFM or OA to the sow and/or nursery diet with ADG being very similar among treatments. Additionally, the DFM+OA fed pigs had both feed additives in the diet, a greater feed intake, and similar ADG resulting in a greater feed cost per kg of BW gain compared to NC fed pigs from CON sows.

3.6 Conclusion

Feeding a *Bacillus licheniformis* DFM to sows may decrease pig born alive weight and subsequent weaning weight but reduce sow BW loss by increasing daily lactation feed intake by 6.4% which quickened the return to estrus. Other than decreasing the number of mummies per litter, feeding the OA alone or in combination did not significantly improve sow reproductive or litter growth performance in this study. Further benefits of feeding the OA alone or in combination with DFM may require a larger sample size to be detected.

Feeding antibiotics to nursery pigs greatly improved growth performance in this study. Feeding a *Bacillus licheniformis* DFM or an OA blend to sows and/or their offspring may improve nursery feed efficiency but not ADG or BW. Feeding the combination diet (DFM+OA) to the sow and nursery pigs tended to increase ADFI. Feeding the DFM+OA combination diet to sows and nursery pigs may reduce the need for therapeutic treatments post-weaning when feeding antibiotic free diets. The intestinal histology of the jejunum in progeny harvested 6 days post-weaning did not provide supporting evidence to improve gut morphology by feeding the DFM or OA to nursery pigs. Feeding the DFM or OA to sows and/or nursery pigs did result in some differences in gene expression but further research is needed to determine the true relevancy of the differences and impacts on gut function.

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Table 3.1 Sow diet composition (as-fed basis)

Item	Gestation[1]	Lactation[2]
Ingredient, %		
Corn	62.213	49.753
Soybean meal, 47% CP	12.850	32.600
DDGS, 7% fat	20.000	10.000
Choice white grease	1.000	3.000
Limestone	1.560	1.430
Monocal. phos., 21.5% P	0.510	1.350
Swine Vitamin premix[3]	0.300	0.300
Sow Vitamin Premix[4]	0.250	0.250
Trace mineral premix[5]	0.125	0.125
Selenium premix[6]	0.050	0.050
Phytase[7]	0.100	0.100
Salt	0.500	0.500
Availa Zn 120[8]	0.042	0.042
Treatment premix[9]	0.500	0.500
Total	100.000	100.000
Calculated analysis[10]		
ME, Kcal/kg	3297.94	3351.58
NE, Kcal/kg	2471.19	2466.17
Crude Protein, %	16.56	22.33
Crude Fat, %	4.98	6.04
Crude Fiber, %	3.08	2.93
Total Lysine, %	0.72	1.18
SID Lysine:ME, g/Mcal	2.19	3.53
Standardized ileal digestible (SID) amino acids, %		
Lysine	0.55	1.00
Methionine	0.26	0.31
Methionine & Cystine	0.50	0.61
Threonine	0.48	0.69
Tryptophan	0.13	0.23
Isoleucine	0.53	0.81
Valine	0.64	0.89
Ca, %	0.75	0.90
P, %	0.52	0.73
Phytase available P, %.	0.35	0.50

(Table continues)

[1] Diets were fed from approximately d 80 of gestation until sows entered farrowing crates on approximately d 112 of gestation.

[2] Diets were fed from d 112 of gestation until weaning.

[3] Provided per kg of diet: vitamin A, 4,961 IU; vitamin D_3, 1984 IU; vitamin E, 53 IU; vitamin K, 4 mg; riboflavin, 9.9 mg; pantothenic acid, 33 mg; niacin, 59 mg; and B_{12}, 0.040 mg.

[4] Provided per kg of diet: biotin, 0.22 mg; folic acid, 1.65 mg; choline, 551 mg; pyridoxine, 4.96 mg; vitamin E, 22 IU; chromium, 0.20 mg; and carnitine 49.6 mg.

[5] Provided per kg of diet: iron, 121.3 mg; zinc, 121.3 mg; manganese, 15.0 mg; copper, 11.3 mg; and iodine, 0.46 mg.

[6] Provided 0.3 ppm selenium.

[7] Phyzyme® (Danisco Animal Nutrition, Morlborough, UK) providing 600 phytase units (FTU)/kg.

[8] Availa Zn 120 (Zinpro Corporation, Eden Prairie, MN) is an organic zinc amino acid complex that provides 50.4 ppm Zn.

[9] Treatment premixes were added to create four dietary treatments. The CON premix contained only ground corn at 0.5% inclusion of the diet. The DFM premix contained the *Bacillus licheniformis* direct-fed microbial (DFM) at a rate of 0.025% plus 0.475% of corn providing 1.6×10^9 CFU/kg of complete feed. The OA premix contained an organic acid blend of medium and short-chain fatty acids (OA; DaaFit Plus, ADM Animal Nutrition, Quincy, IL) at a rate of 0.400% plus 0.100% of corn providing 0.4% OA. The combination diet premix contained 0.025% of DFM, 0.400% of OA, and 0.075% of corn providing 1.6×10^9 CFU/kg and 0.4% OA.

[10] Calculated nutrients were targeted to meet or exceed the NRC 2012. Nutrient Requirements of Swine. 11th ed. Natl. Acad. Press, Washington, DC.

Table 3.2 Nursery diet composition (as-fed basis)[1]

Item	Phase 1	Phase 2	Phase 3	Phase 4
Ingredient, %				
Corn	36.160	42.440	45.600	51.750
Soybean meal, 47% CP	14.500	17.360	25.430	26.250
DDGS, 7% fat	--	5.000	10.000	15.000
Choice white grease	--	--	--	3.000
Soybean oil	5.000	4.000	3.000	--
Limestone	0.820	0.990	1.000	1.480
Monocal. phos, 21.5% P	0.290	0.380	0.280	0.460
Vitamin premix[2]	0.250	0.250	0.250	0.250
Trace mineral premix[3]	0.125	0.125	0.125	0.125
Selenium premix[4]	0.050	0.050	0.050	0.050
Phytase[5]	0.100	0.100	0.100	0.100
Salt	0.250	0.250	0.300	0.350
Plasma protein	5.000	2.500	--	--
Spray-dried blood meal	1.500	1.000	--	--
Soybean concentrate	5.000	3.500	--	--
Fish meal	4.000	3.600	4.000	--
Dried whey	25.750	17.150	8.600	--
Lysine-HCL	0.150	0.300	0.380	0.500
DL-Methionine	0.230	0.215	0.170	0.150
L-Threonine	0.065	0.115	0.135	0.155
L-Tryptophan	0.010	0.025	0.030	0.030
Banminth-48[6]	--	--	--	0.100
Treatment premix[7]	0.750	0.650	0.550	0.250
Total	100.000	100.000	100.000	100.000
Calculated analysis[8]				
ME, Kcal/kg	3542.31	3485.08	3430.88	3405.91
NE, Kcal/kg	2760.84	2675.15	2578.25	2523.35
Crude Protein, %	24.36	23.09	22.72	21.47
Crude Fat, %	7.38	6.82	6.25	6.41
Crude Fiber, %	1.38	1.93	2.63	3.05
Total Lysine, %	1.73	1.62	1.53	1.44
SID Lysine:ME, g/Mcal	4.38	4.16	3.94	3.67
Standardized ileal digestible (SID) amino acids, %				
Lysine	1.55	1.45	1.35	1.25
Methionine	0.54	0.52	0.50	0.44
Methionine & Cystine	0.90	0.85	0.79	0.73
Threonine	0.96	0.90	0.84	0.78
Tryptophan	0.28	0.26	0.25	0.23
Isoleucine	0.86	0.81	0.80	0.73
Valine	1.07	0.97	0.88	0.82
Ca, %	0.85	0.85	0.80	0.75
P, %	0.70	0.66	0.62	0.54
Phytase available P, %.	0.55	0.50	0.45	0.35
Lactose, %	18.03	12.01	6.02	0.00

(Table continues)

[1] All phases were fed for 7 days and in meal form.

[2] Provided per kg of diet: vitamin A, 5,512 IU; vitamin D_3, 551 IU; vitamin E, 37 IU; vitamin K, 1.8 mg; riboflavin, 7.5 mg; pantothenic acid, 18 mg; niacin, 27.5 mg; and B_{12}, 0.03 mg. Wrong inclusion.

[3] Provided per kg of diet: iron, 121.3 mg; zinc, 121.3 mg; manganese, 15.0 mg; copper, 11.3 mg; and iodine, 0.46 mg.

[4] Provided 0.3 ppm selenium.

[5] Phyzyme® (Danisco Animal Nutrition, Morlborough, UK) providing 600 phytase units (FTU)/kg.

[6] Banminth® 48 (Phibro Animal Health, Teaneck, NJ) contains the active ingredient pyrantel tartrate to control internal parasites in pigs.

[7] Treatment premixes were added to create five dietary treatments. The NC premix contained only ground corn. The PC premix contained neomycin-oxytetracycline (Neo-Terramycin; Phibro Animal Health, Teaneck, NJ) in phases 1 and 2 (827 and 551 ppm) and carbadox (Mecadox; Phibro Animal Health, Teaneck, NJ) in phase 3 and 4 (55 ppm). The DFM premix contained the *Bacillus licheniformis* direct-fed microbial (DFM) at 1.6×10^9 CFU/kg for all phases. The OA premix contained an organic acid blend of medium and short chain fatty acids (OA; DaaFit Plus, ADM Animal Nutrition, Quincy, IL) at 0.5, 0.4, 0.3, and 0.0% OA in phases 1-4, respectively. The combination diet premix contained the DFM and OA at the same inclusion as the separated treatments for each nursery phase. All treatment premixes were created using fine ground corn as the carrier for antibiotics, DFMs, and/or OA.

[8] Calculated nutrients were targeted to meet or exceed the NRC 2012. Nutrient Requirements of Swine.11th ed. Natl. Acad. Press, Washington, DC.

Table 3.3 Fatty acid analysis of gestation sow diet (as-fed basis)[1]

Item	CON	DFM	OA	DFM+OA
Fatty Acids				
Crude Fat, %	5.70	5.60	5.57	5.15
C6:0, %	0.00	0.00	0.01	0.01
C8:0, %	0.00	0.00	0.01	0.02
C10:0, %	0.00	0.00	0.01	0.03
C12:0, %	0.00	0.00	0.04	0.04

[1] Samples were collected at the Purdue University Animal Sciences Research and Education Center (ASREC), subsampled in the lab at Purdue University (West Lafayette, IN), and shipped to University of Missouri (Columbia, MO) for fatty acid analysis.

Table 3.4 Fatty acid analysis of lactation sow diet (as-fed basis)[1]

Item	CON	DFM	OA	DFM+OA
Fatty Acids				
Crude Fat, %	5.81	5.94	5.81	5.85
C6:0, %	0.05	0.03	0.07	0.02
C8:0, %	0.00	0.00	0.03	0.02
C10:0, %	0.01	0.01	0.03	0.02
C12:0, %	0.00	0.00	0.15	0.08

[1] Samples were collected at the Purdue University Animal Sciences Research and Education Center (ASREC), subsampled in the lab at Purdue University (West Lafayette, IN), and shipped to University of Missouri (Columbia, MO) for fatty acid analysis.

Table 3.5 Fatty acid analysis of phase 1 and 2 nursery diet (as-fed basis)[1]

Item	Phase 1					Phase 2				
	PC	NC	DFM	OA	DFM+OA	PC	NC	DFM	OA	DFM+OA
Fatty Acids										
Crude Fat, %	7.35	7.25	7.26	7.41	7.63	6.68	6.58	6.78	6.72	6.89
C6:0, %	0.07	0.03	0.03	0.03	0.09	0.03	0.07	0.05	0.05	0.05
C8:0, %	0.02	0.01	0.01	0.03	0.07	0.00	0.02	0.02	0.04	0.03
C10:0, %	0.03	0.02	0.02	0.04	0.08	0.01	0.03	0.02	0.04	0.04
C12:0, %	0.00	0.02	0.02	0.13	0.27	0.02	0.00	0.03	0.15	0.13

[1] Samples were collected at the Purdue University Animal Sciences Research and Education Center (ASREC), subsampled in the lab at Purdue University (West Lafayette, IN), and shipped to University of Missouri (Columbia, MO) for fatty acid analysis.

Table 3.6 Fatty acid analysis of phase 3 and 4 nursery diet (as-fed basis)[1]

Item	Phase 3					Phase 4				
	PC	NC	DFM	OA	DFM+OA	PC	NC	DFM	OA	DFM+OA
Fatty Acids										
Crude Fat, %	6.35	6.42	6.07	6.58	6.42	5.86	6.19	6.29	6.13	5.86
C6:0, %	0.00	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.06	0.03
C8:0, %	0.00	0.00	0.00	0.01	0.01	0.00	0.01	0.00	0.00	0.01
C10:0, %	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.01
C12:0, %	0.01	0.01	0.01	0.04	0.04	0.00	0.00	0.00	0.00	0.00

[1] Samples were collected at the Purdue University Animal Sciences Research and Education Center (ASREC), subsampled in the lab at Purdue University (West Lafayette, IN), and shipped to University of Missouri (Columbia, MO) for fatty acid analysis.

Table 3.7 Gestation and lactation sow diet composition analyzed at Purdue University (as-fed basis)[1]

Item	CON	DFM	OA	DFM+OA
Gestation				
Crude Protein, %	15.26	15.50	16.14	14.27
Gross Energy, kcal/kg	3961.5	3999.4	4008.6	3959.9
Lactation				
Crude Protein, %	22.20	22.16	22.91	21.71
Gross Energy, kcal/kg	4009.6	4022.5	4029.0	4009.1

[1] Samples were collected at the Purdue University Animal Sciences Research and Education Center (ASREC), subsampled in the lab at Purdue University (West Lafayette, IN), and analyzed at Purdue University for gross energy and crude protein as an internal check.

Table 3.8 Nursery diet composition analyzed at Purdue University (as-fed basis)[1]

Item	PC	NC	DFM	OA	DFM+OA
Phase 1					
Crude Protein, %	23.32	23.29	23.86	23.61	24.12
Gross Energy, kcal/kg	4214.9	4162.0	4211.6	4197.5	4171.4
Phase 2					
Crude Protein, %	22.77	22.72	19.77	20.81	21.72
Gross Energy, kcal/kg	4135.9	4123.5	4154.2	4163.0	4107.9
Phase 3					
Crude Protein, %	20.21	20.38	23.11	23.06	23.67
Gross Energy, kcal/kg	4160.3	4168.1	4212.0	4153.0	4167.5
Phase 4					
Crude Protein, %	21.37	21.76	20.67	21.18	21.12
Gross Energy, kcal/kg	4158.0	4147.8	4155.8	4088.9	4110.9

[1] Samples were collected at the Purdue University Animal Sciences Research and Education Center (ASREC), subsampled in the lab at Purdue University (West Lafayette, IN), and analyzed at Purdue University for gross energy and crude protein as an internal check.

Table 3.9 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on sow BW, BCS, and ultrasound scans[1]

	Diet					Probability, $P <$		
	CON	DFM	OA	DFM+OA	SEM	DFM	OA	DFM × OA
Sows, *n*	12	10	12	13	---	---	---	---
Parity	2.58	2.51	2.26	2.36	0.456	0.971	0.577	0.841
Body Weight								
Day 80 of gestation, kg	227.99	226.64	222.77	220.54	7.484	0.797	0.418	0.950
Day 112 of gestation, kg	250.36	248.71	247.25	246.99	7.388	0.889	0.725	0.919
Gestation gain (d 80-112), kg	22.38	22.08	24.48	26.45	2.381	0.707	0.148	0.609
Gestation gain (d 80-112), %	10.31	9.96	11.16	12.13	1.139	0.772	0.159	0.532
Day 1 of lactation, kg	228.71	228.84	226.34	225.49	7.198	0.957	0.670	0.942
BW loss from d 112 - d 1, kg	-21.66	-19.88	-20.91	-21.50	2.849	0.821	0.869	0.655
BW loss from d 112 - d 1, %	-8.75	-8.23	-8.72	-8.83	1.096	0.842	0.779	0.758
Weaning, kg	210.66	216.76	209.47	212.30	7.700	0.534	0.694	0.819
Lactation change (d 1-wean), kg	-18.05	-12.08	-16.87	-13.20	2.827	0.072	0.990	0.662
Lactation change (d 1-wean), %	-8.22	-5.93	-7.83	-6.09	1.212	0.079	0.919	0.806
Body Condition Score[2]								
Day 80 of gestation	3.00	2.90	2.91	3.05	0.188	0.919	0.841	0.486
Day 112 of gestation	2.96	3.14	3.03	3.04	0.196	0.599	0.955	0.645
Gestation change (d 80-112)	-0.04	0.24	0.12	-0.01	0.146	0.565	0.737	0.134
Weaning	2.54	2.93	2.65	2.79	0.174	0.108	0.916	0.430
Lactation change (d 112 -wean)	-0.42	-0.21	-0.38	-0.26	0.166	0.281	0.966	0.774
Backfat thickness[3]								
Day 80 of gestation, cm	1.86	1.84	2.02	1.75	0.130	0.239	0.782	0.324
Day 112 of gestation, cm	1.73	1.86	1.73	1.72	0.111	0.580	0.532	0.479
Gestation change (d 80-112), cm	-0.14	0.02	-0.28	-0.03	0.055	<0.001	0.063	0.367
Gestation change (d 80-112), %	-7.79	2.39	-11.99	-1.33	2.659	<0.001	0.114	0.922
Weaning, cm	1.40	1.58	1.45	1.45	0.117	0.403	0.693	0.407
Lact. change (d 112 -wean), cm	-0.33	-0.27	-0.29	-0.27	0.053	0.487	0.662	0.723
Lact. change (d 112 -wean), %	-16.33	-14.47	-16.52	-15.75	2.609	0.593	0.766	0.825
Loin depth[3]								
Day 80 of gestation, cm	6.08	5.86	5.91	6.09	0.200	0.894	0.879	0.282
Day 112 of gestation, cm	5.67	5.84	5.61	5.75	0.198	0.400	0.664	0.934
Gestation change (d 80-112), cm	-0.41	-0.01	-0.30	-0.34	0.117	0.101	0.320	0.050
Gestation change (d 80-112), %	-6.73	-0.11	-4.80	-5.30	1.902	0.090	0.358	0.049
Weaning, cm	5.09	5.03	4.99	5.21	0.182	0.652	0.824	0.426
Lact. change (d 112 -wean), cm	-0.58	-0.81	-0.61	-0.54	0.171	0.618	0.460	0.347
Lact. change (d 112-wean), %	-9.75	-13.45	-10.50	-8.45	2.939	0.762	0.438	0.295

(Table continues)

[1]A total of 47 sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination improves sow body weight and body condition.

[2] Body condition scores range from 1 (emaciated) to 5 (obese). A body condition score of 3 is optimal.

[3] Live ultrasonic measurements of backfat and loin depth were collected from sows using an Aloka SSD 500V (Aloka Co., Ltd., Tokyo, Japan).

Table 3.10 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on sow reproductive and litter performance[1]

	CON	DFM	OA	DFM+OA	SEM	DFM	OA	DFM × OA
	Diet					Probability, $P <$		
Sows, n	12	10	12	13	---	---	---	---
Parity	2.58	2.51	2.26	2.36	0.456	0.971	0.577	0.841
Litter characteristics								
Total born,[2] n	12.08	12.99	12.74	12.72	0.740	0.520	0.777	0.500
Born alive, n	11.67	11.75	11.96	12.14	0.671	0.831	0.586	0.943
Born alive, %	97.13	90.58	94.79	95.24	2.381	0.174	0.600	0.119
Stillborn,[3] n	0.42	1.24	0.78	0.58	0.334	0.248	0.861	0.269
Stillborn,[3] %	2.87	9.42	5.21	4.76	2.381	0.182	0.871	0.295
Mummies,[3] n	0.83	1.24	0.53	0.12	0.359	0.876	0.010	0.114
Born alive litter weight, kg	20.20	17.24	19.49	18.75	0.902	0.032	0.636	0.190
Post cross-foster measurements[4]								
Litter weight, kg	19.61	17.02	18.71	17.73	0.832	0.025	0.901	0.305
Litter size, n	11.33	11.39	11.16	11.30	0.247	0.670	0.561	0.861
Weaning measurements[5]								
Survival after cross-foster, %	98.66	96.20	95.87	96.32	1.453	0.461	0.330	0.289
Litter weight, kg	70.08	62.65	64.35	64.55	2.237	0.093	0.367	0.074
Litter size, n	11.17	10.94	10.70	10.81	0.262	0.815	0.232	0.486
Weaning age, days	19.00	19.34	19.36	19.46	0.391	0.550	0.501	0.748
Average pig body weight								
Born alive,[6] kg	1.76	1.45	1.65	1.56	0.068	0.003	0.975	0.092
Post cross-foster, kg	1.73	1.49	1.69	1.57	0.070	0.008	0.805	0.364
Weaning, kg	6.33	5.73	6.06	5.95	0.219	0.093	0.898	0.234
Post cross-foster pig gain, kg	4.59	4.23	4.39	4.40	0.178	0.295	0.910	0.269
Post cross-foster ADG,[7] g/d	265	244	254	254	10.254	0.275	0.936	0.285

[1] A total of 47 sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination improves sow reproductive and litter growth performance to weaning.

[2] Total born was calculated as born alive plus stillborn piglets and does not include mummies.

[3] Data were square root-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and P-values represent the square root-transformed data.

[4] Cross-fostering was conducted within dietary treatment after piglet day 1 processing weights were collected.

[5] Weaning weights were collected 1 day before actual weaning age.

[6] Born alive weights were collected during piglet day 1 processing.

[7] Average daily gain was calculated from body weights collected on day 1 and the day before weaning.

Table 3.11 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on lactation feed intake[1,2]

	Diet					Probability, $P <$		
	CON	DFM	OA	DFM+OA	SEM	DFM	OA	DFM × OA
Sows, *n*	12	10	12	13	---	---	---	---
Parity	2.58	2.51	2.26	2.36	0.456	0.971	0.577	0.841
Number of days								
Entering farrowing to d 1	5.00	4.66	4.64	4.54	0.391	0.550	0.501	0.748
Day 14 to weaning	5.00	5.34	5.36	5.46	0.391	0.550	0.501	0.748
Day 1 to weaning	18.00	18.34	18.36	18.46	0.391	0.550	0.501	0.748
Average Daily Feed Intake, kg/d								
Entering farrowing to d 1	2.18	2.26	2.22	2.10	0.198	0.926	0.753	0.600
Day 1 to 3	3.12	3.17	3.24	3.35	0.342	0.804	0.646	0.924
Day 3 to 7	4.00	4.05	4.02	4.04	0.244	0.884	0.995	0.947
Day 1 to 7	3.74	3.76	3.77	3.84	0.245	0.846	0.784	0.916
Day 7 to 14	5.20	5.40	4.83	5.47	0.291	0.124	0.588	0.421
Day 14 to weaning	6.05	6.07	5.67	6.39	0.401	0.328	0.936	0.342
Day 1 to weaning	4.86	5.08	4.76	5.16	0.272	0.234	0.968	0.724

[1] A total of 47 sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination improves sow feed intake in lactation.

[2] Feed disappearance was measured on lactational days 1, 3, 7, 14, and weaning by weighing individual sow feed tubs and using a vacuum to collect and measure the amount of feed left in the feeder each day.

Table 3.12 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on lactation water intake[1,2]

	CON	DFM	OA	DFM+OA	SEM	DFM	OA	DFM × OA
	Diet					**Probability, $P <$**		
Sows, *n*	10	10	9	10	---	---	---	---
Parity	2.30	2.56	1.87	2.10	0.453	0.575	0.312	0.977
Number of days								
Entering farrowing to d 1	4.80	4.65	4.69	4.50	0.407	0.661	0.747	0.961
Day 14 to weaning	5.20	5.35	5.31	5.50	0.407	0.661	0.747	0.961
Day 1 to weaning	18.20	18.35	18.31	18.50	0.407	0.661	0.747	0.961
Average Daily Water Intake, L/d								
Entering farrowing to d 1	18.30	25.27	18.01	17.59	4.489	0.459	0.369	0.405
Day 1 to 3	12.04	13.98	14.64	13.69	1.881	0.788	0.533	0.436
Day 3 to 7	17.78	20.88	19.09	16.83	2.192	0.844	0.526	0.218
Day 1 to 7	15.86	18.58	17.61	15.78	1.924	0.813	0.780	0.235
Day 7 to 14	27.67	34.27	25.19	27.28	4.406	0.318	0.278	0.604
Day 14 to weaning	34.66	40.12	31.57	38.00	5.121	0.241	0.604	0.923
Day 1 to weaning	25.67	30.79	24.77	26.72	3.484	0.306	0.469	0.644

[1]A total of 39 sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination improves sow water intake in lactation.

[2] Water intakes were measured daily using individual water meters (Assured Automation, Clark, NJ). Average daily water intake was calculated to match feed intake time periods.

Table 3.13 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on colostrum Brix value[1]

	Diet					Probability, $P <$		
	CON	DFM	OA	DFM+OA	SEM	DFM	OA	DFM × OA
Sows, *n*	11	8	10	13	---	---	---	---
Parity	2.59	3.00	2.18	2.34	0.509	0.535	0.249	0.775
Colostrum Brix value, %	25.25	24.74	25.19	25.49	0.940	0.903	0.682	0.631

[1] A total of 42 sows were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination improves sow colostrum immunoglobulin G concentration (IgG). The Brix refractometer (MISCO, Cleveland, OH) was used to indirectly measure IgG.

Table 3.14 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on rebreeding rate and wean to estrus interval[1]

	Diet					Probability, $P <$		
	CON	DFM	OA	DFM+OA	SEM	DFM	OA	DFM × OA
Sows weaned, *n*	11	10	12	13	---	---	---	---
Parity	2.29	2.52	2.42	2.43	0.476	0.787	0.961	0.811
Sows returned to estrus, n	9	10	10	12	---	---	---	---
Weaned sows in heat,[2] %	81.82	100.00	83.33	92.31	---	0.155	0.788	---
Wean to estrus interval of sows in heat,[3] d	5.7	4.7	5.1	5.3	0.381	0.312	0.928	0.133

[1] A total of 46 sows were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination improves sow rebreeding rate and wean to estrus interval. One sow was retained as a lactation nurse sow from the CON treatment and therefore was not included in this data set.

[2] The percentage of weaned sows that returned to estrus was calculated as sows returned to estrus / sows weaned * 100 for each dietary treatment. The p-values presented for these calculations are Chi-square statistics.

[3] Wean to estrus interval was included for only those sows that returned to estrus.

Table 3.15 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows on piglet therapies during lactation[1,2]

	Diet				Probability, $P <$		
	CON	DFM	OA	DFM+OA	DFM	OA	DFM × OA
Sows, n	12	10	12	13	---	---	---
Parity	2.58 ± 0.415	2.51 ± 0.456	2.26 ± 0.416	2.36 ± 0.401	0.971	0.577	0.841
Post cross-foster litter size, n	11.33 ± 0.225	11.39 ± 0.247	11.16 ± 0.225	11.30 ± 0.217	0.670	0.561	0.861
Total therapies,[3] %	15.48 ± 5.775	13.06 ± 6.354	8.10 ± 5.796	13.20 ± 5.590	0.722	0.776	0.610
Lameness,[3] %	3.61 ± 2.251	9.62 ± 2.477	6.05 ± 2.259	4.60 ± 2.179	0.606	0.824	0.039
Enteric,[3] %	8.33 ± 4.218	0.48 ± 4.641	1.10 ± 4.233	0.56 ± 4.083	0.230	0.702	0.685
Unthrifty,[3] %	3.54 ± 1.083	1.66 ± 1.192	0.63 ± 1.087	0.55 ± 1.049	0.466	0.086	0.510
Other,[3,4] %	0.00 ± 3.527	1.30 ± 3.880	0.33 ± 3.540	7.50 ± 3.414	0.110	0.533	0.553
Total therapies[5]	4.00 ± 1.535	1.72 ± 1.689	1.10 ± 1.541	2.40 ± 1.486	0.911	0.508	0.630
Lameness, n	0.67 ± 0.355	1.19 ± 0.391	0.66 ± 0.356	0.68 ± 0.344	0.406	0.786	0.161
Enteric, n	2.75 ± 1.390	0.15 ± 1.529	0.21 ± 1.395	0.17 ± 1.345	0.293	0.482	0.470
Unthrifty, n	0.58 ± 0.184	0.20 ± 0.203	0.16 ± 0.185	0.07 ± 0.178	0.278	0.128	0.476
Other, n	0.00 ± 0.715	0.18 ± 0.787	0.07 ± 0.718	1.48 ± 0.692	0.172	0.406	0.445

[1] A total of 47 sows and their progeny were used to determine if feeding gestating and lactating sows a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), or the products in combination decreases the administration rate of therapeutic injections.

[2] Data were log-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and p-values represent the log-transformed data.

[3] Pigs treated at least once as a percent of the post cross-foster litter size.

[4] The other category was comprised of therapies for greasy pig, *Streptococcus suis* infection, and respiratory. Only one litter (DFM × OA treatment) was treated for greasy pig and all pigs in the litter were treated.

[5] The total therapies given in the litter which includes pigs treated multiple times.

Table 3.16 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows and/or nursery pigs on nursery growth performance[1]

Sow Diet	CON	CON	DFM	OA	DFM + OA	DFM	OA	DFM + OA	
Nursery Pig Diet (Treatment Letter)	PC (A)	NC (B)	NC (C)	NC (D)	NC (E)	DFM (F)	OA (G)	DFM + OA (H)	SEM
Pens, n	8	8	8	8	8	8	8	8	---
Day 0-7[2]									
ADG, g	183	126	128	137	113	105	130	122	13.3
ADFI, g	190	155	160	161	152	137	155	162	13.2
G:F	0.984	0.822	0.791	0.849	0.725	0.760	0.822	0.738	0.0436
BW d 0,[3] kg	6.34	6.35	5.98	6.09	6.18	5.95	6.11	6.17	0.415
BW d 7,[3] kg	7.40	7.01	7.02	7.08	6.92	6.86	7.03	6.98	0.091
BW d 7,[4] kg	7.34	7.01	6.97	7.04	6.94	6.84	6.96	6.99	0.091
Day 7-14									
ADG, g	290	190	187	220	204	186	192	193	17.2
ADFI, g	363	286	249	292	292	248	252	284	16.9
G:F	0.795	0.646	0.743	0.735	0.689	0.727	0.751	0.661	0.0358
BW d 14, kg	9.37	8.34	8.27	8.58	8.37	8.14	8.31	8.34	0.159
Day 0-14[2]									
ADG, g	232	155	155	175	155	142	159	155	11.5
ADFI, g	270	215	201	221	216	188	200	218	11.8
G:F	0.867	0.720	0.775	0.783	0.716	0.744	0.796	0.699	0.0268
Day 14-21									
ADG, g	366	220	213	211	208	206	203	242	16.4
ADFI, g	523	395	375	398	397	382	360	442	21.0
G:F	0.713	0.560	0.574	0.538	0.526	0.553	0.556	0.563	0.0340
BW d 21, kg	11.94	9.88	9.76	10.06	9.82	9.59	9.73	10.03	0.218
Day 0-21[2]									
ADG, g	275	176	173	186	172	162	172	182	10.4
ADFI, g	350	272	256	277	273	249	250	289	13.7
G:F	0.794	0.647	0.682	0.673	0.629	0.652	0.689	0.634	0.0205
Day 21-28									
ADG, g	556	465	482	495	484	480	488	422	14.7
ADFI, g	831	675	694	716	728	674	683	704	22.8
G:F	0.670	0.688	0.694	0.691	0.666	0.722	0.712	0.603	0.0201
BW d 28, kg	15.83	13.14	13.14	13.52	13.21	12.94	13.14	12.98	0.264
Day 0-28[2]									
ADG, g	342	245	247	260	245	238	248	240	9.5
ADFI, g	465	369	361	382	380	351	354	388	14.8
G:F	0.740	0.665	0.689	0.681	0.646	0.687	0.700	0.620	0.0151

Table 3.16 Continued

Contrast	DFM in sow diet[5]	OA in sow diet[6]	DFM × OA in sow diet[7]	DFM in sow and nursery diet[8]	OA in sow and nursery diet[9]	DFM × OA in sow and nursery diet[10]	Positive CON vs. Negative CON[11]
Probability, $P <$							
Day 0-7[2]							
ADG, g	0.401	0.839	0.315	0.262	0.434	0.605	0.003
ADFI, g	0.882	0.933	0.589	0.682	0.331	0.327	0.059
G:F	0.077	0.651	0.285	0.096	0.803	0.802	0.010
BW d 0,[3] kg	0.006	0.513	<0.001	0.001	0.858	<0.001	0.888
BW d 7,[3] kg	0.400	0.832	0.314	0.261	0.434	0.604	0.003
BW d 7,[4] kg	0.367	0.988	0.713	0.375	0.573	0.251	0.008
Day 7-14							
ADG, g	0.532	0.113	0.679	0.923	0.744	0.890	<0.001
ADFI, g	0.232	0.106	0.234	0.837	0.926	0.025	0.001
G:F	0.460	0.611	0.040	0.894	0.565	0.015	0.003
BW d 14, kg	0.339	0.257	0.614	0.566	0.581	0.451	<0.001
Day 0-14[2]							
ADG, g	0.359	0.374	0.369	0.438	0.479	0.680	<0.001
ADFI, g	0.403	0.333	0.682	0.702	0.495	0.042	0.001
G:F	0.830	0.949	0.026	0.178	0.547	0.028	0.001
Day 14-21							
ADG, g	0.725	0.594	0.876	0.362	0.497	0.059	<0.001
ADFI, g	0.589	0.523	0.618	0.081	0.511	0.019	<0.001
G:F	0.974	0.170	0.618	0.983	0.897	0.784	<0.001
BW d 21, kg	0.397	0.572	0.780	0.994	0.482	0.155	<0.001
Day 0-21[2]							
ADG, g	0.395	0.659	0.541	0.849	0.408	0.249	<0.001
ADFI, g	0.448	0.380	0.626	0.524	0.469	0.019	<0.001
G:F	0.814	0.462	0.042	0.181	0.525	0.118	<0.001
Day 21-28							
ADG, g	0.818	0.288	0.337	0.089	0.236	0.008	<0.001
ADFI, g	0.486	0.105	0.882	0.650	0.408	0.634	<0.001
G:F	0.636	0.538	0.449	0.067	0.022	0.001	0.532
BW d 28, kg	0.542	0.374	0.532	0.488	0.925	0.946	<0.001
Day 0-28[2]							
ADG, g	0.485	0.499	0.341	0.412	0.824	0.921	<0.001
ADFI, g	0.734	0.252	0.829	0.547	0.416	0.067	<0.001
G:F	0.720	0.349	0.049	0.053	0.300	0.001	0.001

(Table continues)

[1] Progeny from 47 dams fed diets containing a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short chain fatty acids (OA), DFM+OA, or a control (CON) diet from d 80 of gestation until weaning were used to determine if feeding these diets to the dam and/or progeny post-weaning improved nursery growth performance. A total of 64 pens of pigs (6 pigs/pen, 8 pens/treatment) were used in this study and initial BW was used as a covariate.

[2] Interval was calculated including the pig killed on d 6 of the study.

[3] Weight of all 6 pigs in the pen to start.

[4] Weight of the 5 pigs remaining and excluding the pig killed on d 6 of the study.

[5] Main effect of DFM in sow diet: treatment C and E vs. B and D.

[6] Main effect of OA in sow diet: treatment D and E vs. B and C.

[7] DFM×OA interaction in sow diet: treatments B and E vs. C and D.

[8] Main effect of DFM in sow and nursery diet: treatments F and H vs. B and G.

[9] Main effect of OA in sow and nursery diet: treatments G and H vs. B and F.

[10] DFM×OA interaction in sow and nursery diet: treatments B and H vs. F and G.

[11] Positive CON vs. Negative CON of the control sow: treatments A vs. B.

Table 3.17 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows and/or nursery pigs on nursery pig therapies[1,2]

Sow Diet	CON	CON	DFM	OA	DFM + OA	DFM	OA	DFM + OA	
Nursery Pig Diet	PC	NC	NC	NC	NC	DFM	OA	DFM + OA	SEM
(Treatment Letter)	(A)	(B)	(C)	(D)	(E)	(F)	(G)	(H)	
Pens, n	8	8	8	8	8	8	8	8	---
Day 0-7[3]									
Enteric, %	0.0	0.0	0.0	4.2	0.0	0.0	0.0	0.0	0.96
Lameness, %	0.0	2.1	2.1	2.1	2.1	0.0	2.1	0.0	1.65
Unthrifty, %	0.0	0.0	2.1	0.0	0.0	0.0	0.0	0.0	0.74
Other, %	0.0	0.0	0.0	2.1	2.1	0.0	0.0	0.0	1.04
Any reason, %	0.0	2.1	4.2	8.3	4.2	0.0	2.1	0.0	2.05
Day 7-14[4]									
Enteric, %	0.0	0.0	5.0	0.0	0.0	0.0	0.0	0.0	1.16
Lameness, %	0.0	2.5	0.0	5.0	0.0	2.5	2.5	0.0	1.92
Unthrifty, %	2.5	0.0	2.5	3.1	7.5	5.0	0.0	0.0	2.75
Other, %	---	---	---	---	---	---	---	---	---
Any reason, %	2.5	2.5	7.5	8.1	7.5	7.5	2.5	0.0	3.60
Day 14-21[4]									
Enteric, %	0.0	0.0	5.0	2.5	0.0	2.5	0.0	0.0	1.70
Lameness, %	0.0	2.5	0.0	2.5	0.0	0.0	2.5	0.0	1.53
Unthrifty, %	0.0	0.0	2.5	0.0	3.1	2.5	2.5	0.0	1.89
Other, %	0.0	0.0	2.5	0.0	0.0	0.0	0.0	0.0	0.88
Any reason, %	0.0	2.5	10.0	5.0	3.1	5.0	5.0	0.0	3.10
Day 21-28[4]									
Enteric, %	0.0	5.0	0.0	2.5	2.5	10.0	5.0	7.5	3.08
Lameness, %	0.0	2.5	2.5	0.0	0.0	0.0	0.0	0.0	1.25
Unthrifty, %	0.0	5.0	5.0	0.0	2.5	7.5	0.0	0.0	2.27
Other, %	0.0	0.0	2.5	0.0	0.0	0.0	0.0	0.0	0.88
Any reason, %	0.0	12.5	10.0	2.5	5.0	17.5	5.0	7.5	3.60
Day 0-28[3]									
Enteric, %	0.0	4.2	4.2	8.3	2.1	4.2	2.1	4.2	2.70
Lameness, %	0.0	2.1	4.2	4.2	0.0	2.1	4.2	0.0	2.26
Unthrifty, %	2.1	4.2	8.3	0.0	4.2	10.4	2.1	2.1	2.67
Other, %	0.0	0.0	4.2	2.1	4.2	0.0	0.0	0.0	1.55
Any reason, %	2.1	10.4	20.8	14.6	10.4	16.7	8.3	6.3	3.77
Total therapies,[5] n	0.13	1.38	1.63	1.38	1.13	1.88	0.75	0.38	0.49
Average,[6] n	0.02	0.23	0.27	0.23	0.19	0.31	0.13	0.06	0.08

Table 3.17 Continued

Contrast	DFM in sow diet[7]	OA in sow diet[8]	DFM × OA in sow diet[9]	DFM in sow and nursery diet[10]	OA in sow and nursery diet[11]	DFM × OA in sow and nursery diet[12]	Positive CON vs. Negative CON[13]
Day 0-7[3]							
Enteric, %	0.036	0.036	0.036	1.000	1.000	1.000	1.000
Lameness, %	1.000	1.000	1.000	0.212	1.000	1.000	0.376
Unthrifty, %	0.164	0.164	0.164	1.000	1.000	1.000	1.000
Other, %	1.000	0.051	1.000	1.000	1.000	1.000	1.000
Any reason, %	0.602	0.122	0.122	0.299	1.000	1.000	0.462
Day 7-14[4]							
Enteric, %	0.036	0.036	0.036	1.000	1.000	1.000	1.000
Lameness, %	0.056	0.517	0.518	0.518	0.518	0.518	0.362
Unthrifty, %	0.234	0.205	0.934	0.268	0.268	0.268	0.432
Other, %	---	---	---	---	---	---	---
Any reason, %	0.879	0.393	0.393	0.679	0.218	0.218	1.000
Day 14-21[4]							
Enteric, %	0.467	0.467	0.032	0.467	0.467	0.467	1.000
Lameness, %	0.109	1.000	1.000	0.109	1.000	1.000	0.254
Unthrifty, %	0.153	0.961	0.961	1.000	1.000	0.167	1.000
Other, %	0.164	0.164	0.164	1.000	1.000	1.000	1.000
Any reason, %	0.563	0.606	0.161	0.653	0.653	0.181	0.526
Day 21-28[4]							
Enteric, %	0.370	1.000	0.370	0.320	0.921	0.921	0.207
Lameness, %	1.000	0.051	1.000	0.322	0.322	0.322	0.164
Unthrifty, %	0.584	0.104	0.584	0.584	0.008	0.584	0.125
Other, %	0.164	0.164	0.164	1.000	1.000	1.000	1.000
Any reason, %	1.000	0.026	0.447	0.584	0.042	0.831	0.009
Day 0-28[3]							
Enteric, %	0.354	0.923	0.354	0.677	0.677	0.677	0.242
Lameness, %	0.347	0.902	0.237	0.288	1.000	0.288	0.452
Unthrifty, %	0.099	0.099	1.000	0.353	0.082	0.353	0.555
Other, %	0.041	0.488	0.488	1.000	1.000	1.000	1.000
Any reason, %	0.806	0.431	0.101	0.914	0.195	0.549	0.046
Total therapies,[5] n	0.988	0.600	0.262	0.931	0.068	0.384	0.050
Average,[6] n	0.998	0.624	0.448	0.950	0.045	0.363	0.070

(Table continues)

[1] Progeny from 47 dams fed diets containing a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short chain fatty acids (OA), DFM+OA, or a control (CON) diet from d 80 of gestation until weaning were used to determine if feeding these diets to the dam and/or progeny post-weaning decreases the administration rate of therapeutic injections. A total of 64 pens of pigs (6 pigs/pen, 8 pens/treatment) were used in this study. The other category was comprised of therapies for *Streptococcus suis* infection, respiratory, and an infected snout.

[2] Data was log-transformed to meet assumptions of normality; however, means are presented as non-transformed values for ease of interpretation and p-values and standard errors represent the log-transformed data.

[3] Pigs treated at least once as a percent of 6 pigs in the pen.

[4] Pigs treated at least once as a percent of remaining pigs in the pen. One pig per pen was harvested on d 6. One pig on d 7 was pulled off test that was fed NC from an OA fed sow. Two pigs, one on d 14 and another on d 21, were pulled off test that were fed NC from DFM+OA fed sows.

[5] The total number of therapies given in the pen during the whole study (d 0-28).

[6] The total number of therapies given in the pen during the whole study (d 0-28) divided by 6 pigs in the pen.

[7] Main effect of DFM in sow diet: treatment C and E vs. B and D.

[8] Main effect of OA in sow diet: treatment D and E vs. B and C.

[9] DFM×OA interaction in sow diet: treatments B and E vs. C and D.

[10] Main effect of DFM in sow and nursery diet: treatments F and H vs. B and G.

[11] Main effect of OA in sow and nursery diet: treatments G and H vs. B and F.

[12] DFM×OA interaction in sow and nursery diet: treatments B and H vs. F and G.

[13] Positive CON vs. Negative CON of the control sow: treatments A vs. B.

Table 3.18 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows and/or nursery pigs on nursery pig jejunal histology[1,2]

Sow Diet	CON	CON	DFM	OA	DFM + OA	DFM	OA	DFM + OA	
Nursery Pig Diet (Treatment Letter)	PC (A)	NC (B)	NC (C)	NC (D)	NC (E)	DFM (F)	OA (G)	DFM + OA (H)	SEM
Pigs, n	8	8	8	8	8	8	8	8	---
Villus height, μm	336	338	314	332	294	292	364	326	23.0
Crypt depth, μm	251	220	249	247	228	255	230	231	12.4
Villus height/crypt depth	1.42	1.72	1.32	1.46	1.35	1.22	1.53	1.46	0.131

Probability, $P <$

Contrast	DFM in sow diet[3]	OA in sow diet[4]	DFM × OA in sow diet[5]	DFM in sow and nursery diet[6]	OA in sow and nursery diet[7]	DFM × OA in sow and nursery diet[8]	Positive CON vs. Negative CON[9]
Villus height, μm	0.188	0.578	0.778	0.076	0.199	0.856	0.948
Crypt depth, μm	0.704	0.811	0.057	0.156	0.554	0.172	0.085
Villus height/crypt depth	0.058	0.397	0.264	0.035	0.848	0.111	0.106

[1] Progeny from 47 dams fed diets containing a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short-chain fatty acids (OA), DFM+OA, or a control (CON) diet from d 80 of gestation until weaning on d 21 of lactation were used to determine if feeding these diets to the dam and/or progeny post-weaning improved intestinal. A total of 64 pens of pigs (6 pigs/pen, 8 pens/treatment) were used in this study. The gilt with the weight closest to the pen average weaning weight within each pen was harvested 6 days post-weaning.

[2] A minimum of 8 well-oriented and intact villi were measured, and averaged, for villus height and crypt depth of adjacent crypts.

[3] Main effect of DFM in sow diet: treatment C and E vs. B and D.

[4] Main effect of OA in sow diet: treatment D and E vs. B and C.

[5] DFM×OA interaction in sow diet: treatments B and E vs. C and D.

[6] Main effect of DFM in sow and nursery diet: treatments F and H vs. B and G.

[7] Main effect of OA in sow and nursery diet: treatments G and H vs. B and F.

[8] DFM×OA interaction in sow and nursery diet: treatments B and H vs. F and G.

[9] Positive CON vs. Negative CON of the control sow: treatments A vs. B.

Table 3.19 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows and/or nursery pigs on relative fold changes in gene expression of jejunal mucosa from nursery pigs harvested 6 days post-weaning[1,2]

Sow Diet	CON	CON	DFM	OA	DFM + OA	DFM	OA	DFM + OA	
Nursery Pig Diet (Treatment Letter)	PC (A)	NC (B)	NC (C)	NC (D)	NC (E)	DFM (F)	OA (G)	DFM + OA (H)	SEM
Pigs, *n*	8	8	8	7	7	8	8	8	---
Gene of Interest, fold change									
CLDN1[3]	1.25	1.64	2.04	1.58	1.10	0.82	0.60	0.83	0.513
CLDN7[4]	0.88	1.01	0.97	0.83	0.99	0.93	0.85	0.87	0.071
GPx1[5]	1.16	1.01	1.16	1.42	1.14	1.17	1.13	1.81	0.314
IFN-α[6]	1.18	1.51	2.44	1.35	0.90	0.84	0.57	0.69	0.482
IFN-γ[7]	1.89	1.55	2.00	0.66	2.07	5.63	1.54	1.44	1.166
IL-10[8]	0.62	1.13	0.72	0.60	0.96	1.59	0.94	0.86	0.241
OCLN[9]	0.93	1.01	0.72	0.74	0.73	0.99	0.78	0.77	0.084
TGF-β1[10]	0.80	1.04	0.91	0.91	1.09	1.35	0.81	1.04	0.167
TJP1[11]	0.89	1.03	1.05	0.70	0.96	1.04	0.78	0.89	0.112
TNF-α[12]	1.56	1.19	0.88	0.94	1.10	1.42	1.17	1.41	0.446

Probability, *P* <

Contrast	DFM in sow diet[13]	OA in sow diet[14]	DFM × OA in sow diet[15]	DFM in sow and nursery diet[16]	OA in sow and nursery diet[17]	DFM × OA in sow and nursery diet[18]	Positive CON vs. Negative CON[19]
Gene of Interest							
CLDN1[3]	0.658	0.171	0.608	0.383	0.533	0.089	0.212
CLDN7[4]	0.248	0.104	0.086	0.697	0.057	0.334	0.133
GPx1[5]	0.718	0.511	0.370	0.150	0.501	0.306	0.834
IFN-α[6]	0.772	0.198	0.499	0.333	0.438	0.089	0.376
IFN-γ[7]	0.595	0.140	0.884	0.695	0.256	0.547	0.974
IL-10[8]	0.653	0.165	0.287	0.923	0.164	0.481	0.046
OCLN[9]	0.104	0.094	0.115	0.734	0.010	0.773	0.349
TGF-β1[10]	0.793	0.464	0.365	0.341	0.268	0.734	0.145
TJP1[11]	0.229	0.072	0.353	0.916	0.087	0.680	0.192
TNF-α[12]	0.826	0.471	0.422	0.848	0.905	0.651	0.268

(Table continues)

[1] Progeny from 47 dams fed diets containing a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short chain fatty acids (OA), DFM+OA, or a control (CON) diet from d 80 of gestation until weaning were used to determine if feeding these diets to the dam and/or progeny post-weaning changes gene expression in nursery pig jejunal mucosa. A total of 64 pens of pigs (6 pigs/pen, 8 pens/treatment) were used in this study. One gilt per pen was harvested 6 days post-weaning.

[2] Statistical analysis of the gene expression data was performed on the ΔCt values, and results are presented as fold changes with treatments held relative to the average expression of the Negative CON group. One sample was eliminated due to low RNA integrity from the treatment group where the sow was fed DFM+OA and pig was fed NC. Another sample was eliminated due to persistently low amplification from the treatment group where the sow was fed OA and the pig was fed NC.

[3] Claudin 1.

[4] Claudin 7.

[5] Glutathione peroxidase 1.

[6] Interferon alpha.

[7] Interferon gamma.

[8] Interleukin 10.

[9] Occludin.

[10] Transforming growth factor beta 1.

[11] Tight junction protein 1, formerly known as zonula occludens 1.

[12] Tumor necrosis factor alpha.

[13] Main effect of DFM in sow diet: treatment C and E vs. B and D.

[14] Main effect of OA in sow diet: treatment D and E vs. B and C.

[15] DFM×OA interaction in sow diet: treatments B and E vs. C and D.

[16] Main effect of DFM in sow and nursery diet: treatments F and H vs. B and G.

[17] Main effect of OA in sow and nursery diet: treatments G and H vs. B and F.

[18] DFM×OA interaction in sow and nursery diet: treatments B and H vs. F and G.

[19] Positive CON vs. Negative CON of the control sow: treatments A vs. B.

Table 3.20 Primers for quantitative polymerase chain reaction

	Source	Forward Primer (5' – 3') Reverse Primer (5' – 3')	Amplicon Length (bp)	Annealing Temp (°C)
Gene of Interest				
CLDN1[1]	NM_001244539.1	AGAAGATGCGGATGGCTGTC CCCAGAAGGCAGAGAGAAGC	193	60
CLDN7[2]	NM_001160076.1	ATCGTGGCAGGTCTTTGTG CTCACTCCCAGGACAAGAGC	192	60
GPx1[3]	NM_214201.1	TGGGGAGATCCTGAATTG GATAAACTTGGGGTCGGT	184	58
IFN-α[4]	NM_001166311.1	GGCTCTGGTGCATGAGATGC CAGCCAGGATGGAGTCCTCC	150	60
IFN-γ[5]	NM_213948.1	GCTCTGGGAAACTGAATGAC TCTCTGGCCTTGGAACATAG	167	60
IL-10[6]	NM_214041.1	GGTTGCCAAGCCTTGTCAG AGGCACTCTTCACCTCCTC	202	60
OCLN[7]	NM_001163647.2	GAGTACATGGCTGCTGCTGA TTTGCTCTTCAACTGCTTGC	102	60
TGF-β1[8]	NM_214015.2	GGACCTTATCCTGAATGCCTT TAGGTTACCACTGAGCCACAAT	133	60
TJP1[9]	XM_021098856.1	ACGGCGAAGGTAATTCAGTG CTTCTCGGTTTGGTGGTCTG	111	60
TNF-α[10]	NM_214022.1	CCCCCAGAAGGAAGAGTTTC TTGGCCCCTGAAGAGGAC	256	60
Housekeeping Gene				
ACTB[11]	XM_003124280.5	CCAGCACGATGAAGATCAAG AGTCCGCCTAGAAGCATTTG	171	60
GAPDH[12]	NM_001206359.1	CTTCACGACCATGGAGAAGG CCAAGCAGTTGGTGGTACAG	170	60
STX5[13]	NM_001243381.1	TGCAGAGTCGTCAGAATGGA CCAGGATTGTCAGCTTCTCC	144	60

(Table continues)

[1] Claudin 1; sequence order (Hu et al., 2013a).

[2] Claudin 7; sequence order (Pasternak et al., 2018).

[3] Glutathione peroxidase 1; sequence order (Cao et al., 2018).

[4] Interferon alpha, sequence order (Pasternak et al., 2020). This primer sequence targets a common region among sequenced transcripts for all current known IFN-α genes.

[5] Interferon gamma; sequence order (Pasternak et al., 2020).

[6] Interleukin 10; sequence order (Pasternak et al., 2018).

[7] Occludin; sequence order (Pasternak et al., 2018).

[8] Transforming growth factor beta 1; sequence order (Hu et al., 2013b).

[9] Tight junction protein 1, formerly known as zonula occludens 1; sequence order (Pasternak et al., 2018).

[10] Tumor necrosis factor alpha; sequence order (Ballweg et al., 2016).

[11] Actin beta; sequence order (Pasternak et al., 2020).

[12] Glyceraldehyde-3-phosphate dehydrogenase; sequence order (Pasternak et al., 2020).

[13] Syntaxin 5; sequence order (Pasternak et al., 2020).

Table 3.21 Effects of feeding a direct-fed microbial and organic acid blend, alone or in combination, to gestating and lactating sows and/or nursery pigs on nursery feed and therapeutic drug cost[1,2,3]

Sow Diet	CON	CON	DFM	OA	DFM + OA	DFM	OA	DFM + OA	
Nursery Pig Diet	PC	NC	NC	NC	NC	DFM	OA	DFM + OA	SEM
(Treatment Letter)	(A)	(B)	(C)	(D)	(E)	(F)	(G)	(H)	
Day 0-7									
Feed cost/pig, $	1.461	1.134	1.165	1.173	1.109	0.996	1.148	1.206	0.0970
Feed cost/kg gain, $	1.150	1.323	1.373	1.224	1.539	1.535	1.301	1.518	0.1404
Drug cost/pig, $	0.000	0.018	0.035	0.058	0.054	0.000	0.024	0.000	0.0208
Drug cost/pig/d, $	0.000	0.003	0.005	0.008	0.008	0.000	0.004	0.000	0.0030
Day 7-14									
Feed cost/pig, $	2.087	1.581	1.309	1.560	1.581	1.294	1.375	1.565	0.1269
Feed cost/kg gain, $	1.003	1.235	1.090	1.085	1.134	1.101	1.088	1.207	0.0757
Drug cost/pig, $	0.013	0.018	0.040	0.078	0.042	0.085	0.028	0.000	0.0283
Drug cost/pig/d, $	0.002	0.003	0.006	0.011	0.006	0.012	0.004	0.000	0.0040
Day 14-21									
Feed cost/pig, $	1.943	1.379	1.259	1.352	1.363	1.282	1.254	1.554	0.0927
Feed cost/kg gain, $	0.760	0.900	0.858	0.933	0.969	0.917	0.926	0.924	0.0605
Drug cost/pig, $	0.000	0.058	0.061	0.063	0.013	0.026	0.047	0.000	0.0335
Drug cost/pig/d, $	0.000	0.008	0.009	0.009	0.002	0.004	0.007	0.000	0.0048
Day 21-28									
Feed cost/pig, $	2.145	1.587	1.562	1.631	1.678	1.513	1.559	1.625	0.0952
Feed cost/kg gain, $	0.540	0.477	0.478	0.476	0.496	0.461	0.462	0.552	0.0151
Drug cost/pig, $	0.000	0.119	0.066	0.017	0.033	0.127	0.037	0.054	0.0285
Drug cost/pig/d, $	0.000	0.017	0.009	0.002	0.005	0.018	0.005	0.008	0.0041
Day 0-28									
Feed cost/pig, $	7.635	5.681	5.294	5.716	5.730	5.085	5.337	5.950	0.3368
Feed cost/kg gain, $	0.782	0.813	0.776	0.791	0.839	0.771	0.775	0.887	0.0206
Drug cost/pig,[4] $	0.013	0.216	0.209	0.233	0.170	0.238	0.141	0.054	0.0781
Drug cost/pig/d, $	0.000	0.007	0.007	0.008	0.005	0.008	0.005	0.002	0.0026

Table 3.21 Continued

Contrast	DFM in sow diet[5]	OA in sow diet[6]	DFM × OA in sow diet[7]	DFM in sow and nursery diet[8]	OA in sow and nursery diet[9]	DFM × OA in sow and nursery diet[10]	Positive CON vs. Negative CON[11]
			Probability, $P <$				
Day 0-7							
Feed cost/pig, $	0.8668	0.9304	0.6229	0.6785	0.2486	0.3145	0.0196
Feed cost/kg gain, $	0.1300	0.7761	0.2665	0.0759	0.8707	0.9800	0.3052
Drug cost/pig, $	0.7363	0.1583	0.6033	0.3100	0.8703	0.8703	0.5451
Drug cost/pig/d, $	0.7363	0.1583	0.6033	0.3100	0.8703	0.8703	0.5451
Day 7-14							
Feed cost/pig, $	0.1277	0.1293	0.0780	0.5528	0.6879	0.0050	<0.0001
Feed cost/kg gain, $	0.4249	0.3845	0.1110	0.9031	0.7318	0.0394	0.0087
Drug cost/pig, $	0.7905	0.2727	0.2976	0.4873	0.1799	0.0948	0.9001
Drug cost/pig/d, $	0.7905	0.2727	0.2976	0.4873	0.1799	0.0948	0.9001
Day 14-21							
Feed cost/pig, $	0.4187	0.5731	0.3351	0.1410	0.2833	0.0050	<0.0001
Feed cost/kg gain, $	0.9525	0.0938	0.3607	0.8545	0.6973	0.8166	0.0232
Drug cost/pig, $	0.4837	0.5286	0.4407	0.2424	0.5851	0.8222	0.2263
Drug cost/pig/d, $	0.4837	0.5286	0.4407	0.2424	0.5851	0.8222	0.2263
Day 21-28							
Feed cost/pig, $	0.8368	0.1273	0.4857	0.9430	0.4199	0.1826	<0.0001
Feed cost/kg gain, $	0.5123	0.5651	0.5314	0.0191	0.0157	0.0010	0.0050
Drug cost/pig, $	0.5290	0.0216	0.2385	0.6624	0.0092	0.8828	0.0050
Drug cost/pig/d, $	0.5290	0.0216	0.2385	0.6624	0.0092	0.8828	0.0050
Day 0-28							
Feed cost/pig, $	0.4269	0.3165	0.3920	0.9701	0.2693	0.0124	<0.0001
Feed cost/kg gain, $	0.7764	0.2983	0.0363	0.0829	0.0556	0.0003	0.2755
Drug cost/pig,[4] $	0.6607	0.8858	0.7235	0.6755	0.1036	0.4869	0.0727
Drug cost/pig/d, $	0.6145	0.8047	0.6770	0.6682	0.0958	0.4766	0.0663

(Table continues)

[1] Progeny from 47 dams fed diets containing a *Bacillus licheniformis* direct-fed microbial (DFM), an organic acid blend of medium and short chain fatty acids (OA), DFM+OA, or a control (CON) diet from d 80 of gestation until weaning were used to determine if feeding these diets to the dam and/or progeny post-weaning improved cost of nursery diets and therapeutic drugs administered in the nursery. A total of 64 pens of pigs (6 pigs/pen, 8 pens/treatment) were used in this study.

[2] The basal diet cost was $1.043/kg, $0.770/kg, $0.489/kg, and $0.328/kg of complete feed for phases 1-4, respectively. The antibiotic PC diet contained neomycin-oxytetracycline ($14.330/kg) in phases 1 and 2 and carbadox ($13.669/kg) in phases 3 and 4. The *Bacillus licheniformis* DFM cost $3.638/kg and OA blend cost $3.748/kg.

[3] Therapeutic drugs administered in the nursery as individual therapies included enrofloxacin ($0.72/mL), ceftiofur crystalline free acid ($1.34/mL), lincomycin hydrochloride ($0.52/mL), isoflupredone acetate ($0.649/mL), flunixin meglumine ($0.30/mL), penicillin ($0.129/mL), ampicillin ($0.305/mL), and dexamethasone ($0.095/mL).

[4] Total therapeutic drug cost for the pen divided by the number of pigs remaining in the pen on d 28.

[5] Main effect of DFM in sow diet: treatment C and E vs. B and D.

[6] Main effect of OA in sow diet: treatment D and E vs. B and C.

[7] DFM×OA interaction in sow diet: treatments B and E vs. C and D.

[8] Main effect of DFM in sow and nursery diet: treatments F and H vs. B and G.

[9] Main effect of OA in sow and nursery diet: treatments G and H vs. B and F.

[10] DFM×OA interaction in sow and nursery diet: treatments B and H vs. F and G.

[11] Positive CON vs. Negative CON of the control sow: treatments A vs. B.

CHAPTER 4. GROWTH PERFORMANCE AND CARCASS CHARACTERISTICS OF PROGENY FROM FROZEN SEMEN COLLECTED FROM DUROC BOARS IN 2000 TO 2005 VS. 2011 TO 2017

4.1   Abstract

Maternal line gilts (Topigs Norsvin TN70) were bred with frozen semen from Duroc boars born from 2000 to 2017 divided into two genetic groups: semen from boars born in 2000 to 2005 and 2011 to 2017. These genetic groups had different terminal sire indexes (TSI) of 88.2 and 112.0 for 2000 to 2005 and 2011 to 2017, respectively. A total of 155 pigs were weaned into 44 pens in a wean-to-finish facility to determine if genetics from two decades of sires and sex of the progeny impact progeny growth performance and carcass characteristics. Pens were comprised of 3 or 4 pigs per pen with each pen containing pigs from one sex and one sire from a genetic group. All pens received the same diets formulated for gilts with an average of 155 g/d protein accretion for SID lysine, calcium, and available phosphorus. Standardized ileal digestible (SID) amino acid levels were adjusted to the recommended ratios to lysine and all other nutrients were added at a level equal to or greater than 110% of the 2012 NRC requirements. Days 0 to 42 of the study represent the time period when the four nursery phases of feed were consumed. During this time there was a tendency for a genetics × sex interaction for G:F ($P < 0.054$) where in the 2000-2005 genetic group, gilts had a G:F advantage of 0.01 over barrows and in the 2011-2017 genetic group, gilts maintained a G:F advantage of 0.11. This consistent effect resulted in gilts being more feed efficient ($P < 0.018$) during the nursery period (d 0 to 42). Barrows consumed more feed than gilts during the grow-finish period (d 42 to the end of the study; $P < 0.022$) and weaning to market (d 0 to the end; $P < 0.031$). Gilts had a greater G:F than barrows from d 0 to the end of the study ($P < 0.006$). The ending body weights (BW) were collected on d 150 and 168 due to the pigs being

harvested at two separate time points. There was no difference in ending BW between genetic groups or sexes ($P < 0.529$). A subset of 20 pigs were harvested at the Purdue Meats Lab for each harvest group (d 150 and 168) totaling 40 pigs balanced for sex, genetic group, and sire within genetic group. When the carcass backfat was measured at the last lumbar, the 2000-2005 genetic group ($P < 0.048$) and barrows ($P < 0.004$) had thicker backfat than the 2011-2017 genetic group and gilts. Barrows had thicker bellies than gilts ($P < 0.031$). Gilts had a greater fat-free lean percentage than barrows ($P < 0.015$) and the 2011-2017 genetic group tended to have a greater fat-free lean percentage ($P < 0.090$). In conclusion, using frozen semen allowed the genetics of two different time periods of sires and sex of the progeny to be evaluated in this study. The expected large growth performance differences indicated by the TSI's of the two genetic groups were not observed. However, barrows had greater feed intake and fatter carcasses than the more feed efficient and leaner gilts in this study. Modern swine genetics have been selected to be leaner and results from this study agree, although the differences in live scan and carcass measurements were not as large as expected.

4.2   Introduction

The switch from live hog grading systems, based on live body weight (BW), to lean value-based marketing systems in the early 1990's has driven producers to raise leaner pigs to meet consumer demands of leaner pork. Carcasses sold to packers based on carcass leanness evaluation increased from 28 to 78% in 1988 to 1997 (Brorsen et al., 1998). Additonally, U.S. pork packer surveys conducted in 1992 and 2002 found that average hog backfat thickness decreased by 36% and the percentage of lean muscle increased from 49.5 to 55.5% (Martinez and Zering, 2004). Advances in measuring carcass leanness and genetic selection over many decades has resulted in leaner and more feed efficient pig production to supply the growing demand of pork products.

Inseminating a common genetic line of gilts with frozen semen collected from boars born in different years or even different decades is one method of evaluating genetic improvement over time (Canario et al., 2007; Foury et al., 2009). Progeny from these matings can then be followed from birth to market to assess differences in live growth and carcass characteristics due to genetic selection (Tribout et al., 2010).

The objective of this study was to determine if genetics from two decades of sires with different terminal sire indexes (TSI) and sex of the progeny impact progeny growth performance and carcass characteristics. Growth performance parameters included ADG, ADFI, and G:F. Carcass characteristics included measurements of backfat, loin muscle depth, fat-free lean percentage, and carcass yield on all pigs marketed. Additionally, loin muscle area, belly thickness, loin ultimate pH and drip loss percentage, and meat quality characteristics of meat color, marbling, and firmness were evaluated on a subset of carcasses. Live serial ultrasound scans were conducted at seven different time points throughout the grow-finish period, paired with BW, and were used to model the predicted growth of pigs in each genetic group and sex as time progressed and live weight increased.

4.3 Materials and Methods

4.3.1 General

The procedures for this experiment were approved by the Purdue University Animal Care and Use Committee (PACUC # 1112000366)**.** This study was conducted at the Purdue University Animal Sciences Research and Education Center (ASREC) where pigs were born in the farrowing facilities and weaned into the Swine Environmental Research Building (SERB) wean-to-finish facility.

4.3.2 Animals and Diets

Maternal line gilts (Topigs Norsvin TN70) were bred with frozen semen from Duroc boars born from 2000 to 2017. The progeny were divided in two genetic groups: semen from boars born in 2000 to 2005 genetics and 2011 to 2017 genetics. A total of 155 pigs were weaned into 44 pens in the wean-to-finish facility. Pens were comprised of 3 or 4 pigs per pen with each pen containing pigs from one sex and one sire from a genetic group. There were 15 pens comprised of 51 barrows and 17 pens with 62 gilts for 2000-2005 genetics. For the 2011-2017 genetics, there were 7 pens comprised of 24 barrows and 5 pens with 18 gilts.

All pens received the same diets (nursery phases 1 to 4 and grow-finish phases 1 to 6). Nursery and grow-finish diets were formulated to 110% of the swine nutrient requirement (NRC, 2012) for gilts with an average of 155 g/d protein accretion for SID lysine, calcium, and available phosphorus. Standardized ileal digestible (SID) amino acid levels were adjusted to the recommended ratios to lysine and all other nutrients were added at a level equal to or greater than 110% of the NRC requirements (Table 4.1 and 4.2). The last finishing phase of feed did not contain ractopamine. Feed samples were collected at Purdue University (West Lafayette, IN), subsampled, and shipped to University of Missouri (Columbia, MO) for analysis of crude protein, moisture, crude fat, crude fiber, ash, and amino acids. Subsamples were also analyzed for energy, calcium, and phosphorus at Purdue University swine nutrition lab (Table 4.3).

Pigs and feeders were weighed at weaning (d -1), d 14, 28, and 42 post-weaning which completed the nursery period. For the grow-finish period, pigs and feeders were weighed approximately every 3 weeks on d 63, 87, 108, 120, and 150 post-weaning. Pigs not marketed on the first marketing day (d 150) were also weighed on d 168 post-weaning along with feeders. Pig and feeder weights were used to calculate pen ADG, ADFI, and G:F. All therapeutic injectable antibiotic treatments were recorded and divided into six categories: enteric, lameness, Strep.,

unthrifty, respiratory, and other. The other category was comprised of therapies for salmonella and skin irritation. Pigs treated at least once within a period was calculated as a percent of the pigs remaining in the pen.

Serial ultrasound scanning was performed initially from 74 pigs in an attempt to scan about half of the pigs on test. The 74 scan pigs were comprised of 20 barrows and 22 gilts from the 2000-2005 genetic group and 16 barrows and 16 gilts from the 2011-2017 genetic group with all sires in a genetic group represented in each sex. An Aloka model 500V (Aloka Co., Ltd., Tokyo, Japan) ultrasound was used and fitted with an Aloka 3.5 MHz (Aloka Co., Ltd., Tokyo, Japan) probe. With the probe positioned perpendicular to the pig's spine, a backfat measurement was taken approximately 5 to 6 cm off the midline at the last rib and measurements of the backfat, loin depth, and loin muscle area were taken at the 10th rib (NPPC, 2000). Live serial ultrasound scanning was performed on the same group of pigs on d 42, 61, 88, 108, 120, and 150 post-weaning. All pigs (125 total) were live scanned before being harvested either on d 150 or 169 post-weaning.

A total of 20 pigs per marketing group (2 groups), balanced for genetic group and sex, were selected to be harvested at the Purdue Meats Lab (West Lafayette, IN). The remaining pigs that achieved market weight were harvested at Tyson Fresh Meats (Logansport, IN). The first group of pigs were harvested at the Purdue Meats Lab on d 154 post-weaning and at Tyson on d 155 post-weaning. The second group of pigs were harvested at the Purdue Meats Lab and at Tyson on d 170 post-weaning. All pigs were weighed at the farm 2 days prior to harvest.

At the Purdue Meats Lab, hot carcass weights were collected and carcass yield calculated (hot carcass weight divided by live weight multiplied by 100) by using the last live weight from the farm about 2 days before marketing. After approximately a 24-hour chill, each carcass was ribbed between the 10th and 11th rib, allowed 20 minutes to bloom, and evaluated for color (1 to

6), marbling (1 to 10), and firmness scores (1 to 5; National Pork Producers Council; NPPC). Scores of 1 indicate the lightest color, the most devoid of marbling, and the least firm. A Hunter MiniScan EZ colorimeter (Hunter; Reston, VA, USA) was used to collect L*, a*, and b* values from 3 different areas of the same longissimus dorsi sample and averaged (Hunt and King, 2012). Other measurements included the last lumbar backfat, last rib backfat, tenth rib backfat, belly thickness, and loin muscle area. The fat-free lean percentage was calculated as: 11.08 + (0.218*live weight, kg) + (-0.715*tenth rib backfat, cm) + (-3.31*last rib backfat, cm) + (0.346*loin muscle area, cm^2) / (live weight, kg* 0.74) * 100 (Schinckel et al., 2001). Ultimate pH measurements were taken using a calibrated meat pH probe directly inserted into the longissimus muscle on the 11th rib side of the 10th/11th rib carcass split (HANNA HI 99163, Hanna Instrument, Inc., Warner, NH, USA) calibrated with pH 4 and 7 buffers. Three meat samples (19 mm cores) were collected from the same longissimus dorsi sample (approximately 25 mm thick) from each pig. The sample cores were placed in pre-weighed EZ-DripLoss tubes (Danish Technological Institute, Taastrup, Denmark), reweighed, and stored in a cooler (4°C) for 24 hours. At 24 hours post-collection, the sample core was removed from the tubes and remaining purge was weighed. Drip loss percentage was calculated by dividing the weight of the purge by the weight of the original meat sample times 100. The drip loss percentage of the 3 meat cores were averaged.

At Tyson Fresh Meats, individually tattooed pigs had hot carcass weights collected, and carcass yield calculated by also using the last live weight from the farm (2 days before marketing). Carcass backfat and loin depth were measured using the Animal Ultrasound Services (Ithaca, NY) Carcass Value Technology System (CVT system) with a linear measurement from last to tenth rib. The standardized fat-free lean percent was calculated as: [3.0767 - (2.8117*average fat depth, cm)

+ (0.7156*average muscle depth, cm) + (0.47*hot carcass weight, kg)]/ (hot carcass weight, kg) *

100 (NPPC, 2000).

4.4   Statistical Analysis

Data were analyzed as a 2 × 2 factorial design of genetics group and sex using the PROC

MIXED procedure in SAS 9.4 (SAS Institute, Inc., Cary, NC). Growth performance data and

therapeutic injectable antibiotic treatment rate data were analyzed with pen as the experimental

unit. Live ultrasonic measurements and carcass measurements of pigs harvested at the Purdue

Meats Lab (West Lafayette, IN) and Tyson Fresh Meats (Logansport, IN) were analyzed with the

individual pig as the experimental unit.

For growth performance, carcass measurements, ultrasonic measurements, and therapeutic

injectable antibiotic treatment rates, the genetic group, sex, and the genetic group × sex interaction

were fixed effects. Carcass measurements of individual pigs harvested at Purdue and Tyson

included kill day in the model as a random effect. The PROC CORR procedure was used to

determine Pearson correlation coefficients between terminal sire index and average daily tissue

accretion rates. Therapeutic injectable antibiotic treatment rate data was log-transformed to meet

assumptions of normality. The PROC FREQ procedure in SAS 9.4 was used to conduct a chi-

square analysis of the mortality percentage from weaning to harvest. Differences were considered

significant at $P \leq 0.05$ and trends at $0.05 < P \leq 0.10$.

Individual live weights and ultrasonic backfat and loin muscle measurements were used to

model growth of the pigs over time, changes in body composition, and changes in nutrient

requirements as the pigs increased in BW or age. The PROC NLMIXED procedure in SAS 9.4

(SAS Institute, Inc., Cary, NC) was used to fit BW to the generalized Michaelis-Menten (GMM)

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function of time described as days of age (López et al., 2000; Schinckel et al., 2009a), ADFI to the Bridges function as a function of BW (Bridges et al., 1986; Schinckel et al., 2009b), and ultrasonic backfat depth and loin muscle area measurements to the mixed model allometric function: $Y = (A + a_i) BW^B$; where a_i is the pig specific random effect with variance σ_a^2 as a function of BW (Schinckel et al., 2009c). All models included the fixed effect of genetic group and sex. Functions were fit for each genetic group (2000 to 2005; 2011 to 2017) and sex (barrows; gilts).

Body components of empty body protein accretion, empty body lipid accretion, total carcass fat, and total fat-free lean were estimated using equations previously developed (Schinckel and de Lange, 1996; Wagner et al., 1999). The predicted body components were calculated on an average daily gain rate (g/d) for the grow-finish period. This data was analyzed using PROC MIXED procedure in SAS 9.4 (SAS Institute, Inc., Cary, NC) with pig as the experimental unit. The model included the fixed effects of genetic group, sex, and the genetic group × sex interaction. The analysis was also completed using the terminal sire index (TSI) as a covariate.

The ages or BW in which genetic population means differed for modeled growth, ultrasound measurements (backfat, loin muscle area, and loin muscle depth), and ADFI variables were evaluated. The variation of the random effect for each pig or pen, for feed intake, was used to estimate the variance in the pigs or pens within each genetic group and sex at each age or BW. The means of interest on the growth or feed intake curves were evaluated using a t-test with unequal variances using the within group variation and number of observations.

4.5 Results

4.5.1 Growth Performance

At weaning (d 0), there was a tendency for progeny born to 2011-2017 sires to be slightly heavier than progeny from 2000-2005 sires ($P < 0.085$; Table 4.4). There were no genetic or sex

differences during the early and mid-nursery, d 0 to 14 and d 14 to 28. There was a genetics $\times$ sex interaction for G:F during d 28 to 42 of the study ($P < 0.002$) and a tendency for an interaction for ADG in the same period ($P < 0.054$). This interaction and tendency was due to 2000-2005 barrows having greater ADG and G:F than 2000-2005 gilts, whereas in 2011-2017 progeny, gilts outperformed barrows by an average of 0.14 kg/d in ADG and 0.11 increase in G:F.

Days 0 to 42 of the study represent the time period when the four nursery phases of feed were consumed. From d 0 to 42, there was a tendency for a genetics $\times$ sex interaction for G:F ($P < 0.054$) where the magnitude of difference between barrows and gilts was greater for the 2011-2017 sired progeny compared to the 2000-2005 sired progeny. In the 2000-2005 genetic group, gilts had a G:F advantage of 0.01 over barrows, where in the 2011-2017 genetic group, gilts maintained a G:F advantage of 0.11. This consistent effect resulted in gilts being more feed efficient ($P < 0.018$) during the nursery period (d 0 to 42).

There were no genetics $\times$ sex interactions ($P > 0.107$) for the time period where the six grow-finish phases of feed were consumed (d 42 to 168) nor the overall study (d 0 to 168). From d 63 to 87 ($P < 0.066$) and d 120 to 150 ($P < 0.083$) the 2011-2017 genetic group tended to have a greater G:F compared to the 2000-2005 genetic group. The 2011-2017 genetic group had a greater ADG from d 120 to 150 ($P < 0.037$). Barrows gained and ate more than gilts from d 87 to 108, d 108 to 120, and d 120 to 150 ($P < 0.047$). From d 108 to 120, gilts had a greater G:F than barrows ($P < 0.043$).

Between d 150 and 168, the heaviest pigs were harvested in the first group at d 150 and therefore were removed from the pens. Of the remaining pigs on test, the gilts of both genetic groups had a greater ADG ($P < 0.004$) and G:F ($P < 0.002$) than barrows from d 150 to 168. Barrows consumed more feed than gilts during the grow-finish period (d 42 to the end of the study;

$P < 0.022$) and weaning to market (d 0 to the end; $P < 0.031$). Gilts had a greater G:F than barrows from d 0 to the end of the study ($P < 0.006$). The ending BW were collected on d 150 and 168 due to the pigs being harvested at two separate time points. There was no difference in ending BW between genetic groups or sexes ($P < 0.529$).

4.5.2   Carcass Characteristics

A subset of 40 pigs were harvested at the Purdue Meats Lab, 20 for each harvest group balanced for sex, genetic group, and sire within genetic group (Table 4.5). The National Pork Producers Council (NPPC) color scores measured from the longissimus dorsi ranged from 2.75 to 3.05 with no significant interaction or main effects of genetics or sex ($P > 0.05$). The L* values ranged from 51-54 with no significant differences ($P > 0.05$) between genetics or sexes. The a* value, ranging from 8.1 to 8.6, and the b* value, ranging from 5.7 to 6.6, were not different ($P > 0.05$) between genetics or sexes.

The NPPC firmness scores ranged between 2.40 and 2.85 with no significant differences ($P > 0.05$) among genetics or sexes. The NPPC marbling scores ranged between 2.05 and 2.75. The 2000-2005 genetics had higher marbling scores ($P < 0.006$) than the 2011-2017 genetics. However, there was a genetics × sex interaction for the loin muscle marbling score ($P < 0.002$). The barrows of the 2000-2005 genetic group had a greater average marbling score than the gilts, whereas in the 2011-2017 genetic group, the gilts had a greater marbling score than the barrows. The ultimate longissimus dorsi pH average ranged from 5.5 to 5.6 with no significant differences ($P > 0.05$) among genetics or sexes. The average loin muscle drip loss ranged from 6 to 9% and there was a tendency ($P < 0.076$) for average drip loss percentage to be greater in the 2011-2017 sired progeny compared to the 2000-2005 progeny.

Barrows had thicker backfat at the tenth rib measured on the carcass and via live ultrasound before harvest ($P < 0.005$). The main effect of genetics also existed for the live ultrasound scan data of these pigs for last rib backfat ($P < 0.023$), tenth rib backfat ($P < 0.021$), and a tendency for scanned tenth rib loin muscle depth ($P < 0.073$) and carcass tenth rib backfat ($P < 0.107$), with the 2000-2005 genetic group having thicker backfat and the 2011-2017 genetic group tending to have a greater loin muscle depth. When the carcass backfat was measured at the last lumbar, the 2000-2005 genetic group ($P < 0.048$) and barrows ($P < 0.004$) had thicker backfat than the 2011-2017 genetic group and gilts. Barrows had thicker bellies than gilts ($P < 0.031$). Gilts had a greater calculated fat-free lean percentage than barrows ($P < 0.015$) and the 2011-2017 genetic group tended to have a greater fat-free lean percentage ($P < 0.090$).

The remaining pigs that were not harvested at Purdue were harvested at Tyson Fresh Meats (Logansport, IN). Analysis of carcass characteristics paired with live ultrasound scan data were analyzed with the individual pig due to unequal animal numbers remaining in the pens (Table 4.6). There was a genetics × sex interaction for carcass yield ($P < 0.052$) and a tendency for an interaction for tenth rib loin muscle depth measured by live ultrasound scanning ($P < 0.083$). Carcass yield was greater for 2000-2005 barrows compared to gilts in that genetic group by 2.32%, however in the 2011-2017 genetic group, the gilts had a greater carcass yield by 1.33% compared to barrows. Ultrasound scanning revealed a tendency for an interaction where barrows in the 2000-2005 genetic group had a 0.15 cm deeper loin measured at the tenth rib compared to gilts and the difference was greater (0.25 cm) for the 2011-2017 genetic group.

Barrows had a heavier live weight ($P < 0.009$) and hot carcass weight ($P < 0.015$) compared to gilts across both genetic groups. The tenth rib loin muscle area measured by live ultrasound scan of these pigs tended to be larger for barrows ($P < 0.078$). The 2000-2005 genetics

tended to have a deeper loin muscle on the carcass ($P < 0.073$). Barrows had greater backfat depth collected from the carcass ($P < 0.002$) as well as last rib ($P < 0.001$) and tenth rib ($P < 0.001$) backfat depths measured by live ultrasound scanning than gilts. Gilts had a greater carcass percent lean than barrows ($P < 0.023$).

4.5.3   Live Serial Ultrasound Scans

Live serial ultrasound scans were conducted on a subset of initially 74 pigs at seven different timepoints throughout the grow-finish phases beginning on d 42 post-weaning. All pigs were scanned on d 150 post-weaning before the first group of pigs were marketed. Pigs that were not marketed in the first group were also scanned on d 169 post-weaning. The time point designated as the last scan day includes a total of 125 pigs that were scanned before harvest. The measures of last rib backfat, tenth rib backfat, tenth rib loin muscle depth, and tenth rib loin muscle area were collected at each time point.

Analysis of live ultrasound scans paired with live weight data were analyzed with the individual pig as the experimental unit (Table 4.7). The only genetics × sex interaction observed was for tenth rib loin muscle depth on d 108 post-weaning ($P < 0.019$). The 2000-2005 barrows had a 0.28 cm greater loin muscle depth than the 2000-2005 gilts. However, in the 2011-2017 genetic group, the gilts had a 0.17 cm greater loin muscle depth than the barrows. This same trend was observed on d 61 ($P < 0.112$), d 88 ($P < 0.105$), and last scan day ($P < 0.118$), however the genetics by sex interaction for loin muscle depth was not significantly different on these scan days.

On d 42 ($P < 0.039$) and d 150 ($P < 0.035$), the 2000-2005 genetic group had a thicker tenth rib backfat depth than the 2011-2017 genetic group with a similar genetic tendency on d 108 ($P < 0.059$). Also, on d 150, the 2011-2017 genetic group had less backfat at the last rib ($P < 0.043$) with a deeper loin muscle ($P < 0.040$) compared to the 2000-2005 genetic group. There was a

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tendency for the 2011-2017 genetics to have less last rib backfat ($P < 0.062$) on d 108 post-weaning as well as greater tenth rib loin muscle depth on the last scan day ($P < 0.088$).

Barrows had a heavier live weight than gilts on d 108, 120, and 150 ($P < 0.031$). Barrows had greater last and tenth rib backfat than gilts on d 88, 108, 120, 150, and last scan day ($P < 0.017$). On d 61, barrows tended to have greater tenth rib backfat ($P < 0.081$). Barrows had greater d 120 and d 150 tenth rib loin muscle depth ($P < 0.045$) and d 120 tenth rib loin muscle area ($P < 0.040$) than gilts. On d 150 post-weaning, there was a tendency for barrows to have greater tenth rib loin muscle area ($P < 0.062$).

4.5.4   Average Daily Tissue Accretions and Correlations with TSI

Average daily tissue accretions were calculated by subtracting the predicted tissue composition at d 42 post-weaning from their ending predicted tissue composition on either d 150 or d 169 post-weaning then dividing by the number of days between ultrasonic measurements (Table 4.8). The tissue compositions were predicted using data published by Wagner et al. (1999) and used to develop curves using equations published by Schinckel and de Lange (1996). Barrows had a greater average daily empty body lipid accretion ($P < 0.001$) and average daily total carcass fat gain ($P < 0.001$) per day than gilts. The average daily empty body protein and total fat-free lean gained per day was not different between genetic groups or sex ($P > 0.13$).

Pearson correlation coefficients were calculated between the terminal sire index and three variables which include average daily BW gain, protein deposition, and total fat-free lean gain (Table 4.9). These three correlations with an R value of 0.073, 0.040, and 0.067, respectively, were not significant ($P > 0.55$).

4.5.5   Therapeutic Injectable Antibiotic Treatment Rate and Mortality

The injectable therapeutic antibiotcs administered in this study are presented as a percentage of pigs remaining in the pen (Table 4.10). During the first 14 days post-weaning, there was no difference in total therapies given ($P > 0.552$) ranging from 16.7 to 21.1% of pigs being treated. There was a tendency ($P < 0.058$) for 2000-2005 genetics to have a greater percent of the pigs treated for lameness than 2011-2017 genetics (11.9 vs. 1.8%). There was a genetics × sex interaction ($P < 0.047$) for the percent of the pigs treated for *Streptococcus suis* infection (Strep.) where barrows of the 2000-2005 genetics had a greater treatment rate than gilts but gilts of the 2011-2017 genetics had a greater treatment rate than barrows. There was a tendency ($P < 0.075$) for the same type of interaction when pigs were treated for respiratory issues between d 0-14.

There was no difference in total therapies administered for d 14-28 ($P > 0.488$) or d 28-42 ($P > 0.269$) with ranges of 10 to 19.4% and 11.7 to 28.6% of pigs treated, respectively. For the d 28-42 period, 2011-2017 genetics had a greater treatment rate for respiratory issues than 2000-2005 genetics ($P < 0.013$). Thereafter, there were no differences between genetic groups or sex for total therapies or any treatment category ($P > 0.05$). The ranges of total treatments given generally decreased as the pigs grew, however another spike of treatments occurred during d 120-150 where 6.7 to 11.9% of pigs were treated once ileitis was diagnosed in some pigs at the end of the study.

Initially, 44 pens of pigs were put on test comprising 155 pigs. The number of pigs remaining at the end of the study was 123 (20.65% mortality rate). The percent of pigs lost in each group ranged from 13.7 to 29.2% but there was no difference between genetic group or sex ($P > 0.298$) in mortality.

4.5.6 Predicted Growth Curves

Each prediction function is divided into two figures by sex. Within each figure, the solid line represents pigs from the 2000-2005 genetic group and the dashed line represents pigs from the 2011-2017 genetic group.

4.5.6.1 BW, ADG, ADFI, and G:F

Predicted BW based on days of age for barrows and gilts increased as the days of age increased from 42 to 189 days of age (Figure 4.1 and 4.2). The 2000-2005 barrows started 1.15 kg heavier at 42 days of age. While this observation is the inverse of actual measured BW on d 14 in the nursery (Table 4.4), this is a function of the model predicting different mature body weights for each genetic group. Although genetic selection has created larger framed pigs that grow better at heavier weights, the 2011-2017 barrows were still predicted to be 2.97 kg lighter at the end of the grow-finish period. The predicted genetic difference in ending live BW for barrows in this subset of pigs, 2.97 kg, was two times greater than the observed difference of 1.37 kg when all of the pigs were included with the 2000-2005 genetics being heavier in both cases (Table 4.4; BW end). At the point of greatest separation between predicted BW curves, 142 days of age, the 2000-2005 barrows were predicted to weigh 92.8 kg whereas the 2011-2017 barrows were predicted to weigh 86.6 kg (trend, $P < 0.10$).

The 2000-2005 gilts were predicted to start 1.16 kg lighter and grow slower than the 2011-2017 gilts ending with a 6.28 kg difference in BW (ending 129.4 kg vs. 135.6 kg, respectively). The predicted genetic difference for gilts in this subset of pigs, 6.28 kg, was much greater than the observed difference of 2.53 kg when all of the pigs were included (Table 4.4; BW end). The ending difference in BW at 189 days of age, 6.28 kg, although not statistically different ($P > 0.10$) was also the point of the most separation.

The predicted average daily gain (ADG) for barrows was greater for the 2000-2005 genetic group compared to the 2011-2017 genetic group until about 92 kg BW where the 2011-2017 genetic group started and continued to grow faster compared to the 2000-2005 genetic group (Figure 4.3). This switch is evident because ADG for the 2011-2017 barrows peaked 12.1 kg later than the 2000-2005 barrows. The maximum ADG of 1.084 kg/d for the 2000-2005 barrows was achieved at 77.7 kg BW, whereas the maximum ADG of 1.065 kg/d for the 2011-2017 barrows was achieved at 89.8 kg BW. The curves separated the most ($P < 0.05$) at about 135 kg BW (the end of the study) where 2011-2017 barrows were predicted to gain 0.96 kg/d and the 2000-2005 barrows gained 0.85 kg/d.

For gilts, the same relationship occurred where the predicted ADG is greater for the 2000-2005 genetic group compared to the 2011-2017 genetic group until about 81 kg BW where the 2011-2017 genetic group started and continued to grow faster compared to the 2000-2005 genetic group (Figure 4.4). The peak predicted ADG for 2011-2017 gilts occured 15.3 kg later. The maximum ADG of 1.001 kg/d for the 2000-2005 gilts was achieved at 76.3 kg of BW, whereas the maximum ADG of 1.006 kg/d for the 2011-2017 gilts was achieved at 91.6 kg of BW. The curves separated the most ($P < 0.05$) at about 129 kg BW (the end of the study) where 2011-2017 gilts were predicted to gain 0.95 kg/d and the 2000-2005 gilts gained 0.82 kg/d.

Average daily feed intake (ADFI) increased as BW increased. Predicted ADFI was greater for 2000-2005 barrows for the grow-finish period starting from 20 kg BW until 105 kg BW after which ADFI was greater for 2011-2017 barrows until the end of the experiment (Figure 4.5). The greatest degree of separation in ADFI occured at 55 kg BW, where the 2000-2005 barrows were predicted to eat 0.109 kg/d more than 2011-2017 barrows (2.397 kg/d vs. 2.288 kg/d, respectively; $P < 0.05$). In agreement, when all pigs were included in the growth performance analysis, barrows

weighed an average of 54.8 kg, the 2000-2005 barrows consumed 0.14 kg/d more between post-weaning days 63 and 87 (2.04 kg/d vs. 1.90 kg/d; Table 4.4; d 87 weight).

At a starting difference of 0.054 kg of ADFI at 20 kg BW (0.94 kg/d vs. 0.89 kg/d), predicted ADFI was greater for 2000-2005 gilts until the 2011-2017 gilts started and continued to eat more at 40 kg BW (Figure 4.6). In contrast, the growth performance analysis which contained all pigs in the study (Table 4.4) indicated the 2000-2005 gilts weighed 18.09 kg on d 42 and consumed 0.74 kg/d which is less than the 20.65 kg 2011-2017 gilts which consumed 0.79 kg/d. Not only are the ADFI comparisons reversed between genetic groups of gilts but also the ADFI curves predicted 0.1-0.2 kg/d less than the growth performance analysis during this initial grow-finish period. Although not statistically different ($P > 0.10$), the greatest degree of separation in predicted ADFI occured at 85 kg of BW, where the 2000-2005 gilts were predicted to eat 0.048 kg/d less than 2011-2017 gilts (2.689 kg/d vs. 2.737 kg/d, respectively). This prediction is not in agreement with the growth performance analysis difference of 0.05 kg/d greater intake for 2000-2005 gilts when all gilts weighed an average of 88.8 kg BW (Table 4.4; d 108-120 post-weaning). At the end of the prediction curve at about 140 kg BW, the 2011-2017 gilts were only eating 0.01 kg/d more than the 2000-2005 gilts.

The ratio of kg BW gain to kg of feed consumed (G:F) decreased as BW increased (Figures 4.7 and 4.8). In other words, the pig was less feed efficient, gaining less BW per unit of feed consumed, as the pig grew and time progressed. Combining the prediction of ADG and ADFI, at about 20 kg BW the barrows had a very similar G:F, 0.694, between genetic groups. The genetic groups began to separate in G:F at about 85 kg BW and at 125 kg of BW, the 2000-2005 barrows were predicted to have a 0.018 poorer G:F ratio than 2011-2017 barrows (0.280 vs. 0.298, respectively; $P < 0.05$). This is comparatively similar to the growth performance analysis where

the 2000-2005 barrows had a G:F of 0.30 which was slightly less than the 2011-2017 barrows (0.32) and barrows of both groups weighed an average of 122.2 kg (Table 4.4; d 120-150 post-weaning).

For gilts, there were two BW where the genetic groups had a common G:F, 32 and 89 kg BW. The 2011-2017 gilts were more feed efficient until about 32 kg BW and after 89 kg BW with the largest difference of 0.041 at 129 kg BW (0.264 for 2000-2005 vs. 0.305 for 2011-2017; $P <$ 0.05). The observed difference in G:F of 0.01 at an average of 116.4 kg BW when all of the pigs were included in the growth performance analysis was less than the predicted difference of 0.041 at 129 kg BW (Table 4.4; d 120-150 post-weaning). The d 120-150 post-weaning time period for the growth performance analysis comparison was optimal because the d 150-168 time period was comprised of the slower growing pigs and a smaller number of pigs due to the first cut of pigs being marketed on d 150. However, the 2000-2005 gilts were predicted to be more feed efficient between 32 and 89 kg BW with the largest difference of 0.018 at about 50 kg BW (0.447 vs. 0.465). In contrast, the growth performance analysis had gilts weighing an average of 54.6 kg BW across genetic groups on d 87 post-weaning and they had a 0.02 difference in G:F with 2011-2017 gilts being more feed efficient (Table 4.4; d 63-87 post-weaning).

4.5.6.2 Backfat depth, LMA, and loin muscle depth

Last rib backfat depth (Figures 4.9 and 4.10) and tenth rib backfat depth (Figures 4.11 and 4.12) increased as BW increased. Last rib and tenth rib backfat thickness was greater for the 2000-2005 genetic groups for both sexes with the greatest difference in genetic groups at the end of the grow-finish period. The predicted last rib backfat thickness started to separate at about 55 kg BW for both sexes. At 130 kg BW, barrows had a predicted last rib backfat of 1.71 cm and tenth rib backfat of 1.80 cm for the 2000-2005 genetic group and 1.56 cm and 1.65 cm for the 2011-2017

genetic group, last rib and tenth rib, respectively. Both last rib and tenth rib backfat was greater for 2000-2005 barrows compared to 2011-2017 barrows at 130 kg BW by 0.15 cm ($P < 0.05$). This prediction difference of 0.15 cm was slightly lower than the observed differences of 0.23 for last rib and 0.25 cm for tenth rib backfat at about 129 kg, but in agreement that 2000-2005 barrows had more backfat (Table 4.7; Day 150 post-weaning). The predicted last rib and tenth rib backfat was 0.14 cm (1.36 cm vs. 1.22 cm) and 0.15 cm (1.45 cm vs. 1.30) greater, respectively, for 2000-2005 gilts compared to 2011-2017 gilts at 130 kg BW ($P < 0.05$). This prediction difference of 0.14 cm for last rib and 0.15 cm for tenth rib backfat was slightly greater than the observed differences of 0.09 and 0.10 cm for last rib and tenth rib backfat at about 127 kg, but in agreement that 2000-2005 gilts had more backfat (Table 4.7; Day 150 post-weaning). Barrows of both genetic groups had more predicted backfat than gilts. The differences due to sex where barrows had statistically thicker last rib and tenth rib backfat started at d 88 post-weaning and continued for the remainder of the study compared to gilts (Table 4.7).

Predicted loin muscle area (LMA; Figures 4.13 and 4.14) and loin muscle depth (Figures 4.15 and 4.16) measured at the tenth rib increased as BW increased for both sexes. The predicted LMA was very similar between genetic groups and sexes. The 2000-2005 barrows and gilts had a predicted LMA of 11.69 cm^2 and 11.45 cm^2 at 20 kg BW and 50.18 cm^2 and 50.30 cm^2 at 130 kg BW. The 2011-2017 barrows and gilts had a predicted LMA of 11.89 cm^2 and 11.43 cm^2 at 20 kg BW and 50.55 cm^2 and 50.67 cm^2 at 130 kg BW. There was no difference in LMA between genetic groups at any BW for barrow or gilts ($P > 0.10$). The observed means for each day of the live ultrasound scans by genetic group and sex agree that there was no difference in LMA between genetics in this study (Table 4.7).

The largest difference of loin depth for barrows occured at 20 kg BW with 2000-2005 barrows having a loin depth of 1.92 cm and 2011-2017 barrows having a loin depth of 1.86 cm, which is a 0.06 cm difference ($P < 0.05$). After 40 kg BW, this difference in predicted loin depth was less than 0.05 cm for barrows for the rest of the grow-finish period. The predicted loin depth for gilts was 0.20 cm greater for 2011-2017 gilts compared to 2000-2005 gilts at 130 kg BW (5.15 cm vs. 4.95 cm; $P < 0.05$). When compared to the observed live ultrasound scans, at about 121 kg BW, 2011-2017 gilts had similar 0.22 cm deeper loin muscles than 2000-2005 gilts (4.96 cm vs. 4.74 cm) and there was a statistical difference in loin depth between genetics only on d 150 post-weaning, about 125 kg BW, where the 2011-2017 genetics had deeper loin muscles (Table 4.7; d 150 post-weaning). There were also differences due to sex where barrows had statistically deeper loin muscles at d 120 post-weaning compared to gilts (Table 4.7). However, there was no difference in the model predicted loin depth at 125 kg BW: 4.95 and 4.93 cm for gilts and barrows, respectively.

4.5.6.3 EBP and EBL accretion, FFLG, and TCFP

Predicted empty body protein (EBP) accretion rate increased for 2000-2005 barrows until 63.7 kg BW with the peak EBP accretion rate of 187 g/d and then EBP accretion decreased until the end of the grow-finish period where the EBP accretion rate was 123 g/d at 120 kg BW (Figure 4.17). For 2011-2017 barrows, the peak EBP accretion rate was achieved at 179 g/d at 65.5 kg BW and ended at 124 g/d at 120 kg BW. The 2000-2005 barrows reached a predicted 8 g/d greater peak EBP accretion 1.8 kg BW sooner than 2011-2017 barrows. Although not statistically different ($P > 0.10$), the greatest difference between 2000-2005 and 2011-2017 barrow predicted EBP accretion rates occured at 54 kg BW (181 vs. 173 g/d, respectively).

The EBP accretion rate increased for 2000-2005 gilts until 59.4 kg BW with the peak EBP accretion rate of 177 g/d and then EBP accretion decreased to 117 g/d at 111 kg BW (Figure 4.18). For 2011-2017 gilts, the peak EBP accretion rate was achieved at 172 g/d at 61.9 kg BW and decreased to 128 g/d at 109 kg BW. The 2000-2005 gilts reached a predicted 5 g/d greater peak EBP accretion 2.5 kg BW sooner than 2011-2017 gilts. At 111 kg BW, before EBP accretion began to increase again, the 2011-2017 gilts had a greater ($P < 0.05$) predicted EBP accretion than 2000-2005 gilts (128 vs. 117 g/d, respectively). Although these pigs were not fed ractopamine in the late finishing diets, the predicted EBP accretion rates increased at the very end of the grow-finish period. This increase was due to a relatively large standard error at the end and is a function of a polynomial in the prediction, which makes this uptick in EBP at the end likely an anomaly.

Predicted empty body lipid (EBL) accretion rate increased as BW increased (Figures 4.19 and 4.20). The barrows maintain a similar EBL accretion between genetic groups until they reach a BW of about 60 kg when the 2000-2005 barrows maintained a steeper increased slope compared to the 2011-2017 barrows that leveled off their EBL accretion rate. At about 100 kg BW, the 2011-2017 barrows started to increase their EBL until the end of the grow-finish period, maximized at 450 g/d at 123 kg BW. The 2000-2005 barrows had a maximum predicted EBL of 452 g/d at 127 kg BW. The largest difference in genetic groups of the barrows occured at about 96 kg BW where the 2000-2005 barrows are predicted to gain 337 g EBL/d and the 2011-2017 barrows are predicted to gain 294 g EBL/d ($P < 0.05$). The gilts of both genetic groups had a similar EBL until about 70 kg BW where 2000-2005 gilts began to have a greater daily EBL accretion. The largest difference between the gilts genetic groups was predicted to be at about 96 kg BW where 2000-2005 gilts have an EBL accretion rate of 273 g/d and 2011-2017 gilts 252 g/d ($P < 0.05$).

Fat-free lean gain (FFLG) predicted for gilts and barrows increased as the pigs grew until about 70 kg BW where predicted FFLG started to decrease (Figures 4.21 and 4.22). For 2000-2005 barrows, the peak FFLG was achieved at 475 g/d at 66.9 kg BW and ended at 297 g/d at 120 kg BW. The peak FFLG for 2011-2017 barrows was achieved at 459 g/d at 70.7 kg BW and ended at 330 g/d at 120 kg BW. The 2000-2005 barrows reached a predicted 16 g/d greater peak FFLG at 3.8 kg BW sooner than 2011-2017 barrows. The greatest difference between 2000-2005 and 2011-2017 barrow predicted FFLG occured at 120 kg BW (297 vs. 330 g/d, respectively; $P < 0.05$).

For 2000-2005 gilts, the peak FFLG was achieved at 436 g/d at 64.3 kg BW and ended at 326 g/d at 120 kg BW. The peak FFLG for 2011-2017 gilts was achieved at 437 g/d at 70.6 kg BW and ended at 345 g/d at 120 kg BW. The 2000-2005 gilts reached a predicted 1 g/d less peak FFLG at 6.3 kg BW sooner than 2011-2017 gilts. The biggest difference between the gilts occured at about 100 kg BW where the 2011-2017 gilts averaged 29 g/d greater FFLG than the 2000-2005 gilts (384 vs. 355, respectively; $P < 0.10$).

Predicted total carcass fat percentage (TCFP) increased as BW increased (Figures 4.23 and 4.24). At 40 kg BW, 2000-2005 barrows had a predicted TCFP of 11.6%, whereas 2011-2017 barrows had a predicted TCFP of 11.4%. Although not statistically different ($P > 0.10$), the largest difference of 0.7% in TCFP was at 120 kg BW (26.5% vs. 25.8%) where the 2000-2005 barrows had a slightly fatter carcass. For gilts at 40 kg BW, the 2000-2005 genetic group had a predicted TCFP of 11.3% and the 2011-2017 genetic group had a predicted TCFP of 11.0%. The largest difference, 0.9%, in TCFP was at 120 kg BW where the predicted TCFP was 25.4% and 24.5% for the 2000-2005 and 2011-2017 gilts, respectively. However, this difference in TCFP was not statistically different ($P > 0.10$).

4.5.6.4 NE intake, SID lysine requirement, SID lysine:NE, and SID lysine% of diet

Predicted net energy (NE) intake in kcal/d increased as BW increased (Figures 4.25 and 4.26). The barrows maintained a similar daily NE intake until they reached about 60 kg BW when the 2000-2005 barrows maintained a steeper increasing slope compared to the 2011-2017 barrows that leveled off their NE intake. At about 100 kg BW, the 2011-2017 barrows started to increase their NE intake at a greater rate than 2000-2005 barrows until the end of the grow-finish period maximized at 8210 kcal NE/d at 123 kg BW for 2011-2017 barrows. The 2000-2005 barrows had a maximum predicted NE intake of 8291 kcal NE/d at 127 kg BW. Although not statistically different ($P > 0.10$), the largest difference in NE intake between genetic groups of the barrows occured at about 95 kg BW where the 2000-2005 barrows were predicted to eat 6763 kcal NE/d which is 407 kcal NE/d more than 2011-2017 barrows, 6356 kcal NE/d. The predicted NE intake for gilts was very similar between genetic groups. The greatest difference, although not significant ($P > 0.10$), between gilt genetic groups in predicted NE intake occured at about 94 kg BW where 2000-2005 gilts are predicted to consume 158 kcal more NE/d (6037 vs. 5880 kcal/d) than 2011-2017 gilts.

The standardized ileal digestible (SID) lysine requirement prediction in grams per day increased and then decreased as pigs grew with peak SID lysine requirements at different BW for barrows and gilts (Figures 4.27 and 4.28). For 2000-2005 barrows, the peak SID lysine requirement was 20.5 g/d at 66.9 kg BW and ended at 15.5 g/d at 120 kg BW. The peak SID lysine requirement for 2011-2017 barrows was 20.0 g/d at 69.7 kg BW and ended at 15.8 g/d at 120 kg BW. The 2000-2005 barrows reached a 0.5 g/d greater predicted peak SID lysine requirement at 2.8 kg BW sooner than 2011-2017 barrows. The greatest difference (not significant, $P > 0.10$) between 2000-2005 and 2011-2017 barrow predicted SID lysine requirement occured at 55 kg BW (19.6 vs. 18.9 g/d, respectively).

For 2000-2005 gilts, the peak SID lysine requirement was 19.6 g/d at 62.3 kg BW and ended at 15.5 g/d at 120 kg BW. The SID lysine requirement for 2011-2017 gilts was 19.1 g/d at 65.7 kg BW and ended at 17.0 g/d at 120 kg BW. The 2000-2005 gilts reached a 0.5 g/d greater predicted peak SID lysine requirement at 3.4 kg BW sooner than 2011-2017 gilts. The greatest difference between gilt genetic groups in predicted SID lysine requirement was 1.1 g SID lysine/d at 110 kg BW ($P < 0.10$); 14.8 g/d for 2000-2005 vs. 15.9 g/d for 2011-2017.

The predicted ratio of SID lysine requirement to NE intake decreased as pigs grew (Figures 4.29 and 4.30). The 2000-2005 barrows and gilts had a greater ratio to start and a steeper negative slope than the 2011-2017 barrows and gilts. At 35 kg BW, 2000-2005 barrows had a predicted SID lysine:NE of 4.10 g/Mcal, whereas 2011-2017 barrows had a 0.17 g/Mcal lower predicted SID lysine:NE of 3.93 g/Mcal. The SID lysine:NE at 95 kg BW was predicted to be 2.59 g/Mcal for 2000-2005 barrows and 2.77 g/Mcal for 2011-2017 barrows (0.18 g/Mcal difference; $P < 0.05$). The prediction curves of the two genetic groups crossed at 3.65 g/Mcal at 64.6 kg BW and again at 1.96 g/Mcal at 123.0 kg BW. At 35 kg BW, 2000-2005 gilts had a predicted SID lysine:NE of 4.20 g/Mcal, whereas 2011-2017 gilts had a 0.18 g/Mcal lower predicted SID lysine:NE of 4.02 g/Mcal. The prediction curves of the two genetic groups crossed at 3.50 g/Mcal at 71.4 kg BW. The biggest difference ($P < 0.05$), 0.22 g/Mcal, for the gilts in SID lysine:NE occured at 98 kg BW and was predicted to be 2.50 g/Mcal for 2000-2005 gilts and 2.72 g/Mcal for 2011-2017 gilts.

The SID lysine requirement as a percentage of the diet was calculated as g/d of SID lysine requirement divided by ADFI in g/d multiplied by 100. The predicted SID lysine percentage decreased as BW of the pigs increased (Figures 4.31 and 4.32). For barrows, the two genetic groups had similar predicted SID lysine percentages. At 30 kg BW, the genetic groups had a common predicted SID lysine percentage of 0.84% and at 120 kg BW, the 2011-2017 barrows required

slightly more SID lysine (0.48% vs. 0.47%; not significant, $P > 0.10$). The greatest difference between genetic groups of the gilts in predicted SID lysine requirement occured at 48.9 kg BW and was predicted to be 0.93% for 2000-2005 gilts and 0.88% for 2011-2017 gilts ($P < 0.05$). Thereafter, the 2000-2005 gilts had a steeper negative slope than the 2011-2017 gilts. The prediction curves of the two genetic groups crossed at 0.67% at 82.4 kg BW and ended with a difference of 0.04% at 120 kg BW with 2011-2017 gilts requiring more SID lysine (0.55% vs. 0.51%).

4.6   Discussion

The objective of this study was to determine if genetics from two decades of sires with different terminal sire indexes (TSI) and sex of the progeny impact progeny growth performance and carcass characteristics. Using frozen semen to create the two decades of Duroc sires (2000-2005 and 2011-2017), maternal line gilts of common genetics were inseminated. Low density housing and excess nutrient availability (110% of the current swine 2012 NRC recommendations) was provided to minimize differences due to environmental limitations.

Initially, investigation of the genotype × feeding regimen interaction was a goal of this experiment to encompass the different nutrient requirements of swine from the national research council (NRC) published in 1998 and 2012 similarly to Fix et al. (2010). However, a large limitation of using frozen semen is reduced conception rates and therefore smaller litter sizes. Gilts, used in this experiment, also typically have smaller litter sizes compared to older parity sows (van Wettere et al., 2006) due to lower ovulation rates and lower embryo survival. Therefore, in this study, gilts inseminated with frozen semen had too few pigs in total (155 pigs in 44 pens) to consider adding a dietary treatment factor to the experimental design.

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In addition to reduced conception rates, using frozen semen to look at genetic trends of two time periods has other limitations previously discussed by Tribout et al. (2010). The current experiment looked at two fixed periods of 2000-2005 and 2011-2017 and therefore, the shape of the genetic trends between the two fixed time periods is unknown. Another limitation is that all animals were raised under the same environmental and management conditions. The more modern genotypes may be favored compared to older genotypes who could have performed differently under their typical environment and health management conditions. This would be known as a genotype × environment (G×E) interaction which may exist for pig growth and carcass characteristics (Schinckel et al., 1999; Wallenbeck et al, 2009).

At weaning, there was a tendency for progeny born to 2011-2017 sires to be slightly heavier than progeny from 2000-2005 sires. This difference in weaning weight was likely related to the smaller litter size of the 2011-2017 genetics (6.4 vs. 9.4 born alive). Typically, pigs that are heavier at weaning grow quicker and take fewer days to get to the same market weight (Schinckel et al., 2009d). This was not observed in this experiment because there was no difference in d 150 post-weaning or ending BW between genetic groups.

Overall, although the terminal sire indexes (TSIs) of the two genetic groups were different (88.2 vs. 112.0; Table 4.4), we did not observe large performance differences between the genetic groups that we were expecting. In the nursery period, d 0 to 42, there was a tendency for a genetics × sex interaction for feed efficiency where the magnitude of difference between barrows and gilts increased from 2000-2005 to 2011-2017 and gilts maintained a greater G:F advantage over barrows in the more modern genetic group. There were no differences between genetic groups or sex in the overall nursery period (d 0 to 42) for ADG or ADFI with the average of the four groups being 0.33 kg/d ADG and 0.47 kg/d ADFI.

According to Knauer and Hostetler (2013) the US nursery production means of 0.367 kg ADG and 0.637 G:F in 2005 improved to 0.382 kg ADG and 0.655 G:F in 2010. However, when Stalder (2018) used a different subset of farms/companies, the nursery ADG was the same in 2012 as it was in 2017 at 0.372 kg/d. This experiment is similar to Stalder (2018) where the ADG in the nursery period (d 0 to 42) averaged 0.33 kg/d for both 2000-2005 and 2011-2017 genetic groups.

In the grow-finish perod (d 42 to the end of the study) and from weaning to market (d 0 to the end) barrows consumed more feed than gilts. Chen et al. (1999) and Yang et al. (2020) also observed that barrows had a higher feed intake than gilts. However, gilts were more feed efficient than barrows from weaning to market in this study, which is in agreement with results reported by Yang et al. (2020). Ultimately, there was no difference in ending BW between genetic groups or sexes, which is in agreement with results between sexes reported by Chen et al. (1999) when they harvested pigs around 110 kg empty BW. However, in contrast, Yang et al. (2020) harvested pigs when the average BW of the pen reached 120 kg and observed barrows had a heavier ending BW than gilts.

In literature using frozen semen to compare different genetic groups over time, Schwab et al. (2007) did not observe overall ADG differences between mid 1980's Duroc sires and 2000's Duroc sired pigs (0.84 vs. 0.85 kg/d, respectively) from 34 kg BW until marketed at an average BW of 109 kg. This observation is consistant with the current study where there was no overall difference in growth rate between pigs sired by Duroc boars born in 2000-2005 and 2011-2017 (1.01 vs. 1.05 kg/d, respectively) from approximately 19.2 kg BW pigs on d 42 of age until marketed between 114 and 125 kg BW. Although it might be expected that the heavier pigs at the start of the experiment by Schwab et al. (2007) would have a greater overall ADG compared to

the current study, the current experiment harvested pigs at heavier weights which may have impacted the observed overall ADG.

Modern swine genetics have been selected to be leaner and results from this study agree, although the differences in live scan and carcass measurements were not as large as expected between genetics. When pigs were live ultrasound scanned on d 150 post-weaning, they weighed from 120-130 kg BW. At this time, the modern genetics (2011-2017) had statistically less backfat and a greater loin muscle depth than the older genetics (2000-2005) and barrows were heavier, had more backfat, and had deeper loin muscles than gilts by live ultrasound scan. Similarly, when Schwab et al. (2007) ultrasonically measured pigs off test at 109 kg BW, the 2000's sired pigs had greater loin muscle area and less backfat measured at the tenth rib compared to pigs sired by 1980's sires.

Live serial ultrasound scans were conducted at seven different time points throughout the grow-finish period on a subset of the pigs and were used with BW to model the predicted growth of pigs in each genetic group and sex as time progressed and live weight increased. Although we did not see large performance differences, the model predicted peak ADG occurred later for barrows and gilts of the more modern genetics (2011-2017). The barrows of the older and slightly fatter genetics (2000-2005) ate more at the beginning resulting in a faster growth rate and peak ADG at a lighter BW. Consequently, the leaner modern genetics with the higher ADG and protein deposition at heavier BW didn't eat enough at the beginning to grow as fast as the older genetics did. Feed efficiency was very similar between genetic groups of barrows and slightly different between genetic groups of gilts. One limitation in this study from a live serial ultrasound modeling standpoint is that the sample size for feed intake models was limited due to the pen being the experimental unit for feed intake.

The diet in this study was formulated to be unlimiting at 110% of the 2012 NRC requirements and for the highest average protein accretion rate for gilts at 155 g/d. The average empty body protein accretion rate achieved in this study was 147 g/d. In retrospect, the amino acid formulation used in this study was only 5.4% above the observed average protein accretion. Comparable to the current study, Schiavon et al. (2019) fed two different modern terminal genetic lines two different amino acid levels from 60 to 145 kg BW. Authors observed an average protein accretion rate of 182 g/d during the growing period (60-104 kg BW), 102 g/d during the finishing period (104-145 kg BW), and 139 g/d overall (60-145 kg BW), uninfluenced by amino acid level or genetic line (Schiavon et al., 2019). The reason for their overall protein accretion rate, 139 g/d, being 5.4% (8 g/d) less than the average in the current experiment could have been due to pigs reaching heavier weights before harvest (145 kg vs. 128 kg in the current experiment). Heavier pigs have a reduced protein accretion rate and prolonging this period likely decreased the average protein accretion rate in the study by Schiavon et al. (2019). Additionally, in the current study, the average peak empty body protein accretion rate of 179 g/d was achieved at an average of 62.6 kg BW. This peak is similar to the average protein accretion rate of 182 g/d at an average of 81.6 kg BW during the growing period observed by Schiavon et al. (2019).

Schwab et al. (2007) serially ultrasounded tenth rib backfat of pigs sired by 2000's Duroc boars vs. 1980's Duroc boars. The cumulative backfat accretion predicted at 118 kg BW was approximately 2.7 cm and 2.1 cm backfat accretion for 1980's and 2000's sired pigs, respectively. While we also serially ultrasounded tenth rib backfat in the current study, the predicted tenth rib backfat depth at 115 kg BW was approximately 1.5 cm and 1.3 cm for 2000-2005 and 2011-2017 sired pigs, respectively. Although the general trend of gilts having less backfat than barrows holds

true between these two studies, Schwab et al. (2007) had much fatter pigs than the current experiment.

Of the 40 pigs harvested in Purdue's Meats Lab, there was only a tendency for the older genetics to have more backfat and modern genetics to have a greater calculated percent fat-free lean. However, there was a statistical difference between sexes where barrows had more backfat than gilts and a lower calculated fat-free lean percentage, which is in agreement with results reported by Yang et al. (2020). The ribbed carcass measurement for 10th rib backfat was about 0.5 cm greater than the same measurement collected using live ultrasound. However, barrows consistently had thicker backfat at the tenth rib regardless of method. The last live ultrasound scan of the same subset of pigs was in agreement with carcass measurements of the same pigs as the 2000-2005 genetics had greater last rib and tenth rib backfat than the 2011-2017 genetics. The tenth rib loin depth measured by ultrasound also tended to be greater for the 2011-2017 genetics.

The carcasses of 73 pigs harvested at Tyson Fresh Meats were measured for HCW, backfat, loin muscle depth, and the standardized fat-free lean percentage was calculated. Mirroring the 40 pigs harvested at Purdue Univeristy, barrows had greater backfat depths and gilts had greater fat-free lean percentages. In contrast, the barrows in this group of pigs were heavier on a final live weight and HCW basis than gilts, where there was no difference in weight of the pigs harvested at Purdue University. Also, there was a tendency for 2000-2005 genetics to have deeper loin muscles measured on the carcass at Tyson's than 2011-2017 genetics which is opposite of the ultrasound loin muscle depths measured in the Purdue University harvested pigs. The last live ultrasound scan of the Tyson subset of pigs was in agreement as the barrows had more backfat and tended to have larger LMA than gilts. In contrast, Yang et al. (2020) found that gilts had a greater LMA than barrows although barrows had a heavier ending BW than gilts.

The subset of 40 pigs harvested at the Purdue Meats Lab were also evaluated for pork quality. Longissiumus dorsi color was scored visually using the National Pork Producers Council (NPPC) color scores and objectively using a Hunter MiniScan EZ colorimeter. The NPPC color scores range from 1 to 6, with 1 being the lightest color and 6 being the darkest (NPPC, 2000). The Hunter MiniScan EZ colorimeter measures CIE lightness (L*), redness (a*) and yellowness (b*) values (CIE, 2004). The a* value measures greenness (lower value) to redness (higher value) and the b* value measures blueness (lower value) to yellowness (higher value).

There were no differences between genetics and sex in this study for NPPC color scores nor CIE L*, a* and b* values. The loin muscles evaluated in this experiment had NPPC color scores of 2.75 to 3.05. Norman et al. (2003) grouped loins based on NPPC color scores into 3 categories: category A (NPPC color score 1 and 2), B (score 3 and 4), and C (score 5 and 6). Authors measured CIE L*, a* and b* and found category B to have an average of 50.24 for L*, 7.68 for a*, and 14.57 for b*. These values are fairly similar to results in the present study (L* of 51 to 54 and a* of 8.1 to 8.6) with the exeption of b* values (5.7 to 6.6) indicating loin muscle samples in the current experiment were bluer than what Norman et al. (2003) observed.

Loin muscle firmness and marbling were subjectively measured in this experiment using the NPPC scoring systems. The loin firmness scores range from 1 to 5, with 1 being very soft and 5 being very firm (NPPC, 1991). The NPPC marbling scores visually estimate the amount of intramuscular fat (marbling) in the meat and range from 1 to 10, with 1 being devoid of marbling and 10 having abundant marbling (NPPC, 2000).

Lowell et al. (2017) evaluated differences in NPPC color, marbling, and firmness scores of the longissiumus dorsi muscle approximately 1 d postmortum between barrows and gilts. Authors found no difference in color score between sexes with an average of 3.64. In agreement, the current

study found no difference between sexes but had lower color scores closer to 3.0. Lowell et al. (2017) found that there was a significant difference between sexes for firmness where barrows had a firmness score of 3.40 vs. a score of 3.19 for gilts. In contrast, the average loin firmness scores in this experiment ranged between 2.40 and 2.85 with no differences among genetics or sexes. Lowell et al. (2017) found that barrows and gilts tended to have different marbling scores where barrows scored 2.46 and gilts scored 2.32. The average marbling scores in this experiment ranged between 2.05 and 2.75 and the 2000-2005 genetics had higher marbling scores than the 2011-2017 genetics. Interestingly, there was a genetics × sex interaction where barrows of the 2000-2005 genetic group had a greater average marbling score than the gilts, whereas in the 2011-2017 genetic group, the gilts had a greater marbling score than the barrows.

The pH of the loin muscles was measured within 24 hours of harvest and is therefore called the ultimate pH. The preferred range of ultimate pH is 5.45 to 6.04 (Boler et al., 2010), in this experiment, the ultimate pH average ranged from 5.5 to 5.6 with no differences between genetics or sexes. The pH of the meat also affects the fresh meat's ability to hold water, called water holding capacity (WHC; Huff-Lonergan et al., 2002). The ability for meat to hold water is desirable for fresh consumption and further processing because it allows seasonings and marinades to be maintained within the meat product. A drip loss greater than 5% may indicate an issue with pork quality (Torres Filho et al., 2017). In this experiment, average loin drip loss ranged from 6 to 9% and the the 2011-2017 sired progeny tended to have greater drip loss compared to the 2000-2005 progeny. Bee et al. (2007) also measured drip loss after 1 day of storage in 2 different genetic lines. Bee et al. (2007) observed no difference in drip loss between genetic groups and average drip loss ranged from 2 to 3% which is less than the 6 to 9% drip loss observed in this experiment.

Additionally, loin muscles with a quicker pH decline 3 hours post-mortum had significantly more drip loss than loin muscles with a slower pH decline 3 hours post-mortum (Bee et al., 2007).

Overall, meat quality evaluated at Purdue's Meats Lab was good for both genetic groups and 2000-2005 genetics had a greater NPPC marbling score (NPPC, 2000) compared to 2011-2017 genetics. Schwab et al. (2007) modeled daily and cumulative intramuscular fat accretion by using serial ultrasound measurements and determined 1980's sired pigs had greater predicted cumulative intramuscular fat at about 4.75% at 118 kg BW than 2000's sired pigs at about 4.25%.

One potential reason for not observing large differences in growth and carcass characteristics between genetic groups could be due to how the pigs were fed. Potentially, the pigs that were used to develop the assigned TSI's could have been restricted in their nutrition or growing environment as sires, preventing their optimal performance and altering their true TSI. In this study when pigs were in the same low-density environment and fed in excess of their estimated requirements, they grew similarly. It is also important to note that there was a greater death rate in this experiment, 20.65%, compared to the industry wean-to-finish average of 7.99% in 2017 (Stalder, 2018) but the pigs that survived in this experiment grew well. There was also no difference in mortality rate between genetic group or sex. Generally, we believe the pigs had poorer health early due to being born to gilts with poorer colostrum quality providing less passive immunity to their litters (Quesnel, 2011). Mortality rate of 2000-2005 genetic group was consistant in the nursery and grow-finish phases, however the 2011-2017 genetic group had a greater percent mortality in the grow-finish phase compared to the nursery period. Many of the finishing pigs also had ileitis prior to marketing when the final diet was antibiotic free, which affected growth and increased variability during the last couple weeks of the study. This is evident when one compares the numerical BW differences at d 150 to the very similar BW at d 168.

4.7 Conclusion

In conclusion, using frozen semen allowed the genetics of two different time periods of sires and sex of the progeny to be evaluated in this study. The expected large growth performance differences indicated by the terminal sire indexes of the two genetic groups, 88.2 and 112.0 for 2000 to 2005 and 2011 to 2017, respectively, were not observed. However, barrows had greater feed intake and fatter carcasses than the more feed efficient and leaner gilts in this study. Modern swine genetics have been selected to be leaner and results from this study agree, although the differences in live scan and carcass measurements were not as large as expected. One potential reason for the lack of genetic differences could be that these pigs were housed in near ideal conditions (low stocking density and no heat stress) and fed unlimiting diets which may have been different than the sires' rearing conditions that determined their terminal sire index.

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Table 4.1 Diet composition of nursery phases (as-fed basis)[1]

Item	Phase 1	Phase 2	Phase 3	Phase 4
Ingredient, %				
Corn	34.590	32.435	45.350	52.625
Soybean meal, 48% CP	15.350	16.950	22.300	29.900
DDGS, 7% fat	------	5.000	7.500	10.000
Soybean oil	5.000	5.000	3.000	------
Choice white grease	------	------	------	3.000
Plasma protein	6.500	2.500	------	------
SD blood meal	1.500	1.500	------	------
Soy concentrate	4.000	4.000	4.000	------
Fish meal	5.000	4.000	4.000	------
Dried whey	25.000	25.000	10.000	------
Limestone	0.810	0.880	1.075	1.420
Monocal. Phos, 21.5% P	0.330	0.340	0.310	0.660
Salt	0.250	0.250	0.300	0.350
Lysine-HCL	0.070	0.280	0.450	0.520
DL-Methionine	0.220	0.250	0.230	0.200
L-Threonine	0.030	0.110	0.180	0.180
L-Tryptophan	------	0.030	0.040	0.030
L-Valine	------	0.020	0.040	0.030
L-Isoleucine	------	0.020	------	------
Vitamin premix[2]	0.250	0.250	0.250	0.250
Trace mineral premix[3]	0.125	0.125	0.125	0.125
Selenium premix[4]	0.050	0.050	0.050	0.050
Phytase[5]	0.100	0.100	0.100	0.100
Carbadox – 10[6]	0.250	0.250	0.250	0.250
Zinc oxide	0.375	0.280	0.280	------
Copper sulfate	------	0.050	0.050	0.100
Hemicell enzyme[7]	------	0.040	0.040	0.040
Clarify[8]	------	0.090	0.080	0.070
Banminth 48[9]	------	------	------	0.100
Kemgest[10]	0.200	0.200	------	------
Total	100.000	100.000	100.000	100.000
Calculated analysis[11]				
ME, Kcal/kg	3518.7	3573.5	3493.1	3471.1
SID Lysine:ME, g/Mcal	4.53	4.34	4.27	3.89
Crude Protein, %	25.53	24.25	23.64	22.27
Total Lysine, %	1.79	1.73	1.66	1.53
Crude Fat, %	7.40	7.58	6.10	6.09
Crude Fiber, %	1.38	1.72	2.32	2.87
Standardized ileal digestible (SID) amino acids, %				
Lysine	1.59	1.55	1.49	1.35
Methionine:Lysine	34.06	36.71	37.99	36.64
Methionine & Cystine:Lysine	58.29	58.20	58.15	58.42
Threonine:Lysine	61.88	62.08	62.72	62.25
Tryptophan:Lysine	17.95	18.49	18.28	18.38
Isoleucine:Lysine	55.83	56.15	57.49	57.15
Valine:Lysine	71.18	67.96	65.35	65.48
Ca, %	0.90	0.88	0.85	0.77
P, %	0.75	0.70	0.63	0.57
Phytase available P, %.	0.60	0.55	0.45	0.37

(Table continues)

[1] Phase 1 diets were fed for 7 days, phase 2 diets were fed for 7 days, phase 3 diets were fed for 12 days, and phase 4 diets were fed for 16 days. All diets were fed in meal form.

[2] Provided per kg of diet: vitamin A, 6,614 IU; vitamin D_3, 661 IU; vitamin E, 44 IU; vitamin K, 2.2 mg; riboflavin, 9 mg; pantothenic acid, 22 mg; niacin, 33 mg; and B_{12}, 0.04 mg.

[3] Provided per kg of diet: iron, 121.3 mg; zinc, 121.3 mg; manganese, 15.0 mg; copper, 11.3 mg; and iodine, 0.46 mg.

[4] Provided 0.3 ppm selenium.

[5] Phyzyme® (Danisco Animal Nutrition, Morlborough, UK) providing 600 phytase units (FTU)/kg.

[6] Carbadox 10 (Phibro Animal Health, Teaneck, NJ) included at 55 ppm.

[7] Hemicell® enzyme (Elanco Animal Health, Greenfield, IN) contains a source of β-mannanase enzyme.

[8] Clarify® Larvicide (Central Life Sciences, Schaumburg, IL).

[9] Banminth® 48 (Phibro Animal Health, Teaneck, NJ) contains the active ingredient pyrantel tartrate to control internal parasites in pigs.

[10] Kemgest™ (Kemin Industries, Inc., Des Moines, IA) contains a blend of organic and inorganic acids to acidify swine feed.

[11] NRC. 2012. Nutrient Requirements of Swine. 11th ed. Natl. Acad. Press, Washington, DC.

Table 4.2 Diet composition of grow-finish phases (as-fed basis)[1]

Item	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5	Phase 6
Ingredient, %						
Corn	53.420	57.725	62.035	66.120	70.200	75.880
Soybean meal, 48% CP	24.900	20.750	16.650	12.700	8.900	8.350
DDGS, 7% fat	15.000	15.000	15.000	15.000	15.000	10.000
Choice white grease	3.000	3.000	3.000	3.000	3.000	3.000
Limestone	1.480	1.420	1.350	1.300	1.220	1.140
Monocal. Phos, 21.5% P	0.480	0.380	0.330	0.220	0.170	0.210
Salt	0.350	0.350	0.350	0.350	0.300	0.300
Lysine-HCL	0.480	0.480	0.480	0.450	0.440	0.400
DL-Methionine	0.135	0.120	0.110	0.085	0.055	0.055
L-Threonine	0.155	0.155	0.150	0.135	0.135	0.130
L-Tryptophan	0.030	0.030	0.035	0.035	0.040	0.035
Vitamin premix[2]	0.150	0.150	0.150	0.150	0.150	0.150
Trace mineral premix[3]	0.100	0.100	0.100	0.100	0.100	0.100
Selenium premix[4]	0.050	0.050	0.050	0.050	0.050	0.050
Phytase[5]	0.100	0.100	0.100	0.100	0.100	0.100
Clarify[6]	0.070	0.090	0.070	0.080	0.090	0.100
Lincomix 50[7]	0.100	0.100	0.040	------	------	------
Tylan 40[8]	------	------	------	0.125	0.050	------
Total	100.000	100.000	100.000	100.000	100.000	100.000
Calculated analysis[9]						
ME, Kcal/kg	3408.0	3416.6	3427.5	3432.9	3444.5	3450.4
SID Lysine:ME, g/Mcal	3.53	3.22	2.92	2.57	2.26	2.09
Crude Protein, %	20.91	19.27	17.67	16.06	14.56	13.41
Total Lysine, %	1.39	1.28	1.16	1.03	0.92	0.84
Crude Fat, %	6.44	6.52	6.61	6.69	6.78	6.57
Crude Fiber, %	3.03	2.95	2.88	2.81	2.74	2.50
Standardized ileal digestible (SID) amino acids, %						
Lysine	1.20	1.10	1.00	0.88	0.78	0.72
Methionine:Lysine	35.06	35.33	36.11	36.33	35.19	35.86
Methionine & Cystine:Lysine	58.35	59.13	60.54	62.14	62.25	63.50
Threonine:Lysine	63.39	64.11	64.49	65.53	67.39	67.98
Tryptophan:Lysine	18.55	18.18	18.22	18.23	18.52	18.42
Isoleucine:Lysine	58.75	57.88	56.85	57.11	56.38	56.33
Valine:Lysine	66.50	66.53	66.57	68.38	69.36	69.12
Ca, %	0.75	0.70	0.65	0.60	0.55	0.52
P, %	0.54	0.50	0.47	0.43	0.40	0.38
Phytase available P, %.	0.35	0.32	0.30	0.27	0.25	0.23

(Table continues)

[1] Phase 1 diets were fed for 21 days, phase 2 diets were fed for 24 days, phase 3 diets were fed for 21 days, phase 4 diets were fed for 20 days, phase 5 diets were fed for 22 days, and phase 6 diets were fed either 4, 5, or 20 days depending on which day pigs were marketed. All diets were fed in meal form.

[2] Provided per kg of diet: vitamin A, 3,968 IU; vitamin D_3, 397 IU; vitamin E, 26.5 IU; vitamin K, 1.3 mg; riboflavin, 5.3 mg; pantothenic acid, 13.2 mg; niacin, 19.8 mg; and B_{12}, 0.02 mg.

[3] Provided per kg of diet: iron, 97.0 mg; zinc, 97.0 mg; manganese, 12.0 mg; copper, 9.04 mg; and iodine, 0.37 mg.

[4] Provided 0.3 ppm selenium.

[5] Phyzyme® (Danisco Animal Nutrition, Morlborough, UK) providing 600 phytase units (FTU)/kg.

[6] Clarify® Larvicide (Central Life Sciences, Schaumburg, IL).

[7] Lincomix® 50 (Zoetis, Parsippany, NJ) contains the active ingredient lincomycin included at 110 ppm for phase 1 and 2 and included at 44 ppm for phase 3. Lincomycin is intended to reduce respiratory disease associated with *Mycoplasma hyopneumoniae*.

[8] Tylan™ 40 (Elanco Animal Health, Greenfield, IN) contains the active ingredient tylosin phosphate included at 110 ppm in phase 4 and 44 ppm in phase 5. Tylosin phosphate is intended to control swine dysentery, porcine proliferative enteropathies (ileitis), and reduce severity of effects of atrophic rhinitis.

[9] NRC. 2012. Nutrient Requirements of Swine. 11th ed. Natl. Acad. Press, Washington, DC.

Table 4.3 Analyzed diet compositions (as-fed basis)[1]

Item	Nursery				Grow-Finish					
	Phase 1	Phase 2	Phase 3	Phase 4	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5	Phase 6
Crude Protein, %	25.85	23.90	22.07	21.74	20.37	18.08	16.92	15.13	14.77	12.56
Moisture, %	7.93	8.13	9.30	11.13	9.67	10.83	12.00	12.38	11.63	12.11
Crude Fat, %	5.74	4.69	4.14	4.47	6.15	5.40	5.70	5.47	6.06	5.70
Crude Fiber, %	1.57	1.81	2.24	2.86	2.71	2.89	2.84	2.72	2.72	2.35
Ash, %	6.86	7.00	6.47	5.53	5.29	4.86	4.45	4.24	4.02	3.59
Gross Energy, Kcal/kg	4117.6	4140.1	4109.3	4112.5	4129.0	4109.4	4148.1	4074.9	4078.0	4070.8
Calcium, %	0.95	0.91	0.94	0.92	1.18	0.93	0.86	0.66	0.80	0.91
Phosphorus, %	0.69	0.73	0.65	0.58	0.53	0.48	0.44	0.44	0.39	0.39
Total amino acid, %										
Lysine	1.76	1.65	1.59	1.44	1.32	1.24	1.18	1.00	0.90	0.84
Methionine	0.55	0.53	0.58	0.44	0.39	0.40	0.35	0.33	0.30	0.25
Cystine	0.50	0.38	0.34	0.32	0.33	0.31	0.29	0.28	0.27	0.24
Threonine	1.19	1.10	1.04	1.00	0.82	0.81	0.72	0.68	0.58	0.53
Tryptophan	0.34	0.28	0.29	0.27	0.26	0.24	0.19	0.18	0.17	0.14
Isoleucine	1.08	1.03	0.97	0.92	0.88	0.78	0.73	0.66	0.58	0.49
Valine	1.42	1.29	1.10	1.08	0.98	0.90	0.85	0.76	0.69	0.60

[1] Samples were collected at Purdue University (West Lafayette, IN), subsampled, and shipped to University of Missouri (Columbia, MO) for analysis of crude protein, moisture, crude fat, crude fiber, ash, and total amino acids. Subsamples were also analyzed for energy, calcium, and phosphorus at Purdue University swine nutrition lab.

Table 4.4 Effect of genetics and sex on growth performance[1]

	2000-2005		2011-2017		Probability, $P <$		
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Pens, n	15	17	7	5	---	---	---
Pigs, n	51	62	24	18	---	---	---
Avg TSI	87.51 ± 3.107	88.86 ± 2.919	113.12 ± 4.913	110.79 ± 5.382	0.0001	0.908	0.665
Weaning weight, kg	5.41 ± 0.285	4.70 ± 0.268	5.87 ± 0.417	5.57 ± 0.493	0.085	0.194	0.593
Day 0-14							
ADG, kg	0.19 ± 0.020	0.18 ± 0.019	0.17 ± 0.029	0.21 ± 0.035	0.735	0.584	0.457
ADFI, kg	0.21 ± 0.019	0.21 ± 0.017	0.22 ± 0.027	0.25 ± 0.032	0.390	0.606	0.470
G:F	0.83 ± 0.042	0.85 ± 0.040	0.74 ± 0.062	0.84 ± 0.073	0.423	0.293	0.507
BW d 14, kg	7.99 ± 0.441	7.23 ± 0.414	8.32 ± 0.645	8.50 ± 0.763	0.177	0.627	0.425
Day 14-28							
ADG, kg	0.31 ± 0.020	0.30 ± 0.018	0.30 ± 0.029	0.33 ± 0.034	0.707	0.871	0.384
ADFI, kg	0.44 ± 0.029	0.42 ± 0.028	0.44 ± 0.043	0.48 ± 0.051	0.502	0.768	0.393
G:F	0.73 ± 0.038	0.72 ± 0.036	0.68 ± 0.055	0.68 ± 0.066	0.404	0.906	0.877
BW d 28, kg	12.36 ± 0.599	11.37 ± 0.563	12.54 ± 0.877	13.10 ± 1.038	0.239	0.791	0.334
Day 28-42							
ADG, kg	0.53 ± 0.036	0.48 ± 0.034	0.41 ± 0.052	0.55 ± 0.062	0.614	0.305	0.054
ADFI, kg	0.77 ± 0.045	0.74 ± 0.043	0.67 ± 0.066	0.79 ± 0.079	0.649	0.490	0.231
G:F	0.68 ± 0.016	0.65 ± 0.015	0.59 ± 0.024	0.70 ± 0.028	0.342	0.075	0.002
BW d 42, kg	19.75 ± 1.016	18.09 ± 0.954	18.24 ± 1.487	20.65 ± 1.760	0.701	0.782	0.139
Day 0-42[2]							
ADG, kg	0.33 ± 0.022	0.32 ± 0.020	0.30 ± 0.032	0.36 ± 0.037	0.915	0.340	0.174
ADFI, kg	0.48 ± 0.029	0.45 ± 0.027	0.46 ± 0.043	0.49 ± 0.051	0.820	0.858	0.407
G:F	0.70 ± 0.018	0.71 ± 0.017	0.63 ± 0.026	0.74 ± 0.031	0.565	0.018	0.054

Table 4.4 Continued

	2000-2005		2011-2017		Probability, $P <$		
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Day 42-63							
ADG, kg	0.70 ± 0.033	0.67 ± 0.031	0.59 ± 0.048	0.65 ± 0.057	0.163	0.684	0.318
ADFI, kg	1.22 ± 0.066	1.19 ± 0.062	1.05 ± 0.097	1.16 ± 0.115	0.254	0.654	0.417
G:F	0.58 ± 0.009	0.57 ± 0.009	0.57 ± 0.013	0.56 ± 0.016	0.674	0.642	0.802
BW d 63, kg	34.36 ± 1.650	32.25 ± 1.550	30.58 ± 2.415	34.25 ± 2.858	0.688	0.724	0.193
Day 63-87							
ADG, kg	0.93 ± 0.028	0.86 ± 0.027	0.89 ± 0.041	0.91 ± 0.049	0.852	0.478	0.236
ADFI, kg	2.04 ± 0.077	1.82 ± 0.072	1.90 ± 0.113	1.87 ± 0.134	0.634	0.210	0.367
G:F	0.46 ± 0.007	0.47 ± 0.007	0.47 ± 0.011	0.49 ± 0.013	0.066	0.107	0.913
BW d 87, kg	57.09 ± 2.204	53.23 ± 2.070	52.55 ± 3.226	56.02 ± 3.817	0.766	0.947	0.217
Day 87-108							
ADG, kg	1.21 ± 0.031	1.07 ± 0.029	1.19 ± 0.045	1.11 ± 0.054	0.745	0.014	0.412
ADFI, kg	2.75 ± 0.090	2.27 ± 0.085	2.56 ± 0.132	2.42 ± 0.157	0.853	0.013	0.162
G:F	0.44 ± 0.010	0.47 ± 0.010	0.47 ± 0.015	0.46 ± 0.018	0.517	0.322	0.199
BW d 108, kg	83.03 ± 2.669	76.74 ± 2.507	78.36 ± 3.907	79.40 ± 4.623	0.778	0.462	0.306
Day 108-120							
ADG, kg	0.96 ± 0.030	0.91 ± 0.029	0.99 ± 0.044	0.88 ± 0.053	0.946	0.047	0.513
ADFI, kg	3.09 ± 0.093	2.64 ± 0.088	2.93 ± 0.147	2.59 ± 0.161	0.419	0.003	0.693
G:F	0.31 ± 0.006	0.34 ± 0.006	0.34 ± 0.010	0.34 ± 0.011	0.299	0.043	0.108
BW d 120, kg	94.57 ± 2.942	87.60 ± 2.764	90.21 ± 4.307	89.99 ± 5.096	0.801	0.362	0.392
Day 120-150							
ADG, kg	0.96 ± 0.023	0.89 ± 0.021	1.03 ± 0.033	0.95 ± 0.039	0.037	0.021	0.992
ADFI, kg	3.16 ± 0.080	2.79 ± 0.075	3.19 ± 0.117	2.88 ± 0.138	0.581	0.002	0.761
G:F	0.30 ± 0.007	0.32 ± 0.006	0.32 ± 0.010	0.33 ± 0.012	0.083	0.162	0.715

Table 4.4 Continued

| | 2000-2005 | | 2011-2017 | | Probability, $P <$ | | |
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
BW d 150, kg	123.46 ± 3.106	114.28 ± 2.918	120.99 ± 4.547	118.58 ± 5.380	0.825	0.167	0.416
Day 150-168[3]							
Pens remaining, n	13	16	7	3	---	---	---
Pigs remaining, n	20	34	9	9	---	---	---
ADG, kg	0.76 ± 0.049	0.93 ± 0.044	0.76 ± 0.067	1.02 ± 0.102	0.492	0.004	0.490
ADFI, kg	3.11 ± 0.095	2.95 ± 0.090	2.99 ± 0.140	3.10 ± 0.185	0.912	0.906	0.317
G:F	0.25 ± 0.014	0.31 ± 0.013	0.26 ± 0.020	0.33 ± 0.030	0.513	0.002	0.796
BW d 150,[4] kg	120.89 ± 3.131	113.22 ± 2.822	120.99 ± 4.267	112.14 ± 6.518	0.913	0.071	0.894
BW d 168, kg	125.63 ± 2.976	123.24 ± 2.683	124.16 ± 4.056	125.32 ± 6.195	0.943	0.885	0.676
Day 42-End[5]							
ADG, kg	1.06 ± 0.037	0.96 ± 0.035	1.04 ± 0.055	1.05 ± 0.065	0.541	0.363	0.244
ADFI, kg	2.57 ± 0.067	2.29 ± 0.063	2.45 ± 0.107	2.27 ± 0.131	0.485	0.022	0.609
G:F	0.41 ± 0.009	0.42 ± 0.009	0.42 ± 0.014	0.44 ± 0.018	0.257	0.300	0.515
Day 0-End[6]							
ADG, kg	0.79 ± 0.018	0.75 ± 0.017	0.77 ± 0.027	0.78 ± 0.032	0.810	0.467	0.317
ADFI, kg	2.03 ± 0.053	1.84 ± 0.050	1.96 ± 0.084	1.82 ± 0.103	0.531	0.031	0.716
G:F	0.39 ± 0.006	0.41 ± 0.005	0.39 ± 0.009	0.42 ± 0.011	0.277	0.006	0.613
BW end,[7] kg	129.97 ± 2.257	126.12 ± 2.120	128.60 ± 3.304	128.65 ± 3.909	0.848	0.529	0.518
Day 0-42							
Initial pigs on test, n	51	62	24	18	---	---	---
End pigs on test, n	48	54	22	17	---	---	---
Mortality, %	5.9	12.9	8.3	5.6	---	---	---
Day 42-End							
Initial pigs on test, n	48	54	22	17	---	---	---
End pigs on test, n	44	48	17	14	---	---	---
Mortality, %	8.3	11.1	22.7	17.6	---	---	---

(Table continues)

Table 4.5 Effect of genetics and sex on carcass characteristics from pigs harvested at Purdue's Meats Lab[1]

| | 2000-2005 | | 2011-2017 | | | Probability, $P <$ | | |
	Barrows	Gilts	Barrows	Gilts	SEM	Genetics	Sex	Genetics × Sex
Pigs, n	10	10	10	10	---	---	---	---
Avg TSI	88.26	83.35	115.31	107.87	4.675	0.001	0.167	0.774
Live farm weight,[2] kg	134.88	132.95	136.35	133.92	2.131	0.569	0.314	0.908
Hot carcass weight, kg	103.43	101.35	103.62	101.56	1.581	0.899	0.200	0.994
Carcass yield,[3] %	76.71	76.24	76.00	75.84	0.421	0.202	0.460	0.717
Carcass Characteristics								
Loin muscle color score[4]	3.05	2.75	2.75	2.75	0.178	0.404	0.404	0.404
Loin muscle marbling score[4]	2.75	2.25	2.05	2.30	0.110	0.006	0.265	0.002
Loin muscle firmness score[4]	2.85	2.45	2.40	2.50	0.150	0.192	0.325	0.105
Loin muscle L*[5]	51.45	51.37	53.25	52.21	0.948	0.171	0.559	0.615
Loin muscle a*[5]	8.33	8.11	8.59	8.12	0.407	0.735	0.408	0.754
Loin muscle b*[5]	6.20	5.69	6.58	6.10	0.412	0.334	0.237	0.970
Last lumbar backfat,[6] cm	2.36	2.08	2.20	1.75	0.119	0.048	0.004	0.477
Last rib backfat,[6] cm	2.92	2.57	2.79	2.37	0.121	0.189	0.003	0.775
Tenth rib backfat, cm	2.58	2.06	2.24	1.96	0.134	0.107	0.005	0.375
Loin muscle area, cm^2	46.64	49.06	50.69	50.60	1.949	0.161	0.554	0.522
Calculated fat-free lean,[7] %	45.24	47.85	47.13	49.09	0.898	0.090	0.015	0.716
Loin muscle 24-hour pH[8]	5.59	5.57	5.58	5.57	0.029	0.865	0.635	0.973
Avg loin muscle drip loss,[9] %	6.70	7.74	8.07	8.73	0.646	0.076	0.197	0.777
Belly thickness, cm	5.26	4.84	5.07	4.88	0.136	0.578	0.031	0.405
Live ultrasound scan data[10]								
Last rib backfat, cm	1.84	1.58	1.62	1.35	0.094	0.023	0.008	0.958
Tenth rib backfat, cm	1.98	1.67	1.74	1.44	0.097	0.021	0.004	0.959
Tenth rib LD,[11] cm	5.00	5.11	5.19	5.30	0.103	0.073	0.291	1.000
Tenth rib LMA,[12] cm^2	49.77	52.27	51.66	51.41	1.137	0.651	0.330	0.235

(Table continues)

[1] A total of 40 pigs were harvested at the Purdue Meats Lab to determine genetics and sex impacts on carcass characteristics. Frozen semen from Duroc boars were divided into 2 genetic groups: boars born from 2000 to 2005 vs. 2011 to 2017. The boars were mated to maternal line gilts (TN70; Topigs Norsvin). Pigs were harvested on 2 different days; d 154 and 170 post-weaning.

[2] Live weights measured at the farm two days before harvest.

[3] Carcass yield was calculated as the hot carcass weight divided by live weight multiplied by 100.

[4] The NPPC color scores range from 1 to 6, with 1 being the lightest color and 6 being the darkest (NPPC, 2000). The NPPC marbling scores visually estimate the amount of intramuscular fat (marbling) in the meat and range from 1 to 10, with 1 being devoid of marbling and 10 having abundant marbling (NPPC, 2000). The NPPC firmness scores range from 1 to 5, with 1 being very soft and 5 being very firm (NPPC, 1991).

[5] Average of 3 readings per longissimus dorsi sample using a Hunter MiniScan EZ colorimeter (Hunter; Reston, VA, USA).

[6] Average measurements presented are averages of 2 sides of the carcass.

[7] Fat-free lean percent was calculated as $11.08 + (0.218*\text{live weight, kg}) + (-0.715*\text{tenth rib backfat, cm}) + (-3.31*\text{last rib backfat, cm}) + (0.346*\text{loin muscle area, cm}^2) / (\text{live weight, kg}* 0.74) * 100$ (Schinckel et al., 2001).

[8] Ultimate pH measurements taken using a calibrated meat pH probe directly inserted into the loin muscle tissue on the 11th rib side of the 10th/11th rib carcass split (HANNA HI 99163, Hanna Instrument, Inc., Warner, NH, USA).

[9] Three meat samples (19 mm cores) were collected from the same longissimus dorsi sample (approximately 25 mm thick) from each pig. The sample cores were placed in pre-weighed EZ-DripLoss tubes (Danish Technological Institute, Taastrup, Denmark), reweighed, and stored in a cooler (4°C) for 24 hours. At 24 hours post-collection, the sample core was removed from the tubes and remaining purge was weighed. Drip loss percentage was calculated by dividing the weight of the purge by the weight of the original meat sample times 100. The drip loss percentage of the 3 meat cores were averaged.

[10] Live ultrasonic measurements of backfat depth, loin muscle depth, and loin muscle area were collected at the farm using an Aloka SSD 500V (Aloka Co., Ltd., Tokyo, Japan).

[11] Loin muscle depth.

[12] Loin muscle area.

Table 4.6 Effect of genetics and sex on carcass characteristics from pigs harvested at Tyson[1,2]

| | 2000-2005 | | 2011-2017 | | Probability, $P <$ | | |
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Pigs, n	27	35	6	4	---	---	---
Average TSI	87.51 ± 1.857	89.53 ± 1.709	118.07 ± 3.828	119.83 ± 4.688	0.001	0.569	0.969
Live weight,[3] kg	132.72 ± 1.537	128.95 ± 1.414	134.57 ± 3.168	123.60 ± 3.880	0.518	0.009	0.192
HCW, kg	100.41 ± 1.496	95.06 ± 1.307	99.30 ± 3.071	92.08 ± 3.395	0.412	0.015	0.712
Carcass yield,[4] %	75.64 ± 0.519	73.32 ± 0.463	73.40 ± 1.035	74.43 ± 1.133	0.508	0.449	0.052
Backfat depth,[5] cm	1.80 ± 0.072	1.44 ± 0.066	1.78 ± 0.150	1.26 ± 0.183	0.430	0.001	0.524
Loin muscle depth,[5] cm	6.58 ± 0.105	6.52 ± 0.099	6.07 ± 0.212	6.36 ± 0.260	0.073	0.539	0.329
Percent lean,[6] %	54.99 ± 0.302	55.57 ± 0.261	54.02 ± 0.617	55.83 ± 0.688	0.480	0.023	0.230
Live ultrasound scan data[7]							
Last rib backfat, cm	1.75 ± 0.063	1.36 ± 0.058	1.72 ± 0.131	1.28 ± 0.160	0.599	0.001	0.792
Tenth rib backfat, cm	1.83 ± 0.063	1.47 ± 0.058	1.82 ± 0.130	1.30 ± 0.159	0.411	0.001	0.491
Tenth rib LD,[8] cm	5.03 ± 0.073	4.88 ± 0.067	5.00 ± 0.151	4.75 ± 0.185	0.541	0.132	0.083
Tenth rib LMA,[9] cm^2	50.81 ± 0.721	49.03 ± 0.659	51.16 ± 1.493	48.34 ± 1.829	0.895	0.078	0.685

[1] A total of 73 pigs were harvested at Tyson Fresh Meats (Logansport, IN) to determine genetics and sex impacts on carcass characteristics. Frozen semen from Duroc boars were divided into 2 genetic groups: boars born from 2000 to 2005 vs. 2011 to 2017. The boars were mated to maternal line gilts (TN70; Topigs Norsvin). Pigs were harvested on 2 different days; d 155 and 170.

[2] One very heavy weight early (2000-2005) genetics barrow was removed from the data completely due to carcass values being 3 standard deviations from most mean carcass values. Hot carcass weight, yield, and percent lean data points from 9 pigs were removed due to trim loss or an obvious data collection error. The 8 pigs for trim loss include 4 early (2000-2005) genetics barrows, 3 early genetics gilts, and 1 late genetics barrow. The pig with a data collection error was an early genetics barrow.

[3] Live weights measured at the Purdue farm 2 days prior to harvest.

[4] Carcass yield was calculated as the hot carcass weight divided by live weight multiplied by 100.

[5] Carcass backfat and loin muscle depth were measured using the Animal Ultrasound Services (Ithaca, NY) Carcass Value Technology System (CVT system) with a linear measurement from last to tenth rib.

[6] The standardized fat-free lean percent was calculated as: [3.0767 - (2.8117*average fat depth, cm) + (0.7156*average muscle depth, cm) + (0.47*hot carcass weight, kg)]/ (hot carcass weight, kg) * 100 (NPPC, 2000).

[7] Live ultrasonic measurements of backfat depth, loin muscle depth, and loin muscle area were collected using an Aloka SSD 500V (Aloka Co., Ltd., Tokyo, Japan). Last scans were completed on d 150 and 169 post-weaning.

[8] Loin muscle depth.

[9] Loin muscle area.

Table 4.7 Effect of genetics and sex on live serial ultrasound scans[1]

	2000-2005		2011-2017		Probability, $P <$		
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Day 42 post-weaning							
Pigs, *n*	20	22	16	16	---	---	---
Average TSI	87.88 ± 1.606	88.13 ± 1.457	112.43± 2.503	113.52± 2.782	0.001	0.758	0.846
Live weight, kg	21.99 ± 0.960	20.75 ± 0.915	20.83 ± 1.073	21.89 ± 1.073	0.992	0.933	0.256
Last rib backfat, cm	0.50 ± 0.025	0.45 ± 0.024	0.44 ± 0.028	0.46 ± 0.028	0.401	0.548	0.189
Tenth rib backfat, cm	0.55 ± 0.025	0.50 ± 0.024	0.47 ± 0.028	0.46 ± 0.028	0.039	0.285	0.406
Tenth rib LD,[2] cm	2.04 ± 0.069	2.05 ± 0.066	1.97 ± 0.077	2.07 ± 0.077	0.768	0.449	0.539
Tenth rib LMA,[3] cm^2	12.21 ± 0.548	11.82 ± 0.523	11.77 ± 0.613	12.04 ± 0.613	0.847	0.926	0.566
Day 61 post-weaning							
Pigs, *n*	20	22	16	15	---	---	---
Live weight, kg	38.79 ± 1.612	36.75 ± 1.537	35.28 ± 1.802	36.13 ± 1.861	0.231	0.729	0.400
Last rib backfat, cm	0.64 ± 0.028	0.59 ± 0.027	0.62 ± 0.032	0.59 ± 0.033	0.756	0.221	0.696
Tenth rib backfat, cm	0.72 ± 0.029	0.63 ± 0.027	0.64 ± 0.032	0.61 ± 0.033	0.118	0.081	0.334
Tenth rib LD, cm	2.67 ± 0.082	2.53 ± 0.078	2.43 ± 0.092	2.57 ± 0.095	0.231	0.984	0.112
Tenth rib LMA, cm^2	19.80 ± 0.628	18.88 ± 0.599	19.31 ± 0.702	19.18 ± 0.725	0.882	0.434	0.554
Day 88 post-weaning							
Pigs, *n*	20	22	15	15	---	---	---
Live weight, kg	62.79 ± 2.026	58.39 ± 1.932	59.23 ± 2.340	58.46 ± 2.340	0.424	0.238	0.405
Last rib backfat, cm	0.83 ± 0.040	0.71 ± 0.038	0.78 ± 0.046	0.69 ± 0.046	0.434	0.017	0.793
Tenth rib backfat, cm	0.87 ± 0.040	0.73 ± 0.039	0.82 ± 0.047	0.70 ± 0.047	0.375	0.003	0.794
Tenth rib LD, cm	3.42 ± 0.076	3.27 ± 0.072	3.33 ± 0.088	3.45 ± 0.088	0.565	0.867	0.105
Tenth rib LMA, cm^2	27.50 ± 0.802	25.51 ± 0.765	26.43 ± 0.926	25.98 ± 0.926	0.730	0.161	0.373
Day 108 post-weaning							
Pigs, *n*	20	22	15	15	---	---	---
Live weight, kg	88.80 ± 2.282	81.50 ± 2.175	85.51 ± 2.634	82.07 ± 2.634	0.580	0.031	0.430
Last rib backfat, cm	1.16 ± 0.049	0.91 ± 0.047	1.05 ± 0.057	0.83 ± 0.057	0.062	0.001	0.803

Table 4.7 Continued

	2000-2005		2011-2017		Probability, $P <$		
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Tenth rib backfat, cm	1.19 ± 0.048	0.93 ± 0.045	1.07 ± 0.055	0.86 ± 0.055	0.059	0.001	0.614
Tenth rib LD, cm	4.09 ± 0.086	3.81 ± 0.082	3.89 ± 0.099	4.06 ± 0.099	0.788	0.552	0.019
Tenth rib LMA, cm^2	36.38 ± 0.980	33.28 ± 0.934	34.58 ± 1.131	34.74 ± 1.131	0.871	0.165	0.124
Day 120 post-weaning							
Pigs, *n*	20	22	14	15	---	---	---
Live weight, kg	100.05 ± 2.547	92.72 ± 2.428	98.55 ± 3.044	93.19 ± 2.941	0.853	0.024	0.721
Last rib backfat, cm	1.33 ± 0.061	0.96 ± 0.058	1.23 ± 0.073	0.93 ± 0.070	0.319	0.001	0.589
Tenth rib backfat, cm	1.43 ± 0.061	1.04 ± 0.058	1.26 ± 0.073	1.01 ± 0.070	0.154	0.001	0.324
Tenth rib LD, cm	4.49 ± 0.095	4.10 ± 0.091	4.38 ± 0.114	4.31 ± 0.110	0.605	0.032	0.124
Tenth rib LMA, cm^2	40.18 ± 0.901	37.35 ± 0.859	39.82 ± 1.077	38.58 ± 1.040	0.652	0.040	0.415
Day 150 post-weaning							
Pigs, *n*	20	22	14	15	---	---	---
Live weight, kg	128.67 ± 2.818	120.25 ± 2.687	129.79 ± 3.368	122.03 ± 3.254	0.635	0.010	0.916
Last rib backfat, cm	1.79 ± 0.075	1.30 ± 0.072	1.56 ± 0.090	1.18 ± 0.087	0.043	0.001	0.521
Tenth rib backfat, cm	1.92 ± 0.077	1.37 ± 0.074	1.67 ± 0.092	1.25 ± 0.089	0.035	0.001	0.443
Tenth rib LD, cm	4.96 ± 0.098	4.74 ± 0.093	5.18 ± 0.117	4.96 ± 0.113	0.040	0.045	0.983
Tenth rib LMA, cm^2	49.17 ± 1.021	47.15 ± 0.974	50.84 ± 1.221	48.66 ± 1.179	0.154	0.062	0.944
Last scan day[4]							
Pigs, *n*	44	48	17	16	---	---	---
Average TSI	87.17	87.86	114.09	112.36	0.001	0.829	0.614
Live weight, kg	129.74 ± 1.864	125.82 ± 1.784	130.19 ± 2.998	128.50 ± 3.090	0.536	0.266	0.657
Last rib backfat, cm	1.74 ± 0.050	1.38 ± 0.048	1.64 ± 0.081	1.29 ± 0.084	0.162	0.001	0.907
Tenth rib backfat, cm	1.84 ± 0.052	1.47 ± 0.049	1.75 ± 0.083	1.37 ± 0.085	0.166	0.001	0.940
Tenth rib LD, cm	5.01 ± 0.062	4.88 ± 0.059	5.02 ± 0.100	5.15 ± 0.103	0.088	0.953	0.118
Tenth rib LMA, cm^2	50.29 ± 0.634	49.20 ± 0.607	50.59 ± 1.019	50.59 ± 1.051	0.325	0.526	0.528

[1] Ultrasonic measurements (Aloka SSD 500V; Aloka Co., Ltd., Tokyo, Japan) were collected initially from 74 pigs to determine genetics and sex impacts on backfat, loin muscle depth, and loin muscle area of the live animal over the grow-finish period. Frozen semen from Duroc boars were divided into 2 genetic groups: boars born from 2000 to 2005 vs. 2011 to 2017. The boars were mated to maternal line gilts (TN70; Topigs Norsvin).

[2] Loin muscle depth.

[3] Loin muscle area.

[4] A total of 125 pigs were live scanned before harvest. Last scans were completed d 150 and 169 post-weaning.

Table 4.8 Effect of genetics and sex on average daily gain and tissue accretions[1]

| | 2000-2005 | | 2011-2017 | | Probability, $P <$ | | |
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Pigs, n	20	22	14	15	---	---	---
Avg TSI	87.8 ± 2.60	87.1 ± 2.47	116.3 ± 3.35	111.5 ± 3.10	0.001	0.339	0.475
ADG,[2] g/d	1115 ± 43.9	1021 ± 41.9	1119 ± 52.5	1072 ± 50.7	0.563	0.144	0.613
Avg daily tissue accretions[3]							
Empty body protein, g/d	147 ± 3.2	143 ± 3.0	150 ± 3.8	147 ± 3.7	0.311	0.265	0.941
Empty body lipid, g/d	274 ± 8.6	236 ± 8.2	276 ± 10.3	234 ± 9.9	0.952	0.001	0.846
Fat-free lean, g/d	345 ± 8.2	338 ± 7.9	355 ± 9.8	355 ± 9.5	0.132	0.698	0.725
Carcass fat, g/d	329 ± 9.9	293 ± 9.4	330 ± 11.8	293 ± 11.4	0.961	0.001	0.994

[1] Ultrasonic measurements (Aloka SSD 500V; Aloka Co., Ltd., Tokyo, Japan) were collected on the first day of scanning (post-weaning d 42) and on the last weigh day before marketing. Last scans were completed on d 150 or 169 post-weaning. A total of 71 pigs were scanned on both days and were used to determine genetics and sex impacts on average daily gain and average daily tissue accretions. Frozen semen from Duroc boars were divided into 2 genetic groups: boars born from 2000 to 2005 vs. 2011 to 2017. The boars were mated to maternal line gilts (TN70; Topigs Norsvin).

[2] Average daily gain was calculated by subtracting each pig's weight at d 42 post-weaning from their ending weight on either d 150 or 169 post-weaning then dividing by the number of days between weights.

[3] Average daily tissue accretions were calculated by subtracting the predicted tissue composition at d 42 post-weaning from their ending predicted tissue composition on either d 150 or 169 post-weaning then dividing by the number of days between ultrasonic measurements. The tissue accretions were predicted using equations published by Wagner et al. (1999) and used to develop curves based on Schinckel and de Lange (1996) equations.

Table 4.9 Correlations of growth performance and tissue accretion with terminal sire index[1]

	Average daily body weight gain	Average daily protein deposition	Average daily fat-free lean gain
Terminal Sire Index	0.073 (0.56)	0.040 (0.75)	0.067 (0.59)

[1] Pearson correlation coefficients were calculated using PROC CORR in SAS 9.4 (SAS Institute, Inc., Cary, NC). The data is presented as the correlation coefficient (*P*-value).

Table 4.10 Effect of genetics and sex on therapeutic injectable antibiotic treatment rate [1,2,3]

| | 2000-2005 | | 2011-2017 | | Probability, $P <$ | | |
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Pens, n	15	17	7	5	---	---	---
Pigs, n	51	62	24	18	---	---	---
Avg TSI	87.51 ± 3.107	88.86 ± 2.919	113.12 ± 4.913	110.79 ± 5.382	0.0001	0.908	0.665
Weaning weight, kg	5.41 ± 0.285	4.70 ± 0.268	5.87 ± 0.417	5.57 ± 0.493	0.085	0.194	0.593
Day 0-14							
Total Therapies,[4] %	21.11 ± 5.397	21.08± 5.070	20.24 ± 7.901	16.67 ± 9.348	0.552	0.907	0.623
Enteric, %	3.33 ± 3.402	2.94 ± 3.196	13.10 ± 4.980	6.67 ± 5.893	0.288	0.646	0.730
Lameness, %	10.00 ± 4.139	13.73 ± 3.888	3.57 ± 6.059	0.00 ± 7.169	0.058	0.863	0.492
Strep., %	5.56 ± 1.999	0.00 ± 1.878	0.00 ± 2.927	5.00 ± 3.463	0.977	0.977	0.047
Unthrifty, %	0.00 ± 2.356	4.41 ± 2.213	3.57 ± 3.449	0.00 ± 4.080	0.945	0.945	0.155
Respiratory, %	2.22 ± 1.601	0.00 ± 1.503	0.00 ± 2.343	5.00 ± 2.772	0.397	0.397	0.075
Other,[5] %	---	---	---	---	---	---	---
Day 14-28							
Total Therapies, %	19.44 ± 5.693	15.20 ± 5.348	10.71 ± 8.334	10.00 ± 9.861	0.489	0.797	0.488
Enteric, %	---	---	---	---	---	---	---
Lameness, %	4.44 ± 2.671	2.94 ± 2.509	0.00 ± 3.909	0.00 ± 4.625	0.287	0.714	0.714
Strep., %	3.89 ± 3.452	8.33 ± 3.242	0.00 ± 5.053	5.00 ± 5.978	0.484	0.254	0.751
Unthrifty, %	8.89 ± 3.879	3.92 ± 3.644	7.14 ± 5.679	0.00 ± 6.719	0.805	0.175	0.510
Respiratory, %	2.22 ± 1.859	0.00 ± 1.746	3.57 ± 2.721	5.00 ± 3.219	0.140	0.933	0.475
Other, %	---	---	---	---	---	---	---
Day 28-42							
Total Therapies, %	23.33 ± 6.624	25.98 ± 6.222	28.57 ± 9.696	11.67 ± 11.473	0.269	0.875	0.668
Enteric, %	---	---	---	---	---	---	---
Lameness, %	8.33 ± 3.495	5.88 ± 3.283	0.00 ± 5.116	0.00 ± 6.053	0.113	0.593	0.593
Strep., %	2.22 ± 2.089	1.96 ± 1.962	3.57 ± 3.058	0.00 ± 3.618	0.972	0.429	0.482

Table 4.10 Continued

	2000-2005		2011-2017		Probability, $P <$		
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Unthrifty, %	12.78 ± 4.949	14.71 ± 4.649	15.48 ± 7.245	0.00 ± 8.573	0.148	0.380	0.317
Respiratory, %	0.00 ± 3.015	1.47 ± 2.832	9.52 ± 4.413	11.67 ± 5.222	0.013	0.169	0.409
Other, %	0.00 ± 1.320	1.96 ± 1.240	0.00 ± 1.933	0.00 ± 2.287	0.578	0.578	0.578
Day 42-63							
Total Therapies, %	8.33 ± 4.494	9.80 ± 4.222	7.14 ± 6.579	5.00 ± 7.785	0.610	0.720	0.848
Enteric, %	2.22 ± 1.315	0.00 ± 1.235	0.00 ± 1.925	0.00 ± 2.277	0.527	0.527	0.527
Lameness, %	6.11 ± 3.382	2.94 ± 3.177	0.00 ± 4.951	0.00 ± 5.858	0.288	0.696	0.696
Strep., %	---	---	---	---	---	---	---
Unthrifty, %	0.00 ± 0.990	1.47 ± 0.930	0.00 ± 1.449	0.00 ± 1.715	0.578	0.578	0.578
Respiratory, %	0.00 ± 2.639	3.43 ± 2.479	7.14 ± 3.863	5.00 ± 4.571	0.254	0.490	0.662
Other, %	0.00 ± 1.320	1.96 ± 1.240	0.00 ± 1.933	0.00 ± 2.287	0.578	0.578	0.578
Day 63-87							
Total Therapies, %	17.22 ± 5.495	14.71 ± 5.161	9.52 ± 8.043	16.67 ± 9.517	0.406	0.587	0.385
Enteric, %	---	---	---	---	---	---	---
Lameness, %	2.22 ± 2.377	2.94 ± 2.233	0.00 ± 3.480	0.00 ± 4.117	0.404	0.994	0.994
Strep., %	---	---	---	---	---	---	---
Unthrifty, %	11.11 ± 3.474	3.43 ± 3.263	0.00 ± 5.085	10.00 ± 6.017	0.401	0.994	0.101
Respiratory, %	3.89 ± 4.158	8.33 ± 3.906	9.52 ± 6.087	6.67 ± 7.202	0.986	0.624	0.779
Other, %	---	---	---	---	---	---	---
Day 87-108							
Total Therapies, %	4.44 ± 3.020	6.37 ± 2.837	0.00 ± 4.421	0.00 ± 5.231	0.171	0.841	0.841
Enteric, %	---	---	---	---	---	---	---
Lameness, %	2.22 ± 2.377	2.94 ± 2.233	0.00 ± 3.480	0.00 ± 4.117	0.404	0.994	0.994
Strep., %	---	---	---	---	---	---	---

Table 4.10 Continued

	2000-2005		2011-2017		Probability, *P* <		
	Barrows	Gilts	Barrows	Gilts	Genetics	Sex	Genetics × Sex
Unthrifty, %	0.00 ± 1.320	1.96 ± 1.240	0.00 ± 1.933	0.00 ± 2.287	0.578	0.578	0.578
Respiratory, %	---	---	---	---	---	---	---
Other, %	2.22 ± 1.646	1.47 ± 1.546	0.00 ± 2.409	0.00 ± 2.851	0.401	0.931	0.931
Day 108-120							
Total Therapies, %	2.22 ± 2.609	1.96 ± 2.450	0.00 ± 3.819	10.00 ± 4.518	0.600	0.248	0.216
Enteric, %	---	---	---	---	---	---	---
Lameness, %	---	---	---	---	---	---	---
Strep., %	---	---	---	---	---	---	---
Unthrifty, %	---	---	---	---	---	---	---
Respiratory, %	2.22 ± 2.609	1.96 ± 2.450	0.00 ± 3.819	10.00 ± 4.518	0.600	0.248	0.216
Other, %	---	---	---	---	---	---	---
Day 120-150							
Total Therapies, %	8.89 ± 4.441	7.84 ± 4.171	11.90 ± 6.501	6.67 ± 7.692	0.869	0.795	0.696
Enteric, %	---	---	---	---	---	---	---
Lameness, %	3.33 ± 1.972	0.00 ± 1.852	0.00 ± 2.887	0.00 ± 3.416	0.527	0.527	0.527
Strep., %	---	---	---	---	---	---	---
Unthrifty, %	1.67 ± 3.419	7.84 ± 3.212	7.14 ± 5.005	6.67 ± 5.922	0.811	0.411	0.611
Respiratory, %	3.89 ± 2.026	0.00 ± 1.903	4.76 ± 2.965	0.00 ± 3.508	0.931	0.118	0.931
Other, %	---	---	---	---	---	---	---
Day 150-168							
Pigs remaining, *n*	20	34	9	9	---	---	---
Total Therapies, %	---	---	---	---	---	---	---

(Table continues)

[1] A total of 44 pens of pigs were used to determine genetics and sex impacts on growth performance and carcass characteristics. Frozen semen from Duroc boars were divided into 2 genetic groups: boars born from 2000 to 2005 vs. 2011 to 2017. The boars were mated to maternal line gilts (TN70; Topigs Norsvin).

[2] The percent of pigs within a pen treated with therapeutic injectable antibiotics was calculated for enteric, lameness, *Streptococcus suis* infection (Strep.), unthriftiness, respiratory challenges and other conditions. The total therapies is a sum of all categories.

[3] Data was log-transformed to meet assumptions of normality; however, means and standard errors are presented as non-transformed values for ease of interpretation and p-values represent the log-transformed data. Data is presented as the LS mean ± SEM.

[4] Pigs treated at least once for any reason as a percent of pigs remaining in the pen.

[5] The other category included therapies for salmonella and skin irritation.

[6] The mortality percentage was calculated as ending pigs on test / initial pigs on test * 100 for each genetic group and sex. The p-values presented for these calculations are Chi-square statistics.
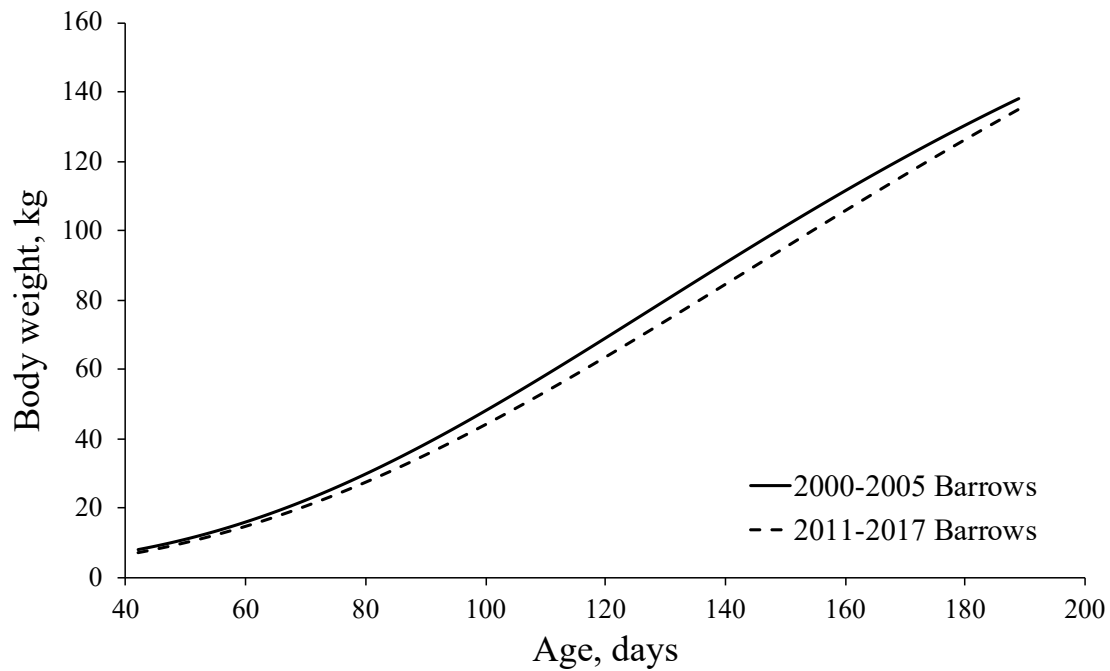
Figure 4.1 Prediction of body weight based on age in barrows. The generalized Michaelis-Menten (GMM) equation was used to predict the mean BW, kg: $WT_{i,t} = WT_0 + \{[(WF - WT_0)(t/K)^C]/[1 + (t/K)^C]\}$, where $WT_{i,t}$ is the BW in kg of the ith pig at age t, WT_0 is the mean birth BW (1.6 kg in this experiment), WF is mean mature BW, and K is a parameter equal to the days of age at which one-half of the mature BW is achieved. The C is a unitless parameter related to the changes in proportional growth and the shape of the growth curves (López et al., 2000; Schinckel et al., 2009a).
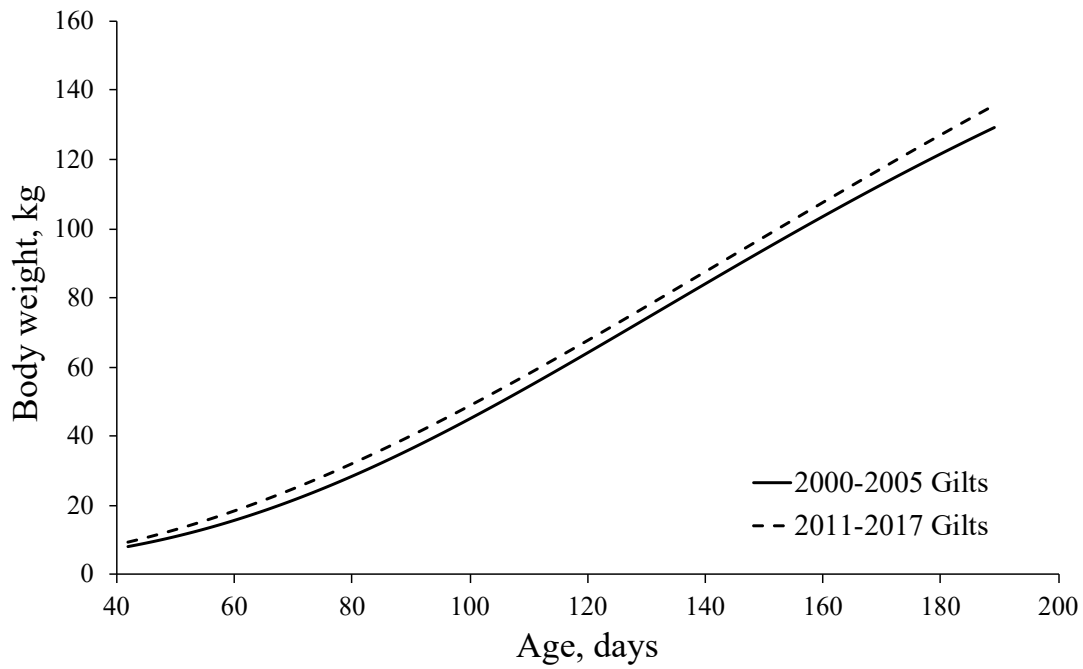
Figure 4.2 Prediction of body weight based on age in gilts. The generalized Michaelis-Menten (GMM) equation was used to predict the mean BW, kg: $WT_{i,t} = WT_0 + \{[(WF - WT_0)\,(t/K)^C]/[1 + (t/K)^C]\}$, where $WT_{i,t}$ is the BW in kg of the ith pig at age t, WT_0 is the mean birth BW (1.6 kg in this experiment), WF is mean mature BW, and K is a parameter equal to the days of age at which one-half of the mature BW is achieved. The C is a unitless parameter related to the changes in proportional growth and the shape of the growth curves (López et al., 2000; Schinckel et al., 2009a).
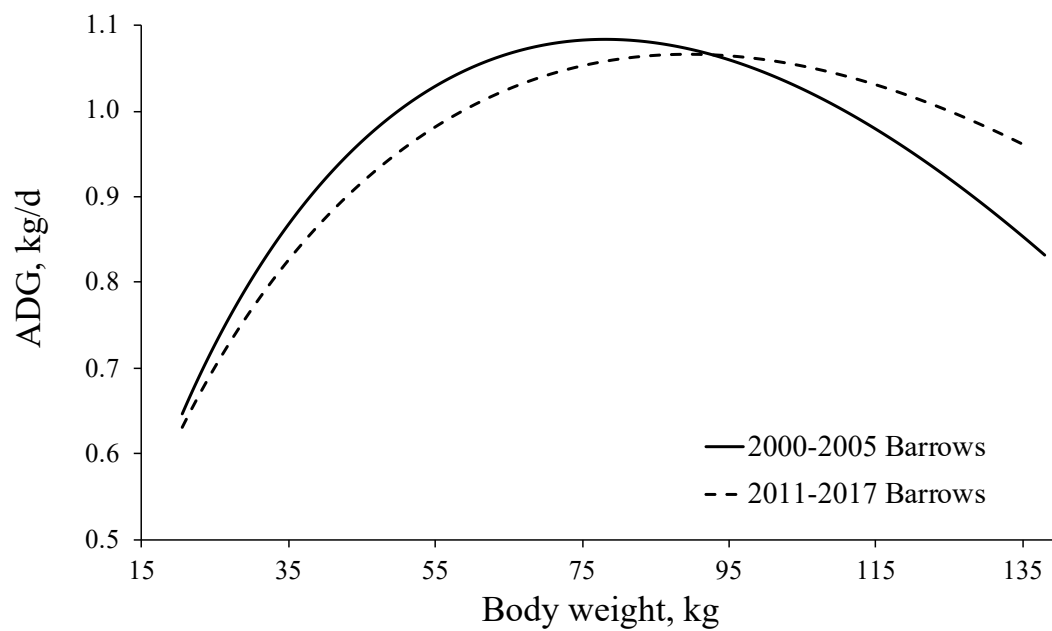
Figure 4.3 Prediction of average daily gain (ADG) based on body weight for barrows. Average daily gain was calculated as the derivative of the generalized Michaelis-Menten (GMM) function to predict BW in kg on time (ADG = ∂WT/∂T; Schinckel et al., 2009a).
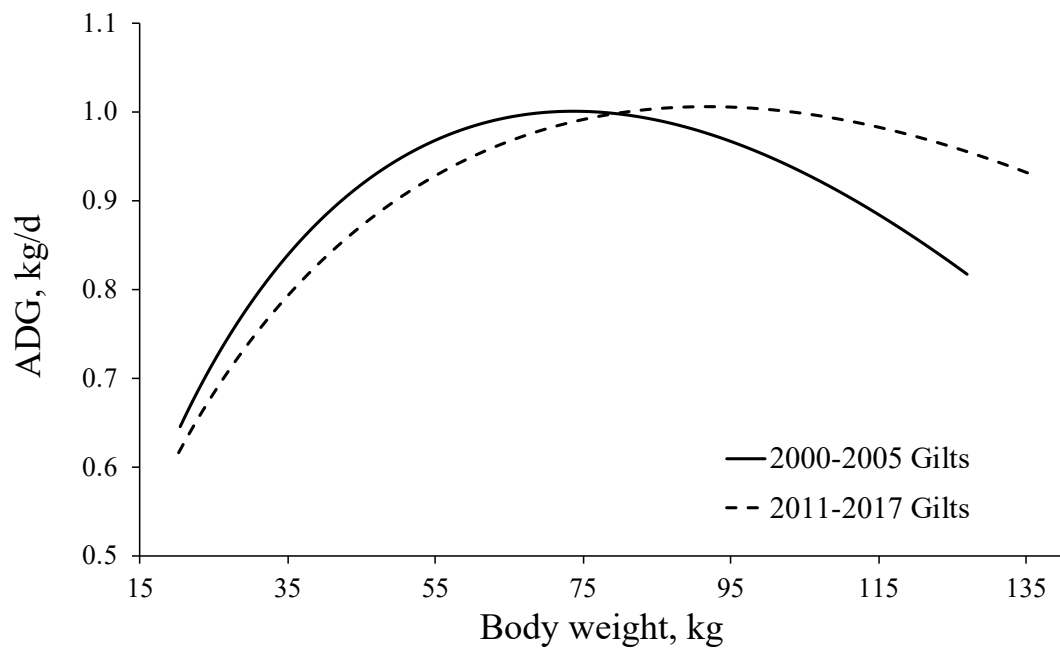
Figure 4.4 Prediction of average daily gain (ADG) based on body weight for gilts. Average daily gain was calculated as the derivative of the generalized Michaelis-Menten (GMM) function to predict BW in kg on time (ADG = ∂WT/∂T; Schinckel et al., 2009a).
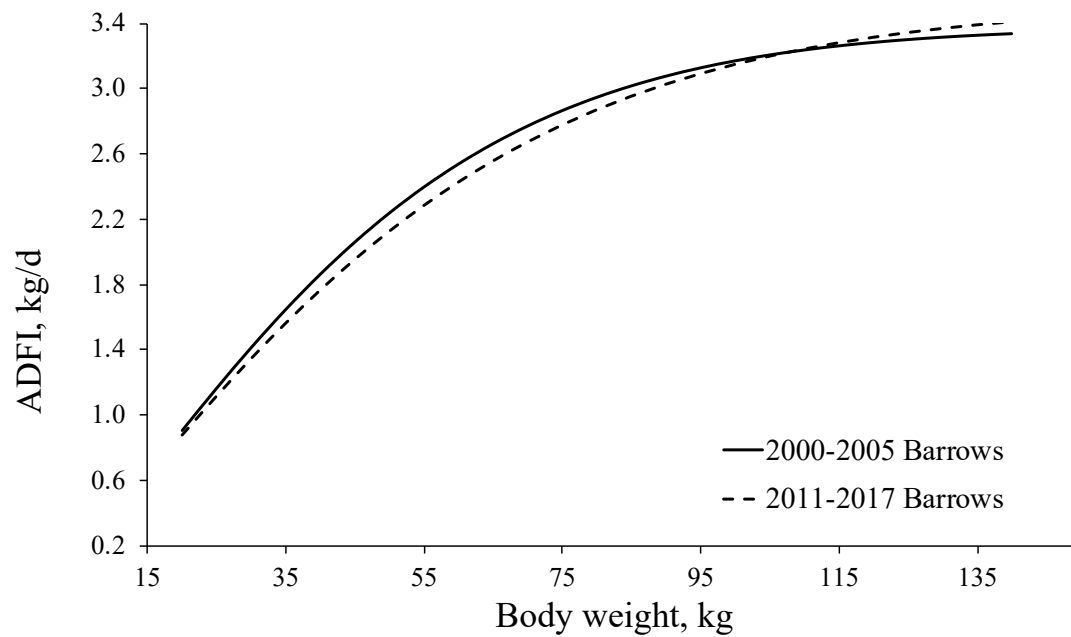
Figure 4.5 Prediction of average daily feed intake (ADFI) based on body weight for barrows. Average daily feed intake (ADFI, kg/d) was predicted using the Bridges function: $DFI_{i,t} = C\{1-\exp[-\exp(M')t^A)]\} + e_{i,t}$, where t is the BW in kg of the ith animal, C is the mean mature daily feed intake (DFI), M' is the log of the exponential growth decay constant, and A is the kinetic order constant (Bridges et al., 1986; Schinckel et al., 2009b).
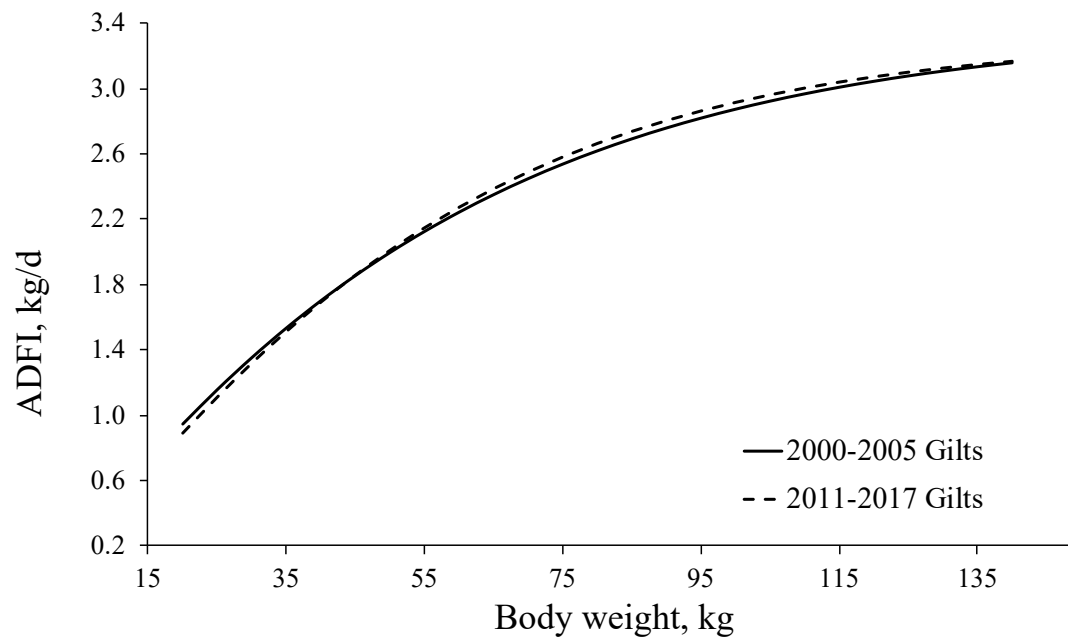
Figure 4.6 Prediction of average daily feed intake (ADFI) based on body weight for gilts. Average daily feed intake (ADFI, kg/d) was predicted using the Bridges function: $DFI_{i,t} = C\{1-exp[-exp(M')t^A)]\} + e_{i,t}$, where t is the BW in kg of the ith animal, C is the mean mature daily feed intake (DFI), M' is the log of the exponential growth decay constant, and A is the kinetic order constant (Bridges et al., 1986; Schinckel et al., 2009b).
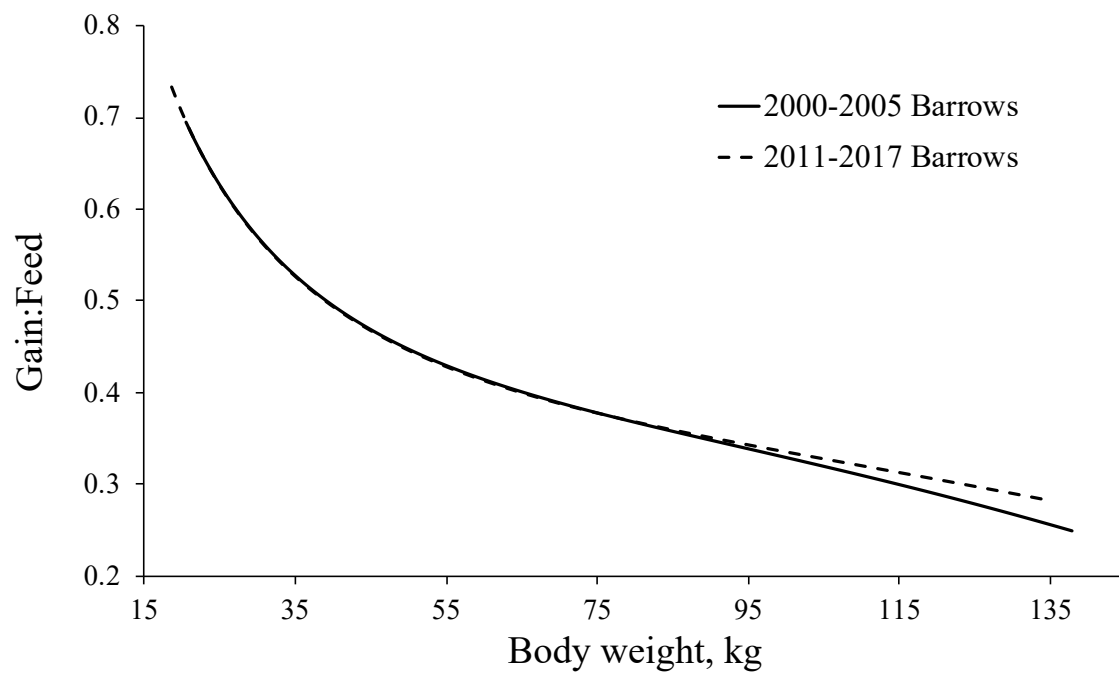
Figure 4.7 Prediction of feed efficiency (Gain:Feed) based on body weight for barrows. Gain:Feed was calculated by dividing ADG by ADFI for each day to produce the Gain:Feed prediction curve based on body weight.
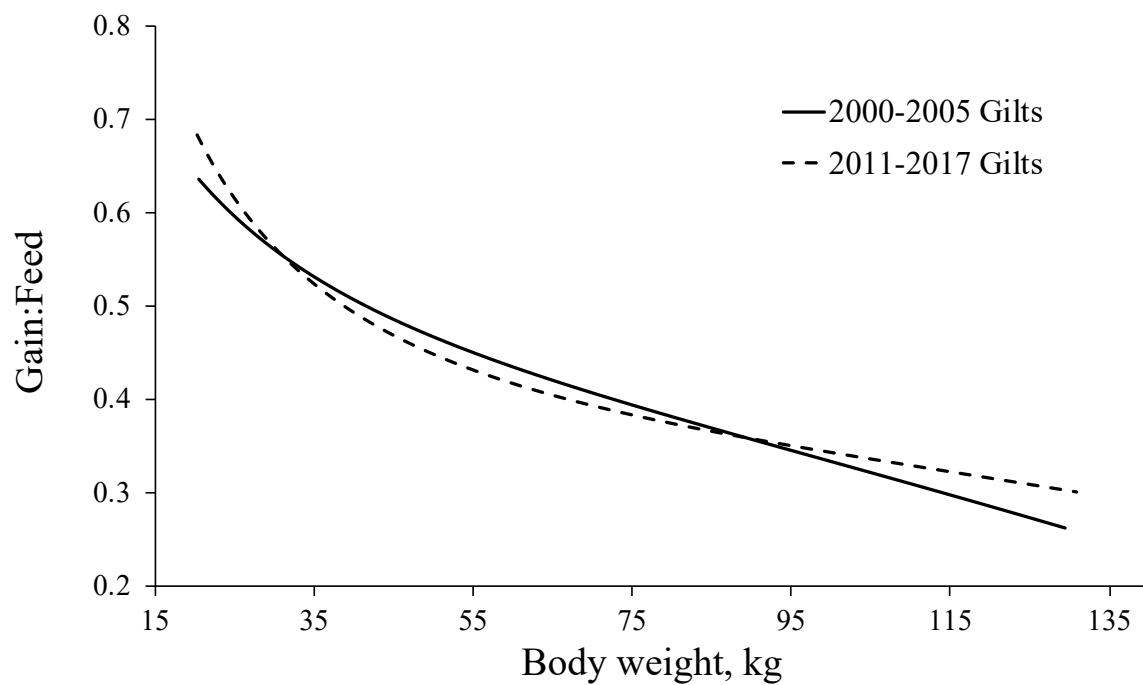
Figure 4.8 Prediction of feed efficiency (Gain:Feed) based on body weight for gilts. Gain:Feed was calculated by dividing ADG by ADFI for each day to produce the Gain:Feed prediction curve based on body weight.
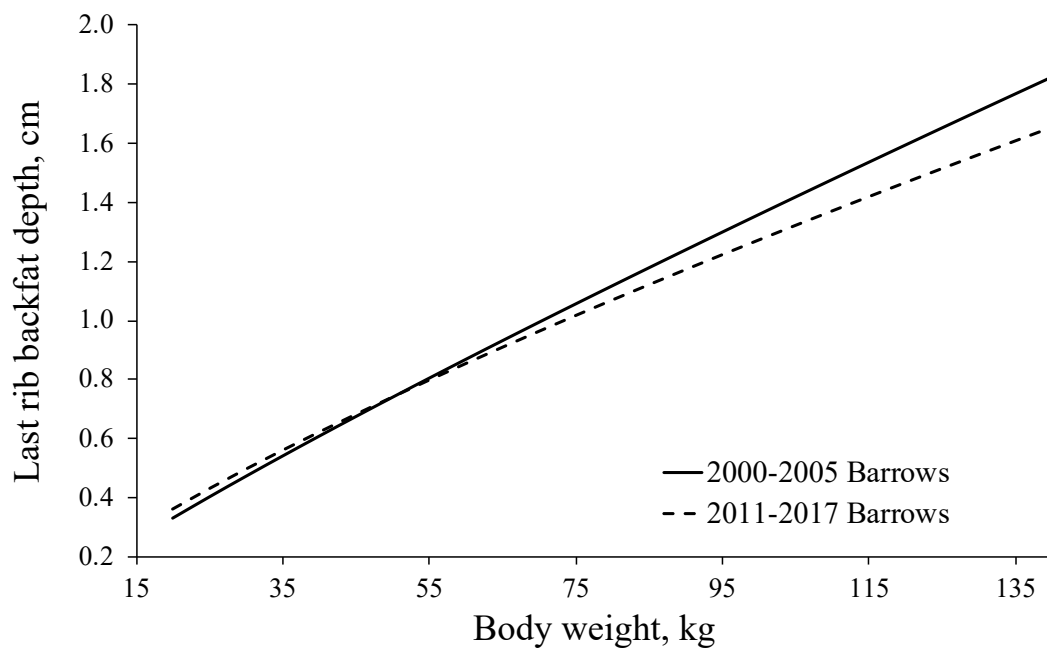
Figure 4.9 Prediction of last rib backfat depth based on body weight for barrows. Live serial ultrasonic last rib backfat depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).
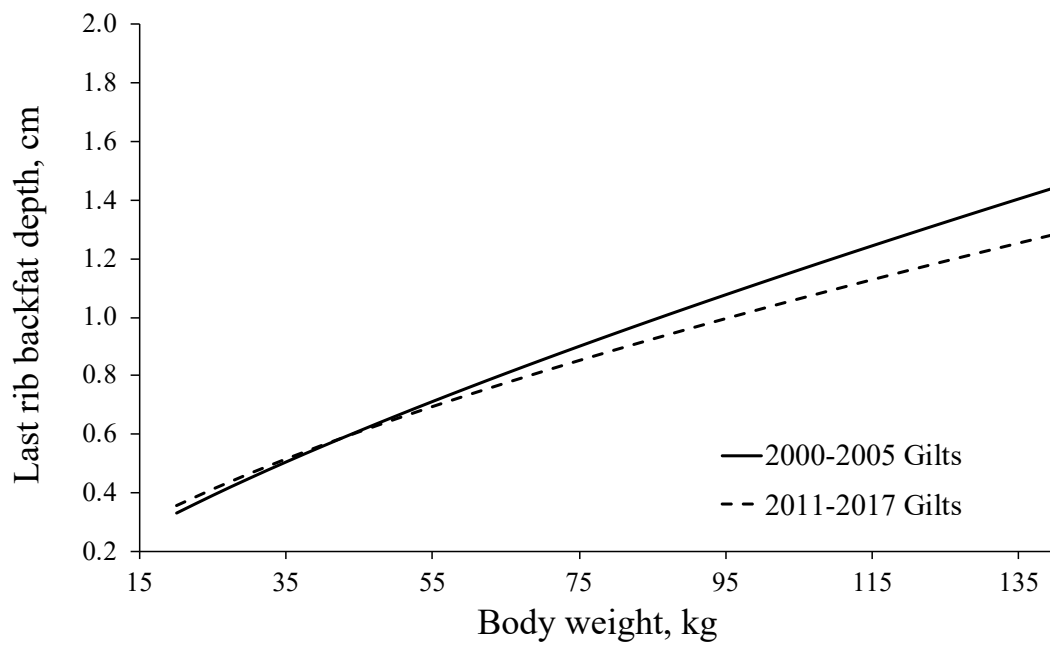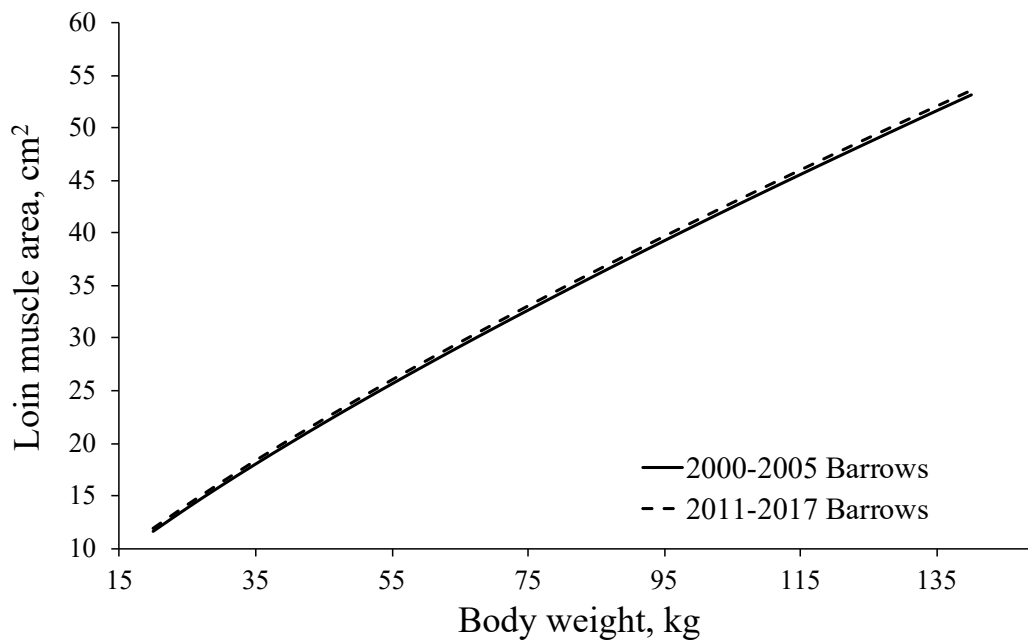
Figure 4.10 Prediction of last rib backfat depth based on body weight for gilts. Live serial ultrasonic last rib backfat depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).
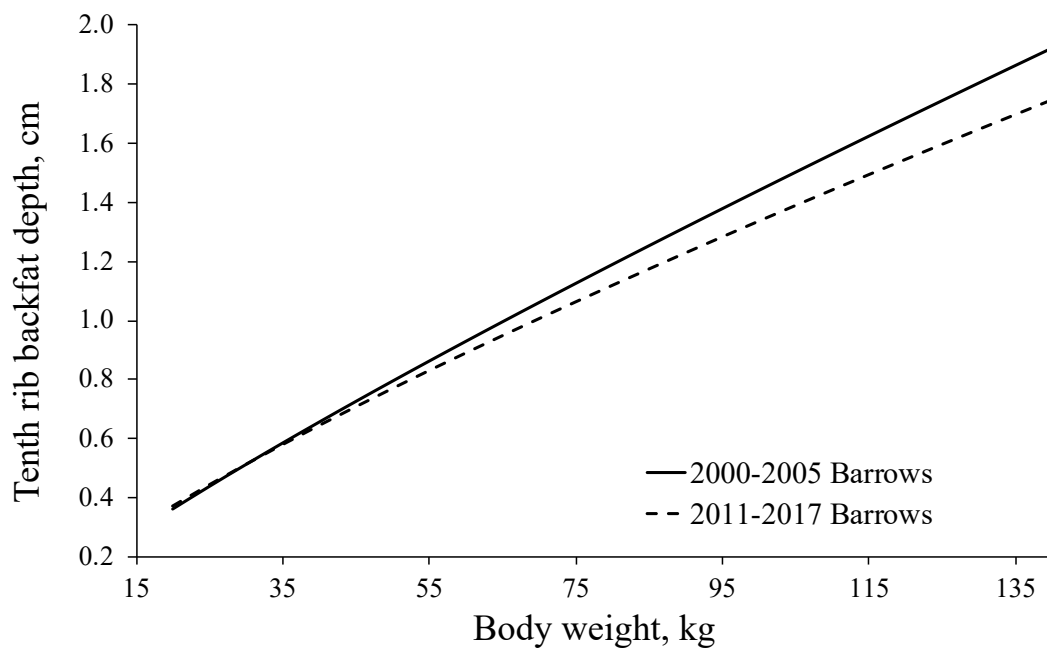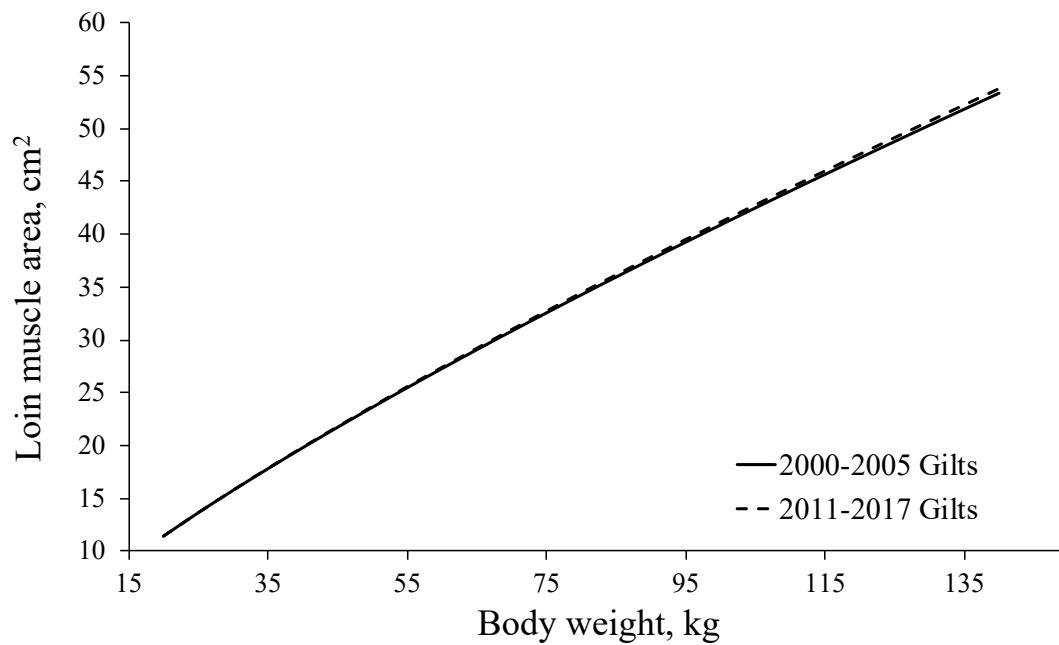
Figure 4.11 Prediction of tenth rib backfat depth based on body weight for barrows. Live serial ultrasonic tenth rib backfat depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).
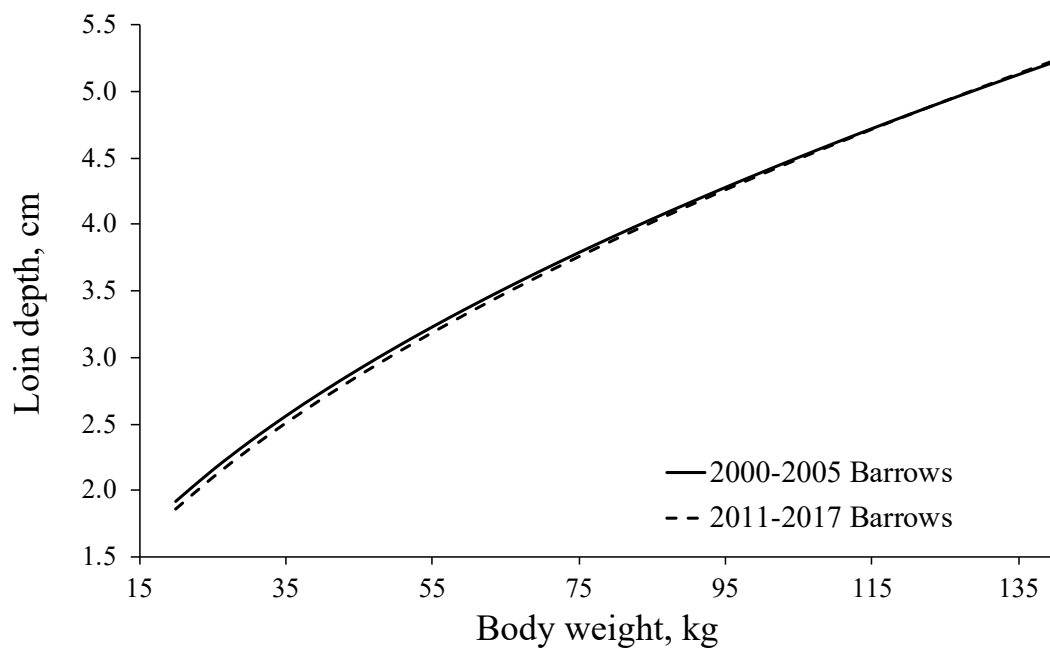
Figure 4.12 Prediction of tenth rib backfat depth based on body weight for gilts. Live serial ultrasonic tenth rib backfat depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).

Figure 4.13 Prediction of loin muscle area based on body weight for barrows. Live serial ultrasonic loin muscle area measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).
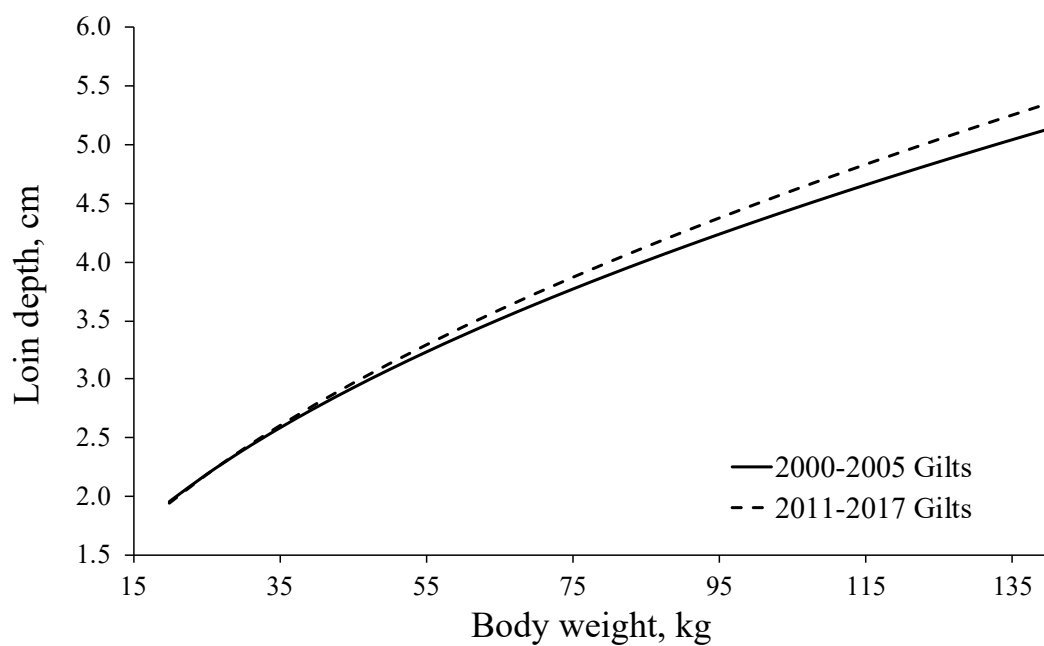
Figure 4.14 Prediction of loin muscle area based on body weight for gilts. Live serial ultrasonic loin muscle area measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).

Figure 4.15 Prediction of loin muscle depth at the tenth rib based on body weight for barrows. Live serial ultrasonic loin depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).

Figure 4.16 Prediction of loin muscle depth at the tenth rib based on body weight for gilts. Live serial ultrasonic loin depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).
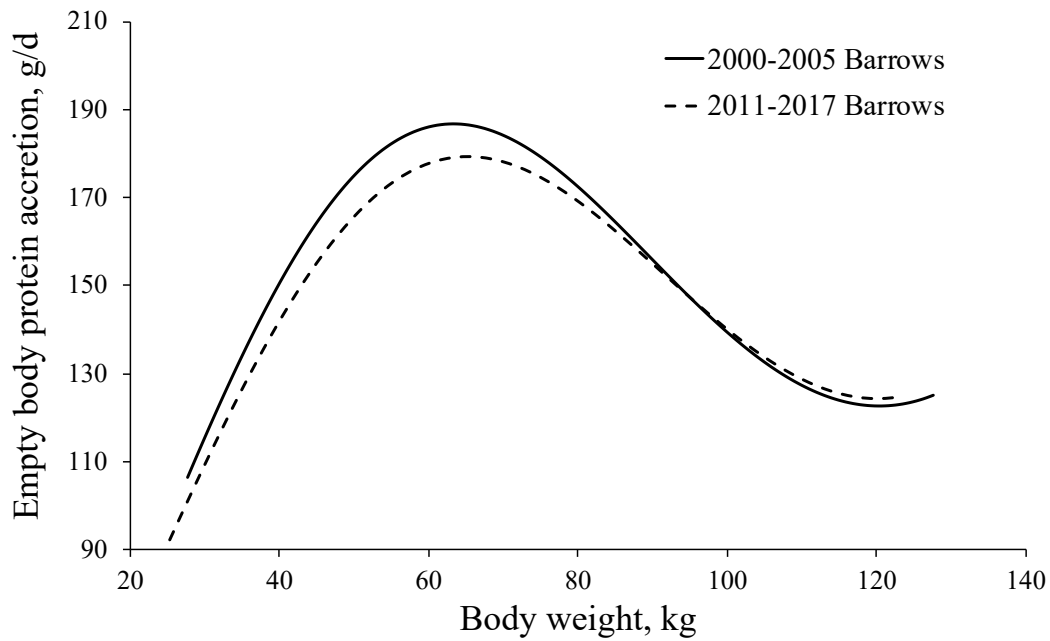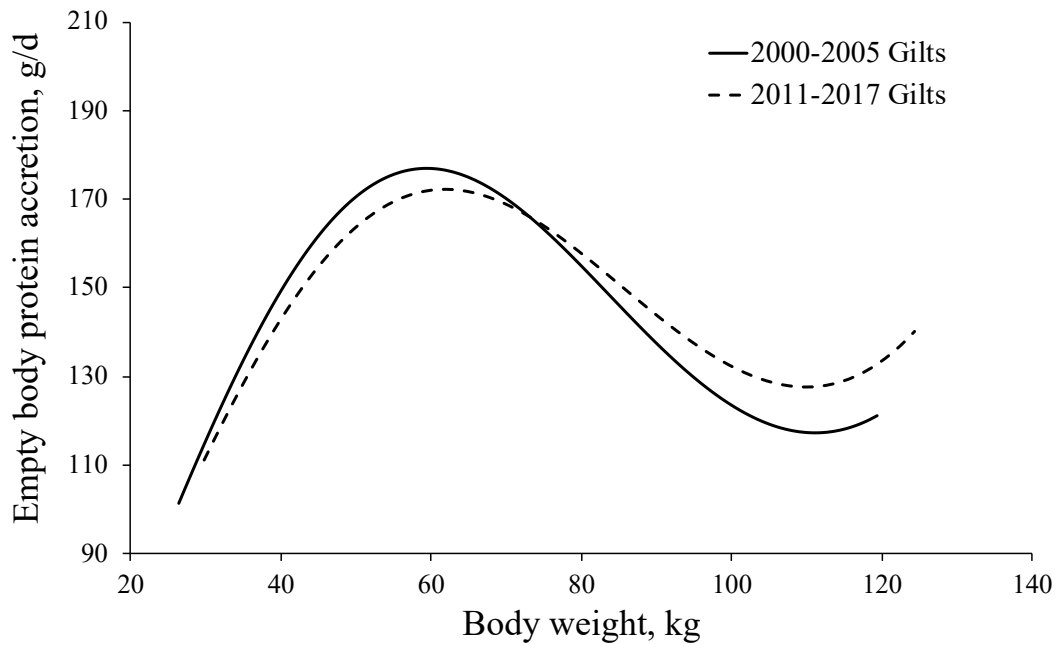
Figure 4.17 Prediction of daily empty body protein accretion rate based on body weight for barrows. An exponential function (Wagner et al., 1999) was used to predict empty body protein accretion: $Y = \exp(b_0 + b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily empty body protein accretion rates were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg.
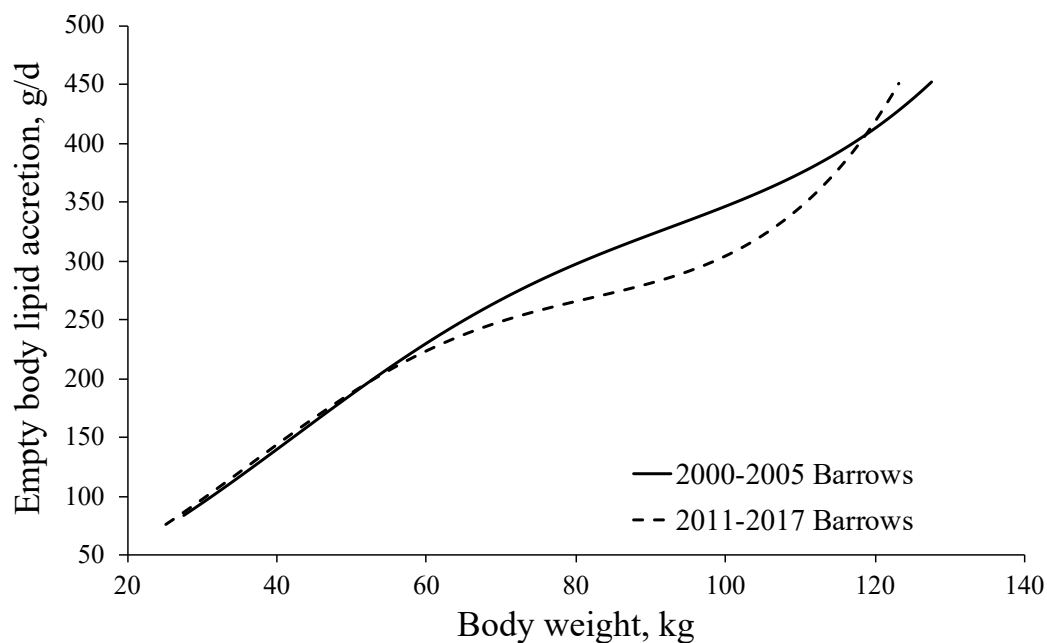
Figure 4.18 Prediction of daily empty body protein accretion rate based on body weight for gilts. An exponential function (Wagner et al., 1999) was used to predict empty body protein accretion: $Y = exp(b_0 + b_1BW + b_2BW^2 + b_3BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily empty body protein accretion rates were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg.
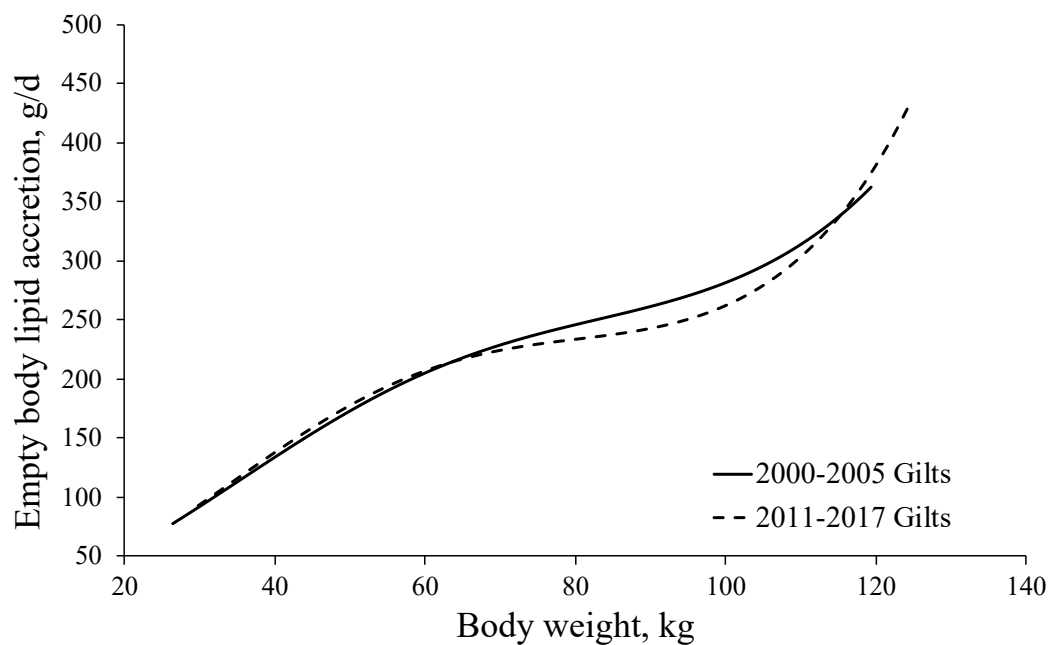
Figure 4.19 Prediction of daily empty body lipid accretion rate based on body weight for barrows. An exponential function (Wagner et al., 1999) was used to predict empty body lipid accretion: $Y = \exp(b_0 + b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily empty body lipid accretion rates were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg.

Figure 4.20 Prediction of daily empty body lipid accretion rate based on body weight for gilts. An exponential function (Wagner et al., 1999) was used to predict empty body lipid accretion: $Y = \exp(b_0 + b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily empty body lipid accretion rates were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg.
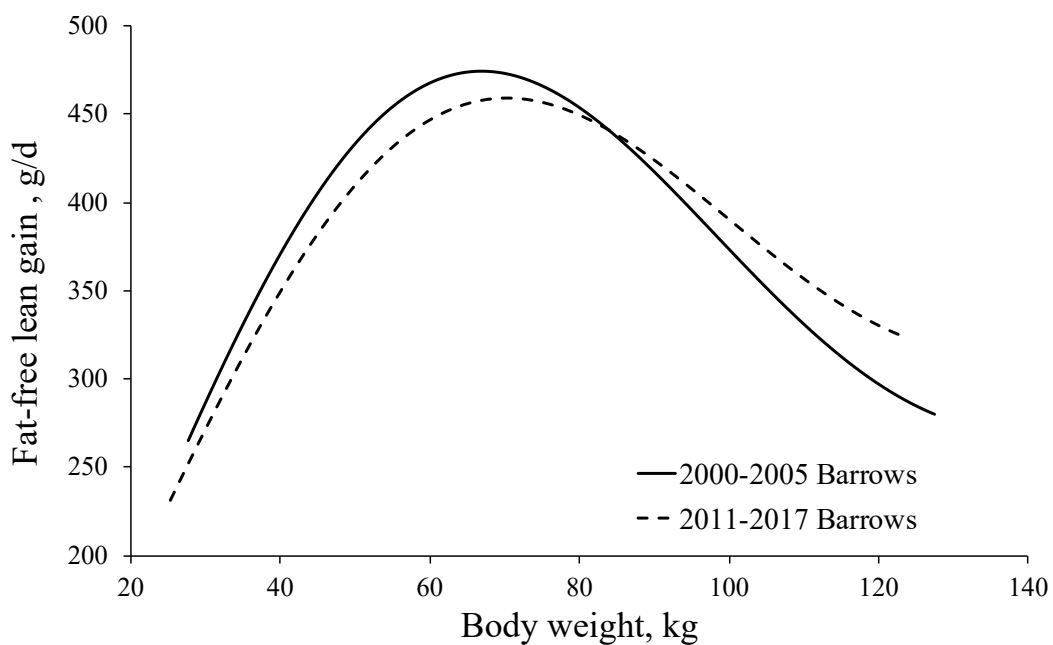
Figure 4.21 Prediction of daily fat-free lean gain based on body weight for barrows. An exponential function (Wagner et al., 1999) was used to predict total fat-free lean gain: $Y = \exp(b_0 + b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily total fat-free lean gain rates were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg.
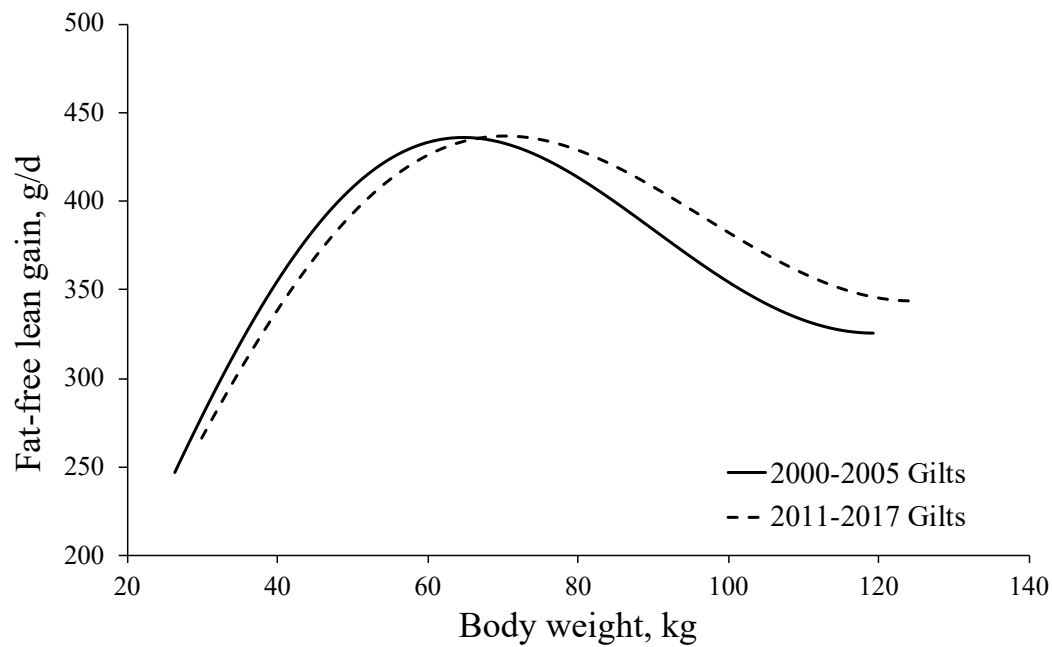
Figure 4.22 Prediction of daily fat-free lean gain based on body weight for gilts. An exponential function (Wagner et al., 1999) was used to predict total fat-free lean gain: $Y = \exp(b_0 + b_1BW + b_2BW^2 + b_3BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily total fat-free lean gain rates were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg.
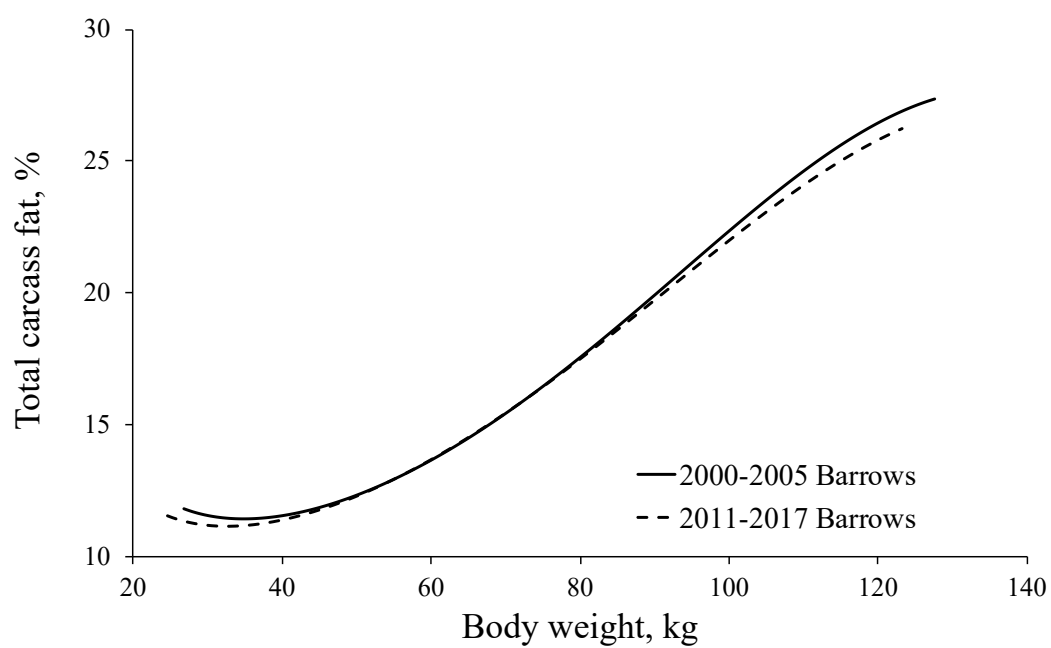
Figure 4.23 Prediction of total carcass fat percentage based on body weight for barrows. An exponential function (Wagner et al., 1999) was used to predict total carcass fat: $Y = \exp(b_0 + b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. The total carcass fat was calculated as a percentage of the live BW in kg.
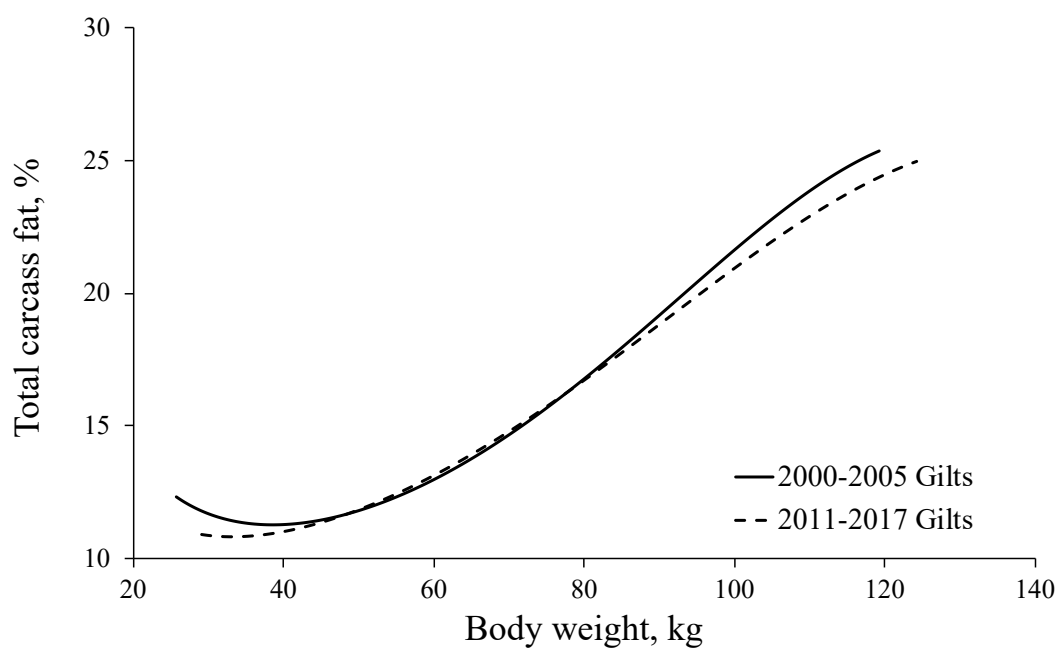
Figure 4.24 Prediction of total carcass fat percentage based on body weight for gilts. An exponential function (Wagner et al., 1999) was used to predict total carcass fat: $Y = \exp(b_0 + b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. The total carcass fat was calculated as a percentage of the live BW in kg.
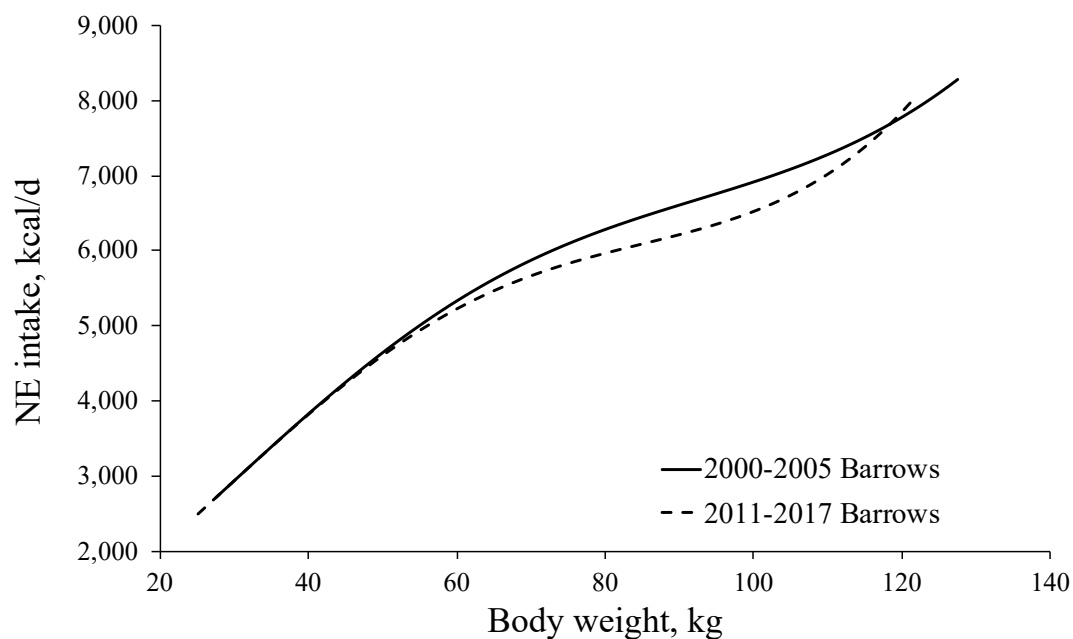
Figure 4.25 Prediction of daily net energy intake based on body weight for barrows. Daily net energy intake was predicted as NE intake, Kcal/d = $((0.179 \times BW^{0.60}) + (5.6863 \times$ Empty body protein accretion, kg/d) + $(9.509 \times$ Empty body lipid accretion, kg/d)) $\times$ 1,000, where $0.179 \times BW^{0.60}$ is the NE required for maintenance, 5.6863 is the energy content of empty body protein accretion, and 9.509 is the energy content of empty body lipid accretion (Noblet et al., 1999).

Figure 4.26 Prediction of daily net energy intake based on body weight for gilts. Daily net energy intake was predicted as NE intake, Kcal/d = $((0.179 \times BW^{0.60}) + (5.6863 \times$ Empty body protein accretion, kg/d) + $(9.509 \times$ Empty body lipid accretion, kg/d)) $\times 1,000$, where $0.179 \times BW^{0.60}$ is the NE required for maintenance, 5.6863 is the energy content of empty body protein accretion, and 9.509 is the energy content of empty body lipid accretion (Noblet et al., 1999).
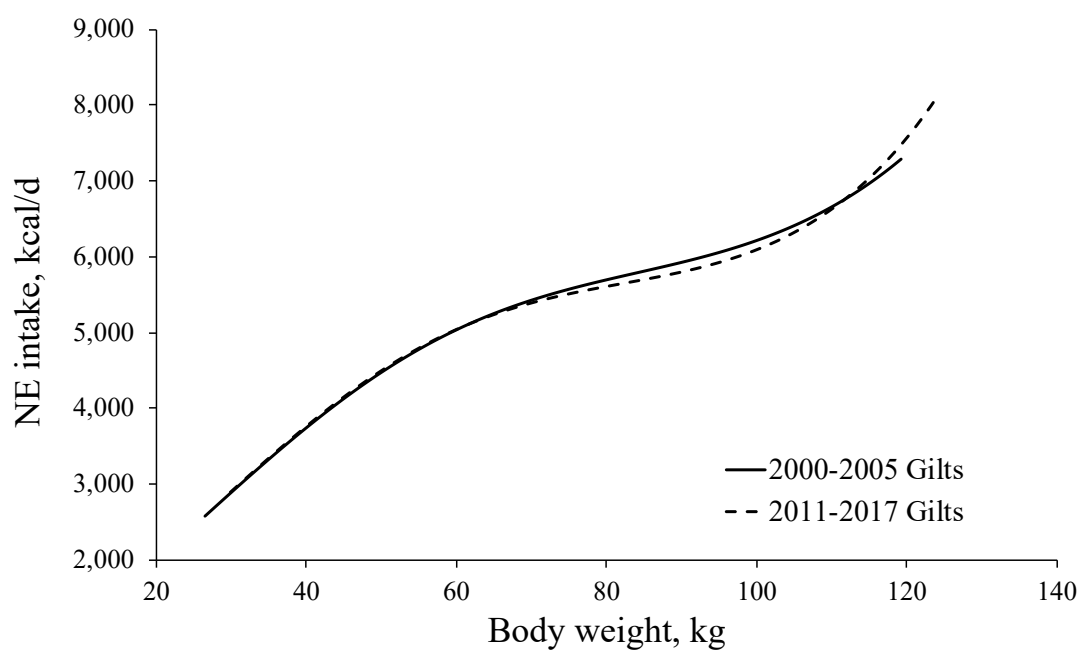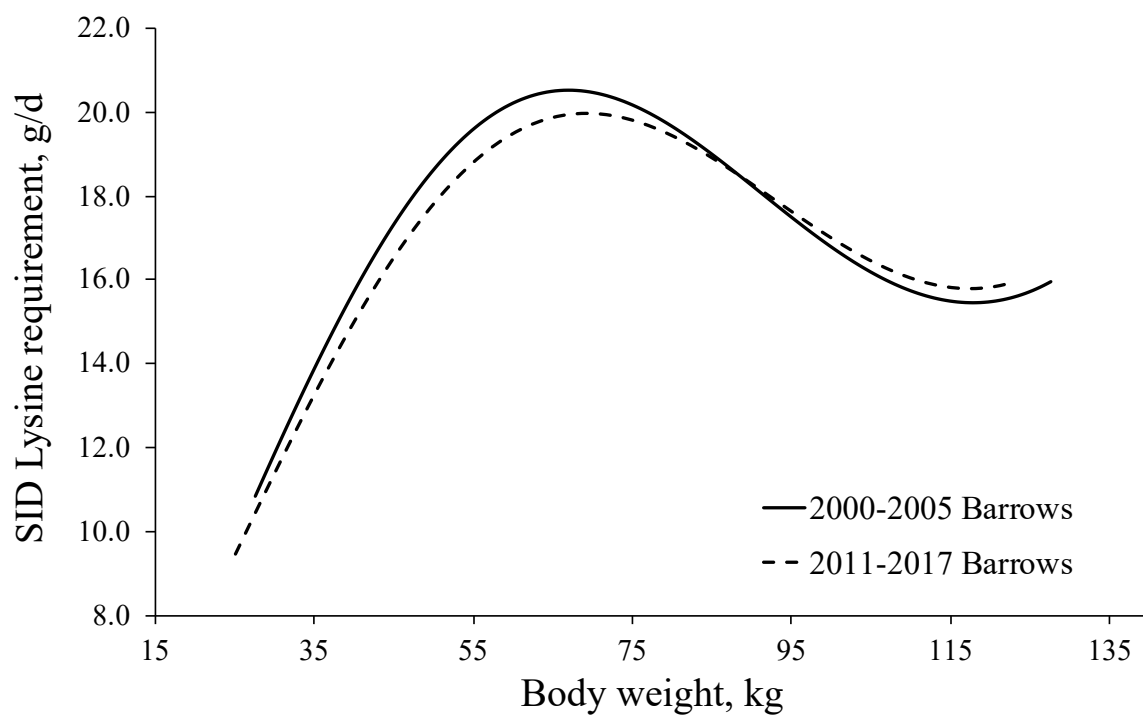
Figure 4.27 Prediction of standardized ileal digestible (SID) lysine requirement based on body weight for barrows. The SID lysine requirement was estimated using the following equation: SID lysine requirement, g/d = SID lysine requirement for protein deposition (Pd), g/d + endogenous lysine losses, g/d + integument lysine losses, g/d. The SID lysine requirement for Pd, g/d = {Lysine retained in Pd / [0.75 + 0.002 × (maximum Pd − 147.7)]} × (1 + 0.0547 + 0.002215 × BW, kg). Endogenous lysine losses, g/d = feed intake × (0.417/1000) × 0.88 × 1.1. Integument lysine losses, g/d = 0.0045 × BW$^{0.75}$ (NRC, 2012).
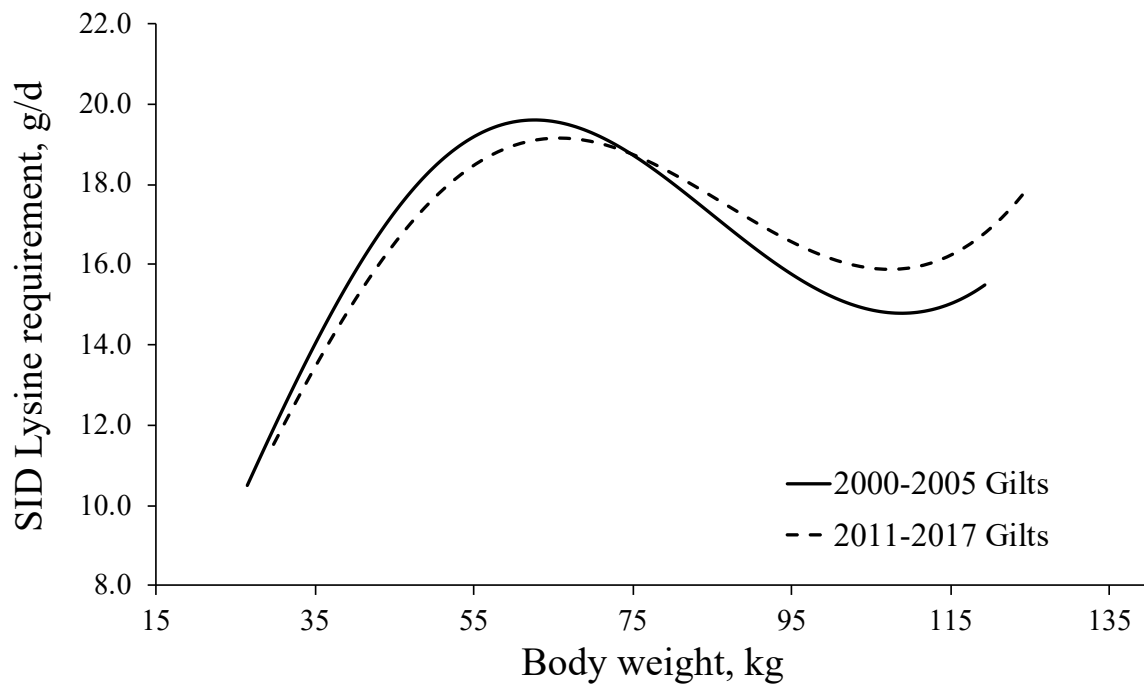
Figure 4.28 Prediction of standardized ileal digestible (SID) lysine requirement based on body weight for gilts. The SID lysine requirement was estimated using the following equation: SID lysine requirement, g/d = SID lysine requirement for protein deposition (Pd), g/d + endogenous lysine losses, g/d + integument lysine losses, g/d. The SID lysine requirement for Pd, g/d = {Lysine retained in Pd / [0.75 + 0.002 × (maximum Pd – 147.7)]} × (1 + 0.0547 + 0.002215 × BW, kg). Endogenous lysine losses, g/d = feed intake × (0.417/1000) × 0.88 × 1.1. Integument lysine losses, g/d = 0.0045 × BW$^{0.75}$ (NRC, 2012).

Figure 4.29 Prediction of standardized ileal digestible (SID) lysine requirement:Net energy (NE) in g/Mcal based on body weight for barrows (Noblet et al., 1999; NRC, 2012). The SID Lysine:NE prediction was calculated by dividing the previously predicted SID lysine, g/d, by the previously predicted NE intake, Mcal/d (Mcal/d = kcal/d × 1,000) for a given BW in kg.
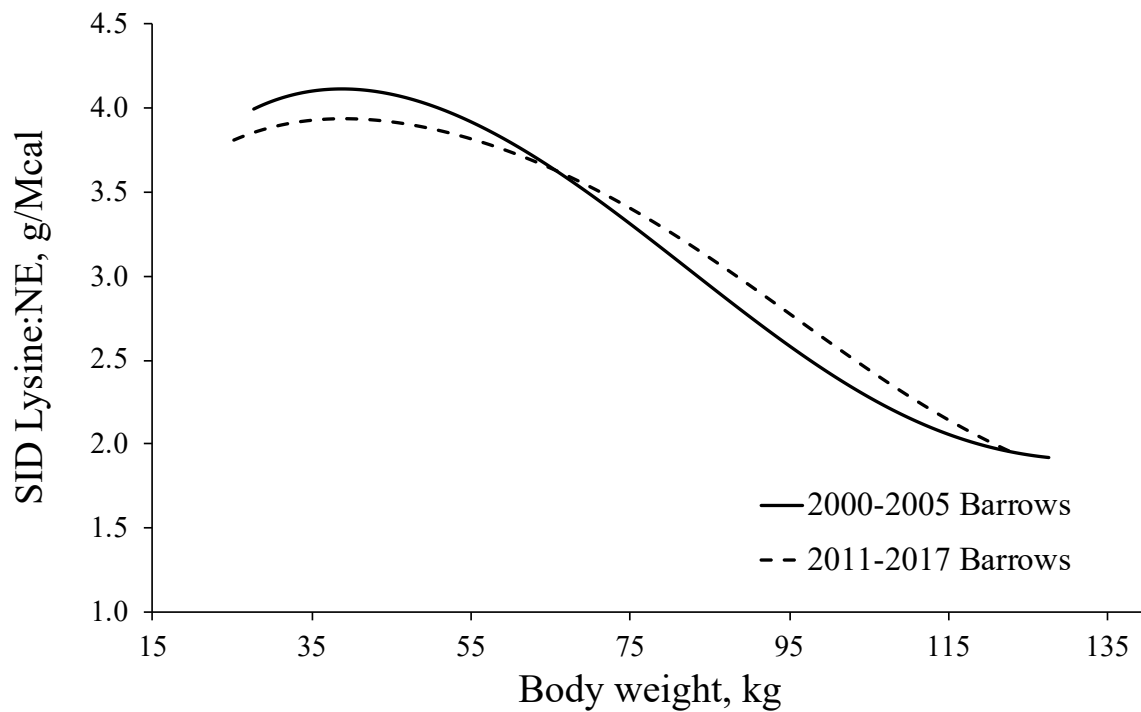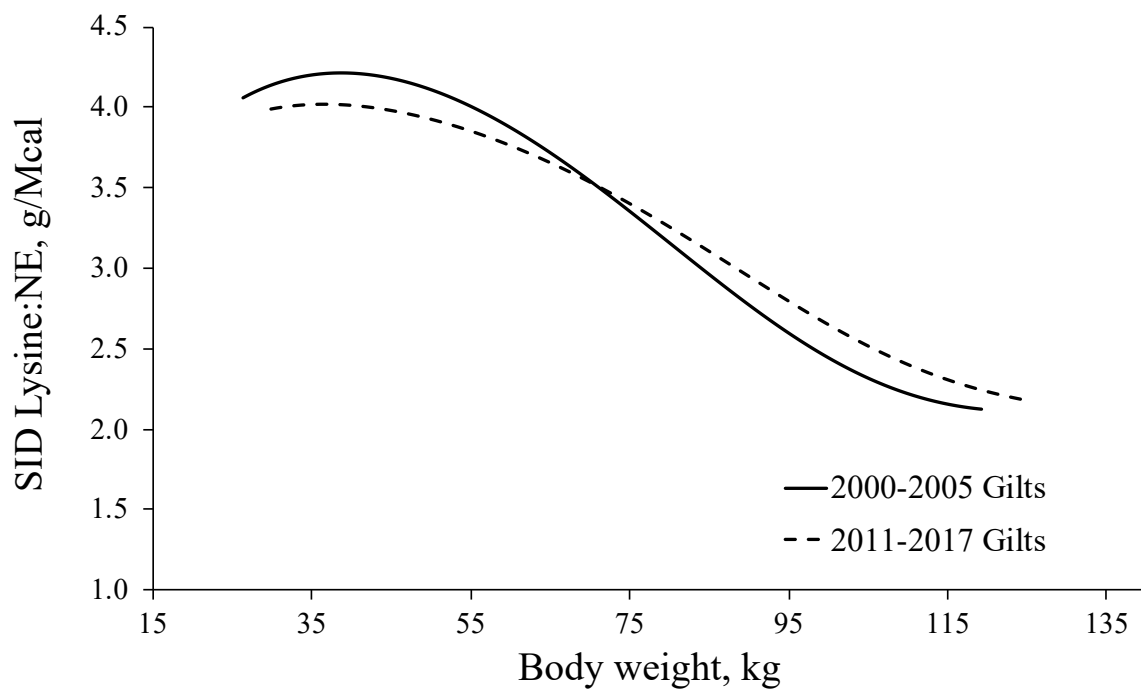
Figure 4.30 Prediction of standardized ileal digestible (SID) lysine requirement:Net energy (NE) in g/Mcal based on body weight for gilts (Noblet et al., 1999; NRC, 2012). The SID Lysine:NE prediction was calculated by dividing the previously predicted SID lysine, g/d, by the previously predicted NE intake, Mcal/d (Mcal/d = kcal/d × 1,000) for a given BW in kg.

Figure 4.31 Prediction of standardized ileal digestible (SID) lysine requirement as a percent of diet based on body weight for barrows (NRC, 2012). The SID lysine requirement as a percent of the diet was calculated by dividing the previously predicted SID lysine requirement, g/d, by the previously predicted ADFI, g/d, times 100.
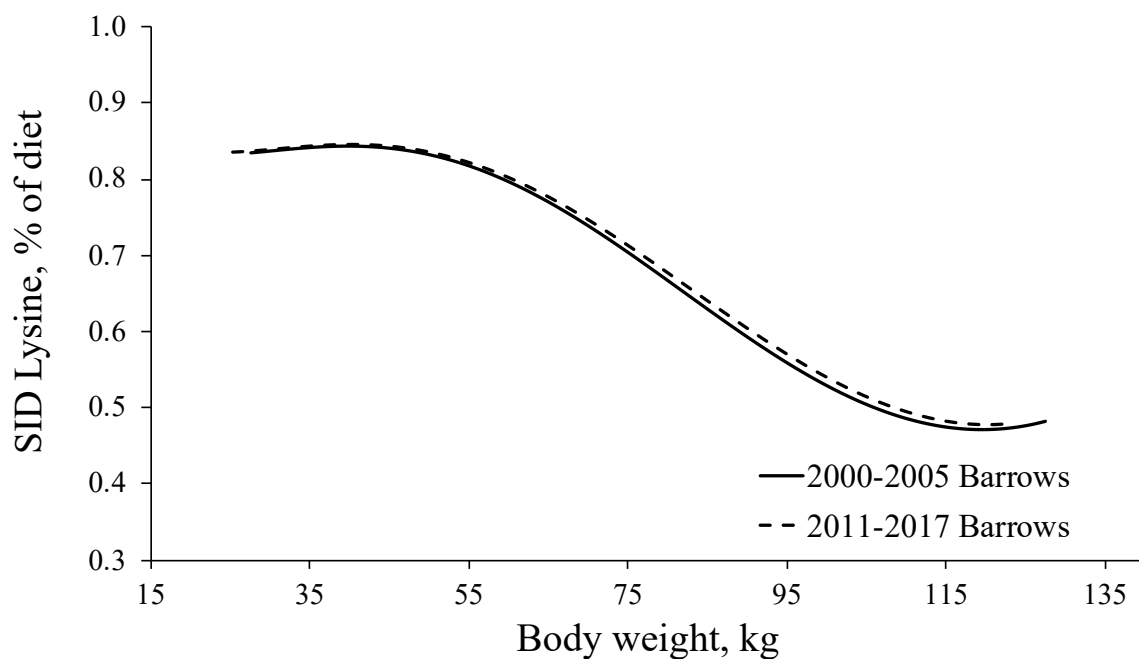
Figure 4.32 Prediction of standardized ileal digestible (SID) lysine requirement as a percent of diet based on body weight for gilts (NRC, 2012). The SID lysine requirement as a percent of the diet was calculated by dividing the previously predicted SID lysine requirement, g/d, by the previously predicted ADFI, g/d, times 100.
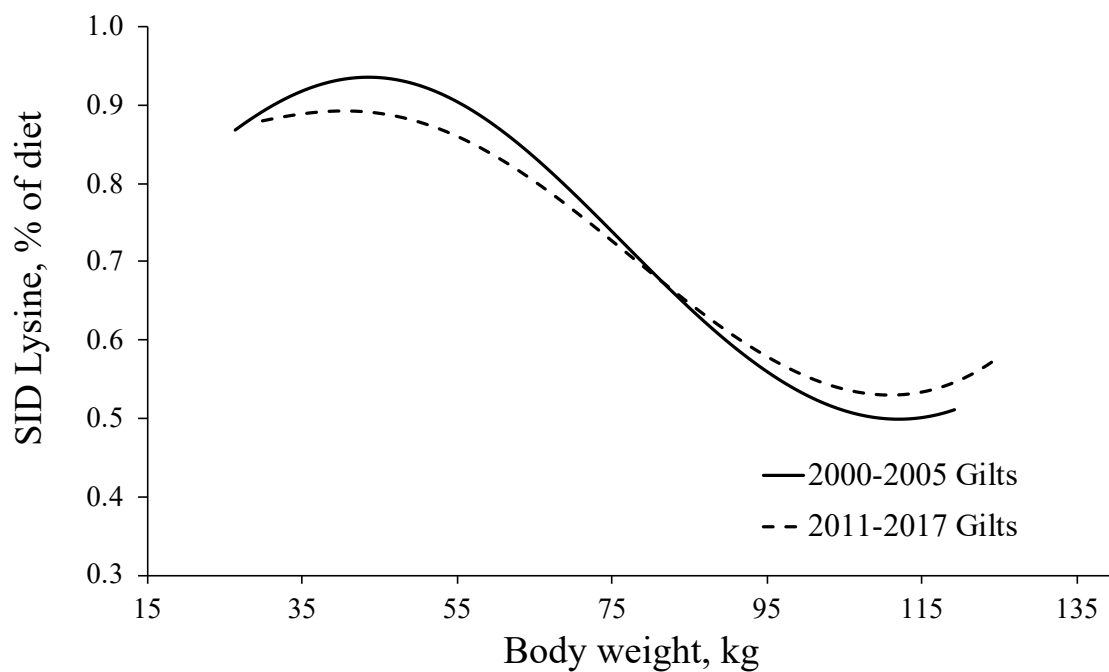
CHAPTER 5. EFFECTS OF SOW GUT MODIFYING FEED ADDITIVES ON REPRODUCTIVE CHARACTERISTICS AND PROGENY GROWTH PERFORMANCE: CONCLUSIONS AND FUTURE DIRECTION

5.1 Conclusions

Feeding gestating and lactating sows a proprietary strain of *Pichia guilliermondi* as a whole-cell inactivated yeast (WCY) product from d 35 of gestation through lactation increased the number of piglets born and weaned by about one-half of a pig. However, pigs born to WCY sows had lighter weaning weights compared to CON which may be due to their increased litter size, nearly 1 day shorter lactation, and potential limits of this sow's genetics or nutrition fed to meet the increased milk demand of the increased litter size. The percent of litters treated for scours decreased dramatically due to feeding WCY, adding to the economic, labor savings, and piglet welfare potential benefits of feeding WCY.

Feeding a *Bacillus licheniformis* DFM to sows from d 80 of gestation through a 21-d lactation may decrease pig born alive weight and subsequent weaning weight but reduce sow BW loss through 6.4% more lactation feed intake, quickening the return to estrus. Other than decreasing the number of mummies per litter, feeding the OA alone or in combination did not improve sow reproductive or litter growth performance.

Feeding antibiotics to nursery pigs greatly improved growth performance. Feeding DFM or OA to sows and/or their offspring may improve nursery feed efficiency but did not result in a difference in ADG or final BW. Feeding the combination diet (DFM+OA) to the sow and nursery pigs tended to increase ADFI. However, feeding the DFM+OA combination diet to sows and nursery pigs may reduce the need for therapeutic treatments post-weaning when feeding antibiotic free diets. Also, there was no additive benefit to feeding DFM and/or OA to nursery pigs in

addition to their dams from d 80 of gestation through lactation. The intestinal histology of the jejunum in progeny harvested 6 days post-weaning did not provide supporting evidence to feed the DFM or OA to nursery pigs for improved gut function. Feeding the DFM or OA to sows and/or nursery pigs did result in some differences in gene expression for immune and gut barrier function indicators but further research is needed to determine the true relevancy of the differences and impacts on gut health.

Lastly, using frozen semen allowed the genetics of two different time periods of sires and sex of the progeny to be evaluated. The expected large growth performance differences indicated by the terminal sire indexes of the two genetic groups, 88.2 and 112.0 for 2000 to 2005 and 2011 to 2017, respectively, were not observed. However, barrows had greater feed intake and fatter carcasses than the more feed efficient and leaner gilts. Modern swine genetics have been selected to be leaner and results from this study agree, although the differences in live scan and carcass measurements were not as large as expected. One potential reason for the lack of genetic differences could be that these pigs were housed in near ideal conditions (low stocking density and no heat stress) and fed unlimiting diets which may have been different than the sires' rearing conditions that determined their terminal sire index.

5.2   Future Direction

Future research of feed additive technologies will likely not be paired with in feed antibiotics or pharmacological levels of zinc and copper. Antibiotics used for growth promotion in swine has already been eliminated in the U.S. effective January 2017 (GFI #213; U.S. Food and Drug Administration, 2020). It is also likely that the regulations made in the European Union (EU) on zinc and copper use will eventually be adopted in the U.S. As of August 13th, 2019, the EU

decreased the maxiumum level of copper in pig diets up to 4 weeks post-weaning from 170 to 150 mg/kg with further reductions expected in the future (Regulation (EU) 2018/1039). Additionally, the EU is banning the use of medicinal levels of zinc oxide by June 2022 (Directive 2001/82/EC on veterinary medicinal products + Regulation (EC) No 726/2004). Therefore, future research on feed additives fed to nursery pigs should consider formulating a basal diet without antibiotics and without pharmacological levels of zinc and copper. In doing so, the true potential of the feed additive will be investigated, and results will more clearly translate to feeding pigs in the future when more restrictive regulations are implemented.

While we will be better prepared for the future with this philosophy, it is also important to conduct research on newly developed technologies within the current guidelines and regulations. Producers and nutritionists need to test new technologies within the producer's specific production system using current formulations that may still contain higher levels of zinc and copper. As technologies consistently show improvements in desired production criteria, producers should consider modeling the response to the technology and conducting a detailed economic evaluation to support the risk of replacing an existing technology or adding a new one before making the decision to adopt the new technology.

The large number of sows used in the WCY sow study provides quite a bit of confidence in the observed results. Additionally, the experiment was conducted in a commercial production system with approximately 300 sows per treatment and therefore results will translate more closely to swine producers in the industry than if the study were conducted on a smaller scale. In the future, research could be done to investigate the mode of action for the *Pichia guilliermondi* WCY specifically concerning the increased number of pigs born alive. Sows weren't fed the WCY until after d 35 of gestation. Therefore, it is logical to assume the CON and WCY sows had similar

ovulation rates and conceptus implantation success because both of these events occur before d 35 of gestation. Future research could focus on the impacts of WCY (β-glucans, mannan-oligosaccharides, and other undiscovered active *Pichia guilliermondi* culture components) on the uterine environment and uterine capacity which may influence the number of pigs born alive. Research techniques used may include harvesting sows at multiple time points throughout gestation and evaluating embryonic and fetal viability and growth. In addition, researchers could histologically study the uterine and placental interface to evaluate placental surface area and transportation of histrotroph from uterine glands through the placental areolae and fetal capillaries into fetal target tissues. Histotroph composition (enzymes, growth factors, cytokines, nutrients, and other regulatory molecules) may also be evaluated by tools like proteomics to investigate potential composition differences due to feeding the sow WCY.

There has been quite a bit of research around different species, inclusion levels, and efficacies of DFMs on the market as well as combinations of OA and specifically, MCFA. I expect this research to continue as some results have shown these feed additives to be potential replacements of antibiotics under certain conditions. Specifically, for the *Bacillus licheniformis* DFM fed to sows in Chapter 3, more research with a larger number of sows is necessary to investigate the potential increase in total born, increased lactation feed intake, reduced BW loss, and quickened the return to estrus interval. If these are confirmed in a large-scale study, investigation of the microbiome may be valuable to evaluate which microorganisms may be positively impacting these responses through mechanisms potentially involving microbial secretions or advancements in immune stimulation and function.

The expected large growth performance differences indicated by the terminal sire indexes of the two genetic groups, 88.2 and 112.0 for 2000 to 2005 and 2011 to 2017, respectively, were

not observed. Future and continued research is needed to validate terminal sire indexes across generations to allow for a more robust evaluation in order to support genetic selection. Additionally, the role of nutrition provided to the different groups of pigs would be interesting to evaluate. This study fed 110% of the nutrients recommended for the highest lean accretion in the 2012 NRC. The intention was to provide nutrients, and pen space, in excess in order to eliminate nutritional and crowding restrictions on the pigs to allow them to express their true genetic potential. However, research has shown that adding too much protein in a pig's diet has a metabolic cost and therefore we may have artificially reduced the differences in fat deposition between genetic groups by overfeeding protein to these pigs.

5.3 Literature Cited

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APPENDIX

Table A.1 Model parameter estimates and standard errors[1]

parameter estimates (standard error)	2000-2005		2011-2017	
	Barrows	Gilts	Barrows	Gilts
Live weight,[2,3] kg				
C	2.7243 (0.02426)	2.6166 (0.02214)	2.5195 (0.04486)	2.2244 (0.06276)
K	169.59 (4.4350)	179.42 (4.7074)	201.25 (14.5038)	221.98 (15.1408)
WF	238.25 (6.0598)	239.55 (6.6572)	291.12 (18.9932)	327.34 (30.6525)
residual variance	3.2757 (0.2215)	2.9857 (0.1942)	4.9245 (0.5542)	9.8669 (1.0578)
Variance (wf_i)	882.51 (229.81)	1250.77 (298.16)	2644.68 (1432.96)	1933.55 (773.41)
Covariance (wf_i, k_i)	512.12 (174.66)	807.09 (235.20)	2216.80 (1285.74)	N/A
Variance (k_i)	670.06 (168.83)	801.55 (225.23)	2613.32 (1197.67)	N/A
ADFI,[4] kg/d				
A	1.3586 (0.04016)	1.0962 (0.04091)	1.2767 (0.05372)	1.1956 (0.08494)
M	-5.2305 (0.1302)	-4.3964 (0.1073)	-5.0690 (0.1582)	-4.7402 (0.2437)
C	3.3758 (0.08295)	3.3644 (0.1329)	3.5232 (0.1297)	3.2981 (0.2155)
residual variance	0.02136 (0.002758)	0.02470 (0.002998)	0.01602 (0.003062)	0.02814 (0.006370)
Variance (a_i)	0.05822 (0.02336)	0.03738 (0.01595)	0.02084 (0.01417)	0.04746 (0.03579)
Last rib backfat,[5] cm				
A	0.02427 (0.006170)	0.03486 (0.006589)	0.03541 (0.005880)	0.05101 (0.009154)
B	0.8742 (0.05344)	0.7530 (0.04031)	0.7777 (0.03534)	0.6522 (0.03930)
residual variance	0.02648 (0.003605)	0.01924 (0.002517)	0.01591 (0.002608)	0.01598 (0.002507)
Variance (a_i)	0.000019 (0.000015)	0.000020 (0.000013)	0.000019 (0.000014)	0.000030 (0.000022)
Tenth rib backfat,[5] cm				
A	0.02783 (0.005792)	0.03777 (0.006978)	0.03401 (0.006172)	0.04500 (0.008022)
B	0.8569 (0.04365)	0.7494 (0.03941)	0.7972 (0.03883)	0.6905 (0.03896)
residual variance	0.02725 (0.003698)	0.02033 (0.002649)	0.01808 (0.003029)	0.01577 (0.002475)
Variance (a_i)	0.000025 (0.000016)	0.000020 (0.000013)	0.000010 (0.000010)	0.000023 (0.000017)

parameter estimates (standard error)	2000-2005		2011-2017	
	Barrows	Gilts	Barrows	Gilts
Tenth rib loin muscle area,[5] cm^2				
A	1.1367 (0.07135)	1.0716 (0.05806)	1.1740 (0.07616)	1.0552 (0.06520)
B	0.7781 (0.01317)	0.7907 (0.01134)	0.7730 (0.01373)	0.7954 (0.01333)
residual variance	3.9686 (0.5663)	3.0351 (0.3870)	3.2514 (0.5300)	2.9234 (0.4573)
Variance (a_i)	0.004555 (0.001534)	0.005609 (0.001487)	0.004684 (0.002028)	0.002258 (0.000989)
Tenth rib loin muscle depth,[5] cm				
A	0.4111 (0.02117)	0.4421 (0.01902)	0.3813 (0.02335)	0.4088 (0.01895)
B	0.5145 (0.01092)	0.4961 (0.009116)	0.5299 (0.01325)	0.5205 (0.009955)
residual variance	0.04950 (0.006790)	0.03643 (0.004627)	0.05115 (0.008209)	0.03056 (0.004830)
Variance (a_i)	0.000418 (0.000143)	0.000575 (0.000166)	0.000344 (0.000157)	0.000443 (0.000190)
Empty body protein Accretion,[6] kg				
Intercept, b_0	0.17569 (0.03034)	0.06082 (0.02984)	0.19393 (0.03559)	0.12114 (0.03629)
b_1	0.05359 (0.00141)	0.0594 (0.00146)	0.05267 (0.00168)	0.05699 (0.00178)
b_2	-0.0003845 (0.00001843)	-0.00046036 (0.00002038)	-0.00037125 (0.00002242)	-0.00042953 (0.00002466)
b_3	0.00000106 (7.23578E-8)	0.00000136 (8.480576E-8)	0.000001 (8.93693E-8)	0.00000124 (1.01886E-7)
Empty body lipid accretion,[6] kg				
Intercept, b_0	-0.57461 (0.10774)	-0.70746 (0.08922)	-0.92460 (0.11879)	-1.09902 (0.10125)
b_1	0.06393 (0.00463)	0.07116 (0.00389)	0.07920 (0.00537)	0.08581 (0.00477)
b_2	-0.00040092 (0.00005810)	-0.00051287 (0.00005040)	-0.00059683 (0.00006999)	-0.00068177 (0.00006465)
b_3	0.00000113 (2.21387E-7)	0.0000016 (1.998377E-7)	0.00000187 (2.74426E-7)	0.00000219 (2.63072E-7)
Total fat-free lean,[6] kg				
Intercept, b_0	1.35833 (0.04914)	1.34524 (0.05284)	1.39822 (0.04904)	1.42058 (0.04831)
b_1	0.04561 (0.00211)	0.04761 (0.00230)	0.04433 (0.00222)	0.04446 (0.00228)
b_2	-0.00029361 (0.00002650)	-0.00032927 (0.00002985)	-0.00027971 (0.00002889)	-0.00028575 (0.00003085)
b_3	0.0000007273 (1.00980E-7)	0.0000009059 (1.183556E-7)	0.000000684 (1.13284E-7)	0.000000726 (1.25523E-7)

parameter estimates (standard error)	2000-2005		2011-2017	
	Barrows	Gilts	Barrows	Gilts
Total carcass fat,[6] kg				
Intercept, b_0	0.44327	0.67620	0.28338	0.27972
	(0.13363)	(0.14004)	(0.15475)	(0.13214)
b_1	0.02429	0.01331	0.03044	0.02934
	(0.00575)	(0.00611)	(0.00699)	(0.00623)
b_2	0.00010663	0.00024276	0.00003297	0.0000425
	(0.00007206)	(0.00007911)	(0.00009118)	(0.00008438)
b_3	-8.30347E-7	-0.00000136	-5.66108E-7	-5.98529E-7
	(2.74582E-7)	(3.136581E-7)	(3.57512E-7)	(3.43343E-7)

[1] These parameter estimates and standard errors for each model were obtained from SAS outputs.

[2] The generalized Michaelis-Menten (GMM) equation was used to predict mean live weight: $WT_{i,t} = WT_0 + \{[(WF - WT_0)(t/K)^C]/[1 + (t/K)^C]\}$, where $WT_{i,t}$ is the BW in kg of the ith pig at age t, WT_0 is the mean birth BW (1.6 kg in this experiment), WF is mean mature BW, and K is a parameter equal to the days of age at which one-half of the mature BW is achieved. The C is a unitless parameter related to the changes in proportional growth and the shape of the growth curves. The variance (wf_i) is the variance of the random effect of pig (López et al., 2000; Schinckel et al., 2009a). Average daily gain was then calculated as the derivative of the BW function on time (ADG = $\partial WT/\partial T$; Schinckel et al., 2009a).

[3] The 2000-2005 barrows and gilts GMM model fit the data better (lower AIC and lower se1) when 1.5 kg was added to the individual pig's body weight. The two random effects model did not converge for the GMM equation to estimate body weight of the 2011-2017 gilts, so these parameter estimates are from the mixed effects model with only 1 random variable.

[4] Average daily feed intake (ADFI) was predicted using the Bridges function: $DFI_{i,t} = C\{1-\exp[-\exp(M')t^A)]\} + e_{i,t}$, where t is the age or BW of the ith animal (BW, kg in this experiment), C is the mean mature daily feed intake (DFI), M' is the natural log of the exponential growth decay constant, and A is the kinetic order constant. The variance (a_i) is the variance of the random effect of pen (Bridges et al., 1986; Schinckel et al., 2009b).

[5] Live serial ultrasonic loin muscle area, last rib backfat, tenth rib backfat, and tenth rib loin muscle depth measurements were fit to the mixed model allometric function: $Y = (A + a_i) BW^B$; where A is an overall parameter, B is an allometric growth coefficient, and a_i is the pig specific random effect with variance σ_a^2 as a function of body weight in kg (Schinckel et al., 2009c).

[6] An exponential function (Wagner et al., 1999) was used to predict empty body protein accretion, empty body lipid accretion, total fat-free lean, and total carcass fat: $Y = \exp(b_0+b_1 BW + b_2 BW^2 + b_3 BW^3)$ where b_0, b_1, b_2, and b_3 are regression coefficients. Daily rates of empty body protein, empty body lipid, and total fat-free lean were determined as the product of the derivatives of two functions by $\partial C/\partial T = ((\partial C/\partial BW) \times (\partial BW/\partial T))$, (Whittemore et al., 1988; Schinckel and De Lange, 1996), where C is the body component mass, T is time, and BW is body weight in kg. The total carcass fat was calculated as a percentage of the live BW.