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DIETARY FIBER AND ACTIVE IMMUNIZATION AGAINST CHOLECYSTOKININ (CCK) DURING GESTATION INCREASES LACTATION FEED INTAKE AND PRODUCTIVITY OF SOWS

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By

Daniel Arthur Nelson

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ABSTRACT

DIETARY FIBER AND ACTIVE IMMUNIZATION AGAINST CHOLECYSTOKININ (CCK) DURING GESTATION INCREASES LACTATION FEED INTAKE AND PRODUCTIVITY OF SOWS

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Three experiments were conducted to evaluate gestation (G) management practices employed to enhance the average daily feed intake (ADFI) of sows in lactation (L). In Experiment 1, thirty York x Landrace (YxL) gilts were assigned to a corn-soybean meal (control), a corn-soybean meal-wheat straw (WS), or a corn-soybean meal-soybean hull (SBH) G diet for three parities. Animals within each treatment were offered the same daily amount of metabolizable energy, crude protein, lysine, calcium, and phosphorus. During L, all sows had ad libitum access to the same diet. The ADFI of SBH and WS sows was greater ($P<.10$) than control sows in L. The WS sows farrowed more ($P<.05$) live pigs than SBH sows, and weaned more ($P<.05$) pigs than either the SBH or control sows. The SBH sows were less constipated at d 109 of G ($P<.001$) and d 10 of L ($P<.05$). In Experiment 2, thirty three YxL gilts were assigned to either the control or SBH diet in G. In L, all sows had ad libitum access to the same diet containing .13% flavor. The ADFI of SBH sows was greater ($P<.10$) in parity 1, but not in parity 2 or overall. During G, SBH sows digested less ($P<.001$) dietary gross energy and crude protein and less ($P<.10$) ether extract, but more ($P<.05$) neutral detergent fiber and more ($P<.001$) acid detergent fiber than control sows. The SBH sows were less ($P<.001$)

constipated at d 110 of G. In Experiment 3, twenty four gilts immunized on d 64 of G farrowed litters; 20 were immunized with the desulfated C-terminal octapeptide of cholecystokinin (CCK) conjugated to Keyhole limpet hemocyanin (KLH) and 4 control females with KLH alone. Booster doses of immunogen (B1, B2, B3) were administered at 14 d intervals. Prior to each vaccination and on d 7, 14 and 21 of L (LD7, LD14, LD21 respectively), blood samples were taken from all sows. Colostrum and milk samples were collected from all sows at farrowing and on LD7, LD14, and LD21. Blood samples were collected from 2 piglets of each litter at LD7. Mean log CCK-antibody (CCK-AB) titers of sow serum at B2, B3, LD7, LD14, and LD21 was .27, 1.10, 1.77, 1.67, and 1.58 respectively. Mean log CCK-AB titer of colostrum was 2.42, and that of milk was .50, -.003, and -.20 at LD7, LD14 and LD21 respectively. These log titer were highly ($P<.005$ in all cases) interrelated to each other. Mean log CCK-AB titer of piglet serum (n=30) at LD7 was 1.22. The ADFI of sows over the 21 d L increased ($r^2=.37$, $P<.05$) with the average of the seven samples taken from the sow during L (ALTSS). Total litter gain also increased with the ALTSS ($r^2=.41$, $P<.05$) and with the ADFI of sows in week 3 ($r^2=.28$, $P<.05$). Nutrient digestibility was not impaired as a result of CCK immunization, and increased with CCK-AB titer in G. Dietary fiber from WS or SBH fed to sows in G increased L ADFI. The L ADFI of sows vaccinated against CCK increased with the anti-CCK-AB log titer of the sow.

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"Any one man really owes no more than an exceedingly small amount to his own efforts. To over emphasize the importance of a persons own efforts is to belittle the contributions of other".

From:
J. Wilson. 1954. How legitimate are names on papers? Science. 120:276.

I know this to be true and acknowledge everyone who helped me attain this degree. Thank you Dr. Josep Garcia-Sirera for countless "bleeds", infusions, vaccinations, weigh-days, surgeries, clean-ups, computer help. For days that began at 4:00 A.M. and for nights that lasted until 2:00 A.M. You are truly a friend and I thank you from the bottom of my heart. This would never have gotten done without your help - of that I am certain. Thank you Ross Santell for your friendship and support. Thank you fellow graduate students: Kim Howard, Dan Jennings, Mark Edwards, Scott Krammer and Michell Mater for help, even when I seemed unappreciative. Thank you Ph.D. committee: M.G. Hogberg, D.W. Rozeboom, J.C. Pekas, M.S. Allen, R. Fogwell, and M. Bennink for guidance and patience. Thank you for sharing your wisdom and friendship E.R. Miller and D.E. Ullrey. Thank you Pau Ku, Sharon DeBarr, David Main and Jane Link for your laboratory help and instruction. Thank you Ellie, Emily, Adam and Abbey for the time we gave up together then, for something better now. Thank you God. Without you I am, and can do, nothing.

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INTRODUCTION

Current swine production practices mandate that sows farrow and wean large litters, return to estrus promptly, conceive and repeat the process again. Herd averages of 2.5 litters/sow/year and 25 pigs/sow/year weaned, unheard of a decade ago, are now being achieved. Maximum lactation feed intake is essential to maintain this level of production. However, the producer is in a catch-22. Consumer demand for lean pork has forced the swine industry to select for animals with reduced fat reserves. Inadvertently, there has been a concurrent reduction in voluntary feed intake of sows of lean-type genetics (Riley, 1989). Although daily voluntary feed intake increases as lactation progresses, most lactating sows do not eat enough to maintain body weight during the average 3 to 4 week lactation period. Lactation weight loss is most rapid during the latter stages of lactation as milk output peaks. Excessive weight loss can lead to delayed return to estrus (> 10 days) following weaning, especially in the primiparous sow. In lactating sows (Matzat, 1990) and growing pigs (Pekas, 1985), voluntary feed intake limits productivity. Lactating sows which were superalimentated via a gastric fistula and received 20% more feed than those sows allowed ad libitum access to feed were able to preserve a greater amount of body mass and were also able to synthesize greater quantities of milk (Matzat, 1990). Gastric fistulation revealed the potential of swine to

utilize more feed, but it's implementation as a production practice has no practical merit.

Several researchers (Pollmann et al., 1979; Pond et al., 1985; Holzgraefe et al., 1986; Mroz et al., 1986; Carter et al., 1987; Hagen, 1988; Nelson et al., 1992) have fed different fiber sources to sows and gilts during gestation and monitored their subsequent lactational performance. All observations indicate that moderate levels of dietary fiber do not adversely affect sow performance. Some research groups have even reported enhanced litter size and lactation feed intake of sows fed fibrous gestation diets. Additionally, fibrous feed ingredients are usually available and are not expensive.

Fibrous ingredients do have some disadvantages. The low bulk density of fibrous ingredients often makes them difficult to handle with conventional feed-mixing equipment. Due to their lower digestibility, fibrous ingredients can increase manure volume. Additionally, the insolubility of the resulting manure may decrease the degree of solid removal from pits and lagoons, and reduce the effectiveness of manure handling practices that employ water flush systems.

Certainly other techniques that enhance the feed intake of lactating sows must be explored. In young growing swine, feed intake has been consistently increased by immunoneutralization of cholecystokinin (CCK). In a series of studies (Pekas and Trout, 1990; Pekas, 1993; Pekas, 1996) with growing pigs, active immunization against CCK stimulated feed intake and growth in proportion to the anti-CCK antibody (CCK-AB) titer elicited by the animal. Recently, superior CCK immunogens have been developed and reported by Pekas (1996), which elicited greater antiserum titers in swine. Immunoneutralization of CCK to enhance the feed intake of lactating swine has not been

reported. If gestating sows were vaccinated against CCK, the resulting CCK-antibodies may increase voluntary feed intake during lactation. If so, sows may respond similarly to super-alimentated sows which synthesized greater quantities of milk while simultaneously preserving a greater amount of body mass (Matzat, 1990). Elevated milk production may aid in piglet survival and enhance litter weaning weights. Preservation of body mass during lactation may contribute to timely rebreeding (< 10 d post-weaning) following weaning.

Enhancing lactation feed intake through gestation management practices such as increasing dietary fiber intake and vaccinating sows so they develop their own active titers against CCK are two methods, the feasibilities of which need to be further explored.

Due to the inherent differences in the chemical composition of fiber, two fibrous ingredients may be vastly different even though they may possess similar concentrations of crude fiber. This characteristic makes it difficult for the nutritionist to make generalized recommendations as to their use. Recommended inclusion rates of most fibrous ingredients within swine gestation diets have yet to be published. Active immunization against CCK has been observed to increase the voluntary feed intake of growing pigs, but it's use in sows has not been published.

This dissertation is a composite of three experiments conducted to evaluate these two methods of increasing sow lactation feed intake. The duration of these experiments lasted over four years, using over 90 sows and more than 130 litters. Experiment 1 and 2 (Chapter 2 and 3 respectively), investigated the effects of including two dietary fiber sources in gestating swine diets; wheat straw and soybean hulls. Experiment 3 (Chapter

5), evaluated active immunization of sows against CCK as a means of increasing lactation feed intake.

CHAPTER 1

DIETARY FIBER IN SWINE DIETS

Introduction

The majority of sows in the United States are housed in confinement units. However, prior to the popularity of swine confinement units most sow herds were maintained on grass and legume pastures throughout gestation. Even today it is not uncommon to find sow herds on pasture during gestation that forage for the majority of their nutrient demands. In other parts of the world (Southeast Asia) where grains are relatively expensive, fibrous feedstuffs are used exclusively to maintain the gestating sow herd. In these situations, sow performance (ie; litter size, litter birth weight, rebreeding interval) is seldom compromised.

When poor weather conditions cause grain prices to rise sharply, producers often seek alternative ingredients to serve as partial grain replacements. Many of these potential grain replacements contain higher amounts of fiber. Gestating sows have the potential to utilize more dietary fiber than is currently being incorporated into most swine gestation diets. Incorporation of high-fiber ingredients into gestation diets is also attractive as the practice increases feeding volume without increasing nutrient intake. As feed intake is generally restricted during gestation, this procedure may appease sow

appetite resulting in increased animal comfort. As world population continues to grow and place more pressure on grain supply for direct use, swine producers may need to reformulate conventional high-grain diets for animals.

This chapter will review the literature pertaining to the use of dietary fiber in swine diets. It will address the topics of fiber digestibility by pigs and the influence that dietary fiber exerts on the digestibility of other nutrients within the diet. This chapter will also review the results of studies in which fiber was included in high amounts in swine gestation diets and the effects of dietary fiber on sow performance and subsequent lactation feed intake.

Dietary Fiber

What is fiber?

Dietary fiber is defined as that portion of plant material that is not digested by enzymes of the mammalian digestive system (Schneeman and Gallaher, 1990). The majority of the fiber that is found in plant material originates in the cell wall where it gives rigidity and strength to the plant. Dietary fiber is composed of non-starch polysaccharides which include cellulose, *B*- glucans, hemicellulose, pectins, and gums. Cellulose and *B*- glucans are polymers of glucose with *B* 1-4 bonds holding the sugar residues together (Schneeman and Gallaher, 1990). Within a cellulose fiber, long chains of glucose polymers line up parallel to one another to form sheets (glucan) which are held together by hydrogen bonds between oxygen atoms and the hydrogen atoms of hydroxyl groups (Albershein, 1975). These glucan sheets can also line up one above the other, also held in place by hydrogen bonds, in this instance, between the sheets. It is the large number of

hydrogen bonds that gives cellulose it's strength and resistance to degradation (Albershein, 1975). The backbone of hemicellulose is composed of xylans, galactans, and mannans while side-chains are usually arabinose and galactose (Schneeman and Gallaher, 1990). Galacturonic acid is the principal sugar comprising the backbone of pectin, and galactose and arabinose form it's side chains (Schneeman and Gallaher, 1990). Within a typical cell wall, hemicellulose and pectin may bind to one another and serve as the "glue" which holds the cellulose fibers together (Albershein, 1975). Lignin is also a constituent of the cell wall of plants. Lignin is classified as fiber but is actually a non-carbohydrate constituent composed of phenylpropane units arranged in a complex three-dimensional structure.

Methods of fiber analysis

Crude fiber is defined as that portion of feed that remains after boiling in a weak acid and then in a weak alkali solution (Ensminger et al., 1990). The assay was originally developed to imitate the digestive processes that occur in the mammalian gastrointestinal (GI) tract, and to serve as an indicator of the relative indigestibility of the sample. However, this method gives no indication as to the type and amounts of the non-starch polysaccharides within the sample. The Van Soest (1967) sequential fiber analysis technique allocates the fibrous portion of the feed into its different components. Neutral detergent fiber (NDF) is that portion of sample remaining after boiling in neutral detergent and is composed of cellulose, hemicellulose and lignin. Acid detergent fiber (ADF) is that portion of sample remaining after boiling with acid detergent, and is composed of cellulose and lignin. The amount of hemicellulose in a sample can be

calculated by difference (NDF - ADF).

Fiber Digestion

Dietary fiber can be partially digested by mammals. The enzymes responsible for the degradation of dietary fiber are synthesized by the huge microbial population of the GI tract. Savage (1977) estimated that the normal human organism is composed of over 10^{14} cells, of which only about 10% are animal cells! The rest are microbial in nature and largely reside in the GI tract. A similar situation may exist in pigs. Two of the most significant cellulolytics found in ruminants; *Bacteroides succinogenes* and *Ruminococcus flavefaciens* are present in the large intestines of pigs (Varel et al, 1984).

Pigs can adapt to diets high in fiber. A greater amount of hemicellulose is digested by pigs after an 80-day adaptation period to a high fiber diet (Pollmann et al., 1979), and the extent of wood cellulose digestibility by pigs increases with time (Longland et al., 1993). Growing-finishing pigs adapt to high fiber diets by increasing the number of cellulolytic bacteria in the large intestine (Varel and Pond, 1985), as do sows and gilts (Varel and Pond, 1986). The cellulolytic population increase at the expense of other microbial species as the total density of microorganisms changes little with the amount and type of polysaccharide being fed to pigs (Bach Knudsen et al., 1993a). The total number of a given specie of microflora inhabiting the gastrointestinal tract may also be influenced by the genetics of the animal. Lean pigs have been observed to have a trend toward larger numbers of cellulolytic bacteria in their gastrointestinal tract than obese pigs (Varel, 1987).

Carbohydrates are the principle substrate for GI tract microflora in pigs (Bach Knudsen et al., 1991; 1993a). Within the GI tract, the process of fiber fermentation occurs primarily in the cecum and colon (Zebrowska, 1988). Bach Knudsen et al. (1993a) confirmed that more than 92% of all dietary structural carbohydrates were degraded in the cecum, proximal colon and ascending colon. Microbial degradation of fiber within the gastrointestinal tract generates volatile fatty acids (VFA), including acetic, propionic and butyric, lactic acid, and various gases (hydrogen, carbon dioxide and methane) (Bach Knudsen et al., 1991). It has been estimated that the VFA arising from microbial degradation of fiber within the cecum and colon of growing pigs may provide up to 30% of pigs maintenance energy requirements (Varel, 1987). A more conservative range of 10 to 24% was published by Bach Knudsen and Hansen (1991). Nitrogen compounds (ammonia) absorbed from the hind gut are not utilized by pigs (Fernandez et al., 1986).

The extent of degradation of dietary fiber is a function of the rate of passage and the rate of microbial fermentation (Ehle et al., 1982; Fernandez et al., 1986) . Microbial fermentation rates will not be maximized until microbes and their enzymes are attached or are in close proximity to fiber digestion sites. Microbial attachment is dependent on the number of available attachment sites, the mass of fiber digesting microbes in the hind gut, the species composition of the microbial population and the ability of the different species to attach to and colonize the fiber (Allen and Mertens, 1988). Additionally, a coarsely ground fibrous ingredient that exhibited a longer retention time would have a greater digestibility than the same material ground finer that had a shorter retention time.

When comparing growing pigs and sows, the differences that exist in ability to digest different ingredients are largest when comparing fibrous materials (Fernandez et al., 1986). Sows have a larger intestinal capacity and a longer ingesta transit time than growing pigs. These two factors increase the extent of microbial fermentation of structural carbohydrates of fiber by sows as compared to growing pigs (Varel and Pond, 1986). The stage of maturity of the fiber source being fed to sows will influence the degree of microbial fermentation. Allee (1981), reported that third-cutting alfalfa has higher digestibility coefficients (DC) for both energy and protein than more mature first-cutting alfalfa . This observation is probably the result of the greater degree of lignification of the more mature plant. It has been reported that the nonstructural polysaccharides (NSP) of unlignified cell walls from the endosperm and aleurone layers of wheat and oats are much more extensively degraded than those NSP of the secondary lignified cell walls from pericarp or testa (Bach Knudsen and Hansen 1991).

Dietary fiber reduces nutrient digestibility of the diet

The inclusion of a fibrous ingredient into diets fed to pigs reduces the digestibility of other nutrients within the diet (Pollmann et al., 1979; Kornegay, 1981; Frank et al., 1983; Calvert et al., 1985; Bray et al., 1986; Mroz et al., 1986; Pond et al., 1986; Everts and Smits, 1987; Moore et al., 1987; Bach Knudsen and Hansen, 1991; Bach Knudsen et al., 1993b).

Dietary alfalfa meal or corn cobs reduced the DC for dry matter, cell contents, and crude protein of the diet (Pond et al., 1986). Alfalfa inclusion rates of 50 and 90% of gestating sow diets lowered the DC for energy, protein and fiber constituents (Calvert et

al., 1985). Similarly, the DC for nitrogen, dry matter, and digestible energy decreased linearly with increasing amounts of dietary corn cobs (Frank et al., 1983). The DC for nitrogen, energy and DM were also lower when oat hulls were added to sow gestation diets (Mroz et al., 1986). Everts and Smits (1987) reported that for every 1% increase in DM dietary fiber, the DC for dry matter and crude protein were reduced by 1.8 and 1.1% respectively.

The overall reduction in diet DC due to fiber inclusion may be the cumulative result of several factors. If the fiber within a feed ingredient is not soluble when they pass through the small intestine, they may act as a barrier against digestive enzymes and potentially reduce the digestibility of starch and other nutrients in the small intestine (Bach Knudsen et al., 1993a). Pectins within fiber are capable of forming a viscous gel-matrix that may similarly reduce exposure of nutrients to digestive enzymes (Mitaru et al., 1984). This pectin gel may also encapsulate the products of digestion and reduce their access to sites of absorption within the intestine (Mitaru et al., 1984). Due to its ability to hydrophobically bind amino acids, lignin may contribute to reduced protein and amino acid digestibilities when fiber is added to the diet (Mitaru et al., 1984).

Stage of gestation affects nutrient digestibility

The extent of nutrient digestibility by sows is influenced with the stage of gestation. Bray et al. (1986) reported that the DC for nutrients in both the control and test diet tended to increase from early gestation to mid gestation while the lowest DC values were consistently recorded for late gestation. Similarly, nitrogen balance and nitrogen retention decreased from the first to the third trimester of gestation (Nutzback et

al., 1984). Nutrient utilization of sows fed high levels of alfalfa was lowest when measured during the last 10-15 d of gestation (Calvert et al., 1985).

Use of soybean hulls in swine diets

Moore et al. (1987) observed that the addition of soybean hulls at the rate of 15% to a corn-soybean meal diet reduced the DC for DM, but increased the DC for all fibrous components. Similarly, as soybean hulls were substituted for a corn-oat-soybean meal diet at the rate of 7.5, 15.0 and 30.0%, the digestibility of DM, energy, CP, cell content and ADF nitrogen decreased, while digestion coefficients for NDF, ADF, cellulose and lignin increased (Kornegay, 1981). However, when soybean hulls replaced 50% of the basal diet, digestibility of DM and energy were reduced as were the DC for NDF and hemicellulose (Bray et al., 1986). Perhaps the difference between these results and those reported by Kornegay (1981) were due to the larger amount of soybean hulls used by Bray et al (1986). In agreement with Kornegay (1981), the DC for ADF and cellulose were higher for those sows consuming a 50% soybean hull diet than sows consuming the basal diet (Bray et al., 1987). Mineral utilization of growing pigs was not influenced by substituting 15% soybean hulls in the basal diet (Moore and Kornegay, 1987).

Experiments with high-fiber ingredients and performance of sows.

One of the earliest reports of the use of fiber in diets for sows concluded that breeding efficiency was increased by the addition of dietary alfalfa meal (Fairbanks et al., 1945). In the years since, other scientific reports have shown similar results. Sows consuming a diet containing 18% alfalfa had more live pigs and weaned more pigs/litter than those on a legume-free diet, and when examined early in gestation, contemporary

sows on the alfalfa diet possessed a greater number of corpora lutea than those fed the legume-free diet (Teague, 1955). Similarly, sows fed gestation diets balanced for similar daily ADF intakes from either alfalfa or sunflower hulls have a greater number of pigs born alive/litter (Carter et. al., 1987). Gilts were observed to have a higher farrowing rate when consuming a gestation diet containing 96.5% alfalfa, than those consuming diets with less alfalfa (Danielson and Noonan, 1975). Additionally, it was observed that animals fed higher levels of alfalfa during gestation are more docile and easier to handle (Danielson and Noonan, 1975). Allee (1981) suggested that a diet consisting of three pounds of alfalfa haylage dry matter, two pounds of grain, and a vitamin and mineral premix is more than adequate for gestating sows.

Not all reports have shown an improvement in sow performance from dietary fiber. When alfalfa was included at 50 and 90% of the diet of gestating sows, the number of live pigs farrowed and weaned per sow, and the average birth weights of piglets were not affected by dietary treatment (Calvert et al., 1985). However, dietary alfalfa inclusion rates of greater than 95% has been associated with reduced birth weights (Calvert et al., 1985; Pond et al., 1985). In a large trial with 567 sows over five parities, there was no clear evidence that either the inclusion of roughage or higher dietary crude fiber improved reproductive performance (Everts, 1991).

The inconsistencies in the literature may be explained partially by the fact that in many instances, fiber was substituted at the expense of other ingredients resulting in differences in dietary nutrient intakes. In experiments where alfalfa was substituted for the basal diet, B-complex vitamins and protein concentrations may have been elevated.

In experiments where oat hulls or soybean hulls were substituted, protein and energy concentrations may have been reduced. Additionally, fiber composition of an ingredient (alfalfa) changes with plant genetics, maturity, and growing conditions. It is not surprising then, to find some slightly contradictory results.

Dietary fiber and constipation

The laxative effects of dietary fiber have long been confirmed. Fiber may reduce the incidence of constipation through more than one mechanism. In one study, the GI tract transit time was reduced for those sows receiving gestation diets containing oat hulls (Mroz, et al., 1886). As removal of water from ingesta is a major function of the large intestine, a shorter transit time would result in feces with a higher water content (less constipated). This was observed when soybean hulls were included in the basal diet of gestating sows; fecal output increased as fecal DM decreased (Kornegay, 1981). The dietary fiber in wheat was reported to increase fecal bulk by virtue of its water-holding capacity, while the dietary fiber in oats did so by increasing microbial biomass (Bach Knudsen et al., 1991). Similarly, the fecal bulking effect of oat bran is mainly caused by an increase in the excretion of protein and fat of bacterial origin (Bach Knudsen et al., 1993a).

Dietary fiber during gestation and subsequent lactation feed intake

Many factors can influence the ADFI of lactating sows. Data from 3,559 lactations revealed that an increase in lactation ADFI was associated with nursing larger litters, increasing parity and longer lactations (O'Grady et al., 1985).

Table 1. Lactation feed intake (kg hd^{-1} d^{-1}) of sows fed fibrous diets during gestation

Authors	Test diets fed during gestation				Significance
	Control	Alfalfa	Sunflower hulls		
Carter et al., 1987.	5.2	5.7	5.4		Alf.+S.F.hulls>Control
	#1; Control	#2; 46% Alf.+ Orchardgrass	#3; #2+ Lasalocid	#4; #1+ Lasalocid	
Holzgraefe et al., 1986.	4.7[a]	5.3[bc]	5.7[b]	5.0[ac]	P<.05
	Control	97% Alfalfa hay	66% Wheat grass		
Pollman et al., 1979.	4.2	4.7	4.8		N.S.
	Control	40% Dehydrated alfalfa	96% Dehydrated alfalfa	20% Corn cobs	
Pond et al., 1985.	4.3[a]	4.5[b]	4.7[b]	4.7[b]	P<.01
	Control	67% of control + pasture	33% of control + pasture	All pasture	
Prince et al., 1986.	7.7	7.6	8.4	8.5	No Stats
	Control	30% of M.E. from alfalfa haylage	60% of M.E. from alfalfa haylage	90% of M.E. from alfalfa haylage	
Hagen, 1988.	5.3	5.9	6.2	6.4	Linear P<.01
		Control	30% of M.E. from alfalfa haylage		
Nelson et al., 1992.	Wk 1;	5.2	5.8		P=.11
	Total;	6.1	6.3		N.S.

An inverse relationship has been reported between lactation ADFI and gestation weight gain (O'Grady et al., 1985; Weldon et al., 1992), and lactation ADFI and ambient temperature of the farrowing room (O'Grady et al., 1985; Matzat, 1990).

Several research groups have observed increased lactation ADFI of sows fed diets containing "high fiber" during the gestation period (Table 1). The results of some of these studies may have been confounded because nutrient intake per animal per day was different among treatments during gestation. However, in every experiment, those animals that were fed fiber during gestation ate more of a common diet during lactation. Reasons for the elevated lactation ADFI are not known. In growing pigs it has been observed that pigs fed "high fiber" diets have heavier stomach weights (Anugwa et al., 1989) and heavier GI tract weights (Pekas et al., 1983; Anugwa et al., 1989). Similarly, the weights of liver, heart, stomach, small intestine, cecum and colon expressed as a percent of body weight are greater for young adult pigs fed diets high in alfalfa than those fed a diet which is low in alfalfa (Pond et al., 1988). Although it has not been documented, heavier stomach weights of pigs fed diets high in fiber may indicate a greater stomach volume or capacity. Perhaps this is why sows fed "high fiber" gestation diets can eat more in lactation.

Stretch receptors within the stomach (Gonzalez and Deutch, 1981) and within the duodenum (Davis and Campbell, 1978) are reported to limit intake via the vagus nerve. Perhaps these receptors from sows on high fiber gestation diets have become acclimated to the increased volume of feed. Sows fed fiber in gestation would come into lactation with the ability to immediately consume more feed than those sows which consumed the

normal 4 to 5 lb corn-soybean meal diet throughout gestation.

Varel (1987) and Anugwa et al. (1989) found that fibrolytic bacteria increase and replace other bacteria in the intestines of pigs fed "high fiber" diets. The production of tyrosine and tryptophan decrease as the number of proteolytic bacteria decrease. These two amino acids are precursors for the anoretic agents dopamine and serotonin and therefore, it has been hypothesized that pigs fed fiber eat more feed due to lower brain levels of dopamine and serotonin (Bergner, 1981). Anarerobic degredation of tyrosine in the intestine also leads to the formation of volatile phenols such as p-cresol (Scheline, 1968). Para cresol and other volatile phenols are potential toxins (Lumanta, 1987). Dietary fiber has been shown to reduce the production of p-cresol in rats (Scheline, 1968), and may have similar effects in pigs fed fiber . The residual time effect on feed intake after withdrawal of fibrous material from the diet has not been reported.

Summary

As the trend for world population growth continues, the demand for feed grains to be used for direct human consumption will also continue to increase. As feed costs represent approximately from 60 to 70% of the total cost of swine production, and the conversion of grain to animal products is an inefficient process. Alternative feedstuffs must be investigated. Diets high in fiber are not well suited for growing/finishing pigs or for lactating sows where energy intake often limits productivity. However, the literature is convincing that, in moderation, fiber can be added to the diets of gestating sows. As most gestating sows are fed only 1.8 to 2.7 $kg \cdot hd^{-1} \cdot d^{-1}$ they may be able to use fibrous diets more efficaciously than full-fed animals due to both a slower GI tract transit time

and smaller digesta volume. Both of which may facilitate the extent of microbial fermentation of the fibrous component.

Before nutritionists can make recommendations as to the inclusion rates of these alternative feedstuffs, better scientific experimentation must explore and elucidate the repercussions of including the feedstuffs in the diet. To ascertain if the observed results are actually due to the dietary fiber components, the daily intake of other dietary nutrients needs to be balanced during gestation. To establish which fiber component is responsible for the observed effects, the NDF or ADF (not crude fiber) content of the dietary treatments needs to be considered. Chapter 2 and 3 of this dissertation investigate the consequences of the use of two dietary feedstuffs in gestating swine diets: wheat straw and soybean hulls.

CHAPTER 2

DIETARY WHEAT STRAW OR SOYBEAN HULLS DURING GESTATION AND SUBSEQUENT LACTATION PERFORMANCE

Abstract

Thirty crossbred (Yorkshire x Landrace) gilts were randomly assigned to a corn-soybean meal (control), a corn-soybean meal-wheat straw (WS), or a corn-soybean meal-soybean hull (SBH) gestation diet for three consecutive parities. Gestation diets were formulated so that animals within each treatment were offered the same daily amount of metabolizable energy, crude protein, lysine, calcium, and phosphorus. The WS and SBH diets were formulated to provide similar daily intakes of NDF. During lactation, all sows consumed the same corn-soybean meal diet. Sows were weighed at breeding, within 12 hours post-parturition, at d 21 of lactation and at weaning. The total number of pigs born, born alive, and stillborn was recorded. Individual piglet weights were recorded at birth, 21 d of age and at weaning. Fecal hardness of sows was determined on d 109 of gestation and on d 3 and 10 of lactation. The SBH sows gained less ($P<.05$) weight in gestation and lost less ($P<.10$) weight in lactation than WS sows. The weight changes of SBH and control sows were similar. Both SBH and WS sows consumed more ($P<.10$) feed in lactation than the control sows. Control sows lost more ($P<.05$) backfat than either SBH or WS sows. The WS sows farrowed more ($P<.05$) live pigs than SBH sows, and weaned

more (P<.05) pigs than either the SBH or control sows. Consequently, litter weaning weights of the WS sows were greater (P<.05) than for control or SBH sows. The SBH sows had a lower incidence of constipation at d 109 of gestation (P<.001) and d 10 of lactation (P<.05) than the control or WS sows . The WS sows had stools that were significantly firmer than the SBH sows at each time point. These results show that both WS and SBH can increase lactation feed intake when incorporated into gestating swine diets. Dietary SBH can also reduce the incidence of constipation.

Introduction

Several researchers (Pollmann et al., 1979; Pond et al., 1985; Holzgraefe et al., 1986; Mroz et al., 1986; Carter et al., 1987; Hagen, 1988; Nelson et al., 1992) have fed different fiber sources to sows and gilts during gestation and monitored their subsequent lactational performance. All indicate that moderate levels of dietary fiber in gestation does not adversely affect sow performance. Some research groups have even reported enhanced litter size and lactation feed intake of sows fed fibrous diets during gestation.

To study the influence of dietary fiber in gestation on subsequent sow performance, the current experiment was in part undertaken by the North Central Region Committee on Swine Nutrition (NCR-42). This committee's protocol included corn-soybean meal (control), and corn-soybean meal-wheat straw (WS) gestation diets. Wheat straw was chosen as a fiber source due to its availability among research stations in the Midwest. As wheat straw is a fairly non-fermentable fiber source, this experiment also included a diet containing the more fermentable fiber of soybean hulls.

The objectives of this study were to determine the effects of dietary fiber in

breeding and gestating diets on the lactation ADFI, weight change and return to estrus interval of sows and the weight gain of their litters, and to determine if fiber source (wheat straw vs soybean hulls) and composition (measured by neutral detergent fiber [NDF]) distinctly modify these performance variables.

Materials and Methods

Thirty Yorkshire X Landrace gilts were assigned to either a corn-soybean meal (control), a corn-soybean meal-wheat straw (WS), or a corn-soybean meal-soybean hull (SBH) gestation diet for three consecutive parities. Gilts were assigned to dietary treatments in the order they initially expressed estrus (ie; control, WS, SBH, control, WS, SBH).

The amount of each treatment diet was calibrated in gestation so that sows among treatments were offered the same daily amount of metabolizable energy, crude protein, lysine, calcium, and phosphorus (Table 2). The WS and SBH diets provided similar daily intakes of NDF. During gestation in parity 2 and 3, sows were offered 1.8 (Control), 2.1 (WS), or 1.9 (SBH) kg of feed·sow^{-1}·d^{-1}. The amount of each treatment diet offered to parity 1 gilts was 25% greater to allow for continued body growth. The nutrient content of soybean hulls used in diet formulation were from the U.S.-Canadian Feed Tables (1982). Wheat straw was assigned a value of zero for all nutrient contributions when formulating the experimental diets. The wheat straw used in this experiment was ground by hammer mill twice through a one-half inch screen. Resulting wheat straw length ranged from between 6 and 12 mm. Gilts were fed their assigned diets beginning approximately one week prior to breeding and throughout each gestation. Throughout

lactation all sows were fed twice daily an amount of feed to provide constant ad libitum access to the diet which control sows consumed throughout gestation. Sows were fed from individual pre-weighed amounts of feed stored in containers in front of each sow. Feed consumption was determined by difference (initial feed weight - [unused feed weight + orts]) and recorded weekly.

Within each parity during the period from weaning to breeding, sows were housed in their respective treatment groups in an outdoor unit. After supervised mating, sows were moved inside a gestation building and were placed in individual gestation crates over partially slotted flooring. Sows were weighed at breeding, d 109 of gestation, within 24 hours post-farrowing, at d 21 of lactation and at weaning. Backfat depths of sows were measured at breeding, d 109 of gestation, at d 21 of lactation, and at weaning with an ultrasonic backfat probe (Renco Lean Meater,[R] Serial # 4458; Mpls., MN). Backfat depths were measured approximately 3 cm off midline at the last posterior rib and the last posterior lumbar vertebrae, and the average of the two readings was recorded. The number of days from weaning to estrus was recorded throughout the study. Sows that failed to establish or maintain pregnancy were removed from the experiment. An assessment of the effects of wheat straw and soybean hulls on the incidence of constipation was made on d 109 of gestation and on d 3 and 10 of lactation. This was done via tactile appraisal of fecal hardness, and the following subjective scoring system was used;

1. Very loose
2. Moderately loose
3. Normal
4. Moderately hard
5. Very hard

Table 2. Composition and calculated analysis of diets used in Experiment 1 and 2.

Diet	Control	Wheat straw[a]	Soybean hull
Ingredient, % of total			
Corn	79.50	69.20	66.60
Soybean meal (44%)	15.50	13.50	10.00
Soybean oil	1.00	.85	.85
Wheat straw	-	13.05	-
Soybean hulls	-	-	19.10
Dicalcium phosphate	1.85	1.75	1.69
Limestone	1.08	70	.76
Salt	.40	.35	.37
Vitamin TM mix[b]	.50	.45	.45
Folic acid supplement	.02	.02	.02
Biotin supplement	.15	.13	.13
L-lysine HCl	-	-	.03
Calculated nutrient intake			
Metabolizable energy, Kcal/d	5960.0	5960.0	5960.0
Crude protein, g/d	246.0	246.0	246.0
Lysine, g/d	11.7	11.7	11.8
Calcium, g/d	16.0	16.0	16.0
Phosphorus, g/d	12.0	12.0	12.0
Crude fiber, g/d	49.0	162.0	196.0
NDF, g/d	172.0	402.0	403.0
ADF, g/d	71.0	218.0	249.0
Daily intake, kg			
Gilts	2.3	2.6	2.4
Sows	1.8	2.1	2.0

[a]Used in Experiment 1 only.

[b]Vitamin-TM premix, and Folic acid and Biotin supplements were supplied free of charge by Feed Specialties Co., Inc., Des Moines, IA. VTM supplied the following amounts per kg of diet: 6600 IU vit A; 1210 IU vit D; 22 IU vit E; 3.37 mg menadione; 22 mg pantothenic acid; 33 mg niacin; 1 mg folic acid; 5.5 mg riboflavin; 28 mcg B_{12}; 577 mg choline; 122 mg Zn; 126 mg Fe; 61 mg Mn; 12.2 mg Cu; .52 mg I; .3 mg Se. Biotin supplement supplied .33 mg biotin/kg diet. Folic acid supplement supplied 1 mg folic acid/kg of diet.

The total number of pigs born, born alive, and stillborn was recorded. Individual piglet weights were recorded at birth, 21 d of age and at weaning. Cross fostering of pigs was not practiced in this study unless a sow had more pigs than functional teats (and then only within treatment). Creep feed was not offered to piglets.

Data Analysis

Data were analyzed with the General Linear Models (GLM) procedure of SAS to determine significant relationships between main effects (treatment, group and parity) and all interactions. When group, parity or an interaction was not significant, it was removed from the model, and the analysis was performed again. Means were separated by the PDIFF procedure within SAS.

Results and Discussion

Lactation length was not standardized, but was not different among treatments (mean $\pm$ SE = 27.1 $\pm$ .6, 26.6 $\pm$ .7, 26.4 $\pm$ .6 d for control, WS and SBH respectively). As 21 d is a typical lactation length, 21 d and total lactation length means will be presented.

ADFI

Both the WS and SBH sows ate more ($P<.05$) feed during the first 21 d of lactation and more ($P<.10$) total lactation feed than control sows (Table 3). The WS sows ate 14.7 kg/sow more feed, while SBH sows consumed 18.9 kg/sow more feed than control sows during the first 21 d of lactation. When analyzed by parity (Table 4), the treatment differences in lactation ADFI were not apparent until parities 2 and 3. During parity 2, the lactation total ADFI of SBH sows was greater ($P<.05$) than control sows,

while WS and control sows consumed similar amounts. However, during parity 3 lactation, both the WS and SBH sows ate more ($P<.01$) than control sows. In parity 3 lactation, WS and SBH sows consumed and average 18.9 kg/sow more than control sows during the first 21 d of lactation. These results imply that older sows may benefit from dietary fiber more than primiparous sows. The influence that dietary fiber may have on GI tract capacity or function may be time-dependent and may not have had time to exert an influence in the primiparous animals of this experiment.

Table 3. ADFI and constipation scores for sows in Experiment 1[a]

Treatment	Control	Wheat straw	Soybean hull
Mean ADFI, kg			
Gestation	2.0 ± .01[b]	2.2 ± .01[c]	2.1 ± .01[d]
To d 21 lactation	5.2 ± .15[e]	5.9 ± .18[f]	6.1 ± .16[f]
Lactation	5.0 ± .12[g]	5.3 ± .14[h]	5.6 ± .12[h]
Length of phases, d			
Gestation	115.0 ± .4	116.0 ± .5	114.7 ± .4
Lactation	27.1 ± .6	26.6 ± .7	26.4 ± .6
Wean to estrus	6.2 ± .7	5.8 ± .8	5.1 ± .7
Constipation score[i]			
d 109 of gestation	4.4 ± .1[b]	4.6 ± .1[b]	3.4 ± .1[c]
d 3 of lactation	3.9 ± .2[jk]	4.3 ± .2[j]	3.5 ± .2[k]
d 10 of lactation	3.3 ± .1[e]	3.2 ± .1[e]	3.0 ± .1[f]

[a] Least square means ± SE.

[bcd]Means in a row with a common or no superscript do not differ (P<.001).

[ef]Means in a row with a common or no superscript do not differ (P<.05).

[gh]Means in a row with a common or no superscript do not differ (P<.10).

[i]1 to 5: 1=diarrhea; 2=loose; 3=normal; 4=firm; 5=constipated

[jk]Means in a row with a common or no superscript do not differ (P<.01).

Table 4. Lactation ADFI by parity for Experiment 1[a]

Treatment	Control	Wheat straw	Soybean hull
Mean ADFI, kg			
Parity 1 lactation total	4.1 ± .2	4.2 ± .2	4.5 ± .2
Parity 2 to d 21 of lactation	4.8 ± .3	5.1 ± .3	5.6 ± .3
Parity 2 lactation total	4.9 ± .2[b]	5.4 ± .3[bc]	5.8 ± .2[c]
Parity 3 to d 21 of lactation	5.7 ± .1[d]	6.6 ± .2[e]	6.6 ± .1[e]
Parity 3 lactation total	5.8 ± .2[d]	6.6 ± .2[e]	6.6 ± .1[e]

[a] Least square means ± SE.
[bc]Means in a row with a common or no superscript do not differ (P<.05).
[de]Means in a row with a common or no superscript do not differ (P<.01).

Constipation, sow body weight and weight changes

Soybean hulls in the gestation diet decreased the incidence of constipation (Table 3). Constipation scores of SBH sows were lower (P<.001) than control and WS sows at d 109 of gestation and lower than WS sows at d 3 (P<.01) and d 10 (P<.05) of lactation. Although sows from both fiber treatments consumed similar NDF daily, the cellulose content of the SBH diet was greater. The cellulose within the SBH treatment may have provided additional energy for the microbial population, and the reduction in constipation was actually the result of an increase in fecal biomass (Bach Knudsen et al. 1993a). The lower incidence of constipation in late gestation and early lactation of SBH sows may have contributed to their ability to consume larger quantities of feed in lactation. Wetter feces also indicate that the digesta transit time through the GI tract is shorter, allowing

more feed to be consumed over a given amount of time. This would explain the greater ADFI of SBH sows, but is not consistant with the observation that WS sows had firmer feces than control sows, yet a higher ADFI.

The daily intake of nutrients among sows on different treatments were calculated to be similar throughout gestation. However, differences did exist among treatments in sow body weight at several time points throughout the reproductive cycle (Table 5). The WS sows were heavier ($P<.01$) than control sows at d 109 of gestation. The water holding capacity of the dietary fiber consumed by WS sows may have contributed to these differences. The WS and SBH sows were heavier ($P<.10$) than control sows at breeding, post farrowing and at d 21 of lactation. SBH sows were heavier ($P<.05$) than either control or WS sows at weaning. The elevated ADFI of the WS and SBH sows during lactation augmented these differences. The WS sows gained more ($P<.05$) weight in gestation than SBH sows, but lost more ($P<.10$) weight in lactation than either control or SBH sows (Table 5). The majority (mean= 76%) of lactation weight loss by all sows occurred during the last 5 to 6 d of lactation. In light of these data it is easy to see how the currently popular production practice of segregated early weaning (SEW) at 10 to 14 d of lactation can help sows maintain body condition and contribute to greater performance in successive parities. The WS sows also lost more ($P<.05$) subcutaneous backfat in lactation than did the SBH sows (Table 6). These observations may be explained partially by the fact that WS sows farrowed more live pigs ($P<.05$) than SBH sows, and weaned more pigs ($P<.05$) per litter than control and SBH sows (Table 7).

Table 5. Sow weights and weight changes in Experiment 1[a]

Treatment	Control	Wheat straw	Soybean hull
n	24	20	25
Weight, kg			
Breeding	136.3 ± 2.1[b]	143.5 ± 2.4[c]	145.5 ± 2.1[c]
d 109 gestation	179.2 ± 2.8[d]	191.7 ± 3.0[e]	185.2 ± 2.8[de]
Post-farrow	163.6 ± 2.4[d]	175.4 ± 2.5[e]	174.0 ± 2.3[e]
d 21 of lactation	161.2 ± 2.4[f]	167.7 ± 2.7[g]	172.9 ± 2.3[g]
Weaning	154.9 ± 2.4[b]	158.8 ± 2.7[b]	166.5 ± 2.3[c]
Weight changes, kg			
Breeding to d 109 of	42 .3 ± 2.2[bc]	46.6 ± 2.5[b]	39.6 ± 2.1[c]
Post-farrow to d 21	-1.8 ± 2.0[fg]	-5.7 ± 2.3[f]	-.8 ± 1.9[g]
Post-farrow to weaning	-8.1 ± 2.2[f]	-14.6 ± 2.6[g]	-7.3 ± 2.2[f]
Breeding to weaning	18.4 ± 2.3[fg]	15.2 ± 2.5[f]	21.2 ± 2.3[g]
Weight change, kg/d			
Post-farrow to d 21	-.1 ± .1[fg]	-.3 ± .1[f]	<-.1 ± .1[g]
d 21 lactation to weaning	-1.3 ± .3	-2.0 ± .4	-1.4 ± .3
Post-farrow to weaning	-.3 ± .1[b]	-.5 ± .1[c]	-.3 ± .1[b]

[a]Least square means ± SE.
[bc]Means in a row with a common or no superscript do not differ (P<.05).
[de]Means in a row with a common or no superscript do not differ (P<.01).
[fg]Means in a row with a common or no superscript do not differ (P<.10).

Table 6. Sow backfat depths and backfat depth changes in Experiment 1[a]

Treatment	Control	Wheat straw	Soybean hull
Backfat depth, mm			
Breeding	21.6 ± .9	22.6 ± 1.1	21.6 ± .9
d 109 of gestation	21.8 ± .8	22.8 ± .9	21.3 ± .8
d 21 of lactation	21.0 ± .7	21.1 ± .8	20.5 ± .7
Weaning	20.3 ± .7	20.7 ± .8	20.7 ± .7
Backfat depth change, mm			
Breeding to d 109 gestation	.3 ± .7	-.1 ± .8	-.3 ± .6
d 109 gestation to d 21 lactation	-.8 ± .6	-2.3 ± .7	-.6 ± .6
d 109 gestation to weaning	-1.4 ± .6[bc]	-2.5 ± .7[b]	-.5 ± .6[c]
Breeding to weaning	-1.1 ± .9	-2.7 ± 1.1	-.9 ± .9

[a] Least square means ± SE.
[bc]Means in a row with a common or no superscript do not differ (P<.05).

The number of liveborn pigs and the total litter weight of the WS sows was greater (P<.01) than either control or SBH sows. Litter size was not standardized in this trial, but WS sows maintained their litter size advantage. The litter weights of WS sows were significantly larger than those of control or SBH sows at each time point measured. However, as litter weight gains were not different among treatments, the WS litter weight advantage must be attributed to their larger initial birth weight.

The SBH sows weaned heavier (P<.05) piglets than did control or WS sows. This difference is attributed to the significantly smaller number of pigs weaned/litter by SBH sows compared to WS sows. It is assumed that piglets from SBH sows has less

Table 7. Sow reproductive performance in Experiment 1[a]

Treatment	Control	Wheat straw	Soybean hull
n	24	20	25
Number of pigs /litter			
Live born	9.4 ± .6[bc]	10.6 ± .6[b]	8.5 ± .5[c]
Stillborn	.6 ± .1[b]	.2 ± .1[c]	.1 ± .1[c]
Live at d 21	8.3 ± .4[b]	9.6 ± .4[c]	7.4 ± .4[b]
Weaned	8.3 ± .4[b]	9.6 ± .4[c]	7.4 ± .4[b]
Litter weight, kg			
Live born	13.6 ± .8[d]	17.1 ± .9[e]	12.9 ± .8[d]
Total at birth	14.0 ± .9[b]	17.1 ± 1.0[c]	12.8 ± .8[b]
d 21	47.3 ± 2.0[b]	54.8 ± 2.2[c]	48.0 ± 2.0[b]
Weaning	56.5 ± 2.4[b]	64.5 ± 2.6[c]	56.6 ± 2.4[b]
Litter weight gain, kg			
To d 21 lactation	33.6 ± 1.7	37.2 ± 1.8	35.3 ± 1.6
To weaning	43.1 ± 2.1	47.3 ± 2.5	43.8 ± 2.1
Weight/pig, kg			
Born alive	1.5 ± .1	1.6 ± .1	1.5 ± .1
d 21 lactation	5.8 ± .2[d]	5.8 ± .2[d]	6.8 ± .2[e]
Weaning	7.0 ± .3[b]	6.8 ± .3[b]	8.0 ± .3[c]

[a] Least square means ± SE.

[bc]Means in a row with a common or no superscript do not differ (P<.05).

[de]Means in a row with a common or no superscript do not differ (P<.01).

competition from one another and were able to consume greater quantities of milk from the sow.

Conclusions

Both WS and SBH fed in gestation increased ADFI during lactation. Each test diet increased the daily intake of NDF from about 172g (control) to about 400g/sow (WS and SBH) in gestation. The additional volume the NDF provided with its water-holding capacity may have induced changes in the stomach and GI tract of the sows during gestation which enabled them to consume greater quantities of feed than control sows when they entered the lactation period.

The WS increased the number of pigs weaned/litter and litter weaning weights. Because litter size was not standardized in this experiment, and suckling intensity is known to drive feed intake, the observed effects of greater ADFI of WS sows cannot be attributed solely to treatment.

Soybean hulls, but not WS alleviated the incidence of constipation which is common malady in sows near the time of parturition. The elevated feed intake of the SBH sows may have been related to their lower incidence rate of constipation.

The results of this three parity study indicate that wheat straw or soybean hulls can be used in swine gestation diets (at 13.05 and 19.1% respectively) and lactation sow performance will be similar to or greater than that of sows fed a common corn-soybean meal gestation diet.

CHAPTER 3

EFFECT OF FEEDING SOYBEAN HULLS DURING GESTATION ON SOW PERFORMANCE, NUTRIENT DIGESTIBILITY AND CHANGES IN BODY COMPOSITION

Abstract

Thirty-three York x Landrace gilts (about 10/group) were fed a corn-soybean meal (Control) diet or a corn-soybean meal-soybean hull (SBH) diet throughout the gestation periods through two parities. These diets were formulated to provide similar daily intakes of M.E., CP, Lysine, Ca., and Phos. In lactation, sows from both treatments consumed a corn-soybean meal diet containing .13% of a flavoring agent (Kent Feeds). During lactation, sows were fed twice daily and orts were discarded once daily. Average daily feed intake (ADFI) was summarized weekly. Sows and piglets were weighed within 12 hours of parturition, and weekly thereafter. In parity 1, the deuterium dilution technique was employed to ascertain if the normal changes in body composition that occur during a parity were influenced by dietary treatments. In parity 1 and 2 lactation and in parity 2 gestation, dietary nutrient digestibility coefficients (DC) were estimated by the indicator method.

The ADFI of SBH sows was greater ($P<.10$) in parity 1, but not in parity 2 or overall. Sows consuming the SBH diet had a much lower ($P<.001$) incidence of

constipation at d 110 of gestation. There were no differences between treatments in any reproductive parameter measured. There were no statistically significant differences between treatments in the rate of body weight change for any period over the two parities. Similarly, data derived from the deuterium-dilution technique revealed no differences in body composition changes in any period between sows on either treatment in parity 1. However, ultrasonic measurement revealed that SBH sows lost more ($P<.10$) subcutaneous backfat in lactation than control sows. During gestation, SBH sows digested less ($P<.001$) dietary gross energy and crude protein and less ($P<.10$) ether extract. However, SBH sows digested more ($P<.05$) neutral detergent fiber and more ($P<.001$) acid detergent fiber than control sows during gestation. In lactation, sows from both treatments had similar dietary DC. There was no residual effect of the dietary treatment imposed in gestation, on dietary DC measured in lactation.

Introduction

Previous research (Chapter 2), indicated that SBH could be incorporated into swine gestation diets with a concurrent increase in the ADFI in lactation. Litter size was not standardized in that experiment. However, SBH sows consumed more feed than the control sows during lactation, even though they tended to wean almost 1 pig/litter less. To further study the implications of these results, this experiment was initiated. As suckling intensity is known to influence ADFI, litter size was standardized. Because the level of feed intake is known to impact changes in body composition, the deuterium dilution technique was employed to estimate body composition. As dietary fiber has been reported to influence DM digestibility, digestion coefficients (DC) were measured in

late gestation and early lactation. The latter to determine if there may be a residual effect of the dietary gestation treatment in lactation. The dietary changes that the fiber-fed sows make as they are switched from a high fiber gestation diet to a corn-soybean meal lactation diet is often radical in terms of physical form, nutrient compositon and taste. However, the change that control sows make from their corn-soybean meal gestation diet to their corn-soybean meal lactation diet is negligible. It is possible that this difference contributes to the elevated rate of ADFI in lactation of sows fed high fiber diets during gestation. In an attempt to reduce this disparity, a flavoring agent was added to the lactation diet so that all sows had what we perceived to be, a significant change in diet as they were switched from their gestation to lactation diets.

Materials and Methods

Thirty-three York x Landrace gilts were divided into three groups (about 11 gilts/group). To synchronize estrus among gilts, each group was moved from inside a finishing barn to an outdoor unit and were artificially inseminated (AI) with the semen of purebred Hampshire boar on expression of their second estrus. Gilts were heat-checked with boars twice daily and inseminated 12 hours after the onset of standing heat, and at 12 hour intervals thereafter for a total of three inseminations/gilt. After breeding and throughout gestation gilts were housed indoors in individual gestation crates over partially slotted floors. During parity 2, sows were housed in individual gestation crates post-weaning and bred via AI upon expression of estrus.

Table 8. Composition and calculated analysis of lactation diet used in Experiment 2 and 3[a].

Ingredient	% of total
Corn	69.25
Soybean meal (44%)	23.00
Soybean oil	3.00
Monodicalcium phosphate	2.20
Lime	1.15
Vitamin TM mix[b]	.50
Salt	.50
Biotin supplement	.15
Flavor[2]	.13
L-lysine HCL	.10
Folic acid supplement	.02
Calculated analysis	
Metabolizable energy, kcal/kg	3313.50
Crude protein, %	15.70
Lysine, %	.90
Crude fat, %	5.47
Calcium, %	.90
Phosphorus, %	.77

[a]Flavor was used in Experiment 2 only. In Experiment 3, corn replaced the flavor.

[b]Vitamin-TM premix, and Folic acid and Biotin supplements were supplied free of charge by Feed Specialties Co., Inc. VTM supplied the following amounts per kg of diet: 6600 IU vit A; 1210 IU vit D; 22 IU vit E; 3.37 mg menadione; 22 mg pantothenic acid; 33 mg niacin; 1 mg folic acid; 5.5 mg riboflavin; 28 mcg B_{12}; 577 mg choline; 122 mg Zn; 126 mg Fe; 61 mg Mn; 12.2 mg Cu; .52 mg I; .3 mg Se. Biotin supplement supplied .33 mg biotin/kg diet. Folic acid supplement supplied 1 mg folic acid/kg of diet.

Dietary treatments were initiated on the day of breeding and consisted of a corn-soybean meal (control) diet and a corn-soybean meal-soybean hull (SBH) diet. These two diets were also used in Experiment 1 (Chapter 2, Table 2). Gilts were allotted to treatment as they exhibited estrus with every other gilt receiving the SBH diet. On the day of farrowing, sows in both treatment groups were switched to a common corn-soybean meal diet (Table 8). This lactation diet contained .13% of a flavoring agent (Kent Feeds).

Sows were weighed at breeding, d 110 of gestation, within 12 hours post parturition, at d 7, 14 and 21 of lactation and at weaning. Backfat depths of sows were measured at breeding, d 110 of gestation and at weaning with an ultrasonic backfat probe (Renco Lean Meater, Serial #4458; Mpls., MN). Backfat depths were measured approximately 3 cm off midline at the last posterior rib and at the last posterior lumbar vertebrae, and the average of the two readings was recorded. Stillborn and live piglets were individually weighed within 12 hr of birth, at d 7, 14 and 21 of age and at weaning.

Apparant digestibility of dietary nutrients was measured in late gestation and early lactation. In parity 1 and 2, beginning on the day of farrowing and lasting for 12 days, a chromium-mordant fiber made from ground corn stalks (5.4% Cr) was added to the lactation diet of all sows at the rate of .28% to provide a ration concentration of 150 ppm chromium. In parity 2 at an average gestation length of 100 days and lasting for 12 days, the same mordant-fiber was again used to supply a similar dietary chromium concentration. In each lactation and in gestation, a 7 day adjustment period was followed by a 5 day collection period. In the collection period a grab sample of fresh manure was

collected twice daily (AM and PM) from each sow. Daily samples were pooled and frozen during the collection process. Prior to analysis, samples were dried for 72 hours at 55^{0}C and ground in a blender. Feed and fecal nutrient analysis was done using the following proceedures: gross energy content by adiabotic bomb calorimetry, starch by high performance liqued chromatography (HPLC) analysis of free glucose following incubation in sodium hydroxide and amylase, crude protein by a modified Hach total nitrogen procedure, ether extract by Soxhelet ether extraction, and fiber fractions by the Van Sooste detergent sequential fiber analysis technique. For specific protocols detailing laboratory procedures used for the analysis of feed and fecal nutrient content, see the Appendices.

Digestibility coefficients for gross energy (GE), crude protein (CP), ether extract (EE), ash, neutral detergent fiber (NDF), acid detergent fiber (ADF), and lignin were determined using the indicator method equation, Figure 1.

$$\% \text{ Digestibility of nutrient} = 100 - \left(100 * \frac{\% \text{ indicator in feed}}{\% \text{ indicator in feces}} * \frac{\% \text{ nutrient in feces}}{\% \text{ nutrient in feed}}\right)$$

Figure 1. Indicator method equation used to calculate digestibility coefficients.

In parity 1, body composition of gilts was estimated at three different time points via the deuterium oxide dilution technique. Body composition was estimated between

days 7 and 14 of gestation, at two days post-parturition and at 7 days post-weaning. The amount of empty body water, empty body fat, empty body protein, and empty body ash that was estimated at each time point was then used to calculate the change in body composition during each production phase (ie; gestation and lactation).

With the deuterium oxide dilution technique, gilts were infused with deuterium at the rate of .25g/kg body weight. Deuterium was infused through an ear vein following a 24 hour fast and 12 hours without water. Blood samples were collected via external jugular vein puncture at 2.5 and 3.5 hours post infusion. The deuterium concentration in the 3.5 hour post-infusion sample was compared to that in the 2.5 hour post-infusion sample to verify that deuterium equilibrium had occurred within the body. Whole blood samples were sublimated in liquid nitrogen and the resulting water was analyzed for deuterium concentration via high performance liquid chromatography. The equations of Rozeboom et al. (1994) were used to estimate body composition of gilts near breeding. The equations of Sheilds et al. (1984) were used to estimate body composition of sows post-farrowing and post-weaning.

Data Analysis

Data were analyzed with the General Linear Models (GLM) procedure of SAS to determine significant relationships between main effects (treatment, group and parity) and all interactions. When group, parity or an interaction was not significant, it was removed from the model, and the analysis was performed again. Means were separated by the PDIFF procedure within SAS.

Results and discussion

ADFI

In contrast to Experiment 1 where SBH sows had a significantly greater lactation ADFI, the pooled lactation ADFI for the two parities in Experiment 2 did not differ ($P>.19$) between treatments (Table 9). However, the SBH sows did have a numerically larger ADFI at each time point measured. When ADFI was analyzed by parity, statistical differences were apparent in parity 1. The SBH sows tended ($P<.12$) to consume more feed in week 1 of lactation. The ADFI of the SBH sows was significantly greater ($P<.08$) for the period from farrowing to d 21 of lactation and greater ($P<.10$) for the entire parity 1 lactation. During parity 2 there were no statistical differences between treatments in ADFI. These observations were different from those observed in Experiment 1, where differences in ADFI become significant during later parities. The reasons for the different response between experiments are unclear. Interestingly, sows from both treatments in this experiment had an ADFI about 1 kg more/d in each parity, than contemporary sows in Experiment 1.

Table 9. Lactation ADFI for Experiment 2, kg[a]

Treatment	Control	Soybean hull	P value
Mean ADFI, kg			
Week 1	4.7 ± .1	4.9 ± .1	.19
Week 2	5.8 ± .2	5.9 ± .2	.62
Week 3	6.0 ± .2	6.1 ± .1	.71
To d 21	5.5 ± .1	5.6 ± .1	.47
To weaning	5.6 ± .1	5.8 ± .1	.37
Days of lactation	26.1 ± .6	24.9 ± .5	.12
Parity 1			
Week 1	4.3 ± .2	4.6 ± .1	.12
Week 2	5.1 ± .2	5.5 ± .2	.18
Week 3	5.4 ± .2	5.8 ± .2	.14
To d 21	4.9 ± .1	5.3 ± .1	.08
To weaning	5.1 ± .1	5.4 ± .1	.10
Parity 2			
Week 1	5.3 ± .1	5.3 ± .1	.87
Week 2	6.3 ± .4	6.2 ± .3	.93
Week 3	6.6 ± .4	6.3 ± .3	.52
To d 21	6.0 ± .3	5.9 ± .2	.79
To weaning	6.0 ± .3	6.1 ± .2	.94

[a]Least square means ± SE

Constipation, body weight, body weight change and body composition changes

The SBH sows were significantly less (P<.001) constipated than the control sows at d 110 of gestation (Table 10). Differences in the degree of constipation had disappeared by d 3 of lactation and were also similar on d 10 of lactation. These results are nearly identical to what was observed in Experiment 1.

There were no significant differences between treatments in body weight of sows at any time point (Table 10). Similarly, there were no differences between treatments in body weight change of sows during any period. However, SBH sows tended (P<.11) to lose more body weight during week 3 of lactation. Backfat depths of sows on the control and the SBH diet were similar at every time measured. However, the backfat depth of SBH sows fluctuated to a greater extent than did that of the control sows. The SBH sows lost more (P<.09) backfat in lactation and gained more (P<.01) backfat in gestation than control sows.

During parity 1 there were no significant differences between treatments in the changes in body composition of any measured parameter in either gestation or lactation (Table 11). However, the increases during gestation and losses during lactation were numerically larger for all compartments of the control sows. The inclusion of SBH did not influence the body compositional changes of gilts consuming the test diet. Although the ADFI of SBH sows was greater in parity 1 lactation, it was not sufficiently larger to influence the degree of change in body composition.

Table 10. Sow weights, weight changes, and constipation scores in Experiment 2[a]

Treatment	Control	Soybean hull	P value
Weight, kg			
Breeding	151.6 ± 2.2	153.3 ± 1.8	.55
d 110 gestation	197.9 ± 2.9	199.9 ± 2.3	.59
Post-farrowing	179.7 ± 2.1	182.5 ± 1.7	.32
d 7 lactation	185.6 ± 2.3	186.8 ± 1.8	.68
d 14 lactation	183.9 ± 2.4	186.0 ± 1.9	.49
d 21 lactation	183.3 ± 2.7	183.6 ± 2.2	.94
Weaning	181.1 ± 2.9	183.9 ± 2.4	.44
Weight change, kg			
Gestation total	45.4 ± 2.3	45.9 ± 1.8	.88
Week 1 lactation	6.0 ± .9	5.0 ± .7	.38
Week 2 lactation	-2.1 ± 1.0	-1.5 ± .8	.60
Week 3 lactation	-.3 ± 1.1	-2.6 ± .9	.11
To d 21 lactation	3.9 ± 1.9	1.2 ± 1.5	.28
Lactation total	2.3 ± 2.1	1.6 ± 1.7	.81
Backfat depth, mm			
Breeding	20.6 ± .9	20.5 ± .7	.91
d 110 gestation	20.3 ± .8	21.9 ± .7	.15
Weaning	19.6 ± .8	19.9 ± .7	.79
Backfat depth change, mm			
Gestation	-.2 ± .5	1.8 ± .4	.01
Lactation	-1.0 ± .5	-2.2 ± .4	.09
Parity	-1.1 ± .6	-.5 ± .5	.53
Constipation score			
d 110 gestation	4.5 ± .1	3.2 ± .1	.001
d 3 lactation	3.3 ± .1	3.3 ± .1	.90
d 10 lactation	3.0 ± .1	3.0 ± .1	.85

[a]Least square means ± SE

Table 11. Changes in sow body composition in Experiment 2, kg[a]

Treatment	Control	Soybean hull	P value
During gestation			
Body weight	39.5 ± 1.3	37.3 ± 1.2	.21
Empty body weight	32.9 ± 1.2	30.8 ± 1.1	.20
Empty body water	14.4 ± 1.0	12.5 ± 1.0	.21
Empty body protein	1.9 ± .3	1.6 ± .2	.36
Empty body fat	10.0 ± .8	9.9 ± .8	.93
Empty body ash	.76 ± .1	.72 ± .1	.66
During lactation			
Body weight	-23.4 ± 2.3	-20.2 ± 2.1	.31
Empty body weight	-21.4 ± 2.1	-18.5 ± 1.9	.31
Empty body water	-8.4 ± 1.1	-7.4 ± 1.1	.54
Empty body protein	-1.5 ± .2	-1.2 ± .1	.26
Empty body fat	-10.1 ± 1.3	-8.4 ± 1.2	.35
Empty body ash	-.3 ± .03	-.2 ± .03	.70

[a]Least square means ± SE

Table 12. Changes in sow body composition by stage in Experiment 2, kg[a]

	Gestation		Lactation	
Treatment	Control	Soybean hull	Control	Soybean hull
Body weight	39.5 ± 1.8[b]	37.3 ± 1.7[b]	-23.4 ± 1.8[c]	-20.2 ± 1.7[c]
Empty body weight	32.9 ± 1.7[b]	30.8 ± 1.6[b]	-21.4 ± 1.7[c]	-18.5 ± 1.6[c]
Empty body water	14.3 ± 1.1[b]	12.5 ± 1.0[b]	-8.4 ± 1.1[c]	-7.4 ± 1.0[c]
Empty body protein	1.9 ± .2[b]	1.6 ± .2[b]	-1.5 ± .2[c]	-1.2 ± .2[c]
Empty body fat	10.0 ± 1.1[b]	9.9 ± 1.0[b]	-10.0 ± 1.1[c]	-8.6 ± 1.0[c]
Empty body ash	.7 ± .05[b]	.7 ± .05[b]	-.3 ± .05[c]	-.2 ± .05[c]

[a]Least square means ± SE

[bc]Means within a row lacking a common superscript are different (P<.001).

When treatments were compared by stage (gestation verses lactation), the body composition for both the control and the SBH sows changed similarly (Table 12). All sows gained weight of every compartment during gestation, and lost weight of every compartment during lactation.

Reproductive data

There were no differences between treatments in any measured reproductive variable (Table 13). The SBH sows farrowed and weaned a similar number of pigs/litter as the control sows. Additionally, the litter weights and litter weight gains of SBH litters were not different than those recorded for the litters from control sows.

Table 13. Sow reproductive performance in Experiment 2[a]

Treatment	Control	Soybean hull	P value
Number of pigs /litter			
Total born	10.4 ± .7	10.3 ± .6	.90
Live born	9.6 ± .7	9.9 ± .5	.74
Live at d 21	9.4 ± .2	9.7 ± .2	.29
Weaned	9.4 ± .2	9.7 ± .2	.22
Litter weight, kg			
Total at birth	14.9 ± .9	14.4 ± .7	.69
Live born	13.7 ± .8	14.2 ± .6	.67
d 21	54.0 ± 2.1	53.4 ± 1.6	.81
Weaning	66.0 ± 2.5	63.6 ± 2.0	.46
Litter weight gain, kg			
Birth to d 7	11.2 ± .6	11.2 ± 5	.96
d 7 to d 14	14.6 ± .8	14.0 ± .9	.53
d 14 to d 21	15.6 ± .9	15.4 ± .7	.86
d 21 to weaning	14.1 ± 1.4	13.7 ± 1.1	.79
Birth to weaning	52.8 ± 2.3	49.3 ± 1.9	.25

[a]Least square means ± SE

Apparant digestibility

During parity 1 lactation when sows from both treatments were consuming the same diet, there was no indication of a residual affect of the SBH diet on digestibility (Table 14). Both control and SBH sows digested similar amounts of dietary nutrients. During parity 2 gestation, SBH sows digested less ($P<.001$) dietary GE and CP and less ($P<.08$) EE (Table 14). However, the SBH sows digested more ($P<.05$) NDF and more ($P<.001$) ADF than control sows. These results agree with others (Kornegay, 1981; Bray et al., 1986; 1987) who have substituted SBH into the diet of gestating sows. The SBH sows had a lower ($P<.02$) DC for lignin than that for control sows. In parity 2 lactation there appeared to be a residual influence of the dietary soybean hulls that were fed during gestation. The SBH sows digested less ($P<.01$) CP from d 7 to 12 of lactation than the control sows. The SBH sows also had a higher ($P<.06$) DC for the dietary ash than the control sows.

When the apparant digestibility data from parity 1 and 2 lactations were pooled, the differences between treatments for CP and ash DC disappeared (Table 15). The control and SBH sows digested similar amounts of dietary nutrients during lactation. Results for the gestation DC for the pooled data was similar to the previous analysis; SBH sows digested less ($P<.001$) GE, less ($P<.10$) CP, and less ($P<.05$) EE and ash. However, the SBH sows did digest more ($P<.05$) NDF and ADF than the control sows.

The DC measured for EE of this experiment are within the range (36.7% to 57.95%) of those reported by Graham et al. (1985) for growing pigs. Additionally, the 11.7 unit decrease in EE digestibility for gestating sows on the SBH diet was similar to

Table 14. Nutrient digestibility coefficients (DC) for Experiment 2[a]

Treatment	Control	Soybean hull	P value
Parity 1 lactation			
Gross energy	80.6 ± .7	81.3 ± .7	.47
Crude protein	79.7 ± .8	79.9 ± .8	.82
Ether extract	30.1 ± 5.1	35.6 ± 5.1	.46
Ash	39.3 ± 2.1	37.8 ± 2.1	.63
NDF	46.0 ± 2.3	50.0 ± 2.3	.23
ADF	46.9 ± 3.1	52.8 ± 3.1	.20
Lignin	40.4 ± 2.1	43.5 ± 2.1	.30
Parity 2 gestation			
Gross energy	84.7 ± .6	79.8 ± .4	.001
Crude protein	79.9 ± 1.0	68.7 ± .7	.001
Ether extract	64.8 ± 10.7	41.4 ± 6.2	.08
Ash	32.9 ± 2.7	28.7 ± 1.5	.19
NDF	67.5 ± 2.0	72.4 ± 1.1	.05
ADF	62.6 ± 1.8	78.5 ± 1.3	.001
Lignin	26.8 ± 5.3	8.8 ± 3.7	.02
Parity 2 lactation			
Gross energy	81.6 ± .6	80.7 ± .4	.23
Crude protein	81.4 ± .6	79.1 ± .4	.01
Ether extract	34.5 ± 5.2	29.0 ± 3.7	.41
Ash	23.8 ± 4.2	34.6 ± 3.0	.06
NDF	55.9 ± 2.5	55.1 ± 1.7	.78
ADF	57.4 ± 3.3	52.8 ± 2.3	.27
Lignin	22.5 ± 4.3	27.3 ± 3.1	.39

[a]Least square means ± SE

Table 15. Comparison of digestibility coefficients (DC) by stage for Experiment 2

	Gestation		Lactation	
Treatment	Control	Soybean hull	Control	Soybean hull
Gross energy	85.7 ± .8[b]	80.1 ± .6[c]	80.9 ± .5[c]	80.9 ± .4[c]
Crude protein	82.0 ± 1.3[d]	69.5 ± .9[e]	80.3 ± .8[df]	79.5 ± .6[f]
Ether extract	55.8 ± 8.4[g]	44.1 ± 5.9[gh]	32.7 ± 5.0[h]	32.9 ± 4.3[h]
Ash	39.3 ± 3.9[g]	29.0 ± 2.8[h]	33.7 ± 2.3[gh]	36.4 ± 2.0[gi]
NDF	64.1 ± 2.8[g]	72.4 ± 2.0[h]	49.1 ± 1.7[i]	52.4 ± 1.4[i]
ADF	62.6 ± 3.4[g]	78.5 ± 2.4[h]	50.7 ± 2.1[i]	52.8 ± 1.8[i]
Lignin	26.8 ± 7.1[g]	8.8 ± 5.0[h]	32.7 ± 4.3[g]	34.0 ± 3.7[g]

[a]Least square means ± SE

[bc]Means within a row lacking a common superscript are different (P<.001).

[def]Means within a row lacking a common superscript are different (P<.10).

[ghi]Means within a row lacking a common superscript are different (P<.05).

the reduction in digestibility from adding fiber that was reported by Den Hartog et al. (1985), and Just et al. (1985) (10.8 and 8.0% respectively) for growing pigs.

When comparing stages (gestation verses lactation), control sows digested more (P<.001) GE in gestation than in lactation, while the DC of SBH sows for GE did not differ between gestation and lactation (Table 15). The SBH sows digested more (P<.10) CP in lactation than in gestation, while control sows digested similar amounts of CP in the two periods.

The DC for EE was depressed (P<.05) in lactation as compared to gestation for both the control and the SBH sows. Ash digestibility was lower ((P<.05) in lactation for

control sows, but ash digestibility improved (P<.05) in lactation for SBH sows.

The digestibility of structural carbohydrates was reduced in lactation. The DC for NDF and ADF were lower (P<.05) for both treatments during lactation. While the lignin DC in lactation improved (P<.05) for the SBH sows, the extent of lignin digestibility did not change between gestation and lactation for the control sows. Some of the differences observed between gestation and lactation in the degree of digestibility may be partially explained by the differences in the rate of passage between the two periods. As the sows were switched from the restricted amount of feed intake in gestation to the ad-libitum rate in lactation, the rate of transit through the GI tract may have increased. Digestive enzymes in the small intestine had less time to do their job, as did the microbes of the colon.

Conclusions

In many respects data from this experiment mirror observations made in Experiment 1. However, because it was only conducted over 2 parities (in contrast to the 3 in Experiment 1) some relationships may not have been as apparent. SBH sows did consume more feed in parity 1, and had less constipation. The elevated ADFI of SBH of SBH sows may in fact have been the result of the lower incidence of constipation which may be attributed to the shorter transit time of the digesta of SBH sows. This may have enabled SBH sows to consume more feed per unit of time.

During gestation, SBH sows digested less (P<.001) dietary gross energy and crude protein and less (P<.10) ether extract. However, SBH sows digested more (P<.05) neutral detergent fiber and more (P<.001) acid detergent fiber than control sows during

gestation. This observation may reflect the adaptability of the microflora within the GI tract of the SBH sows. The SBH sows may have cultivated a greater number of cellulolytic bacteria in their GI tract, similar to those sows of Varrel and Pond (1985). As sows on each treatment were switched to a common lactation diet, differences in digestibility disappeared. There was no residual effect of the treatment imposed in gestation, on dietary DC measured in lactation. Even if the higher number of cellulolytic bacteria could survive the new environment, the rate of feed passage of the ad-libitum fed sows probably increased to such an extent that they were unable to exert any influence.

At this inclusion rate of SBH (19.1%), dietary digestibility of GE and CP will be reduced. Therefore as was done in the present experiment, actual feeding rates of the gestation diet will need to be increased about 10% to supply similar daily nutrient intakes.

Although the differences in ADFI were significant in parity 1, the additional amount of feed consumed by the SBH sows was not sufficient to significantly influence changes in body weight or changes in body composition during that lactation.

Including SBH in sow gestation diets at the rate of 19.1% will not adversely affect, and may enhance sow performance during lactation. These observations may in part be related to the reduction in the incidence rate of constipation at or near the time of parturition.

CHAPTER 4

REVIEW OF THE LITERATURE PERTAINING TO CHOLECYSTOKININ (CCK)

Introduction

Consumer demand for lean pork has forced the swine industry to select for animals with reduced fat reserves. Inadvertently, there has been a concurrent reduction in voluntary feed intake (Riley, 1989). Although daily voluntary feed intake increases as lactation progresses, most lactating sows do not eat enough to maintain body weight during an average 3 to 4 week lactation period. Lactation weight loss is most rapid during the later stages of lactation as milk output peaks. Some sows have been observed to lose more than 1.5 kg body weight/day from days 21 to 28 of lactation (Chapter 2). Excessive weight loss can lead to delayed return to estrus intervals (> 10 days) following weaning, especially in the primiparous sow.

In lactating sows (Matzat, 1990) and in growing pigs (Pekas, 1985), voluntary feed intake limits productivity. Lactating sows which were superalimentated (SA) via a gastric fistula and received 20% more feed than those sows allowed ad libitum access to feed were able to preserve a greater amount of body mass and were also able to synthesize greater quantities of milk (Matzat, 1990).

Increasing voluntary feed intake of lactating sows could augment sow productivity and increase profit potential for the producer.

Regulation of voluntary feed intake

Voluntary feed intake is regulated by many unknown mechanisms collectively referred to as appetite (Pekas and Trout, 1990). Appetite and hunger are not one and the same. Whereas appetite is satisfied by palatability, hunger is satisfied by calories (ie; animals stop eating long before digestion makes calories available to the animal) (Houpt and Houpt, 1991). Although exact mechanisms of appetite regulation are poorly understood, many endogenous compounds are known to inhibit feed intake. Cholecystokinin (CCK) is one such compound that is known to suppress voluntary feed intake. Other endogenous agents are known to stimulate voluntary feed intake.

Table 16. Endogenous inhibitory and stimulatory agents of appetite.

Inhibitory	Stimulatory
Cholecystokinin	Gamma amino butyric acid (GABA)
Calcitonin	Growth hormone releasing factor (GRF)
Dopamine	Norepinephrine
Epinephrine	Opoid peptides (ie: *B*-endorphin)
Glucagon	Pancreatic peptides (ie: Neuropeptide Y)
Insulin	
Neurotensin	
Serotonin (5-OHT)	
Somatostatin	

A partial list (Leibowitz, 1986) of inhibitory and stimulatory agents of appetite are listed in Table 16.

Cholecystokinin: structure, function and sites of release

Cholecystokinin exists in at least 5 molecular forms containing from 4 to 39 amino acids. The sulfated form of the C-terminal octapeptide (CCK-8) is the most abundant and potent in the circulation (Granner, 1988). CCK peptides stimulate gallbladder contractions and pancreatic enzyme secretions, and inhibit gastric emptying in some species. However, neither endogenously released nor exogenously infused CCK inhibit gastric emptying in pigs (Rayner and Gregory, 1989). Cholecystokinin is synthesized in the I cells of the mucosa of the duodenum and proximal jejunum and is released into the portal blood after a meal in response to peptides, amino acids, long chain fatty acids, calcium and acid entering the small intestine (Granner, 1988). Fat components entering the small intestine are the most potent stimulants of meal-induced CCK release (Walsh, 1981). The release of peripheral CCK is under vagal control, and abdominal vagotomy abolishes the effect of meal-induced release of CCK (Smith et al., 1981; Verbalis et al., 1986; Linden et al., 1989). In addition to peripheral CCK-8, CCK-8 has also been found in central locations in brain and cerebral spinal fluid (Walsh, 1981). Similar to the meal-induced peripheral release of CCK, hypothalamic concentrations of CCK have been elevated post-prandialy in primates and cats (Schick et al., 1987; 1989, respectively). Because CCK does not cross the blood-brain barrier (Walsh, 1981) it may be assumed that there is production and release of CCK in the brain and cerebral spinal fluid.

Mechanism of CCK as a satiety factor

CCK participates in rapid, preabsorptive satiety in pigs (Anika et al., 1981) and other species. Intraperitoneal injections of partially purified CCK produced a dose related suppression of feed intake (Gibbs et al., 1973; Stallone et al., 1989), while lateral cerebral ventricle injections of CCK also depressed feed intake (Tsai et al., 1984) of rats. Systemic infusions of CCK-8 have reduced meal size of pigs (Houpt, 1983). Although the mechanism of CCK action in appetite suppression is not fully understood, evidence indicates that the satiety effects of exogenous (and perhaps endogenous) CCK depend on the activation of central monoaminergic systems (Tsai et al., 1884). Linden (1989) reported that intraperitoneal administration of CCK-8 restored levels of dopamine in the cerebral spinal fluid of rats mildly deprived of food. Dopamine is a known anorexergic (Leibowitz, 1986). Therefore, the satiety effect of CCK may in part result from central release of dopamine in response to peripheral CCK release. Stallone et al. (1989) proposed that exogenous CCK-8 acts at peripheral sites and exerts a satiety effect by increasing serotonin (5-HT) activity in the brain. Serotonin is also an anorexergic (Leibowitz, 1986). Cooper and Dourish (1990) proposed that CCK and 5-HT are mutually interdependent on each other in mediating satiety. Evidence from Kow and Pfaff (1986) suggest that the role of CCK-8 in satiety induction is two-fold: CCK-8 serves as a satiety agent in the periphery, mediated through vagal afferent nerves to the brain, and as a neurotransmitter in the brain to convey the information originating in the periphery. The results of Linden (1989) concur with these findings, indicating that peripheral CCK receptor mechanisms induce a release of CCK in the brain. In the brain

and in the periphery CCK may serve as a neurotransmitter (Kow and Pfaff, 1986; Linden, 1989). Ultimately, the satiety center of the hypothalamus is stimulated by these endogenous compounds, and the animal terminates the meal.

To date, two types of CCK-receptors have been identified. An A-type found in many peripheral locations, and a B-type found in the brain (Cooper and Dourish, 1990). To understand the role of each receptor type in the induction of satiety, Cooper and Dourish (1990) administered CCK receptor antagonists to rats. Devazepide, a selective CCK-A receptor antagonist, and L-365,260, a selective CCK-B/gastrin receptor antagonist, both increased feed intake in well-satiated rats. The CCK receptor antagonist L-365,260 was more potent. Only Devazepide was able to block the satiating effects of exogenously administered CCK. These data provide further evidence that satiety results from a combination of both peripheral and central CCK action.

Role of cholecystokinin in lactation

In lactating rats and sows, daily ad libitum feed intake generally increases as lactation length progresses. Flemming (1976) suggested that the energy expenditure associated with increasing amounts of milk synthesis is responsible for a large portion of this hyperphagia. However, other mechanisms, may also be involved in stimulating the hyperphagia of lactating rats and sows.

Daily feed intake is the sum of the number of meals eaten within a day and the size of each meal. In turn, meal size is a combination of the duration of time spent eating, and the rate of feed ingestion. CCK mediates its anoretic effects by reducing meal size (Leibowitz, 1986). Interestingly, the gradual increase in feed intake of lactating rats

occurs primarily because of an increase in meal size (Strubbe and Gorissen, 1980; McLaughlin et al., 1983) rather than an increase in meal frequency. Meal size becomes larger in lactating rats in spite of the fact that plasma CCK levels are elevated (Linden, 1989; McLaughlin et al., 1983). The elevated CCK concentrations could be partially explained by the fact that peripheral CCK is released in lactating rats (and dogs) in response to suckling stimulus (Linden et al. 1990). This release of CCK is immediate and short lasting, and in rats it is of a smaller magnitude than that seen after feeding (Linden et al. 1990). Nevertheless, even with elevated CCK concentrations, feed intake gradually increases as lactation progresses. Therefore, Linden (1989), McLaughlin et al. (1983), and Wager-Srdar et al. (1986) speculate that the marked hyperphagia of lactating rats reflects an insensitivity of the animals to the inhibitory effects of CCK on food intake as lactation length progresses; CCK has greater anoretic effects during earlier phases of lactation than during later stages of lactation. However, pancreatic hypertrophy, a known biological response to elevated CCK concentrations still occurs in lactating rats (McLaughlin et al., 1983). Therefore, CCK-insensitivity during lactation appears to be restricted to satiety. In contrast to others, Helmreich et al. (1991) did not observe a decrease in sensitivity to the anorexigenic effects of CCK as lactation progressed in rats.

CCK treatment has been shown to stimulate pituitary oxytocin secretion in lactating rats (Verbalis et al., 1986; Helmreich et al., 1991). The effect of CCK administration on oxytocin release was blunted by gastric vagotomy (Verbalis et al., 1986; Linden et al., 1990). Gastric vagotomy also blocks the inhibition of food intake induced by CCK (Smith et al., 1981; Verbalis et al., 1986). Collectively these

observations suggest that peripheral CCK elicits its effects on satiety and oxytocin release through gastric vagal fibers, and not directly on the brain in rats. Because the suckling-induced release of CCK precedes milk-ejection and the associated release of oxytocin in the rat, Linden (1989) speculated that the release of CCK in response to suckling may facilitate the milk-ejection reflex. CCK has also been implicated to play a physiological role in the maintenance of good maternal behavior as intracerebroventricular CCK infusions have been observed to block the disruption of maternal behavior in rats caused by the simultaneous injection of Beta endorphin (Felicio et al., 1991).

Immunization against endogenous CCK

To demonstrate that endogenous CCK participates in feeding behavior several research groups have injected CCK antibodies (CCK-AB) into animals. Circulating CCK-AB do not cross the blood-brain barrier, but are believed to sequester the peripheral, free circulating endogenous CCK making it unavailable to the CCK receptor. This phenomenon is known as immunoneutralization.

Exogenous CCK-AB administered via continuous lateral cerebral ventricular injection increased the feed intake by wethers (Della-Fera et al., 1981). Feed intake of Zucker rats has been increased by exogenous CCK-AB administration, and also by endogenous CCK-AB following active immunization against CCK (McLaughlin et al., 1985). Similarly, the feed intake and growth rate of growing pigs has been elevated (8.2 and 10.6% respectively) following immunization against desulfated CCK-8 conjugated to human serum globulin (Pekas and Trout, 1990). In another experiment Pekas (1993) reported elevated feed intakes of growing pigs following immunization against desulfated

CCK-8 conjugated to one of four different haptens; bovine serum albumin (BSA), human serum globulin (HSG), Keyhole limpet hemocyanin (KLH), or purified protein derivative (PPD).

Antibody titers to CCK-8 have also been successfully raised in sheep immunized against CCK-8, however feed intake of the immunized animals was not different than controls (Trout et al., 1989; Spencer, 1992). Spencer (1992) gave several possible explanations for this anomaly;

1) Although CCK-AB were raised, the amount may have been insufficient to effectively immunoneutralize the endogenously produced CCK.

2) The CCK-AB raised may have had too low an affinity to effectively prevent the hormone from binding to the receptor.

3) There may be an elevated production and secretion rate of endogenous CCK due to decreased negative feedback as CCK-AB sequestered CCK.

4) If endogenous CCK action is primarily by autocrine or paracrine means, it will be less affected by circulating AB.

In both experiments (Trout et al., 1989; Spencer, 1992), feed intake was actually slightly depressed on the days following booster vaccinations. Pekas and Trout (1990) reported similar observations in pigs. Spencer (1992) offered three explanations why the immunization procedure may actually potentiate rather than neutralize CCK action;

1) The CCK-AB may act like a plasma binding protein and protect the hormone from degradation thereby extending it's biological half-life.

2) The AB-bound hormone may be presented to the receptor in such a way as to enhance it's orientation at the receptor binding sites.

3) The AB-bound hormone may extend the hormone's transmembrane effectiveness by inhibiting internalization and clearance of the hormone.

Summary

CCK reduces feed intake both directly and indirectly by stimulating the satiety center of the hypothalamus. Active immunization against CCK increased feed intake in growing rats and pigs presumably through immunoneutralization of the endogenous CCK. Active immunization of lactating animals against CCK has not been examined to date.

Implications

If gestating sows were vaccinated against CCK, the resulting CCK-AB may increase voluntary feed intake during lactation. If so, sows may respond similarly to super-alimentated sows (Matzat, 1990) which synthesized greater quantities of milk while simultaneously preserving a greater amount of body mass. Elevated milk production may aid in piglet survival and enhance litter weaning weights. Preservation of body mass during lactation may contribute to timely rebreeding following weaning. If active immunization against CCK increases voluntary feed intake of lactating sows, it may be adopted by producers and become a standard management procedure in the future.

CHAPTER 5

ACTIVE IMMUNIZATION OF GILTS AGAINST CHOLECYSTOKININ (CCK) IMPROVES LACTATION PERFORMANCE

Abstract

On d 64 of gestation, 20 York x Landrace gilts were immunized with the desulfated C-terminal octapeptide of CCK conjugated to Keyhole limpet hemocyanin (KLH) and 4 gilts with KLH alone (control). Three booster doses of immunogen (B1, B2, B3) were administered at 14 d intervals after the primary dose. Prior to each vaccination and on d 7, 14 and 21 of lactation (LD7, LD14, LD21 respectively), blood samples were taken from all sows via external jugular vein puncture. Serum was harvested and frozen for later determination of CCK antibody (AB) titer. The CCK-AB titer determination followed procedures described by Pekas (1996a). At parturition a colostrum sample was collected from each sow and litter size was standardized to 10 piglets. Blood samples were collected from 2 piglets of each litter at LD7. Milk samples were collected from all sows on LD7, LD14, and LD21. During lactation, sows were fed twice daily and orts were discarded once daily. The average daily feed intake (ADFI) was summarized weekly. Sows and piglets were weighed weekly. The deuterium dilution technique was employed to ascertain if changes in body composition that occur during a parity were influenced by CCK immunization. For a 12 day period in late

gestation and again in early lactation, chromium oxide (Cr_2O_7) was added to the diet of all gilts to provide a ration concentration of .05% chromium oxide. In both gestation and lactation, a 5 day fecal collection period followed a 7 day adjustment period. Dietary nutrient digestibility coefficients (DC) were estimated by the indicator method.

Four CCK immunized sows were removed due to poor health in lactation. Antisera titers reached values greater than 1:10 in 15 of the remaining 16 treated animals. In lactation, specific binding of radio-iodinated CCK was not demonstratable at 1:10 dilution of serum from control sows or their piglets. Mean log titers of sow serum at B2, B3, LD7, LD14, and LD21 was .27, 1.10, 1.77, 1.67, and 1.58 respectively. Mean log titer of colostrum was 2.42, and that of milk was .50, -.003, and -.20 at LD7, LD14 and LD21 respectively. These log titers were highly correlated to each other ($P<.005$ in all cases). Mean log titer of piglet serum (n=30) at LD7 was 1.22. Regression analysis revealed that the ADFI of sows in week 3 (W3) of lactation was correlated with each sow serum titer value and with the titer of colostrum ($r^2 = .43$ to .67, $P<.01$ in all cases). The ADFI in W3 was highly correlated ($r^2=.68$, $P<.001$) with the average of the seven samples taken from the sow during lactation (ALTSS). The ADFI for the entire 21 d lactation was also correlated ($r^2= .37$, $P<.05$) with the ALTSS. Total litter gain was related ($r^2=.31$, $P<.05$) to and increased with the ALTSS and with the ADFI of sows in W3 ($r^2=.28$, $P<.05$) . Body compositional changes were not equivocally related to anti-CCK titer. Nutrient digestibility was not impaired as a result of CCK immunization, and appeared to be enhanced, and increase with titer in gestation.

It is concluded that the elevated feed intake expressed by sows with higher anti-CCK titers was used by the sow to produce more milk which resulted in greater litter weight gain.

Introduction

Studies conducted with swine have shown that performance is limited by voluntary feed intake. Superalimentation (through a gastric fistula) at 20 % above ad libitum intake increased rate of gain in growing pigs (Pekas, 1985), and increased milk secretion and litter weight gain while simultaneously reducing sow weight loss during lactation (Matzat, 1990). A series of studies with young rapidly growing pigs showed that active immunization against CCK stimulated feed intake and/or growth (Pekas and Trout, 1990; Pekas, 1993) in proportion to the titer elicited by the animal. Recently, Pekas (1996) reported that some CCK-antigens increase CCK antiserum titers in swine better than others.

Clinical studies with CCK have shown that exogenous CCK decreases the transit time of digesta through the GI tract (Mutt, 1980). Cholecystokinin is also responsible for pancreatic release of amylases, proteases and lipases. These enzymes function within the small intestine to facilitate digestion of dietary proteins, lipids and starches. The premise behind CCK immunization is that the anti-CCK antibodies generated by the animal sequester endogenous CCK. Therefore, we hypothesized that the nutrient digestibility of gilts with higher anti-CCK titers may be altered. There is no evidence in the literature pertaining to this question. Therefore, the apparent digestibility of several nutrients was estimated in late gestation and again in early lactation in primiparous gilts that had been

immunized against CCK.

Immunization of gestating and lactating swine against CCK has not been reported. Therefore, the present study was undertaken.

Objectives

1) To determine the effect of active immunization against CCK on the voluntary feed intake and productivity of lactating sows and their litters.

2) To determine if CCK antibodies are present in the colostrum and milk of vaccinated sows, and if CCK-AB are passed on to their piglets.

3) To determine if active immunization against CCK affects the magnitude of changes in sow body composition during gestation and lactation.

4) To determine if an active CCK-AB titer would modify nutrient digestion.

Materials and Methods

Vaccine Preparation

Although the sulfated form of CCK-8 is needed for biological activity, antibodies have been raised to the unsulfated form (Pekas and Trout, 1990) at much lower expense. Therefore, the purified C-terminal octapeptide of desulfated cholecystokinin (CCK-8) was used in this study. The conjugated CCK8:KLH antigen was custom prepared (Cambridge Research Biochemicals Inc., Wilmington, DE) using the glutaraldehyde condensation reaction (Harlow and Lane, 1988). Prior to each vaccination, the antigen was allowed to soak overnight in phosphate buffer. The antigen/buffer was then emulsified in Freund's adjuvant with the use of two, hypodermic syringes connected via a double-hub needle.

Sows

In lactating swine, depressed feed intake, extensive weight loss and delayed return to estrus following weaning are more common in primiparous sows. Therefore, thirty-three crossbred gilts were used and were housed in three groups (~10 gilts/group). To synchronize estrus among gilts, each group was moved from inside a finishing barn to an outdoor Cargill unit and were artificially inseminated (AI) with the semen of purebred Hampshire boars on expression of their second estrus. Gilts were heat-checked with boars twice daily and inseminated 12 hours after the onset of standing heat, and at 12 hour intervals thereafter for a total of three inseminations/gilt. Gilts were housed indoors in individual gestation crates over partially slotted floors throughout gestation.

At about d 64 (± 3 d) of gestation gilts were given their primary vaccination. The primary dose contained CCK-8-KLH conjugate emulsified in a solution of 50% Freund's complete adjuvant and 50% phosphate buffer (Pekas and Trout, 1990). Each 1 mL dose contained about 1 mg of antigen. Each dose was administered by subdermal-subcutaneous infusion at three loci in an infusion site on the neck posterior (5 to 10 cm) to the base of the ear. On days 78, 92, and 106 of gestation gilts received booster vaccinations (B1, B2, B3, respectively) containing the same amount of the conjugate, albeit administered in Freund's incomplete adjuvant. The order of vaccine administration among gilts within a group was reversed at each vaccination date. This was done in an attempt to reduce the possibility of the antigen settling-out of the adjuvant within the syringe, thereby resulting in a situation where some gilts would get less or more of the antigen. The time line of this experiment is summarized in Figure 2. Booster doses were

alternately administered on the opposite side of the neck. These vaccination intervals are similar to those used by Pekas and Trout (1990) in growing-finishing pigs. Our hypothesis was that the immune response of the gilts would be similar to that of the growing pigs used by Pekas (1993). Therefore, peak antisera titer should occur about 60 days after the primary vaccine or about 7 days post-parturition. As lactating laboratory animals (rats) become desensitized to the effects of CCK as lactation progresses, our goal was to minimize the inhibitory effect of CCK when it is greatest (early lactation). In support of this strategy, research shows that total lactation feed intake is greater for sows that eat more during early lactation (Moser, 1985). The control gilts were immunized following the same protocol used for the CCK-8 immunized group and the control vaccines were prepared in the same manner to provide equivalent amounts of KLH without conjugation to CCK-8. Both the CCK immunized and the KLH immunized control sows experienced the severe tissue reactions from the Freund's adjuvants. Therefore, values obtained from the KLH immunized control sows would reflect detrimental effects of the vaccination protocol on performance. Prior to each vaccination, and also on d 7, 14 and 21 of lactation (LD7, LD14, LD21), blood samples were taken from all sows via external jugular vein puncture. Serum was harvested and frozen for later determination of CCK antibody titer.

Because active immunization against CCK had not been reported in sows, it was not known if CCK-AB were passed in the colostrum and milk with other immunoglobulins. Therefore, a colostrum sample was taken from all sows within 12 hours post parturition to determine CCK antibody titer. Milk samples were collected

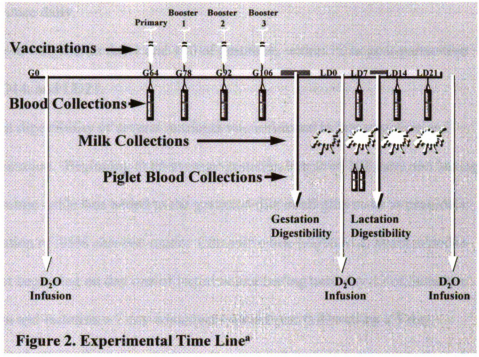

Figure 2. Experimental Time Line[a]

[a]Time code: G0=breeding; G64=d 64 gestation; G78=d 78 gestation; G92=d 92 gestation; G106=d 106 gestation; LD0=farrowing; LD7=d 7 lactation; LD14=d 14 lactation; LD21=d 21 lactation.

from all sows on LD7, LD14, and LD21. Milk samples were collected via manual expression approximately 5 minuets following a 3 mL dose of oxytocin administered intramuscularly.

Throughout gestation, gilts were fed 2.27 kg per head once daily of a common corn-soybean meal diet (See Control diet, Chapter 2, Table 2). In lactation all sows had ad libitum access to a corn-soybean meal diet (Table 12) and water. During lactation, sows were fed twice daily and feed disappearance was recorded at each feeding. Orts

were discarded once daily.

Sows were weighed on day 1 and 110 of gestation, within 12 hr post-parturition and on LD7, LD14, and LD21.

Apparent digestibility of several nutrients was estimated in late gestation and again in early lactation. Beginning at an average gestation length of 100 days and lasting for 12 days, chromic oxide was added to the gestation diet of all gilts so as to provide a ration concentration of .05% chromic oxide. Chromic oxide (.05%) was again added to the lactation diet beginning on day one of lactation and lasting until day 12 of lactation. In both gestation and lactation a 7 day adjustment period was followed by a 5 day collection period. In the collection period a grab sample of fresh manure was collected twice daily (AM and PM) from each sow. All the fecal grab samples from a sow within a collection period were pooled and frozen during the collection process. Prior to analysis, samples were dried for 72 hours at 55^0 C and ground in a blender. Digestibility coefficients (DC) for gross energy (GE), starch, crude protein (CP), ether extract (EE), ash, neutral detergent fiber (NDF), acid detergent fiber (ADF), and lignin were determined using the indicator method equation described in Chapter 3, Figure 1.

Body composition of gilts was estimated at three different time points via the deuterium oxide dilution technique. Body composition was estimated near breeding (d 7 to 14 of gestation), post-parturition (d 2 lactation) and post-weaning (7 d post-weaning). The amount of empty body water, empty body fat, empty body protein, and empty body ash that were estimated at each time point were then used to calculate the change in body composition during gestation and lactation (This technique is described in Chapter 3).

Piglets

Stillborn and live piglets were weighed individually within 12 hr of birth, and at LD7, LD14, and LD21. Because suckling stimulus induces release of both CCK and oxytocin, we attempted to equalize suckling intensity via standardization of litter size among sows. Therefore, the number of pigs per litter was standardized by day 2 of lactation to 10 piglets. Nonexperimental sows provided extra piglets when needed. A blood sample was collected from two piglets (one male and one female) of each litter at LD7. This was done to determine if CCK-AB that may have been present in colostrum were absorbed by the pig. Serum was harvested and frozen for later determination of CCK antibody titer. Creep feed was not offered to piglets during lactation. Litter weight gains were used to assess sow milk production.

Lab analysis

Serum, colostrum and milk anti-CCK serum binding titers were estimated by radioimmunoassay (Pekas, 1996). Bolton-Hunter ^{125}I-labeled CCK8s (sulfated; 2,200 Ci/mmol; Dupont, Boston, MA) was the radiolabeled antigen. Each assay tube contained 4,885 dpm or 1.0 fmol of Bolton-Hunter ^{125}I-labeled CCK8s. Antiserum titers were computed from the specific binding of Bolton-Hunter ^{125}I-labeled CCK8s at four dilutions (1:10, 1:100, 1:1,000, and 1:10,000) in phosphate buffered saline (.01 M, pH 7.2) containing .05% gelatin (Sigma; #G-2500). The antiserum titer is defined as that serum dilution that gives 50% specific binding of Bolton-Hunter ^{125}I-labeled CCK8s, and was computed using a linear regression of the logit of specific binding percentage verses the log of the serial dilution factor. For the specific protocols detailing laboratory procedures

used in the analysis of CCK-AB titer and feed and fecal nutrient analysis, see the Appendices.

Data analysis

Linear regression (SAS for Windows, 1994) was used to estimate the association of CCK-AB titer with sow and litter production data, with sow nutrient digestibility, and with the change in body composition data. This was based on previous reports (Pekas and Trout, 1990; Pekas, 1996) that showed that feed intake and growth were proportional to the anti-CCK-AB elicited, and that comparison of immunized to control animals by analysis of variance was not meaningful because a proportion of immunized pigs had undetectable or very low antiserum titers. The number of KLH immunized (control) sows were few and were included principally to verify that regression intercept coefficients derived from CCK immunized sows were reasonable.

Results and Discussion

Twenty four gilts conceived and farrowed (20 CCK-KLH immunized gilts and 4 KLH-KLH control gilts). Of the 20 CCK:KLH immunized gilts, four became ill and one did not elicit a detectable antiserum titer. These five sows were removed from the data base. Therefore, unless stated otherwise, n=15 in all analyses.

Anti-CCK titers of serum, colostrum, and milk

Basal nonspecific binding of the radiolabelled CCK8 antigen, by serum collected before the primary injection of immunogen, was scarcely detectable and only at the 1:10 dilution. This was true for both the KLH:KLH control gilts and the CCK:KLH immunized gilts. Specific binding of [BH-^{125}I]CCK8s in serum collected from

CCK:KLH sows immediately before the primary (P) dose injection was less than 2% at 1:10 dilution and was not detectable at 1:100, 1:1000, or 1:10,000 dilution; consequently antiserum titers could not be computed. Similarly, antiserum titers could not be computed for serum collected from most CCK8:KLH immunized sows at the first booster (B1). The progression of serum, colostrum and milk titers of the 15 CCK8:KLH immunized sows and the average piglet titer is illustrated in Figure 3. Mean (±SEM) log titers of serum at B2, B3, LD7, LD14, and LD21 was .27 (±.07), 1.10 (± .28), 1.77 (± .46), 1.67 (± .43), 1.58 (± .41) respectively. Variation of serum log titer values was high, with the coefficient of variation ranging from between 67% and 73% during lactation. This wide variation of titer was typical of the variation observed in young pigs immunized against CCK (Pekas and Trout, 1990, 1993; Pekas 1996). The peak average log titer of sow serum, 1.77 (equivalent to linear titer 1:59) occurred at LD7; thereafter the log titer decreased to 1.58 (equivalent to linear titer 1:38) at LD21. The pattern of immune response elicited by these primiparous sows was similar to that of growing pigs where peak titers were observed about 60 days after the primary dose of immunogen (Pekas and Trout, 1990, 1993; Pekas, 1996). Similarly, the peak log titer obtained with the CCK8:KLH immunogen in young pigs was 1.9, (equivalent to linear titer 1:80, Pekas, 1996).

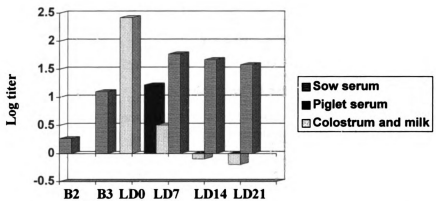

Figure 3. CCKAB titers for sow and piglet serum, colostrum and milk

The mean log titer of defatted colostrum was 2.42 (± .624, CV = 47.9%) which was 2.3-fold higher than the log titer of sow serum at B3, and about 1.3-fold higher than the log titer of sow serum at LD7. On a linear titer basis, where the titer of colostrum is 1:263 and of serum at LD7 is 1:59, colostrum titer is 4.46-fold higher than the peak sow serum titer.

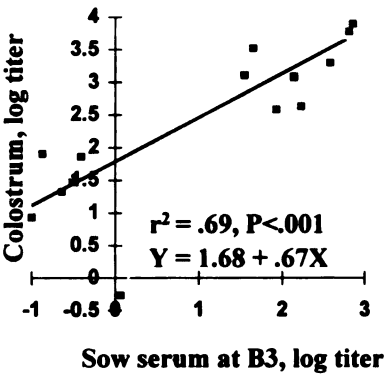

Figure 4. Relationship between log titer of sow serum and colostrum.

The regression equations show that colostrum titer increased .65 log unit for each log unit increase in sow serum titer measured at B2 (R^2= .53, P<.01), and .67 log unit for each log unit increase in sow serum titer measured at B3 (R^2= .69, P<.001) (Figure 4).

Titers of the defatted milk samples were relatively low compared to titers of colostrum or to those of sow serum (Figure 3). Mean log titers of defatted milk at LD7, LD14, and LD21 were .50 (± .130), -.003 (± .009), and -.20 (± .05) respectively. Milk titer at LD7 increased .77 log unit for each log unit of increase of sow serum titer measured at LD7.

Table 17 summarizes the correlation coefficients, r, of the log titers of all sow samples. The correlation coefficients between log titers of serum, colostrum, and milk samples were high, ranging from .67 to .99 (P <.005 in all cases), and demonstrate that the CCK-AB titer of all samples taken from sows are highly interdependent.

These data imply that the concentration of CCK-AB in colostrum and milk are

dependent on the concentration of CCK-AB in sow serum. The CCK immunoglobulins are sequestered by the mammary gland and concentrated in colostrum at considerably greater concentrations than that observed in the circulating serum. The role that anti-CCK immunoglobulins may play in CCK immunoneutralization before being transferred and accumulated in colostrum and milk is not known. For this reason the immunoglobulins in colostrum and milk, accounted for by titer, are included in these analyses. For comparative purposes, the log titer (mean=1.22, CV = 125.3%) of piglet serum (n=30) at LD7 is also presented in Figure 3.

Table 17. Matrix table of correlation coefficients, r between all pairs of log titer values for sow serum (SS), colostrum (C) and milk (M)[a]

Sample	SS					C	M		
Time[b]	B2	B3	LD7	LD 14	LD 21	LD 0	LD7	LD 14	LD 21
SS-B2	1.00	.88	.69	.74	.70	.73	.76	.76	.67
SS-B3		1.00	.84	.89	.84	.83	.87	.78	.74
SS-LD7			1.00	.98	.99	.69	.88	.81	.78
SS-LD14				1.00	.97	.78	.86	.83	.80
SS-LD21					1.00	.68	.86	.84	.81
C						1.00	.74	.76	.74
M-LD7							1.00	.93	.90
M-LD14								1.00	.98
M-LD21									1.00

[a]Probability of greater r value is P <.005 in all cases listed.
[b]Time code: B2=Booster 2; B3=Booster 3; LD0=Farrowing; LD7, LD14, and LD21= d 7, 14 and 21 of lactation respectively.

Anti-CCK titers of piglet serum

Anti-CCK-AB titers in the serum of piglets (two piglets per litter), were observed (mean log titer =1.22) and believed to have been derived passively through absorption following transfer of anti-CCK immunoglobulins from the sow to colostrum. Although piglet blood samples were not taken prior to nursing, it is assumed that the CCK-AB were passively absorbed in the first 12 to 24 hours after birth. Specific binding of radio-iodinated CCK was not demonstratable at 1:10 dilution in serum from piglets nursing

control sows or from non-experimental piglets that had been transferred on to immunized sows at 1 or 2 d of age.

Piglet titers were highly correlated (r^2= .79, P<.001) with the colostrum titers from the sow being suckled (Figure 5), and increased 1.16 log units for each log unit of increase in colostrum .

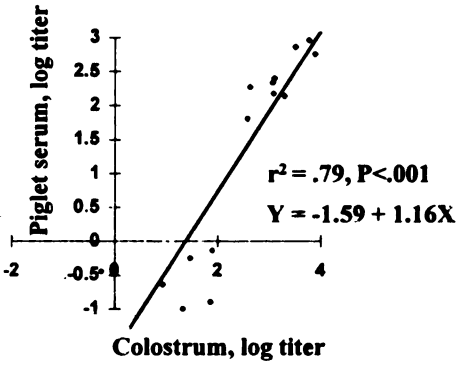

Figure 5. Relationship between log titer of colostrum and piglet serum.

As stated earlier, the titer of colostrum was closely associated with titer of sow serum (Table 17). Therefore, titer of piglet serum was expected to, and found to be associated with sow serum titer measured at each time point (r^2 = .62 to .89, P<.001 in all cases). Because piglets derived their titer from colostrum, which was supplied immunoglobulins from sow serum, the B3 serum titer of the sow should best reveal relationships of sow titer to that of piglet titer. Regression analysis disclosed that this was true. Piglet titer increased 1.0 log unit for every one unit increase in B3 sow serum titer (Figure 6).

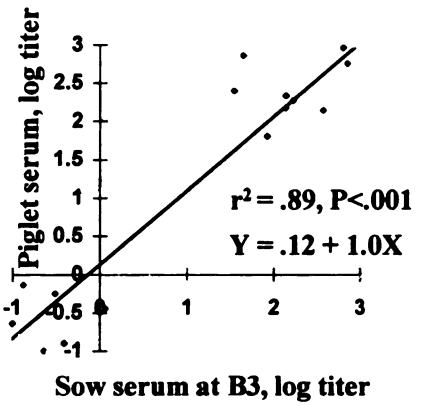

Figure 6. Relationship between log titer of sow serum and piglet serum

Feed intake and response to CCK immunization

The ADFI (± SE) during the first, second, and third week of lactation, and for the entire 21 d period was 4.19 (±.21), 4.98 (±.36), 5.62 (±.34) and 4.93 (±.24) kg/day for the four control sows, and 4.24 (±.10), 4.93 (± .18), 5.07 (± .19), and 4.75 (±.13) kg/day for the CCK immunized sows.

Correlation coefficients, r, were computed to relate ADFI during the first, second, and third week, and for the total 21-day lactation period with the log titers of serum, defatted colostrum, and with the average log titer of the seven samples (ALTSS) taken from each sow during lactation (Table 18). The seven samples included the three samples of sow serum, the one sample of defatted colostrum, and the three samples of defatted milk. The ALTSS is an indicator of the overall immune response executed by sows during lactation.

The ADFI during week 1 and week 2 of lactation were not significantly correlated

with log titer of any of the serum samples, but was correlated with log titer of colostrum ($P < .05$ for week 1; $P < .10$ for week 2). However, the ADFI during week 3 was highly correlated with log titer of each sample of serum ($P < .01$), and especially with colostrum and the ALTSS ($P < .001$). This evidence indicates that the feed intake response to CCK immunization is either dependent on the peak titer, which was not expressed until the second week of lactation, or is subject to a time delay after a minimal threshold titer was expressed. The ADFI over the entire 21-day lactation was correlated with log titers of samples of serum ($P < .10$), and especially with colostrum ($P = .002$), and with the ALTSS ($P = .02$). Although the strongest relationships between ADFI and CCK-AB titer were observed during week 3, the ADFI over the 21-day lactation did increase with CCK-AB titer.

Table 18. Correlations, r of ADFI with the log titer of sow serum and defatted colostrum.

	ADFI, kg			
Period	Week 1	Week 2	Week 3	Weeks 1,2,3
Sow serum				
Booster 2	.157	.177	.655[a]	.432
Booster 3	.172	.167	.718[a]	.462[c]
Lactation, d 7	.056	.132	.716[a]	.415
Lactation, d 14	.128	.123	.734[a]	.440[b]
Lactation, d 21	.065	.143	.705[a]	.417
Colostrum	.543[c]	.433[b]	.819[d]	.725[a]
ALTSS[e]	.294	.312	.822[d]	.608[c]

[a]Probability of greater r value, P<.01.
[b]Probability of greater r value, P<.10.
[c]Probability of greater r value, P<.05.
[d]Probability of greater r value, P<.001.
[e]ALTSS=Average log titer of seven samples taken during lactation; sow serum at d 7, d 14, and d 21 of lactation, colostrum, and milk at d 7, d 14 and d 21of lactation.

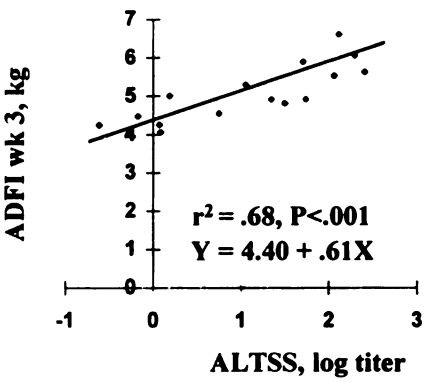

Figure 7. Relationship between average log titer of seven sow samples (ALTSS) and ADFI in week 3.

The relationship between feed intake during week 3 of lactation and the ALTSS taken from sows during the 21-day lactation was examined by linear regression. The quantity of ADFI during week 3 of lactation (Y; kg/day) as a function of ALTSS (X) is described by the equation: Y = a + bX, where a = 4.40 kg/day (intercept) and b = .615 (slope coefficient), illustrated in Figure 7. Using the ALTSS mean (1.1) and standard deviation (SD=1.0), this equation estimates that ADFI during week 3 would be about 4.4, 5.1, 5.7, or 6.3 kg/hd when the ALTSS was 0 (no titer), 1.1 (the mean), 2.1 (mean + 1 SD), or 3.1 (mean + 2 SD) respectively. These estimates represent a 0, 16, 30, or 43% increase of feed intake compared to sows with an anti-CCK titer of zero.

Body weight loss and response to CCK immunization

Means (± SEM) of body weight change of sows during week 1, 2, 3, and for the entire 21-d lactation are presented in Table 19. The CCK immunized sows gained an average 1.67 kg/hd body weight during the 21-day lactation period. This net weight gain was the result of 4.75 kg/sow gain in week 1 of lactation, followed by weight loss in week 2 and week 3 of lactation.

Table 19. Summary of sow body weight changes during lactation by weekly periods and correlations, r with the log titer of serum, colostrum and milk.

	Body weight change (kg/period)			
Period	Week 1	Week 2	Week 3	Weeks 1,2,3
Means	4.75	-2.89	-0.18	1.67
SEM	1.41	1.03	.88	1.76
Correlation coefficients, r				
SS-B2[b]	.276	.099	.374	.464[a]
SS-B3	.101	.114	.242	.268
SS-LD7	-.173	.002	.222	-.027
SS-LD14	-.084	-.106	.288	-.014
SS-LD21	-.174	-.019	.221	-.040
Colostrum	-.064	.156	.242	.159
ALTSS[c]	-.086	.057	.302	.115

[a]Probability of greater r value, P<.10

[b]Time code: B2=Booster 2; B3=Booster 3; LD0=Farrowing; LD7, LD14, and LD21= d 7, 14 and 21 of lactation respectively.

[c]ALTSS=Average log titer of seven samples taken during lactation; SS-LD7, SS-LD14, SS-LD21, Colostrum, M-LD7, M-LD14, MLD21.

Body weight loss of sows during lactation is normal under conventional husbandry practices. The possibility that CCK immunization, and the associated increase in feed intake would suppress, or even reverse body weight loss during lactation was examined by correlation analysis. As for the analysis of feed intake response, correlation coefficients between body weight change variables and log titer variables were computed (Table 19). Although trends of total body weight change during lactation indicated that the weight losses during week 2 and especially those during week 3 were suppressed, the effect was not clearly associated with anti-CCK titers of serum, colostrum or milk. Only total sow body weight change over the 21-d lactation was associated ($P<.10$) with the log titer of sow serum, and then only that titer measured at the second booster (B2).

Response of body composition and backfat thickness to CCK immunization

Body composition of sows was determined both during gestation and lactation. Net change of the various body compartments (including: body weight, empty body weight, empty body water, empty body protein, empty body fat, and empty body ash) is summarized in Table 20. It is imperative to note that the published procedures and associated equations for the deuterium dilution technique of estimating body composition were established for and mandate specific infusion times relative to the life-cycle of the sow. Therefore, results of sow weight changes for a particular period, derived from the D_2O procedure may not mirror previously described sow body weight changes within this text. Nevertheless, as all sows were treated similarly, the collected data are considered informational. All body compartments were enlarged during gestation and all were reduced during lactation.

Table 20. Body weight change and empty body (EB) composition changes of CCK-immunized sows during gestation and lactation, kg[a]

Time	Weight	EB weight	EB water	EB protein	EB fat	EB ash
Gestation	39.4 ± 6.4	33.0 ± 5.9	11.7 ± 4.0	2.7 ± 1.1	12.4 ± 4.8	.9 ± .2
Lactation	-20.1 ± 7.3	-18.5 ± 6.8	-6.2 ± 3.9	-1.3 ± .5	-9.7 ± 5.0	-.2 ± .1

[a]Mean ± SD.

This trend was expected. An important objective was to establish if CCK immunization might influence the magnitude of these changes. Therefore, correlation coefficients between the body compartment mass changes vs. the log titers of a variety of samples were computed. With rare exception (sows with higher LD7 and LD14 serum titers lost more [r=.44, P<.10] EB ash in lactation), there was no association between compartment mass change and log titers, either during gestation nor lactation. This evidence, combined with evidence that body weight change was not associated with log titer, indicates that immunized sows with higher titers utilized the nutrients from their additional feed intake for the synthesis and secretion of milk to feed their piglets.

The possibility that CCK immunization, and the associated increase in feed intake would change the rate of backfat depth dynamics (depletion or deposition) was examined by correlation analysis. Correlation coefficients were computed for backfat depth (measured via ultrasound), and change verses the log titers of serum, colostrum, and ALTSS. The results are summarized in Table 21, and reveal that backfat depth at LD21

Table 21. Sow backfat depth and change, mm and correlations, r with the log titers of serum, colostrum and milk.

	Backfat depth		
Item	Breeding	Lactation d 21	Parity change
Means	22.6	20.4	-2.1
SD	2.4	3.5	2.6
		r	
Sow serum			
Booster 2	.12	-.17	-.34
Booster 3	.05	-.32	-.49[a]
Lactation, d 7	-.03	-.55[b]	-.73[c]
Lactation, d 14	-.06	-.52[b]	-.65[c]
Lactation, d 21	-.08	-.60[b]	-.74[c]
Colostrum	.03	-.30[b]	-.44
ALTSS[d]	.01	-.51[b]	-.71[c]

[a]Probability of greater r value, P<.10.
[b]Probability of greater r value, P<.05.
[c]Probability of greater r value, P<.01.
[d]ALTSS=Average log titer of seven samples taken during lactation; sow serum at d 7, d 14, and d 21 of lactation, colostrum, and milk at d 7, d 14 and d 21of lactation.

is negatively associated with the log titer of samples collected during lactation. The association of backfat depth change and CCK-AB titer was explored by linear regression analysis to obtain an estimate of quantitative response expressed in terms of the log titer. Using sow serum at LD21, the regression equation and coefficients are:$Y = a + bX$, where Y = backfat loss (mm), a = .49, b = -1.67, and X = log titer of serum at LD21, (r^2

= .55, P < .01). Thus sows lost an average 1.67 mm of backfat for each one unit increase in log titer of sow serum at LD21. Substituting the ALTSS in the equation; a = -.09, b = -1.85 (r^2 = .51, P<.01), predicts that sows lost an average 1.85 mm for each 1 unit increase in log titer (Figure 8).

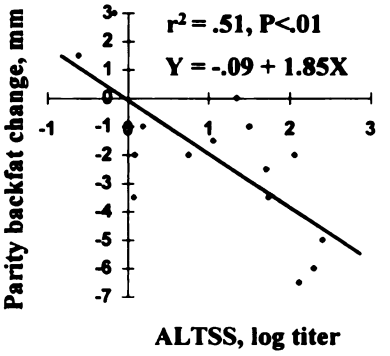

Figure 8. Relationship between average log titer of seven sow samples (ALTSS) and backfat depth change.

Litter performance

Means of litter weight gains and correlation coefficients between litter gain variables and log titer variables of the sow and of the piglets are summarized in Table 22. The litter weight gain that occurred in week 1 and in week 2 of lactation was not associated with any single titer measurement derived from the sow. However, week 3 litter gains were correlated with every sow serum titer, colostrum, and with the ALTSS. Week 3 litter gains were highly correlated ($r=.84$, $P<.001$) with sow serum titers measured at LD7, LD14, and with that measured at LD21. Total litter weight gain was related to every lactation sow serum titer and to the ALTSS, but not to colostrum. Linear regression analysis showed that total litter weight gain increased 4.0 kg for each one unit increase in ALTSS ($r^2=.41$, $P<.01$, Figure 9).

Table 22. Summary of litter weight gains during lactation by weekly periods and correlations, r with the log titer of serum, colostrum and milk.

	Litter weight gains (kg/period)			
Period	Week 1	Week 2	Week 3	Total
Means	10.1	12.8	13.1	35.9
SD	2.9	1.8	3.3	6.2
	Correlation coefficients			
Sow serum				
Booster 2	.04	.10	.59[a]	.27
Booster 3	.15	.07	.70[b]	.47[c]
Lactation, d 7	.39	.36	.84[d]	.73[b]
Lactation, d 14	.36	.24	.84[d]	.69[b]
Lactation, d 21	.40	.32	.85[d]	.73[b]
Colostrum	.29	.06	.51[c]	.39
ALTSS[e]	.35	.23	.77[d]	.64[a]
Piglet titer, d 7	.26	.16	.73[b]	.56[a]

[a]Probability of greater r value, P<.05
[b]Probability of greater r value, P<.01
[c]Probability of greater r value, P<.10
[d]Probability of greater r value, P<.001
[e]ALTSS=Average log titer of seven samples taken during lactation; sow serum at d 7, d 14, and d 21 of lactation, colostrum, and milk at d 7, d 14 and d 21of lactation.

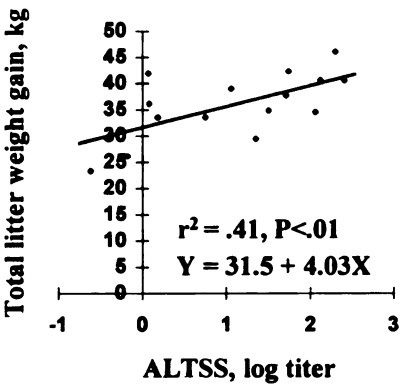

Figure 9. Relationship between average log titer of seven sow samples (ALTSS) and litter weight gain

Although confounded with other variables, litter gain in week 3 of lactation was correlated (r=.73, P<.01) with the log titer of serum from the piglets themselves, as was the total gain of the litter (r= .56, P<.05) (Table 22). Regression analysis showed that litters gained an additional 2.2 total kg in lactation for every 1 log unit increase in their serum titer (Figure 10).

Litter weight gain during week 3 of lactation was also correlated (r^2 =.44, P<.01) with sow feed intake during week 3 of lactation. The linear regression equation shows that litter weight gain increased 3.0 kg per week (.43 kg/d) for each 1 kg increase in ADFI during week 3 (Slope = 3.0). Total litter weight gain over the 21 d lactation period was also correlated (r^2 = .28, P<.05) with sow feed intake during week 3 of lactation (Figure 11) and similarly increased 4.4 kg for every 1 kg increase in ADFI of the sow during week 3 of lactation.

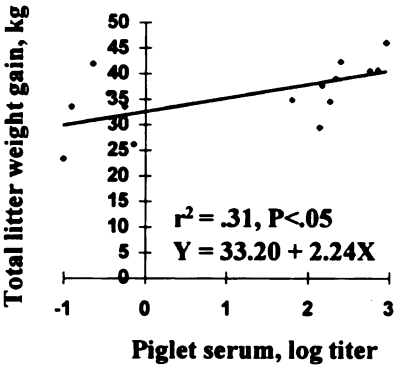

Figure 10. Relationship between log titer of piglet serum and litter gain

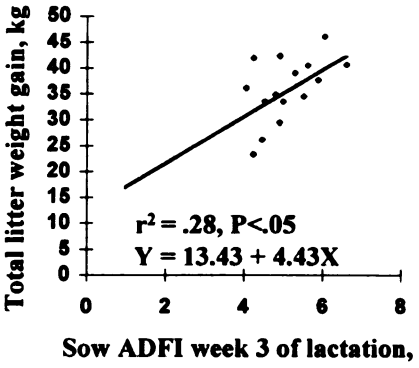

Figure 11. Relationship between sow ADFI and litter weight gain

As stated previously, the feed intake of sows was elevated in proportion to the CCK-AB titer. Because of the co-variation between feed intake of the sow and the CCK-AB titer of the sow, the regression analysis of litter weight gain with either sow feed intake or sow CCK-AB titer is confounded. There is also co-variation between piglet titer and sow titer. Therefore, it can not be said if the elevated rate of litter weight gain can be attributed to the higher rate of ADFI by the sow, to the CCK-AB titer level of the sow, or to the CCK-AB titer of the pig.

However, when considering the strength of the different calculated relationships, several observations can be made: The best association of week 3 ADFI of the sow and sow serum titer is that measured at LD14 (r^2=.54, P<.01). Similarly, the sow serum titers measured at LD14 and LD21 showed the strongest relationships (r^2=.71, P<.001) with week 3 litter gains. The sow's ability to synthesize milk will ultimately limit the growth rate of the pig, no matter what the piglet's appetite may be. Nevertheless, the associations between week 3 litter gain and colostrum (from which the pigs derived their titers) (r^2=.26, P<.05), and week 3 litter gain and piglet titer (r^2=.54, P<.01) are not as robust as the relationship between sow serum titer and litter weight gain. Because the ADFI of the sow was dependent on, and increased as log titer of the sow increased, it seems logical that the increase in litter weight gain is a direct result of the elevated feed intake of the sow. Since piglets in this study did not have access to creep feed, it is assumed that the additional litter gain was derived from an elevated secretion rate of milk by the immunized sows.

Interestingly, litter weight gain was positively associated with feed intake and sow

titer, while sow weight during the same period changed little and was not associated with titer. This implies that the additional nutrients derived from the elevated ADFI were prioritized for milk synthesis over maintenance. Litter weight gains and sow weight change and composition changes are associated negatively. Sows that produced heavier litters at LD21 accumulated less weight themselves in lactation (r^2 = -.35, P<.05). Additionally, sows producing heavier litters lost more (r^2 = -.33, P<.05) EB protein, more (r^2 = -.42, P<.01) EB ash, more (r^2 = -.26, P<.10) EB fat, and more (r^2 = -.81, P<.001) subcutaneous backfat. It appears that the elevated ADFI and associated higher levels of some dietary nutrient(s) may have stimulated milk synthesis. However, other dietary nutrients were insufficient to prevent body catabolism.

Digestibility response to CCK immunization

The gestation fecal samples for one sow were inadvertently not collected. Therefore, gestation DC data include only 14 observations. The relationship between CCK-AB titer and the digestibility coefficients obtained during gestation were best described using those titers obtained in gestation; B2 and B3. For a time reference, the B2 and B3 serum titer values were measured approximately 16 and 1 d respectively, prior to the beginning of the gestation digestibility collection period. The DC measured during gestation for GE, starch, CP, EE, and ash were correlated with CCK-AB titer level and increased proportionably with the titer of the gilt (Table 23). As CCK-AB titer at B3 increased, sows were able to extract more (r^2=.26, P<.10) GE and CP, more (r^2=.21, P<.10) EE, and more (r^2=.25, P<.10) ash from the gestation diet. Similarly, sows with higher anti-CCK-AB titers at B2 and B3 were able to utilize more dietary starch (r^2 =.30,

P<.05; r^2=.36, P<.05 respectively). The ability of sows to utilize fibrous components of the diet was not influenced by CCK-AB titer level. The DC calculated during gestation for NDF, ADF, and lignin were not correlated with any CCK-AB titer of the gilts. This observation seems logical as the enzymes responsible for fiber digestion are not synthesized by the pancreas but are rather microbial in nature. During lactation (LD8 to LD12) dietary nutrient digestion was not influenced by CCK-AB titer. However, there was a trend (r^2 =.19, P=.103) for the ash DC to increase as CCK-AB titer measured at d 14 of lactation increased.

Table 23. Regression coefficients, r^2 for CCK-antibody titer and dietary DC in gestation.

Digestibility coefficients	GE	Starch	CP	EE[a]	Ash
Booster 2	NS[b]	.30[c]	NS	NS	NS
Booster 3	.26[d]	.36[c]	.26[d]	.21[d]	.25[d]

[a]Ether extract
[b]Not statistically significant
[c]P<.05.
[d]P<.10.

The mean calculated DC measured during gestation and lactation for CCK immunized sows are presented in Table 24. In both gestation and lactation, the digestion of dietary starch was nearly complete (DC = 98.9 and 98.3 respectively). These values are close to those observed by Graham et al. (1985) who reported fecal starch DC ranging from between 98.9 to 99.4 in growing pigs. Others (Jorgensen et al., 1985; Just et al.,

1985) have reported whole tract starch DC of 100. During lactation, sows tended (P=.15) to digest less of the dietary GE than they did in gestation, while digestibility of starch was significantly lower (P<.02). These data may reflect increased rate of digesta passage through the GI tract as sows are switched from restricted to ad-libitum feed intake in lactation. Pekas and Trout (1990) did not observe a depression in digestibility (measured by G/F) in pigs which expressed a greater ADFI as a result of immunization against CCK. Whereas Pekas and Trout (1990) looked at the efficiency of utilization of the entire diet, the apparant digestibility method used in this study measured the efficiency of utilization of individual nutrients, and may have been more sensitive. Additionally, whereas Pekas and Trout (1990) reported G/F for the entire trial, thereby allowing time for adaptability, the measurements in this trial were taken in early lactation (LD8 to LD12), probably before a higher rate of digestive enzyme synthesis could manifest itself. Digestibility of ash was greater (P<.02) in lactation than in gestation and may reflect the large mineral demands of milk synthesis. Digestibility of lignin was also greater (P<.02) in lactation.

Table 24. Digestibility coefficients measured in gestation and lactation for CCK immunized sows[a]

Stage	GE	Starch	CP	EE[b]	Ash	NDF	ADF	Lignin
Gestation	82.9± .9	98.9± .2[c]	77.7± 1.0	24.2± 6.0	31.7±1.9[c]	53.9± 2.0	53.9± 3.0	48.1±1.2[c]
Lactation	81.0± .9	98.3± .2[d]	79.9± 1.0	32.3± 5.8	44.6±1.9[d]	51.5± 2.0	53.2± 2.9	54.2±1.2[d]

[a]± SEM.

[b]Ether extract.

[cd]Means within the same column with different superscripts are different, P<.02.

Nutrient digestibility of gilts vaccinated against CCK does not appear to be impeded either in gestation nor in lactation as serum CCK-AB titers increase. These data would suggest that the CCK-AB of the vaccinated gilts are not impairing pancreatic release of digestive enzymes. In gestation, the digestibility of GE, starch, CP, EE, and ash was actually enhanced as CCK-AB increased. Spencer (1992) offered three explanations as to why the immunization procedure may actually potentiate rather than neutralize CCK action;

1) The CCK-AB raised may have had too low an affinity to effectively prevent the hormone from binding to the receptor.

2) The AB-bound hormone may be presented to the receptor in such a way as to enhance it's orientation at the receptor binding sites.

3) The AB-bound hormone may extend the hormone's transmembrane effectiveness by inhibiting internalization and clearance of the hormone.

Within this discussion these mechanisms would be limited to the receptors of the pancreas, because as previously described, the lactation ADFI and litter weight gain of these CCK immunized sows paralleled increases in CCK-AB titer.

It is also possible that production of CCK may have been up-regulated as a result of immunoneutralization. As the pancreas is in close proximity to the site of CCK release, it may have had greater exposure to CCK before it was diluted and neutralized in the periphery.

Other observations

As done in Experiment 1 and 2, an assessment was made of the incidence of constipation among sows. Due to missing observations, the number of sows in this data set totaled nine (n=9). Nevertheless, some interesting observations were made: Using regression analysis it was observed that sows with softer feces at LD3 had a greater (r^2=.39, P<.10) week 2 ADFI. Similarly, sows with softer feces at LD10 had a greater (r^2=.46, P<.05) week 2 ADFI and a greater (r^2=.40, P<.10) overall ADFI. Interestingly, sows with firmer feces at LD3 were able to digest more (r^2=.45, P<.05) starch, and more (r^2=.47, P<.05) CP, and tended (r^2=.29, P=.13) to digest more dietary GE in lactation. These observations may be related to the differences in the rate of digesta transit time through the GI tract. It is presumed that sows with firmer feces (lower water content) had a longer transit time, allowing greater time for digestive enzymes to work.

The relationship between the actual vaccination order of the gilts and their resulting titer was determined via regression analysis. This analysis included complete records on 10 gilts (n=10). The results demonstrated that gilts vaccinated closer to the end of the vaccination order at time B2 had higher (r^2=.43, P<.05) B3 titers, higher (r^2=.31, P<.10) colostrum titers, and higher (r^2=.30, P<.10) M-LD21 titers. Gilts vaccinated closer to the end of the vaccination order at time B2 also tended (r^2=.29, P=.11) to have higher M-LD14 titers, and their pigs tended (r^2=.24, P=.15) to exhibit greater titer values. These limited observations would at least suggest that vaccine preparation and/or vaccination order should be carefully considered prior to execution of the next experiment.

Ensuing Data Analysis

This experiment was designed to be analyzed via linear regression using CCK-AB titer as the dependent variable. Many strong relationships between CCK-AB titer and sow performance and CCK-AB titer and pig performance during lactation were discovered. However, some relationships appeared to lack continuity; consisting of low titer (mean lactation sow serum titer < 1.0), and high titer (mean lactation sow serum titer > 1.0) groups of sows. Therefore, the data was reanalyzed via the GLM procedure of SAS, using only treatment in the model statement. Using this analysis, high titer (HT) sows consumed more (P<.01) feed than low titer (LT) sows during week 3 of lactation (ADFI = 5.41 and 4.40 kg respectively). The mean ADFI for the HT sows was also greater (P<.06) than LT sows throughout the 21 d lactation period (4.93 vs 4.39 kg respectively). Similarly, litter weight gains during week 3 of lactation were greater (P<.03) for HT sows than LT sows (14.38 vs. 10.56 kg respectively), and total litter weight gains were greater (P<.10) for HT sows than LT sows (32.18 vs. 37.83 kg respectively).

Implications of Experiment 3

These results show that CCK immunization has the potential to improve performance of primiparous gilts. CCK immunization increased ADFI during the third week of lactation by about .62 kg for each unit of log titer increase, or about 1.8 kg/hd/d for high titer sows. Effects on sow body weight change and body compositional changes were equivocal. Although changes in body composition of the sows was not clearly influenced by CCK immunization, backfat depth was reduced as anti-CCK titer

increased.

Immunization of first parity gilts against CCK has potential in the swine industry to increase feed intake resulting in increased milk secretion and litter weight gain. This new technology has potential to (1) produce heavier piglets at weaning which would be expected to maintain this weight advantage and attain market weight in less time, or (2) produce heavier piglets which could be weaned earlier (segregated early weaning [SEW]), with less incidence of mortality and morbidity. At this time it is not known what direct (if any) effect the passively derived CCK immunoglobulins may exert on the piglet. Although the half-life of passively derived immunoglobulins is short (3 to 4 weeks), they may have tremendous potential in SEW situations.

The potentially negative affect of impaired or reduced nutrient digestibility as a result of CCK vaccination was not observed in this trial. In gestation, it appeared that nutrient digestibility was actually improved in gilts with higher CCK-AB titers. Additional studies which follow similar protocols should certainly be conducted to confirm these observations.

APPENDIX

APPENDIX

LABORATORY PROCEDURES

Determination of Antisera Titer Against Cholecystokinin

Reagents

1) 0.1% Gel Buffer, pH 7.5 (Used for dissolving tracer)

2.68 g Na_2HPO_4 H_2O (.01 M) (Sigma cat.# S-9390)

0.37 g EDTA Na_2 (.001 M) (Sigma cat.# ED2SS)

1.00 g Na azide (.1% w/v) (Sigma cat. #S-2002)

8.10 g NaCl (.14 M) (Sigma cat. # S-9888)

1.00 g Gelatin (.1% w/v) (Sigma G-2500)

Place in 1000 mL volumetric flask and partially fill to volume with deionized distilled water, Stir on low heat on magnetic stirrer in order to dissolve gelatin. After gelatin is dissolved, solution is cooled to room temperature. Solution is then adjusted to pH 7.5 with 1 N HCL. Finish filling the flask to volume with deionized distilled water. Check to be sure pH is still 7.5.

2) 0.05% Gel Buffer, pH 7.5 (Used as the assay buffer)

2.68 g Na_2HPO_4 H_2O (.01 M) (Sigma cat.# S-9390)

0.37 g EDTA Na_2 (.001 M) (Sigma cat.# ED2SS)

1.00 g Na azide (.1% w/v) (Sigma cat. #S-2002)

8.10 g NaCl (.14 M) (Sigma cat. # S-9888)

0.50 g Gelatin (.1% w/v) (Sigma G-2500)

Place in 1000 mL volumetric flask and partially fill to volume with deionized distilled

water, Stir on low heat on magnetic stirrer in order to dissolve gelatin. After gelatin is dissolved, solution is cooled to room temperature. Solution is then adjusted to pH 7.5 with 1 N HCL. Finish filling the flask to volume with deionized distilled water. Check to be sure pH is still 7.5.

3) 1.25% Dextran-coated Charcoal in 0.05% Gel Buffer

1.25 g Norit A charcoal (Fisher cat. # C176-500)

0.125 g Dextran, average molecular weight = 79100 (Sigma cat. # D-4751)

100 mL of 0.05% Gel Buffer, pH 7.5

Place in 150 mL beaker and stir at room temperature on magnetic stirrer for 30 minuets. Store in refrigerator. Stir continuously on magnetic stirrer over crushed ice while using during the assay procedure.

4) Tracer

[^{125}I]-Bolton Hunter-CCK-8s (DuPont NEN cat. # NEX-203)

The ul of concentrated tracer used will depend on the size of the assay, but will be diluted with 0.1% Gel Buffer, pH 7.5 to give a final radio concentration of 4884 dpm/100 ul assay (1 fmol CCK-8s/100 ul assay). Mix on rocker table for 30 minuets at room temperature. Tracer is then quantitatively transferred to another tube to prevent any bound tracer on walls of tube from leaching into the solution. Fifty ul aliquots are then pipetted into 12 X 75 mm tubes and counted to determine actual dpm being put into assay tubes.

5) Reference Antisera

For assays in Dr. J.C. Pekas' lab (USDA, ARS, MARC), 25 ul of pig 42307 (Expt. #JP8704, CCK immunized, blood collected on 1/28/88) is diluted in 25 mL of 0.05% gel buffer, pH 7.5 (1:1000 dilution))which gives specific binding of approximately 50%. Diluted antisera is then aliquoted into 300 ul amounts into 12 X 75 mm assay tubes and stored at -20^{0}C. These tubes are used directly in each assay run.

6) Antisera Sample Preparation and Dilutions

Defatted colostrum and milk samples were prepared by initially aliquoting 1 mL of each sample into a 12 X 75 glass test tube. Samples were centrifuged at 2,000 X G for 30 minuets at 4^{0}C. The resulting fat "layer" on the top of each sample was gently pierced, and the supernate was poured into another 12 X 75 glass test tube. Raw antisera and defatted colostrum and milk were diluted as follows:

A) 1:10 111 ul of sample + 1,000 ul of 0.05% gel buffer

B) 1:100 111 ul of "A" + 1,000 ul of 0.05% gel buffer

C) 1:1,000 111 ul of "B" + 1,000 ul of 0.05% gel buffer

A) 1:10,000 111 ul of "C" + 1,000 ul of 0.05% gel buffer

Assay Procedure

Total Counts (TC)	100 ul of tracer
Non Specific Binding (NSB)	300 ul of 0.05% gel buffer + 100 ul of tracer
Reference Antisera (RA)	300 ul of reference antisera + 100 ul of tracer
Unknowns	300 ul of unknowns + 100 ul of tracer

1. Pipette 300 ul of each antisera dilution in triplicate into 12 X 75 mm glass test tubes. For "NSB" tubes use 300 ul of 0.05% gel buffer.
2. Add 100 ul of tracer to each tube and gently vortex.
3. Cover racks of assay tubes with aluminum foil and incubate for 48 hours at 4^0C.
4. After 48 hours add 250 ul of ice-cold dextran-coated charcoal to tubes. Shake rack of tubes vigorously to mix. Incubate for 10 minuets at 4^0C. **Do not add charcoal to TC tubes.**
5. After the 10 minuet incubation, add 300 ul of ice-cold 0.05% gel buffer to tubes and shake the rack of tubes vigorously to mix. Incubate an additional 10 minuets at 4^0C.
6. After the second 10 minuet incubation, centrifuge tubes for 5 minuets in 4^0C centrifuge at 2,000 X G.
7. Remove tubes from centrifuge and immediately remove supernate from charcoal pellet using disposable 9" Pasteur pipettes. Place supernatants in corresponding 12 X 75 mm glass test tubes. Keep other tubes on ice until they are ready to be separated.
8. Count both the supernates and charcoals in a gamma counter for 2 minuets.

<u>Calculations</u>

Nonspecific binding = cpm of NSB/cpm of TC

Specific binding = (cpm of unknown supernate - cpm of NSB) / (cpm of TC - cpm of NSB)

Sequential Fiber Analysis

Neutral-Detergent Fiber (NDF)

1) Weigh 1.0g (±.05g) air-dry sample into a Berzelius beaker.
2) Add 100 mL of room temperature neutral detergent (ND), 2 ml of a 20% solution of amylase (Novo Termamyl), and .5g sodium sulfite. Sodium sulfite was used to break disulfide bonds present in the feces.
3) Boil beaker contents under reflux for 60 minuets. As beakers are removed from the heat source, add an additional 2 mL of a 20% solution of amylase (Novo Termamyl).
4) Filter beaker contents under suction through fritted glass crucibles.
   A) While on the suction manifold, rinse the filtrate 3 times with hot water (90-100^0 C). To ensure that all detergent is completely removed, rinse the outside and bottom of each crucible with hot water as it is removed from the filter manifold.
5) While under an exhaust hood, apply suction to filtrates and rinse with acetone to facilitate drying. Allow acetone to completely evaporated.
6) Dry crucibles at 106^0 C for a minimum of 8 hours and hot-weigh.

The NDF can now be subjected to acid-detergent fiber analysis procedures.

Acid-Detergent Fiber (ADF)

1) Loosen the fiber mat remaining in the bottom of each crucible by placing crucibles in a saucepan containing an amount of acid detergent (AD) sufficient enough to cover the NDF mat. Boil for 5 minutes.

2) Rinse the crucibles two times each with 50 mL room temperature AD into a Berzelius beaker. Particles adhering to the crucible can be loosened with the help of a rubber policeman.

3) Place beakers on a heat source and allow to boil under reflux for 55 minutes.

4) As beakers are removed from the heat source, empty beaker contents back into their respective crucibles and filter under suction.

   A) While on the suction manifold, rinse the filtrate 3 times with hot water (90-100^0 C). To ensure that all detergent is completely removed, rinse the outside and bottom of each crucible with hot water as it is removed from the filter manifold.

5) Under an exhaust hood apply suction to filtrates and rinse with acetone to facilitate drying. Allow acetone to completely evaporate.

6) Dry crucibles at 106^0 C for a minimum of 8 hours and hot-weighed.

The ADF can now be subjected to lignin analysis procedures.

Acid Detergent Lignin (ADL) and Ash

Prior to starting this procedure, place 72% H_2SO_4 on ice and allow it to cool to about 15^0 C.

1) Place crucibles containing the ADF in shallow Pyrex pans and add an amount of cooled 72% H_2SO_4 sufficient enough to cover the ADF fiber mat.

2) Place a Teflon rod into each crucible and stir the contents into a paste.

3) Add fresh 72% H_2SO_4 to the samples as it drains through the crucibles.

4) Allow samples to soak in the 72% H_2SO_4 for 3 hours at room temperature.

5) After 3 hours, fill crucibles 2/3 full with hot water (90-100^0 C).

6) Rinse material adhering to the Teflon rods into the respective crucible, and remove the rods.

7) Filter crucible contents under suction and rinse 3 times with hot water (90-100^0 C).

   A) To ensure that all acid is completely removed, rinse the outside and bottom of each crucible with hot water as it is removed from the filter manifold.

8) Dry crucibles at 106^0 C for a minimum of 8 hours and hot-weighed.

9) Place the lignin-containing crucibles in a muffle furnace at 500^0 C for a minimum of 6 hours to ash. Allow crucibles to cool to 200^0 C, place in drying oven and hot-weighed at 106^0 C.

Calculation of Fiber Fractions

Fiber fractions were calculated as a percent of the dry matter of the sample in the following manner where;

N = crucible and NDF residue

A = crucible and ADF residue

L = crucible and LDF residue

X = crucible and ash residue

DM = g of sample dry matter

$$NDF = (N-X)/DM$$

$$ADF = (A-X)/DM$$

$$ADL = (L-X)/DM$$

Determination of ether extract (EE)

1) Using a pencil, sequentially number Whatman filter paper (#4, 11 cm) and place it in a drying oven (106^0 C) overnight, and hot-weigh.
2) Allow the filter paper to cool to room temperature and weigh approximately 2 g of sample onto the filter paper.
3) Fold the paper carefully so as to ensure that all of the sample remains contained. Secure the ends with paper clips.
4) Place the filter papers containing the samples "Lincoln-log" style into the modified Soxhlet ether extraction apparatus under a hood.

In this system, ether is heated to boiling, condenses and fills a container holding the samples. Samples soak in the condensed ether and ether-soluble compounds are released into the solvent. Polluted ether is frequently removed by siphon and replaced by freshly condensed ether. Samples remained in this system overnight (approximately 16 hours).

5) Remove samples from the ether extraction devise, gently remove paper clips and allow the ether to evaporate from the samples under a hood.
6) Place samples in a drying oven (106^0 C) overnight and hot-weighed.

The % EE (fat) of the samples is calculated as follows where;

DM = g of sample dry matter

EX = ether-extracted sample weight

PW = paper weight

EE = ((DM - (EX - PW))/DM) * 100

Determination of crude protein (CP)

Sample CP concentration was determined using a modification of the procedure described by Hach et. al (1987).

1) Weigh .25 g (±.005g) feed or .20 g (±.005g) feces onto a precut tissue paper measuring approximately 6 X 6 cm.
2) Fold the tissue containing the sample and slip it gently into a 100 mL Digestahl flask.
3) Add 4 mL concentrated H_2SO_4 to each flask and swirl gently to allow the paper and sample to become saturated with acid.
4) Attach aspirators with condenser columns and capillary funnels to each flask and place on burners at 440^0 C.
5) After 6 minutes place 20 mL of 50% H_2O_2 into the capillary funnel of each apparatus. Heat flasks continually and allow them to remain on the burners for an additional 1-2 minutes after the H_2O_2 is completely emptied from the capillary funnel.
6) Remove the flasks from the burners and remove condensers from the flasks once fumes are no longer visible in the flasks.
7) When the flasks have cooled to room temperature, diluted flask contents to 100 mL with distilled H_2O. Stopper and mixed thoroughly.
8) Prepare a set of standards using NH_3Cl and distilled H_2O as follows:

mg N/mL:	0.0	0.01	0.02	0.04	0.06	0.08
mL stock solution (0.3056 g NH_3Cl/L distilled H_2O)	0.0	0.1	0.2	0.4	0.6	0.8
mL distilled H_2O	0.8	0.7	0.6	0.4	0.2	0.0

9) Add 25 mL of a .1g/L solution of polyvinyl alcohol and 1 mL of Nessler's reagent to each standard and to each .8 mL aliquot of sample. Mixed thoroughly.

10) Read absorbance values via spectrophotometer at 460 nm visible light within 1 hour of adding the Nessler's reagent.

11) Estimate nitrogen concentration of the samples.

A) Use the linear equation derived from the regression of the standards where:

$$\text{mg N/mL} = \frac{\text{absorbance - intercept}}{\text{slope}}$$

B) Convert mg N/mL to a sample basis:

mg N/sample = mg N/mL * 100

C) Calculate percent N:

%N = ((mg N/sample) / (mg sample DM used)) * 100

12) Calculate crude protein of the samples:

%CP = %N * 6.25.

Determination of starch

1) Weigh a .2 g (±.01g) feed sample or a .6 g (±.01g) fecal sample and place it into a 125 ml Erlenmeyer flask containing 20 mL distilled H_2O. Allow the samples to hydrate for at least 4 hours.
2) Add .5 mL 50% NaOH to each flask while gently swirling. Allowed samples to gelatinize for 15 minutes.
3) Add 15 mL distilled H_2O and 10 mL acetate buffer and gently swirl.
4) Add .8 mL concentrated HCL and 250 ul Amylase (Diazyme L-200, Solvay Enzymes) to each flask and gently swirled.
5) Seal with a cork stopper and set in a 55^0 C water bath for 14 hours.
6) After the incubation period, transfer the flask contents to a 200 mL volumetric flask and diluted with distilled H_2O to 200 mL. Mixing thoroughly.
7) Place a 5 mL aliquot into a centrifuge tube and spin at 26,000 X G for 20 minutes.
8) Transfer a 900 ul aliquot of the resulting supernate to a vial and analyzed for glucose concentration by HPLC.
9) Convert sample glucose concentration to starch concentration.

Preparation of macro amounts of ground corn stalk chromium mordant

By adding distilled water to a known amount of ground corn stalk material it was estimated that 1 g of air dry ground corn stalk material could absorb approximately 8.0 g of water. Based on this information, a 3.4 % solution of $Na_2Cr_2O_7$ (.034 g $Na_2Cr_2O_7$/mL distilled water) was prepared. When applied to the ground corn stalk material as described below, this concentration of $Na_2Cr_2O_7$ provided a chromium concentration equal to approximately 11% of the weight of the fiber (Uden et al 1980).

Procedure

1) Weigh the fiber material into a shallow aluminum pan.
2) Slowly pour the solution of $Na_2Cr_2O_7$ over the fiber until the solution covers the fiber completely. Allow ample time for the fiber to become completely saturated.
3) Cover the aluminum pan with aluminum foil, being careful to stretch the foil tight so as not to have the foil touching the $Na_2Cr_2O_7$ solution (otherwise the solution will run out of the pan via capillary action).
4) Bake in an oven at 100^{0}C for 24 hours. Make sure the foil is sealed tightly around all edges so that the fiber is baked in a moist environment (otherwise the fiber may dry out and burn).
5) Place the fiber in a pillowcase and tie it securely to a water faucet. Manually agitate the fiber within the pillow case while letting the water run vigorously for at least an hour.
6) Remove the fiber from the pillowcase and suspend the fiber in tap water. Add an amount of ascorbic acid equal to one half the weight of the dry fiber, and let stand for

one hour.

7) Again place the fiber in a pillowcase and tie it securely to a water faucet. Manually agitate the fiber within the pillow case while letting the water run vigorously for at least an hour , and until it is free of soluble green matter.

8) Dry the material in an oven at 65^0C.

Determination of chromium (Cr) concentration

1) Weigh approximately 1 g feed or .5 g feces into a 250 mL Phillip beaker.
2) Add 20 mL of nitric acid (14M) and 4 mL of perchloric acid (10M) to each sample.
3) Digest samples on hot plates under a perchloric hood until approximately 1 mL of volume remain in the flask.
4) Remove flasks from the hot plate and wash the interior sides with deionized water.
5) Once flasks have completely cooled, add a sufficient amount of deionized water to attain a net weight of about 50 g. Samples which contained high Cr concentrations were further diluted with distilled water.
6) The concentrations of Cr in the samples were determined by flame atomic absorption spectrophotometry (model I1 951, Instrumentation Laboratory, Wilmington, MA). The final concentration of Cr was calculated using the following formula where:

PPM = concentration of mineral in part per million

D = final dilution

FWF = final weight of the flask

FW = initial weight of the flask

C = concentration of Cr (ppm)

SW = sample weight

$$PPM = [(D)(FWF-FW)(C)] / SW$$

Determination of gross energy (GE)

1) Press approximately 0.8 g of sample into a pellet with the aid of a hydraulic jack, and record weight.
2) Completely combust the sample in an Parr adiabatic bomb calorimeter (Parr Instrument Company, Moline, IL)
3) Titrate the resulting residue with 0.725 N sodium carbonate to correct for heat generated by nitric acid formation.
4) The final GE content was calculated with the following formula where:

   A = temperature change recorded from bomb calorimeter

   B = bomb adjustment factor

   C = kcal energy contained in nitric acid produced during the combustion

   D = kcal energy contributed by ignition wire

   GE, kcal/g = ((A * B) - (C + D))

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