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# THE EFFECT OF DIETARY pH AND PHOSPHORUS SOURCE ON PERFORMANCE, GASTROINTESTINAL DIGESTA, BONE CHARACTERISTICS AND BODY COMPOSITION IN WEANLING PIGS

by

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(ABSTRACT)

Crossbred pigs (n=144, avg age and weight - 28 ± 3 d, 7.5 kg) were used in two 6 wk trials to assess the effects of dietary pH and phosphorus source on performance, gastrointestinal digesta pH and chloride ion concentration ($Cl^-$), bone characteristics and body composition. Pigs were blocked according to weight within sex and litters were balanced across groups. Treatments were randomly allotted within blocks to a 3 x 2 factorial arrangement with three dietary pH levels (5.4, 6.0 and 6.7) and two phosphorus sources: dicalcium phosphate (DCP) and defluorinated phosphate (DFP). Pigs fed the pH 6.7 diet had reduced average daily gain (ADG, $P < .01$) and average daily feed intake (ADFI, $P < .001$) during wk 1-3 and overall compared with pigs fed the pH 6.0 diet, but ADG and ADFI were not effected when the pH 5.4 diet was fed. There was a dietary pH by phosphorus source interaction ($P < .05$) for ADFI. DCP fed pigs had increased ADFI as dietary pH was reduced from 6.7 to 5.4, but DFP fed pigs had similar ADFI as dietary pH decreased from 6.0 to 5.4, and decreased ADFI as dietary pH

increased from 6.0 to 6.7. Dietary pH had little influence on F:G, and phosphorus source had little effect on either ADG, ADFI or F:G.

Dietary pH did not influence the pH and $Cl^-$ of the digesta for any gastro-intestinal section measured, except the $Cl^-$ in the stomach; both pH 5.4 and 6.7 fed pigs had a higher ($P < .01$) $Cl^-$ than the pH 6.0 fed pigs. Only shear force of the fourth metacarpal and specific gravity of the fourth metatarsal were increased ($P < .01$) for pigs fed the DCP compared with DFP diets. Neither dietary pH nor phosphorus source influenced backfat or loin muscle area.

These results suggest that maintaining the acidifity of the diet during the first 3 wk after weaning at 28 d of age is important with the primary response seen in ADFI. Varying dietary pH from 5.4 to 6.7 had little or no effect on gastrointestinal digesta characteristics, bone development and body composition.

(Key words: Pigs, Acidity, Alkalinity, Phosphorus, Hydrochloric acid, Sodium hydroxide.)

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# CHAPTER I

## INTRODUCTION

Current swine management practices involve weaning pigs at three to four weeks of age (early weaning) to optimize utilization of sows and farrowing facilities. The digestive system of early weaned pigs, is however, immature, which often results in scouring and a depression in gain immediately postweaning. Such phenomena are referred to as the postweaning lag phase (Whittemore et al., 1978). Attempts to stabilize the pH of the immature gastrointestinal tract digesta and to enhance digestive enzyme development by feeding a variety of organic acid-producing additives, such as citric and fumaric acids, have met with limited success (Kirchgessner and Roth, 1982; Falkowski and Aherne, 1984; Giesting and Easter 1985; Henry et al., 1985; Risley et al., 1990). Also, the cost of feeding these additives is generally much greater than the value of potential improvements. In addition, the source of minerals used to supply the requirements of the pig are known to influence the acidity/alkalinity of the diet (Patience, 1990). Thus, manipulation of the pH of the diet by varying the mineral sources might be more cost effective than organic additives. An acidic diet is desirable for enhancing digestibility and altering the microflora of the gastrointestinal tract, which may result in reduced scouring

and improved performance. Improved understanding of the maturation of the digestive tract and information on how diet manipulations may improve digestion and performance could help optimize postweaning diet formulation. The objective of this research was to use different mineral sources and inorganic acid and base to alter dietary pH. The effects of the altered dietary pH on performance, gastrointestinal digesta pH and chloride concentration, bone development, loin muscle area and backfat of weanling pigs was investigated.

# CHAPTER II

# REVIEW OF LITERATURE

## *Gastric Function in the Young Pig*

The principal functions of the gastrointestinal tract are assimilation of nutrients and excretion of the waste products of digestion. Most nutrients are ingested in a form that is too complex for absorption. Following mastication, breakdown of ingested feedstuffs occurs in the stomach of the pig, where the mucosa wall is divided into two sections: the nonglandular esophageal and the glandular section. The glandular portion is divided into three regions: cardiac, fundic and pyloric (Bone, 1988). Glands found in the cardiac region produce mucus, primarily. In the fundic or oxyntic region are found simple glands that are the true gastric glands which consist of three types of cells in folds (Frandson, 1986). The constricted neck of each gland contains mucous-producing neck cells, and parietal (oxyntic) cells. These parietal cells are involved in the production of hydrochloric acid (HCl), which is an integral component of gastric juice.

Parietal cell exocrine secretion of the hydrogen ions ($H^+$) for HCl production is accompanied by active secretion of chloride ions ($Cl^-$) obtained from the interstitial fluid in exchange for bicarbonate ($HCO_3^-$) passing from the cell to the interstitium. The $HCO_3^-$ is produced from carbon dioxide

($CO_2$) and water ($H_2O$) via carbonic acid ($H_2CO_3$). The $CO_2$ may be produced in the parietal cell or taken up from plasma or interstitial fluid. Figure 1 illustrates this metabolic activity which has the following formula:

$$OH^- + H^+ \longrightarrow \underset{\text{carbonic anhydrase}}{H_2O + CO_2} \longrightarrow \underset{\text{enters venous blood}}{H_2CO_3} \longrightarrow H^+ + HCO_3$$

For every $H^+$ secreted an electron is removed. The electron is accepted by oxygen to form $OH^-$ which is neutralized by $H^+$ from $H_2CO_3$ (Kaneko, 1980). Carbonic anyhdrase, the enzyme necessary for this reaction, is present in high concentrations within the parietal cells of the stomach (Bone, 1988). Small amounts of $Cl^-$ and sodium ($Na^+$) are secreted continuously by unstimulated parietal cells, with $H^+$ replacing $Na^+$ when secretion is stimulated (Dousa and Dozios, 1977).

Deeper in the lumen of the oxyntic glands lie chief or zymogen cells that produce pepsinogen, the precursor of the endopeptidase pepsin (Kaneko, 1980). Pepsinogen is not an active proteolytic agent, thus protecting the producing zymogen cells from autodigestion. The active form, pepsin, is produced by the action of HCl (produced by parietal cell) on the bonds of the pepsinogen molecule (Currie, 1988). Autocatalytic conversion of pepsinogen to pepsin occurs through dissociation of the inhibitory peptide bond that begins below a pH of 6.0, with optimal pH for the pepsinogen

to pepsin activation occurring between 1.6 and 2.5 (Taylor, 1968). Enzymatic activity of pepsin in the stomach is optimized at two pH levels, pH of 2.0 and 3.5 (Rerat, 1981).

The pyloric gland area, which lies near the exit of the stomach to the small intestine, contains glands producing the hormone gastrin and a slightly alkaline mucus. Gastrin secretion, stimulated by the sight or smell of food or the presence of food in the stomach (Gregory et al., 1964), acts as a stimulator of HCl secretion by the parietal cells. Gastrin appears to act on the parietal cells in part through the production of histamine, a local vasodilatory agent (Kaneko, 1980). The resulting increase in blood supply to the fundic gland cells supports the increased metabolic needs of these cells during secretory episodes. Dousa and Dozios (1977) reported that histamine also acts on the parietal cells directly by activating adenylate cyclase, which results in the synthesis of cyclic 3'5' adenosine monophosphate (cAMP) and ultimately HCl secretion.

The presence of excess HCl or $H^+$ inhibits gastrin secretion, thus reducing parietal cell HCl production (Kaneko, 1980, Currie, 1988). Grossman and Konturek (1974) demonstrated the presence of H+ receptors in the stomach and suggested that there is a direct response of reduced HCl secretion to excess gastric $H^+$. In addition, the small intestine provides some inhibitory control over the

secretion of HCl through the activity of the duodenally-released peptide pancreozymin-cholecystokinin (PZ-CCK). Parietal cell HCl secretion is directly reduced by PZ-CCK released when acidic stomach contents are present in the duodenum (Currie, 1988).

Hydrochloric acid produced by the parietal cells is one of the most important constituents of gastric juices. The acid is important in macerating food, for pepsin activation and protein hydrolysis, and the acidic pH provided by HCl acts as an antibacterial solution for the stomach (Stevens, 1977). The acidity of stomach contents varies with age and diet of the pig, and with the method of sampling. Two types of pH measurement may be made on the gut contents. The pH of mixed contents indicates the extent to which gastric or intestinal secretion has modified the buffering power of the diet. Alternatively, the pH from the outer layers of the food mass in the separated stomach or via a cannula in the live animal can be recorded. Position of the electrode in the latter methods can greatly affect measurements recorded (Manners, 1970). The measurement of free acid in the stomach is difficult because it is affected by the buffering capacity of the feed (Cranwell, 1985). Early workers (Cranwell et al., 1976; Kidder and Maner, 1978) were unable to detect free HCl in the stomach of nursing pigs, probably

due to the buffering capacity of milk, rather than the lack of HCl secretion.

*Development of gastric secretion*. There is a large difference between the pH in the stomach of a piglet and that in the stomach of an adult pig. Mature pigs are able to adjust the stomach pH following food ingestion and their stomachs can reach a very acid pH, allowing for optimum pepsin function. In the very young pig, pH in the stomach remains close to that of the diet, even in the outer layers of the food mass (Manners, 1976). Development of acid secretion in the newborn is fairly rapid, however, with initial acid secretion detected within 24 h of birth in some pigs in work by Forte et al. (1975), Cranwell et al. (1976), and Cranwell and Titchen (1976). The pH in the first day of life of baby pigs was high (5.2-5.3) in work by Smith and Jones (1963). Cranwell et al. (1968) reported that the time of onset of HCl secretion varied widely among pigs and was greatly affected by environment. In subsequent work Cranwell (1985) found that the amount of acid secreted by young pigs was greatly affected by the buffering properties of milk, saliva, regurgitated bile acid and pancreatic juice. Both sow's milk and milk replacer diets have considerable acid buffering power and any HCl that is secreted is probably neutralized by the stomach contents. Consumption of frequent small meals such as occurs during

suckling also reduces the need for acid secretion (Pond and Maner, 1984). Acid secretion was linked to the consumption of milk in work by Cranwell (1984), with the volume and concentration of $H^+$ being reduced and virtually ceasing if the pigs were separated from the dam for 2.5 - 6 h. Following d one, Walker (1959) reported no change in the pH of the stomach during the suckling phase, but, there was considerable variation among pigs. Cranwell (1985) reported a positive correlation between stomach weight (wt), maximal acid and pepsin output and body wt. Cranwell and Stuart (1984) found a significant correlation between acid output and fundic mucosal wt. Earlier, Card and Marks (1960) had reported that HCl-producing parietal cell number was linearly correlated to fundic wt.

Reported research thus indicates that as the pig matures and its body and stomach size increase, there appears to be a concomitant increase in the volume of HCl and perhaps pepsin secretion. There is a gradual increase in acid secretion until three to four wk of age, with a greater increase in volume occurring after four weeks (Cranwell and Titchen, 1976). In 1985, Cranwell reported an average pH of 5.3 to 5.5 in 28 d old pigs, with an increased acidity to a pH of 4.0 by 56 d of age.

Diet and food consumption will greatly affect stomach pH in pigs of any age. Manners (1970) reported pH ranges of

4.75 to 5.24 in two and three wk old pigs fed a casein-based diet, and 2.23 to 3.86 in barley-fed pigs of the same age. Walker (1959) reported the stomach pH in all pigs under five wk of age to be greater than 3.4. The consumption of solid food such as a starter diet results in the production of more acid/unit of stomach wt than does sow's milk (Hartman et al., 1961). The buffering power of sow's milk indicates that it is easier to acidify than is a high-protein starter diet. Cranwell and Stuart (1984) found that five to six wk old creep-fed pigs had significantly more fundic mucosal tissue than milk-fed pigs of the same age and wt. Hartman et al. (1961) and Cranwell (1985) found that pigs given access to solid food before weaning had heavier stomachs and greater acid and pepsin secretory capacity than pigs fed entirely on sow's milk.

*Effect of limited gastric function in the young pig.* Regardless of diet or environment, it appears that the stomach of the young pig is rarely suitable for maximum pepsin activity, and high gastric pH may have a negative effect on protein digestion in the stomach. Easter (1988) reported a negative effect on gastric digestion in young pigs due to the stomach's inability to maintain appropriate gastric pH. There is little or no proteolytic activity evident in the first one to two wk of life, with some increases occurring by three to four wk of age. Liebholz

(1984) reported that six percent of the dietary protein is hydrolyzed in the stomach of 28 d old pigs, increasing to 50% by 56 d and 80% in 120 d old growing pigs whose gastric pH had dropped to a range of one to three.

Reduced proteolytic digestion is not the only effect of high gastric pH levels in the young pig. Manners (1976) states that the main practical result of the lack of gastric acidity is the susceptibility of the young pig to outbreaks of gastrointestinal disease, as evidenced by scouring. Work by Manners (1970) showed that the pH in the stomach remained fairly high until five wk of age, presumably allowing little restriction on microorganism multiplication in the gut of the young pig. The ensuing fermentation of feedstuffs by the gastric microorganisms may be responsible for an increased incidence of gastrointestinal disease and reduced HCl secretion due to negative feedback on HCl production as a result of the presence of excess H+ ions from organic acids (Cranwell et al., 1976).

*Organic acids.* Friend et al. (1963) and Easter (1988) reported that gastric fermentation of lactose in sows milk to lactic acid by resident lactobacillus bacteria provides the major organic acid found in the stomach and small intestine throughout the first nine wk of life. Easter (1988) suggested that lactic acid, not HCl, is the primary source of gastric acidification. The lactobacilli and other

bacteria enter the alimentary canal and colonize it during the first d of life when acid secretion is low and stomach pH is high (Smith and Jones, 1963).

An alkaline diet, overwhelming the ability of the stomach to acidify it, might preserve a high pH during the time the feed was in the stomach, allowing additional bacterial proliferation and passage into the small intestine. Twice- daily feeding would aggravate such a situation because the rate of stomach emptying is greatest when the stomach is full, and a greater number of bacteria would be able to pass through the stomach unaffected by an acidic pH. Under conditions of low HCl levels, the greater the amount of feed consumed at any one meal, the less likely that the pH will fall to levels capable of inhibiting bacterial growth, and the more likely that a considerable part of the contents will pass from the stomach to the small intestine soon after feeding (Manners, 1970). Cranwell et al. (1968) found an inverse relationship between HCl secretion by the stomach and lactic acid production, indicating that the low HCl levels did allow increased bacterial multiplication and fermentation.

*Pancreatic Secretion*

The pancreas is found in the first loop of the duodenum of the small intestine. The pancreas has two functions and

a double structure: an acinar portion, which is exocrine and secretes enzymes known as pancreatic juice, and the islets of Langerhans, which are endocrine structures and secrete hormones (Currie, 1988). Pancreatic juice passes to the small intestine through the bile duct. The basic component of pancreatic juice is a dilute, slightly alkaline sodium bicarbonate ($NaHCO_3$) solution containing electrolytes and pancreatic enzymes added as a result of hormonal control. The juice is iso-osmotic with plasma; it's principal anions are $HCO_3^-$ and $Cl^-$ whose concentration is a function of the rate of secretion (Kaneko, 1980). Bicarbonate concentration varies directly with the flow rate, and $Cl^-$ concentration varies inversely with $HCO_3^-$ concentration.

As shown in figure 2, the $HCO_3^-$ is believed to come from ductal epithelial cells, with carbonic anhydrase catalyzing the reaction ($CO_2 + H_2O \rightarrow H_2CO_3$). Carbonic acid dissociates to $H^+ + HCO_3^-$ at normal intracellular pH (Janowitz and Dreiling, 1959). A ductal epithelial cell exchange pump for $Na^+$ and $H^+$ appears to move $Na^+$ into the cell and $H^+$ out. Thus, in the cellular synthesis of $HCO_3^-$, an equivalent amount of acid is produced and enters the blood (Frandson, 1986). This is the opposite of what happens in the stomach, where $H^+$ required by the cell for HCl production are obtained from the interstitial fluid in

exchange for $HCO_3^-$, which is produced in amounts equal to gastric acid production.

*Control of pancreatic secretion.* A basal rate of pancreatic secretion exists in most animals. With the ingestion of food, however, at least two duodenally-produced hormones alter pancreatic secretion. Secretin, released from the small intestine mucosa by entry of $H^+$ into the duodenum stimulates the pancreas to secrete large amounts of dilute $HCO_3^-$ fluid. The acidity of the chyme entering the small intestine acts as a strong stimulant for the secretion of $HCO_3^-$ (Konturek et al. 1979). The pH threshold for secretin-stimulated $HCO_3^-$ secretion was reported to be four in dogs; a higher pH was not efficient in eliciting pancreatic juice secretion (Llanos et al., 1977). Kaneko (1980) reported that the hormone gastrin produced by the stomach mucosa may also influence pancreatic $HCO_3^-$ secretion. The addition of pancreatic enzymes to the $HCO_3^-$ alkali solution results from the action of PZ-CCK (Currie, 1988), released in response to the products of protein digestion entering the duodenum from the stomach (Konturek et al., 1979).

*Acidosis and alkalosis response.* Alterations in blood pH result in changes in the secretion of pancreatic juice. Metabolic acidosis causes lower than normal $HCO_3^-$ concentrations, and metabolic alkalosis can result in an

increased flow rate of pancreatic juice with increased $HCO_3^-$ concentration (Rawls et al., 1963; Kaneko, 1980). Thus, the body tries to excrete through pancreatic secretions the excess plasma $HCO_3^-$ that occurs during alkalosis and conserve $HCO_3^-$ during acidosis-induced plasma deficits.

*Pancreatic enzyme function.* Pancreatic enzymes are responsible for the hydrolytic processing of the digesta into absorbable nutrients.

The pancreas has particular importance in young pigs because the duodenum and jejunum are thought to be the major site of protein hydrolysis (Liebholz, 1984). Protein and dry matter digestibilities were significantly reduced when the pancreatic duct of young pigs was ligated (Pekas et al., 1964). In work reported by Partridge et al. (1982), the serous $HCO_3^-$ secretions neutralized and buffered the duodenal pH to allow for optimal pH of pancreatic enzymes. The major pancreatic enzymes are secreted in an inactive form, and upon entering the duodenum, are converted to their active forms. Proteolytic enzymes secreted by the pancreas include trypsin and chymotrypsin. Pancreatic amylase splits starch into maltose, and maltase hydrolyzes maltose to glucose. Pancreatic lipase hydrolyzes fats into fatty acids and glycerol. These enzymes have pH optimas in the 8 range (Kaneko, 1980).

*Pancreatic development.* Development of pancreatic secretion in the pig was studied by Corring et al., (1978). They found that the development of enzymatic activity of the pancreas varies according to the enzyme considered. Amylase activity increased after 21 d of age, while lipase remained low until 35 d after birth. In relation to body wt, the pancreas undergoes rapid development during the first wk of life and then again from the fourth to the eighth wk. Major increases in the activities of pancreatic lipase, amylase, trypsin and chymotrypsin were also seen from the fourth to the eighth wk. Manners (1976) reported that the proteolytic activity of pancreatic secretions was very high in 20 d old pigs, and decreased with age. Lindemann et al. (1986), working with pigs weaned at four wk of age, reported a linear increase in body and pancreatic wt with a greater growth rate of the pancreas during the postweaning period.

Partridge et al. (1982) reported variability in pancreatic secretion depending on diet. The mean values for the volume of pancreatic juice secreted in 24 h in pigs adapted to a barley, wheat and fishmeal diet were four times that secreted by pigs on a starch, sucrose and casein diet. There was considerable variability among pigs, however, and no clear response to time of feeding or any diurnal pattern of secretion. Total wt of the secreted enzyme proteins was 45% higher on the barley diet. Bicarbonate was the

predominate anion on both diets but the barley diet had a higher $HCO_3^-$ to $Cl^-$ ratio and higher $Na^+$ and potassium ($K^+$) levels.

Thus the pH of the contents of the small intestine is likely to be relevant to the efficiency of digestion in the young pig because of the effect that pH may have on the secretion and activity of enzymes involved in the hydrolysis of proteins, carbohydrate and fats (Manners, 1970). Walker (1959) found that the pH in the small intestine of young pigs (6.4) was well suited for carbohydrase activity, but minimal for optimum proteolytic and lipase activity.

*Body Acid-Base Balance*

To discuss buffering, there are a few basic principles which must be described. An acid is a compound that donates $H^+$ and a base is a compound that accepts $H^+$ (Masoro, 1982). Strong acids completely dissociate in water and have little or no buffering capacity. Buffers are composed of weak acids and their salts and are important in maintaining a relatively constant $H^+$ concentration. These buffers (anions) combine with $H^+$ and bring about neutralization; thus, $Buffer^-$ + $H^+$ <---> H-Buffer. All buffers are in equilibrium with the same $H^+$ concentration and each other. This characteristic is termed the isohydric principle.

The body contains many physiological molecules in partially ionized states which dissociate into anions and cations in water and tissue fluids (e.g. lactic acid dissociates to lactate and $H^+$). To maintain the physiological pH within narrow limits, the balance between these cations and anions -- the process of physiological buffering -- is imperative to the growth and well being of the animal (Currie 1988).

Contributors of $H^+$ include metabolic reactions (metabolism of sulfur and phosphorus (P) to sulfuric and phosphoric acid), organic acids (absorbed from fermentation digestion) and blood (dissociation of carbonic acid to $H^+$ and $HCO_3^-$), which is the primary source of $H^+$ (Currie, 1988). The major anions which can take up excess $H^+$ are $HCO_3^-$, and the nonbicarbonate buffers: proteins, hemoglobin and phosphates (Masoro, 1982).

The nonbicarbonate buffer systems make up a relatively small amount of the body's buffering potential. Since the $HCO_3^-$ concentration in extracellular fluid is high and it is readily formed from $CO_2$ and $H_2O$ during physiological processes, the $HCO_3^-$ buffer system is responsible for the majority of buffering. It is based primarily on the dissociation of $H_2CO_3$ at physiological pH.

Plasma proteins serve as buffers because they usually carry negative charges at physiological pH, thus acting as

anions that can combine with $H^+$. Hemoglobin of venous blood (which is not carrying oxygen) has a high affinity for $H^+$. Phosphate acts as a buffer by shifting between two forms, $HPO_4^{2-} + H^+ <--> H_2PO_4^-$. These systems allow the normal distribution of acids as $H^+$, and bases as $OH^-$ or $HCO_3^-$ to be strictly regulated. Disturbances can occur for reasons related to metabolism, respiration or diet (Bone, 1988 and Currie, 1988).

*Metabolic acidosis and alkalosis.* Metabolic acidosis is a condition that occurs when the concentration of blood $H^+$ is excessive and the $HCO_3^-$ concentration is subnormal (Currie, 1988). Should the opposite conditions be present then the body is considered to be in metabolic alkalosis. Metabolic alkalosis usually results from vomiting or excess renal excretion, causing excessive acid loss. Metabolic acidosis can be caused by 1) high rates of acid production, ingestion or infusion; 2) the loss of $HCO_3^-$ or other conjugate bases from extracellular fluid; or 3) an impaired ability of the kidney to excrete $H^+$ (Masoro, 1982). Metabolic acidosis and alkalosis are diagnosed by serum $HCO_3^-$ levels. Bicarbonate concentration also reflects the state of intra and extracellular buffers. When acid loading is sustained for extended periods of time, serum $HCO_3^-$ ultimately stabilizes despite continuing acid retention,

which is indicative of additional buffer systems being activated (Lemann et al., 1966).

*Dietary influence on acid-base balance.* An area of interest is the influence of diet on the acid-base status of the animal. Yen et al. (1981), reported that the ingestion of calcium chloride ($CaCl_2$) increased plasma $Cl^-$ concentration, exceeding the $HCO_3^-$ buffering capacity and producing metabolic acidosis. The subsequent effect was reduced growth and feed intake, and depressed blood pH, $HCO_3^-$, total $CO_2$ and base excess. When the dietary electrolyte balance of pigs diets (calculated by Mongin, 1981 as $Na^+ + K^+ - Cl^-$) was reduced from a basal level of 175 millequivalents (meq)/kg, Patience et al. (1987) reported that blood pH and $HCO_3^-$ concentration dropped, indicating metabolic acidosis. Performance, however, was similar for electrolyte balances ranging from 0 to 341, and depressed below zero. More recently, Patience (1990), described the acid potential of the diet in terms of dietary undetermined anion (dUA), which is illustrated as:

$$dUA = (Na^+ + K^+ + Ca^{++} + Mg^{++}) - (Cl^- + H_2PO_4^- + HPO_4^{2-} + SO_4^{2-})$$

or the more easily calculated form

$$dUA = (Na^+ + K^+ + Ca^{++} + Mg^{++}) - (Cl^- + P^- + S^-).$$

An excess of fixed cations yields an equivalent excess of metabolizable anions relative to cations, which represents a contribution to alkalinity (Patience, 1990). If an excess

of anions occur then the associated cations would provide an acid load. This can be taken a step further to utilize the acid-binding properties of each feed. When acid-binding capacity is calculated as the amount of HCl required to bring 1 kg of a feedstuff to a pH of 3, corn is calculated at 160-200 meq. Soybean meal will range from 950-1200 meq, and it requires 20,000 meq of HCl to acidify limestone (Prohaszka and Baron, 1980). Careful selection of dietary feedstuffs could help to maintain an acid pH in the stomach of the young pig.

*Dietary Acidification*

The pig has an immature digestive system at the time of commercial weaning, which may result in a growth phase commonly known as the postweaning lag phase. In attempts to alleviate postweaning lag, many researchers have explored the acidification of diets to compensate for the weanling pig's inability to maintain an appropriate gastric pH. The primary method used for the acidification of diets has been the addition of organic acids, such as fumaric, citric and proprionic acid. Adding acid to the diets of weanling pigs has had varying results, depending on wt and age of pigs, and acids used.

*Effects on performance.* Although variable, the primary response seen with the acidification of diets using organic

acids has been an improvement in average daily gain (ADG) and feed to gain ratio (F:G) (Kirchgessner and Roth, 1982; Falkowski and Aherne, 1984; Giesting and Easter 1985; Henry et al., 1985; Risley et al., 1990). Risley et al. (1990) using 1.5% citric or fumaric acid in diets of pigs weaned at 25 d of age, reported that citric acid increased ADG during wk four and wk one to four postweaning, with no effect seen from the addition of fumaric acid. F:G was improved by 6.1% with citric and 5.3% with fumaric acids during wk one to five postweaning, but no effect on feed intake (ADFI) was observed. Cole et al. (1968) observed improvements in growth rate and feed efficiency with the addition of .8% lactic acid to the diet. Lewis (1981), using four wk old pigs and graded levels of fumaric acid, reported an improvement in ADG with no improvement in ADFI or F:G. Work by Radecki et al. (1988) demonstrated that fumaric acid added to the diet at the 1.5% level improved F:G for pigs wk five and six, but citric acid tended to depress gain. When pigs were allowed free access to either a citric, fumaric or no-acid diet, a significantly smaller amount of acidified diet was consumed compared to the nonacidified diet. Similarly, Giesting and Easter (1985) observed depressed ADFI when pigs were fed a diet containing 2% proprionic acid.

Some of the variation in results obtained among researchers may be partially explained by the acids used and the various levels that have been attempted. Kirchgessner and Roth (1982) suggested an optimum level of 2% organic acid, while Giesting and Easter (1985) reported an optimum 3% inclusion rate. Differences among diets used in research trials may also help explain the variability in results. The use of milk products in certain diets would have provided lactic acid and thus reduced the need for dietary acid, perhaps indicating a diet type and acid interaction. Maner et al. (1962) reported that four wk old pigs fed casein-based diets had significantly faster gastric pH drops than did soy-fed pigs. Giesting (1986) fed pigs either a simple corn-soy diet or a complex diet containing 25% dried skim milk, with acid levels of 0, 2 or 3%. Addition of acid to the simple corn-soy diet resulted in a linear improvement in performance up to the 3% level, whereas the complex diet containing dried skim milk reached a plateau response at the 2% acid level. In addition, nitrogen and dry matter digestibilities were greater in milk-based verses corn-soy diets. Liveweight gain and F:G showed greater improvements as a result of diet acidification for pigs fed soy protein diets over those fed casein-based diets (Giesting, 1986).

*Mode of action.* Recently, extensive research has been conducted to determine the mode of action for dietary

acidification. It has been proposed that by acidifying the diet, gastric pH is decreased and enzyme activity is increased with a subsequent improvement in the digestibility of nutrients. A reduced pH of the chyme leaving the stomach could also increase pancreatic secretion and improve small intestine digestion (Llanos, et al., 1977). In addition, by making a more acidic digesta the gastrointestinal environment may be modified so that it is less compatible for harmful bacteria.

It was hypothesized that due to the lack of HCl secretion, the gastric pH of young pigs was inadequate for the activation of pepsinogen (Manners, 1970). Rerat (1981) reported pepsin has two pH optima (2.0 and 3.5). Thus, elevated gastric pH likely causes a net reduction in efficiency of protein digestion. Fumaric acid supplementation improved protein utilization (Kirchgessner and Roth, 1982 and Falkowski and Aherne, 1984). In work reported by Giesting et al. (1985), the supplementation of 2% fumaric acid tended to improve dry matter and protein digestibility when simple corn-soy diets were fed. In contrast, Giesting and Easter (1985) observed a similar response to dietary acid when pigs were fed either a 16 or 20% crude protein diet, suggesting that acid did not increase protein digestion or utilization.

Dietary acidification may also exert a beneficial effect by limiting bacterial colonization and multiplication. When pigs were contaminated with multiple bacteria, mucosal damage was evident by the presence of stunted and deformed villi (Kenworthy and Allen, 1966). According to Stevens (1977), stomach pH has a role in preventing the movement of viable bacteria from the stomach into the upper small intestine. Drasar et al. (1969) suggested that in humans the flora of the small intestine is derived from the flora of the stomach contents. Smith and Jones (1963) observed that the alimentary tract of the pig was flooded with bacteria in the first d or so of life. When the pig's diet was made alkaline, the researchers observed a multiplication of bacteria that are normally inhibited at gastric pH. Stomach pH values and the incidence of scouring were reduced by the addition of lactic acid to the diet of two d old, artificially reared pigs (White et al., 1969). Similar results were reported by Thomlinson and Lawrence (1981): lactic acid reduced pig mortality as well as the multiplication of E. Coli in the stomach. The addition of .8% lactic acid in drinking water resulted in the reduction of haemolytic E.Coli in the duodenum and jejunum in work by Cole et al. (1968). In contrast, Risley et al. (1990) observed no differences in

gastrointestinal flora due to dietary acidification with 1.5% citric or fumaric acids.

By restricting the flora of the gastrointestinal tract, an increase in the efficiency of absorption of all nutrients may be observed. Kirchgessner and Roth (1982) reported increases in calcium (Ca) and P absorption as a result of dietary acidification. In acidification studies however, it is difficult to differentiate whether observed increased mineral absorption is due to microbial activity or an acid-base effect.

It has also been suggested that the addition of dietary organic acids may have some influence on energy metabolism (Kirchgessner and Roth, 1982). Metabolizable energy was improved 1.8 to 2.5% and digestible energy 1.5 to 2.1% when feeding 1 or 2% fumaric acid. Kornegay et al. (1976) reported no effect on daily energy balance when pigs were fed citric acid.

Giesting (1986), attempting to duplicate diet acidification responses with inorganic acids, demonstrated that the dietary addition of phosphoric acid improves feed efficiency, while HCl additions severely depressed growth. The author attributed this to the high levels of $Cl^-$ contributed by the HCl, which upset the physiological electrolyte balance. As reported by Patience (1990), the acidity or alkalinity of the diet can be influenced by

varying mineral sources and levels. The contribution of different mineral sources to dietary acidity/alkalinity while maintaining equivalent mineral levels has received little attention.

*Bone Metabolism*

Bone continuously undergoes turnover by the combined processes of anabolism (apposition) and catabolism (resorption). Bone is made up of an organic matrix and mineral salts within this matrix. The organic matrix consists of glycosaminoglycan, collagens, citric and ascorbic acid and is formed by bone cells termed osteoblasts (Currie, 1988). Mineralization of the organic matrix occurs with calcium phosphate ($Ca_3(PO_4)_2$) being the primary salt deposited. As the osteoblasts become surrounded by mineral, they become known as osteocytes. This mineralization is termed apposition and occurs very rapidly early in life, and then lessens with age (Frandson, 1986). Bone resorption can occur by two methods. The first is a process known as osteoclasia, which is the result of phagocytic osteoclasic cells resorbing bone minerals on surfaces or following bone injury. Osteolysis is a continuing process directed by the osteocytes deep within the mineralized tissue. These cells resorb mineral, as a major part of the body's maintenance of Ca homeostasis.

Calcium homeostasis is dependent on the movement of Ca from bone tissue to extracellular fluid and back (Currie, 1988). Calcium is the most abundant mineral in the body and approximately 99% of it is stored in the skeletal system. Thus, the process of osteolysis is vital in the maintenance of Ca homeostasis and whole body acid-base balance.

*Calcium deposition and resorption.* Parathyroid hormone (PTH) and Vitamin D are the major factors controlling the level of blood Ca. PTH controls the mobilization of Ca from the skeleton, promotes Ca and P absorption from the intestine and causes the kidneys to excrete P while retaining Ca (Wills, 1970; Boyle et al., 1971). PTH may also release bone citrate, lowering the pH of the bone and thereby increasing Ca solubility (Frandson, 1986). Vitamin $D_3$ (cholecalciferol) is another hormone involved in Ca honeostasis that when activated by the liver to 25-(OH)-$D_3$ and then by the kidneys to 1,25-$(OH)_2D_3$, increases the absorption of Ca from the small intestine, assists in resorption of Ca from the bones and increases the excretion of P from the kidneys (DeLuca, 1980). Therefore, it works in conjunction with PTH to increase the blood Ca levels. PTH is involved in the kidney activation of 1,25-$(OH)_2D_3$ (Currie, 1988). A third hormone involved in Ca homeostasis is calcitonin, it is involved in regulating extracellular Ca and P levels and provides a negative control to PTH. It is

released by the thyroid gland cells when extracellular Ca levels increase. Calcitonin inhibits the resorption of bone and decreases the release of Ca from bone to the blood (Kaneko, 1980). Calcitonin, Vitamin D, PTH and serum Ca concentration form a system to allow the regulation of Ca in the plasma and bone

*Bone as a buffer.* As previously discussed, the primary buffering systems of the body are the bicarbonate/carbonic acid system in extracellular fluid and the phosphate system within the cell. Should these systems be overloaded further anion reserves must be tapped in order to maintain the acid-base homeostasis of the body. As early as 1932, Jaffe et al. noted the dissolution of bone material as a result of an acid environment during an in vitro study. Qualitatively, bone is a minor buffer, but when considering the overwhelming mass of bone within the body the quantitative importance becomes obvious (Winters et al., 1969). Smith and Riggs, (1975) reported that bone acts as a buffer of hydrogen ions by releasing Ca salts, in agreement with Lemann et al. (1966), who found that the release of Ca stores into the body fluids contributes to the alkalinity of the plasma. In addition, bone P, Na, magnesium and carbonate can contribute to the regulation of acid-base balance (Hegsted, 1973). Lemann et al. (1965) found that extra- and intracellular buffers shared equally in the

neutralization of a long term dietary acid load resulting from ammonium chloride ($NH_4Cl$) ingestion. After the extracellular buffers had become significantly titrated, the cell buffers plus the bone and finally only bone continued to provide buffer reserves. Milligan and Evans (1979) reported that acidic diets resulted in bone erosion in rats. In a study using human patients, Lemann et al. (1966) found that losses of calcium carbonate ($CaCO_3$) from the skeleton resulted in the neutralization of 1 meq of acid for each meq of Ca loss. In their work, Ca and P losses from the bone did not occur simultaneously, therefore indicating that the loss from the skeleton was not all $Ca_3(PO_4)_2$. P losses began with acid loading and stopped abruptly when the acid diet was discontinued. Ca losses rose slowly and progressively during acidosis and gradually declined after acid loading stopped. Therefore, some Ca loss must come from $CaCO_3$. Petito and Evans (1984) found in rats bone mineral is used as a buffer in acidosis imposed by $NH_4Cl$ and acidic phosphate. They reported a reduced femur dry wt and specific gravity (sp gr) in $NH_4Cl$ fed rats. Newell and Beauchene (1975) investigated the hypothesis that the lifelong utilization of the buffering capacity of the alkaline salts of the bone for the maintenance of acid-base balance may increase the incidence of osteoporosis. They found that no significant differences existed in bone

analysis for $NH_4CL$-fed rats. They found no difference in tibia wt or composition in rats on the acid stress diets. Petito (1981) produced acidosis in rats with acid phosphate and calcium lactate additions to the diet. They also used an alkaline phosphate and $CaCO_3$ diet to produce a high blood pH. The acid phosphate when combined with a high protein diet resulted in an additive loss of bone density, while rats fed $CaCO_3$ and alkaline phosphate had no significant bone loss. The more acid the phosphate, the greater the metabolic acidosis that was induced and the greater the loss of femur sp gr. In their research, bone was the ultimate buffer after the limitations of the blood carbonic acid system were reached. Barzel and Jowsey (1969) found dietary $NH_4Cl$-induced bone loss in the femur of rats. These researchers concluded that there may be differences among bones in the way they are affected by altered body acid-base balance. Crenshaw et al. (1981a) reported that the metacarpal bones of pigs were more responsive to dietary mineral differences than femurs were.

It has been proposed that metabolic acidosis produced by an acid loading diet or a high protein (amino acid) diet causes Ca mobilization from the bone either by increasing PTH secretion or by augmenting the action of PTH in mobilizing Ca from bone (Barzel, 1969; Wachman and Bernstein, 1970; Beck and Webster, 1976). Zemel and

Linkswiler (1981) showed an increase in cAMP during dietary induced bone resorption, indicating an increase in the PTH secretion. Acidosis has been shown to directly inhibit the kidney tubular reabsorption of Ca by a direct effect of the acidosis on metabolic processes within the renal tubular cells (Garn, 1970, Beck and Webster, 1976, Petito and Evans, 1984). Beck and Webster (1976) reported that acute metabolic acidosis directly increased Ca mobilization from the bone without any vitamin D involvement. Ca balance is, however, affected by urinary Ca excretion and gastrointestinal absorption as well as by Ca mobilization from bone. Thus it is difficult to elucidate all the mechanisms that may be involved in the mobilization of Ca.

Petito and Evans (1981) found that diet acidity could also lead to loss of bone size and mineral content of bone via phosphate released from the bone to buffer $H^+$. Increased serum Ca in this trial was excreted via the urine. Blood pH changes due to the diet were thought to cause PTH involvement in this response. They calculated a buffer or titration volume (TV) of a diet by titrating the diet with HCl and sodium hydroxide, thus mimicking the digestive processes in a closed system. Their research showed that the TV was a good predictor of the physiological response of an organism to an acid diet. Small or negative TV numbers

imply a low acid load, while large numbers indicate an increased acid load that the system must buffer.

*Determination of bone characteristics and strength.* If bone strength is to be used as a criterion of response to dietary acid, standardization of procedures for measuring and reporting bone strength and characteristics is essential. Specific gravity has been used as an index of bone response in some trials (Petito and Evans, 1981) because it serves as a measure of both organic and inorganic content of bone. The measurement of only mineral, as in ashing techniques, is inadequate since bone loss is a loss of organic as well as mineral content (Albanese, 1977).

Bone response has also been measured through assessment of shear strength. As bone mineralization increases, the maximum stress the bone can withstand increases (Crenshaw et al., 1981b). Measurement of stress allows comparisons to be made between bones that differ in size and shape. Crenshaw et al. (1981b) also reported that the inside diameters of bones in growing pigs respond to dietary variations in levels of Ca and P, but the outside diameters change very little. Crenshaw et al. (1981a) concluded that shear stress was a more sensitive indicator of mineralization than percentage of ash on the basis of the responses of bone to stress.

Shear stress according to Harner and Wilson (1985) is best defined by; Shear stress = Maximum Force / (2 X Area). Area of bone may be quite difficult to determine depending on the particular bone. Combs (1989) calculated the area of the metacarpals and metatarsals of swine based on the assumption that the bone resembled the shape of a quarter circle. An equation was derived to determine the area of the bone wall minus the marrow, which has no effect on shear strength.

*Body Composition*

During the first three wk of life much of the tissue that a pig deposits is fat. If suckling continues past this point, growth has a relative constant composition of fat, lean and water (Manners and McCrea, 1963). Pigs gain little wt during the first wk postweaning, however, and in some cases, actually have a net loss from weaning wt. The growth lag that has been observed following weaning has been reported to be a loss of lipid (Whittemore et al., 1978), composed primarily of body fat stores (Whittemore et al., 1981). The researchers postulated that weaned pigs catabolize lipid from subcutaneous fat deposits and the lost weight is replaced by retained water. In their research with pigs weaned at 28 d, they reported that when empty body

weight gain dropped below 193 g/d, young pigs were likely to catabolize body lipid stores.

Once the pig has overcome the postweaning lag phase any wt gain following weaning is expected to be of a relatively constant composition (Manners and McCrae, 1963 and Whittemore et al., 1978) and linear. The slope of the curve, is however, dependent upon genetic, environmental and nutritional factors (Robison, 1975). Standal (1973) suggested that backfat deposition in 56 d old pigs was linear relative to either age or wt. Also, Hendrick (1983) reported that fat depth is positively associated with yield of fat cuts and negatively associated with yield of lean cuts. Based on this information, accurate determination of fat depth and loin muscle area in the older weanling pig or young grower pig could be used to predict final carcass composition as part of an early selection process.

*Backfat and Loin Muscle Determination*

The first technique to determine fat depth in live pigs was the ruler probe (Hazel and Kline, 1952). The ruler probe was accurate in fat depth determination, but involved an incision on the pig. Until the development of ultrasound, however, there were no methods for the determination of loin muscle area.

Wild (1950) first reported the use of ultrasonics to determine density boundaries without destruction of tissue. Since that time, ultrasound equipment has been used in pig selection programs. The instruments used have evolved widely over the years, ranging from small, portable A-mode scanners for measurement of backfat only, to B-mode instruments that are capable of producing two-dimensional images. Reports on the usefulness of backfat thickness and loin muscle area to predict carcass composition are extensive in the literature. Results of studies using real-time (B-mode) ultrasonography, which yields a continuous two-dimensional image, to estimate body composition in live pigs are limited.

Hendrick (1983) demonstrated that the relationships between fat thickness or longissimus (loin) muscle area measured using real-time ultrasonography in the live animal were similar to relationships obtained between the same measurements of the carcass and carcass composition. Work by McLaren et al. (1989) indicated that nursery and growing phase measurements had similar predictive power as preslaughter measures. Their correlations between measurements of preslaughter ultrasound and carcass last rib fat, avg. fat, tenth rib fat and loin muscle area at the tenth rib were reported to be .55, .62 .55 and .61, respectively. They concluded that real-time ultrasound

could prove to be a useful tool in early selection decisions. Correlations reported by Forrest et al. (1986) for tenth rib fat (.71) and loin muscle area (.68) in market hogs were slightly better than those presented by McLaren et al. (1989). Lopes et al. (1987) working with similar age pigs reported tenth rib fat correlations ranging from .8 to .89, last rib fat correlations of .75 to .89 and loin muscle values of .27 to .71.

As is evident there is considerable variation in the results. Although authors reported good prediction accuracy, variability was also high and source of variability must be kept in mind. Operator effects, both in obtaining the scans and interpretation of the same have been indicated as one source of variation (Bailey et al., 1988). Additional variation results from the differences in ultrasound (sound reflected) images and those received by the eye (light reflected) on visual appraisal of the carcass. There is not a one to one correlation between light and sound reflection, therefore a perfect correlation between the two types of measurements cannot be expected. Through close observation to minimize error, ultrasound has the ability to be a beneficial tool for the estimation of body composition.

Dietary Mineral Sources

The mineral or electrolyte composition of the diet can influence the acid potential of that diet. Therefore, by using different mineral sources, the pH of a diet can be manipulated without the addition of organic acids. Ca sources such as Ca lactate and $CaCO_3$ may be used, because Ca lactate acts as a potentially acid molecule and $CaCO_3$ as a potentially basic molecule (Petito and Evans, 1984). In addition, the contribution of P to diet acidity must also be considered. Mono-dicalcium phosphate (DCP) provides an acidic pH but defluorinated rock phosphate (DFP) is potentially a basic molecule. To effectively use these different mineral sources, differences in their metabolic effects must be considered.

Hagemeier et al. (1981) reported no difference in bone breaking strength between pigs fed DFP or DCP, but the DFP-fed pigs had larger metatarsals that were weaker per unit of area than the DCP-fed pigs. In their research, however, there was a significantly higher level of $Na^+$ in the DFP diet. This higher $Na^+$ level may have affected the acid-base balance or electrolyte composition of the diets. Kornegay et al. (1990) found DFP and DCP sources equally effective for bone mineralization and breaking strength in pigs. When metacarpals and metatarsals were examined on a body wt corrected basis, P source did not influence the wt, size or mechanical characteristics. These researchers found no

consistent significant effect from trial to trial of P source on sp gr of fresh bones.

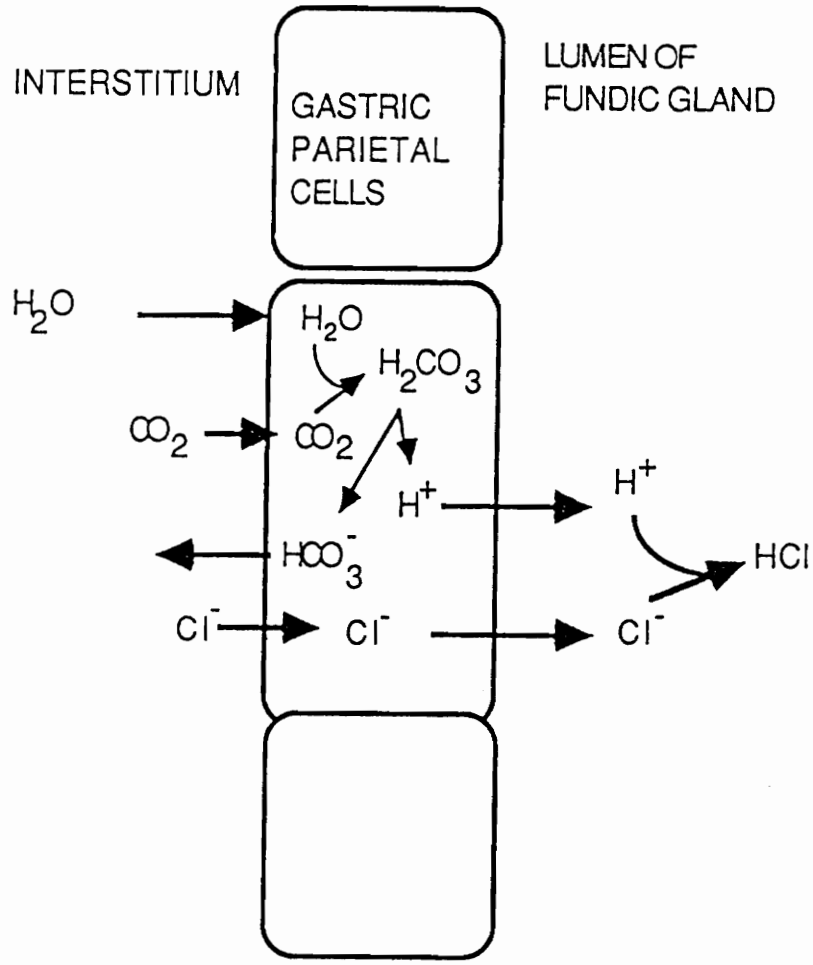


Figure 1. Illustration of hydrochloric acid production in gastric parietal glands (adapted from Currie, 1988).

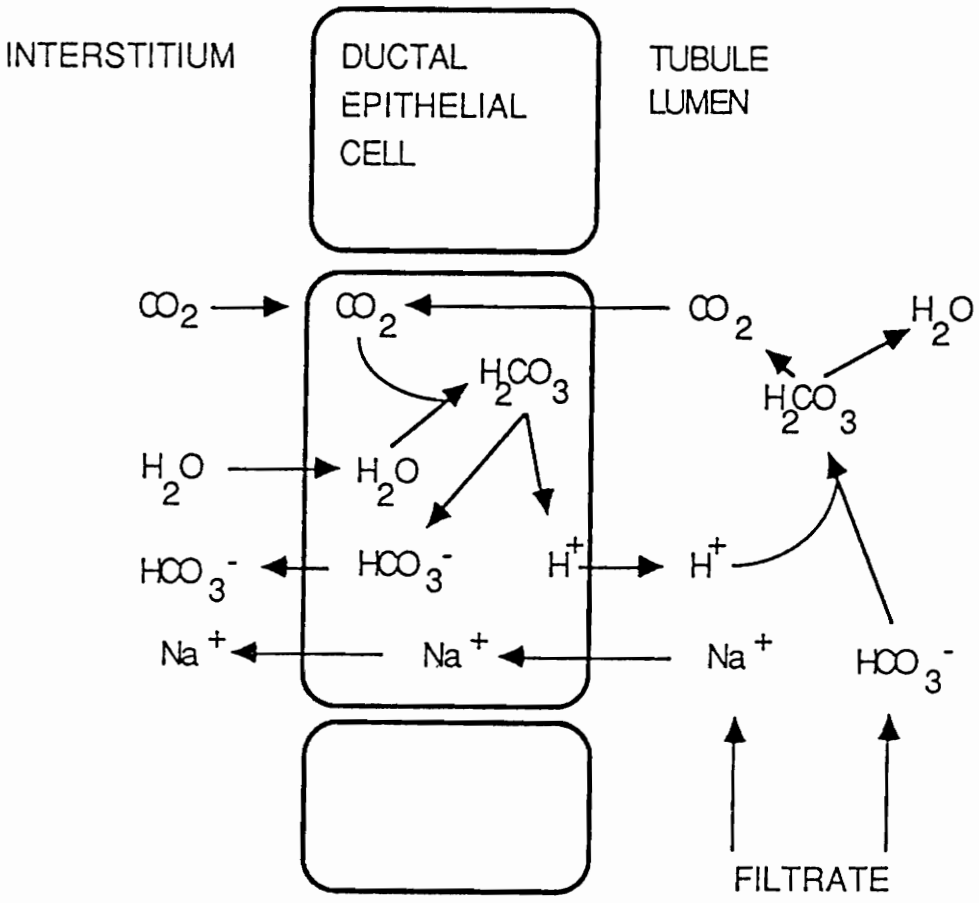


Figure 2. Illustration of pancreatic bicarbonate production (adapted from Currie, 1988).

# CHAPTER III

## RESEARCH JUSTIFICATION

There has been extensive research evaluating the use of organic acids to alter dietary pH in efforts to improve the performance of weanling pigs. There is, however, limited published data on the effects of inorganic acids and mineral sources to vary dietary pH and resultant swine performance. In addition, data on the effects of dietary pH alterations on gastrointestinal parameters are conflicting. Research evaluating the influence of dietary pH on bone characteristics and body composition is also lacking. This research was designed to investigate the effectiveness of varying mineral sources and inorganic acids for changing dietary pH, and to characterize the influence of dietary pH and phosphorus source on postweaning performance, gastrointestinal digesta pH, bone development and body composition of weanling pigs.

# CHAPTER IV

# OBJECTIVES

This study was conducted to examine the effects of altering dietary pH through the manipulation of dietary mineral sources and the addition of either hydrochloric acid or sodium hydroxide on the performance of weanling pigs.

Specific objectives were:

1) to determine the performance of weanling pigs fed diets formulated to different pH levels and using different phosphorus sources;
2) to explore the effects of dietary pH and phosphorus sources on the gastrointestinal digesta;
3) to evaluate bone characteristics of relative to dietary pH and phosphorus source; and
4) to determine whether dietary pH or phosphorus source influences backfat depth and loin muscle area.

# CHAPTER V

## THE EFFECT OF DIETARY pH AND PHOSPHORUS SOURCE ON PERFORMANCE, GASTROINTESTINAL DIGESTA, BONE CHARACTERISTICS AND BODY COMPOSITION IN WEANLING PIGS.

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## ABSTRACT

Crossbred pigs (n=144, avg age and weight - 28 ± 3 d, 7.5 kg) were used in two 6 wk trials to assess the effects of dietary pH and phosphorus source on performance, gastrointestinal digesta pH and chloride ion concentration ($Cl^-$), bone characteristics and body composition. Pigs were blocked according to weight within sex and litters were balanced across groups. Treatments were randomly allotted within blocks to a 3 x 2 factorial arrangement with three dietary pH levels (5.4, 6.0 and 6.7) and two phosphorus sources: dicalcium phosphate (DCP) and defluorinated phosphate (DFP). Pigs fed the pH 6.7 diet had reduced average daily gain (ADG, $P < .01$) and average daily feed intake (ADFI, $P < .001$) during wk 1-3 and overall compared with pigs fed the pH 6.0 diet, but ADG and ADFI were not effected when the pH 5.4 diet was fed. There was a dietary pH by phosphorus source interaction ($P < .05$) for ADFI. DCP fed pigs had increased ADFI as dietary pH was reduced from 6.7 to 5.4, but DFP fed pigs had similar ADFI as dietary pH decreased from 6.0 to 5.4, and decreased ADFI as dietary pH

increased from 6.0 to 6.7. Dietary pH had little influence on F:G, and phosphorus source had little effect on either ADG, ADFI or F:G.

Dietary pH did not influence the pH and $Cl^-$ of the digesta for any gastro-intestinal section measured, except the $Cl^-$ in the stomach; both pH 5.4 and 6.7 fed pigs had a higher ($P < .01$) $Cl^-$ than the pH 6.0 fed pigs. Only shear force of the fourth metacarpal and specific gravity of the fourth metatarsal were increased ($P < .01$) for pigs fed the DCP compared with DFP diets. Neither dietary pH nor phosphorus source influenced backfat or loin muscle area.

These results suggest that maintaining the acidifity of the diet during the first 3 wk after weaning at 28 d of age is important with the primary response seen in ADFI. Varying dietary pH from 5.4 to 6.7 had little or no effect on gastrointestinal digesta characteristics, bone development and body composition.

(Key words: Pigs, Acidity, Alkalinity, Phosphorus, Hydrochloric acid, Sodium hydroxide.)

# INTRODUCTION

The growth check usually observed during the first week after early weaning is primarily due to inadequate feed intake and the lack of an appropriate gastric environment for optimal digestion (Blair et al., 1963). Liebholz (1984) reported that the 28 d old pig had limited ability to hydrolyze dietary protein in the stomach, which according to Rerat (1981) was a result of excesively high gastric pH for adequate activation of pepsin. Manners (1970) reported that the lack of gastric acidity resulted in an increased susceptibility of the young pig to outbreaks of gastrointestinal disease due to an increased multiplication of microorganisms. Thus, the acidification of the diet and the gastrointestinal digesta should improve performance and reduce scouring in the weanling pig.

Several researchers (Kirchgessner and Roth, 1982; Falkowski and Aherne, 1984; Giesting and Easter 1985; Henry et al., 1985; Risley et al., 1990) have shown improvements in performance of weanling pigs with dietary additions of organic acids which lower dietary pH. When inorganic acids have been used to alter dietary pH, the acid-base balance of the pig has been altered and the effect on performance has been inconsistent (Giesting, 1986). Dietary acidity/ alkalinity can also be influenced by using different mineral

sources (Patience et al. 1987), and their reduced cost compared with organic acids may warrant their use for pH modification of practical swine diets.

Evidence in rats and humans suggests that a more acidic diet can result in the dissolution of calcium (Ca) from the bone (Petito and Evans, 1984 and Lemann et al. 1966). Lemann et al. (1966) found that the release of Ca stores into the body fluids contributes to the alkalinity of the plasma and thus acts as a buffer in the maintenance of acid-base homeostasis.

The objective of this research was to determine the effects of altering the pH of the diet through inorganic acid and base additions and by varying the sources of minerals (Ca, P, Na and Cl), and their respective anion or cation components on performance, gastrointestinal digesta, bone development, loin muscle area and backfat depth measurements of weanling pigs.

## MATERIALS AND METHODS

One hundred and forty-four crossbred pigs (equal numbers of barrows and gilts), with an average initial weight of 7.5 kg and age of 28 ± 3 d, were used in two 42 d trials (72 pigs/trial). Pigs were blocked according to weight within sex, and litters were balanced across groups. Treatments were randomly allotted within blocks to a 3 X 2 arrangement of treatments which included three dietary pH levels (5.4, 6.0 and 6.7) and two phosphorus sources (DCP - dicalcium phosphate and DFP - defluorinated phosphate). The three pH levels were attained by the addition of either hydrochloric acid (HCl), no additive or sodium hydroxide (NaOH). All diets (Table 1) consisted of an 18% crude protein corn-soybean meal basal diet and were formulated to meet or exceed NRC (1988) levels for 10 to 20 kg pigs.

Pigs were housed in enclosed temperature controlled nurseries, in .6 x 1.5 m wire bottom pens, two pigs per pen. Temperature (Hinkle et al., 1978) and ventilation rates (Lubinus and Murphy, 1979) were maintained at recommended levels. One pig in each trial died before completion of the trial, but the deaths were not believed to be related to the treatments.

Pen feed consumption (2 pigs) was measured daily and the pigs were weighed weekly. Diets were mixed weekly and sampled. To ensure analysis of a representative feed

sample, a composite sample for each dietary treatment was made based on the average weekly feed consumption for the 12 pens of each treatment group.

Mineral analysis of feeds (Table 2) involved nitric-perchloric digestion and Atomic Absorption Spectophotometry for Ca and Na. Phosphorus was determined by colorimetric procedure as described by Fiske and Subbarow (1925). The remainder of the minerals were determined by inductively coupled plasma spectrophotometry. Dietary undetermined anions (dUA) in meq were calculated according to the procedure of Patience (1990) and dietary electrolyte balance in meq was calculated according to Mongin (1981). Dry matters were determined by placing duplicate .5 g feed samples in preweighed and predried pans, drying at 80$^{o}$ C until no further weight was lost. For the final weighing, the samples were placed in a desiccator until cool so that total dryness could be assured.

Dietary pH was determined on a 5 g subsample of each diet sample which was mixed into 50 ml of deionized water. This slurry was continuously mixed with an electromagnetic mixer and stir bar while the pH was determined with a pH meter (Fischer Accumet model #620) and combination electrode (Fischer Accu-pHast #13-620-281). To determine titration value (TV), this sample was shaken for 30 min, centrifuged (275 X g) and the pH of the supernatant determined. The

supernatant was taken to a pH of 4 using .2N HCl and then back titrated to a pH of 8 with .2N NaOH. The TV was determined by subtracting meq of HCl required from meq of NaOH according to the procedure of Petito and Evans (1981). Dietary TV values are shown in Table 2.

Particle size was determined for DCP and DFP by placing a 100 g sample into a series of sieves (20, 40, 60, 140, 200, 270 and 325 mesh corresponding to .841, .325, .250, .105, .074, .053 and .045 mm respectively) and shaken for 10 min. The mean particle size diameter was then determined using the Kansas State University particle size analysis program.

Pigs were killed by electrocution for 20 seconds and exsanguinated via the vena cava. The gastrointestinal tract was exposed by an incision from the sternum to the rectum and promptly tied off at the posterior end of the stomach. Stomach contents were emptied and weighed, titrated and pH and Cl ion concentrations ($Cl^-$) determined. Samples were taken from the jejunum (.5 m starting at a point .5 m from the proximal end of the duodenum), cecum and distal colon.

Stomach dry matters were determined similarly to feed dry matter, by using 5 g subsamples. A 2 g sample of digesta from each gastrointestinal section was diluted with 20 ml of deionized water and maintained at 37$^{o}$C. The pH was determined in the same manner as dietary pH.

Determination of $Cl^-$ was accomplished with the same 2 g sample using a Cl electrode (Orion Research Ionalyzer #94-17-B) and double junction reference electrode (#90-02). Standards were used to determine reference points. Each sample had 1 ml ionic strength adjuster (5 M sodium nitrate) added and was analyzed in millivolts, with the $Cl^-$ calculated from the standard curve. Stomach digesta TV were determined similarly by placing a 10 g digesta sample in a 100 ml beaker and adding 50 ml of deionized water. The beaker was maintained at physiological temperature (37°C). With continuous stirring, .2N HCl was added until a pH of 2 was reached. The sample was back titrated to a pH of 8 using .2N NaOH.

The left humeri were removed and frozen at -18°C in moisture-proof bags. Also, the left front and rear feet were removed at the carpal joints and frozen at -18°C in airtight bags so that the third and fourth metatarsals and metacarpals could be obtained. Sedlin (1965) and Sedlin and Hirch (1966) reported that freezing before testing did not affect the mechanical properties of the bone.

The metacarpal and metatarsal bones were thawed and excised by manually removing the surrounding soft tissue. Fourth metacarpals and metatarsals were rehydrated in deionized water at room temperature for 1 hr, blotted dry, and weight in air and water taken using a Mettler AE240

balance. Volume of bone was calculated as the weight in air minus the weight in water. Specific gravity (sp gr) was determined as weight in air divided by the volume. Bones were refrozen in moisture-proof bags until shear force could be determined.

For determination of shear force, the fourth metacarpals and metatarsals were thawed in moisture-proof bags at room temperature. Length and diameter at mid point of the bones were taken using a caliper (Figure 1). Bones were then placed in a holding apparatus (Figure 2) in the position at which the highest force would be achieved (Figure 1), as decribed by Crenshaw et al. (1981a). Shear force was determined as the force (kg) required to shear the bone using an Ingstron Universal Testing Machine (Model TM 1123, Ingstrum Co., Cantron MA, 02021) with a head speed of 20 mm per min. Once the bone was sheared, it was cut near the midpoint with a band saw and wall thickness was determined as the average of three thickness measurements. Bone area was calculated according to the procedures of Comb (1989) (Figure 3). Shear stress was calculated as the maximum force divided by twice the area (Harner and Wilson, 1985).

Ultrasonic measurements of fat and loin muscle area were recorded on the empty body in Trial 1 and on the live animal in Trial 2. Trial 1 backfat measurements were taken

at the tenth rib using the Equisonic LS300A real-time ultrasound equipped with a 3.5 MHz probe and an Aloka 210 real-time ultrasound equipped with 5.0 MHz and 7.5 MHz probes. Only the 3.5 MHz probe was used for loin muscle measurements. In Trial 2, both tenth rib backfat thickness and loin muscle area were determined using the Aloka 210 machine and a 3.5 MHZ probe (UST.5021-3). The two dimensional images were recorded on VHS video tapes and played back on a VCR which allowed stop action and accurate display of single ultrasound image frames. This image was then traced on acetate paper from a 31.5 cm monitor. Backfat depth and loin muscle area were calculated from the acetate using a computerized distance and area program, a digitizer. Conversion factors were used to adjust for screen size.

After slaughter, grid and ruler measurements of the chilled tenth rib section were recorded and tracings of the loin muscle area and fat were transcribed using the digitizer.

*Statistical analysis.* Data were analyzed using the GLM program of the statistical analysis system (SAS, 1985) with pen as the experimental unit for performance. Gastrointestinal, bone and body composition measurements were also analyzed using the GLM program but with individual pigs as the experiemtnal unit. All sex and trial

interactions were tested and removed if not significant. Comparisons between dietary pH treatments were made using non-orthogonal contrasts. Body composition and ultrasound data were analyzed by a multivariate analysis of variance with partial correlations.

## RESULTS AND DISCUSSION

The majority of the dietary pH by phosphorus source interactions were nonsignficant. Therefore, main effect means will be compared and significant ($P < .05$) dietary pH by phosphorus source interactions will be pointed out in the discussion. Treatment means, however, are presented in the tables. Diets with a pH of 5.4 or 6.7 were compared with the pH 6.0 diet by using contrasts, since the pH 6.0 diet received no pH manipulation by adding HCl or NaOH to lower or raise the pH, and it therefore more closely resembles that of a typical swine diet. The data reported as overall is that obtained for wk 1-6 with the exception of two replications (12 total pens) in Trial 1, which were slaughtered at the completion of wk 5, so that their overall data is actually wk 1-5. Also, wk 4-6 does not include wk 6 of two replicates in Trial 1. Tables including performance means for each week, main effects and trial by main effects interactions are present in the appendix.

### *Performance measurements*

During wk 1-3, pigs fed pH 6.7 diets had reduced average daily gain (ADG) ($P < .01$) via a reduction in average daily feed intake (ADFI, $P < .001$) compared with pigs fed pH 6.0 diets (Table 3). Across phosphorus source, ADG and ADFI of pigs fed pH 5.4 diets were not significantly

different from those of pigs fed pH 6.0 diets. The phosphorus source by dietary pH interaction was significant ($P < .05$) for ADFI. When DCP was used, decreasing the pH of the diet from 6.0 to 5.4 increased ADFI and increasing the pH from 6.0 to 6.7 decreased the ADFI, whereas when DFP was used, decreasing the pH of diet from 6.0 to 5.4 had no effect on ADFI, but increasing the pH from 6.0 to 6.7 also decreased the ADFI. A similar numerical relationship was observed for ADG, but the phosphorus source by dietary pH interaction was nonsignificant. Feed to gain ratio (F:G) tended to increase ($P < .10$) as dietary pH increased from 5.4 to 6.0 to 6.7, with the phosphorus source by dietary pH interaction being significant ($P < .05$). The F:G increased as dietary pH increased when DCP was used as the phosphorus source, but dietary pH had little effect on F:G when DFP was used.

During wk 4 - 6, the influence of dietary pH on performance was not as great as that observed during wk 1-3. Numerically, the highest ADG, ADFI and F:G were observed for pigs fed pH 6.0 diets; the largest differences were observed between pigs fed 6.0 and 6.7 diets, and for pigs fed diets using DFP. The smallest differences were observed between pigs fed the pH 5.4 and 6.0 diets containing DCP.

Overall, ADG and ADFI were approximately the same for pigs fed pH 5.4 and 6.0 diets, but were decreased ($P < .01$

and $P < .001$) when pH 6.7 diets were fed. Although the dietary pH by phosphorus source interactions were not significant, the patterns observed during wk 1-3 were evident for the overall values. That is, ADG and ADFI decreased as dietary pH increased from 5.4 to 6.0 to 6.7 when DCP was used, whereas, when DFP was used ADG and ADFI increased as dietary pH changed from 5.4 to 6.0 and then decreased as dietary pH increased from 6.0 to 6.7. Dietary pH did not influence F:G.

With the exception of F:G during wk 4-6, and overall, which tended to be improved ($P < .10$) for pigs fed diets using DCP compared with DFP (1.92 vs. 1.98), performance was similar for pigs fed different phosphorus sources during all time periods.

The improvement in ADG and ADFI observed with decreasing dietary pH during the first three weeks after weaning are in agreement with Falkowski and Aherne (1984) and Giesting and Easter (1985). Risley et al. (1990) reported that organic acid addition to DFP diets resulted in improvements in ADG and F:G for the first four weeks following weaning at 24 d of age and Giesting et al. (1985) reported improved performance for the first two weeks following weaning at 28 d of age when fumaric acid was added to DCP diets. Contrary to work by Radecki et al. (1988) who used organic acids, and Giesting (1986) who uses HCl additions to weanling pig

diets, ADFI was increased with decreasing dietary pH in our study. The Giesting (1986) work using HCl treatment of diets resulted in a 1.3% $Cl^-$ level which may have affected dietary electrolyte balance (dEB) and thus intake and performance. The dEB for that work was -6.7 meq, while in our trials, $Cl^-$ concentration and dEB for all treatment groups averaged .49%, and 118 meq, respectively. Work by Patience et al. (1987) found that dEB in the low and negative ranges adversely affected performance.

Giesting et al. (1985) suggested that dietary acidification of diets for weanling pigs improves performance by improving protein and energy digestibilities. Fumaric acid at the 1.5 to 2% level improved energy and protein digestibility by 2 to 3% and N retention by 5 to 7% in weanling pigs in work by Kirchgessner and Roth (1982). Tschierschwitz et al. (1982) reported that the activity of aspartate transferase, alanine transferase and succinate dehydrogenase were increased with the addition of fumaric acid in rats suggesting a modification of intermediary metabolism of protein and energy. Liebholz (1984) reported that only 6% of the dietary protein is hydrolyzed in the stomach of the 28 d old pig, increasing to 50% by 56 d when the pH of the stomach decreased to about 4. Her research showed that the major site of dietary protein hydrolysis in young pigs is not the stomach, but the duodenum and jejunum

(44 and 34% respectively), with most of the hydrolysis due to pancreatic proteolytic enzymes. Proteolytic digestion by pancreatic enzymes was greatly reduced in growing and finishing pigs. Thus, one proposed method of action for performance improvement with dietary acidification is a reduction in gastrointestinal pH, which may increase the amount of protein digested in the stomach by increasing the activity of pepsin.

*Selected gastrointestinal measurements*

Manipulation of dietary pH above or below 6.0 generally did not significantly affect stomach TV values and pH and $Cl^-$ concentrations of the various gastrointestinal sections measured with the exception of stomach pH and $Cl^-$ concentration (Table 4). Stomach pH tended to be lower ($P < .06$) for pigs fed the pH 5.4 diet as compared to the pH 6.0 fed pigs but was not different from the pH 6.7 fed pigs. A higher $Cl^-$ concentration was observed for pigs fed pH 5.4 diets ($P < .003$) and pH 6.7 diets ($P < .005$) compared with pH 6.0 diets.

Phosphorus source also had no effect on stomach TV values, or pH and $Cl^-$ concentrations of the digesta from the various gastrointestinal sections, with the exception of stomach DM and colon pH (Table 4). Dry matter values were lower ($P < .01$) for pigs fed DFP diets compared with DCP

diets. Pigs fed DFP diets also had a lower colon pH ($P < .001$) than did DCP fed pigs.

It has been suggested (Scipioni et al., 1978; Redecki et al., 1988) that the mode of action for improved performance of organic acid supplemented pigs is due to the acidification of gastrointestinal digesta. Scipioni et al. (1978) reported a reduction in stomach pH's from 4.5 to 4.3 and 3.5 in 42 d old pigs with the addition of either .7 and 1% citric or fumaric acid. Working with 70 d old pigs, Risley et al. (1990) found no differences in the pH of any gastrointestinal section when pigs were fed 1.5% of either citric or fumaric acid, although dietary pH was reduced by the addition of organic acid (pH 4.9 and 4.7 vs 6.4). Our results are in agreement with Risley et al. (1990) and suggest that reduced pH of the gastrointestinal digesta may not be the mode of action for improved performance seen when diets are acidified. It should, however, be pointed out that gastrointestinal digesta samples in our study and those of Risley et al. (1990) were taken at the termination of the trial. During the last week of both studies, no differences in performance due to dietary treatment was observed.

As the pig matures and its body and stomach size increase, there is a concomitant increase in the volume of HCl secreted (Cranwell, 1985). Lindemann et al. (1986) reported an increase in pancreatic and gastric enzyme

activity with age, due to increases in tissue weight and enzyme activity per g of tissue. They also found a depression in pancreatic enzyme activity but not gastric proteolytic activity during the first week following weaning. Liebholz (1984) reported that the hydrolysis of soy protein diets improves with increasing age of the pig, particularly from 28 to 56 d. Few researchers have reported dietary acidifiation results past 4 wk. Scipioni et al. (1978) reported that improvements observed during the first 3 wk postweaning with dietary acidification were lost during wk 4-6. With these data in mind, it appears that as the pig ages and digestive capabilities mature, any improvement in protein and energy digestibilities due to dietary acidification would be minimized, as would performance improvements. Our results have shown improvements due to dietary acidification in ADG and F:G during the first three wk post weaning, when the pigs were approximately 50 d of age. After this time, pigs fed the acidified diet had less of an advantage over the other dietary pH levels. These results would support the hypothesis that the young weanling pig may be unable to efficiently digest certain nutrients. Lack of adequate levels of HCl in the stomach (Cranwell, 1985) may limit pepsinogen activation and protein utilization. Digesta samples taken at slaughter indicate little, if any, gastrointestinal pH alteration as a result

of HCl or NaOH addition. This may be due to increased maturation of the digestive tract by slaughter age (63 ± 2 d) and thus the pigs were better able to adjust their stomach pH.

*Bone measurements*

Volumes were greater ($P < .03$ and $P < .06$) and sp gr values were lower ($P < .05$ and $P < .06$) for the metacarpals and metatarsals, respectively, from barrows compared with gilts (Table 5). Also sp gr of the humerus was lower ($P < .04$) for barrows compared with gilts. Force and stress measurements for metacarpals were smaller ($P < .05$) for barrows compared with gilts, but force and stress values for metatarsals were not different between barrows and gilts.

Crenshaw et al (1981b) reported that bones from barrows withstood less stress than those of gilts when force was expressed per unit of bone area. Lower stress values of bone are indicative of less mineralization. It can be concluded from shear stress and sp gr measurements in these trials that barrows have less mineralized, therefore, weaker bones than gilts. This is in agreement with work by Liptrap et al. (1970) who found that when stress of bones was calculated, boars were found to have weaker bones than gilts.

As shown in Table 6, the various bone measurements were not influenced by dietary pH with two exceptions: volume of metacarpals of pigs fed pH 6.0 diets tended to be larger than those of pigs fed pH 5.4 diets ($P < .06$) and pH 6.7 diets ($P < .10$), and shear stress of metatarsals of pigs fed pH 5.4 diets was less, ($P < .10$) than those of pigs fed pH 6.0 diets. There was no difference in shear stress of metatarsals of pigs fed pH 6.7 diets as compared to pH 6.0 diets.

Phosphorus source generally did not affect the various bone measurements with the exception of metacarpal force ($P < .10$) and metatarsal sp gr ($P < .01$), both of which tended to be lower for pigs fed diets with DFP compared with DCP diets. No phosphorus source by dietary pH interactions were observed for any bone measurements.

Hagemeier et al. (1981) reported decreases in bone strength per unit area of the bone cross section for DFP compared to DCP in finishing pigs fed .3% P. They suggested that the observed decrease in bone strength in DFP fed pigs may have been due to higher dietary Na levels. In our research, however, the level of Na was .22% and .23% for DCP and DFP respectively. This suggests that differences due to phosphorus source were not due to dietary Na variations. In agreement, Kornegay et al. (1990) reported no differences in

bone strength with diets of varying Na levels and wider P to Na ratios than those fed by Hagemeier et al. (1981).

Work by Cromwell et al. (1987) found the bioavailabilty of P from DCP to be 105%, while that of DFP was only 87%. In our research, analyzed dietary P levels for DCP diets averaged .60% while those for DFP diets were .65%. These meet or exceed the suggested NRC (1988) requirements for both total P (.60%) and available P (.32%) for the the 10 - 20 kg weight pig. Evans (personal communication) indicated that their research has shown a reduction in net P retention in rats with decreased particle size of DFP due to an increased rate of excretion. In our research DCP mean particle size was .55 $\pm$ .002 mm while DFP mean particle size was much smaller at .06 $\pm$ .002 mm. Burnell et al. (1988) reported no differences in growth rate, F:G, femur strength or metacarpal and metatarsal strength in pigs fed DFP particles sizes from .05 to 2.0 mm. Likewise, Crowmell et al. (1987) reported no differences in gain or F:G when pigs were fed DCP or DFP (coarse or fine). Femur strength was, however, less in pigs fed DFP as compared to DCP or monosodium phosphate. They concluded that particle size did not influence the availability of P from DCP or DFP and that DFP was approximately 20% les bioavailable than DCP. In our research, increases observed in metacarpal force and metatarsal sp gr for pigs fed DCP compared with DFP suggests

a lower biological availablity of P in DFP. However, it should be noted that other bone measurements were not different between phosphorus sources.

According to Petito and Evans (1984) acidic diets can cause bone erosion and resultant decreased stress values and sp gr due to the use of the bone as a buffer for the body acid-base system. Kirchgessner and Roth (1982), however, found that in pigs acidification of the diet with fumaric acid improved the retention and balance of Ca and P by 14 and 13%, respectively. Differences observed by different researchers may be due to the levels of dietary acidification that occurred. In the trials reported here no differences in bone parameters due to dietary acidity were observed, suggesting that the acid-base equilibrium of these pigs was not influenced sufficiently by dietary acidity to incur an acidotic condition and thus require the utilization of bone as a buffer.

*Body Composition*

Neither dietary pH nor phosphorus source influenced backfat or loin muscle area as measured by ultrasound or grid and ruler at slaughter (Table 7). There was, however, a significant ($P < .05$) dietary pH by phosphorus source interaction observed for loin muscle area. This relationship was similar to that observed in performance

measurements with the loin muscle area increasing as dietary pH decreased from 6.7 to 6.0 to 5.4 for DCP fed pigs. When DFP was used as the P source, the pH 6.0 pigs had larger loin muscle areas than did the pH 5.4 or 6.7 fed pigs. Published research is limited on the effect of dietary pH on lean and fat tissue growth in pigs. However, from these results it appears that additional work on the effect of dietary pH alterations should include investigations of body composition and whether any effect would continue to be expressed at normal slaughter weights. There was also a significant effect of trial on backfat depth ($P < .01$) and loin muscle area ($P < .001$). We suggest that these are genetic differences related to sire and dam effects.

There is limited data available on ultrasound measurements for feeder pigs. To determine the usefulness of real-time ultrasound in determining body composition of pigs used in nursery trials, partial correlations between ultrasound values and carcass measurements were calculated for Trial 1, in which ultrasonic measurements were calculated on the empty body and for Trial 2, in which, the ultrasonic measurements were calculated on the live animal. The correlations were based on 72 (Trial 1) and 59 (Trial 2) observations. Our research showed significant correlations between real-time ultrasound and grid and ruler measurements at slaughter (Tables 8 and 9).

In Trial 1, loin muscle area ultrasound values were significantly correlated ($P$ <.01) with carcass tracing measurements with a coefficient of .63 for the 3.5 MHz probe (Table 8). Tenth rib fat depth ultrasound measurements were also significantly ($P$ < .01) correlated to carcass backfat measurements, with coefficients of .50, .39 and .43 for the 3.5, 5.0 and 7.5 MHz probes respectively. Only the 3.5 MHz probe was used in Trial 2, where carcass backfat and loin muscle area were both significantly correlated ($P$ < .05 and $P$ < .001, respectively) to live animal ultrasound values (Table 9). Lopes et al. (1987) reported correlations of .80 to .89 for tenth rib fat and .27 to .71 for loin muscle area, while Forrest et al. (1986) reported the correlations between live and carcass measurements to be .71 for tenth rib fat and .68 for loin muscle area of market weight pigs. Neither of these researchers reported the frequency of probe used, therefore, direct comparisons are difficult. Partial correlations of .60 and .61 for tenth rib fat and loin muscle area of feeder pigs using a 3.5 MHz probe were reported by McLaren et al. (1989).

In our trials the correlations were comparable to those of McLaren et al. (1989). Operator effects, both in obtaining the scans and interpretation of the same has been indicated as one source of variation (Bailey et al., 1988). Additional variation results from the differences in

ultrasound (sound reflected) images and those received by the eye (light reflected) on visual appraisal of the carcass (Bailey et al., 1988). There is not a one to one correlation between light and sound reflection, therefore a perfect correlation between the two types of measurements cannot be expected. Work by McLaren et al. (1989) also indicated that nursery and growing period measurements had similar predictive power as preslaughter measures. Including ultrasound data in growth prediction equations for barrows increased the $R^2$ values across dependent variables by 68% for grower data. These results indicate that if error in interpretation of ultrasound images is minimized, this procedure may be a useful tool in estimating carcass composition in the live animal at various stages of growth.

# CONCLUSIONS

A decrease in dietary pH resulted in an increase in the performance of weanling pigs by improving average daily gain and feed intake during the first three weeks postweaning. The effects were more prominent for pigs fed DCP diets compared with DFP diets. This effect diminished as the pigs aged and was absent after approximately 50 days of age. Gastrointestinal digesta pH measurements taken at the end of the 6-wk trial were generally unaffected, which suggests that improvements in performance might not have been elicited through a reduction in gastric pH or that the timing of slaughter at the end of the trial missed any changes in the gastrointestinal digesta measurements that might have occured earlier. Dietary pH ranges from 5.4 to 6.7 had no effect on bone characteristics or body composition measurements that were taken. Dicalcium phosphate tended to improve F:G and bone mineralization when compared with DFP.

Further research into the determination of the mode of action for diet acidification is needed. One possible area of research is in the continuation of work by Kirchgessner and Roth (1982) and Giesting et al. (1985) into the effect of acidification on protein and energy digestibility. Also, it would be of benefit to understand the effects of dietary buffering capacity on the absorption of nutrients across brush border membranes in the gastrointestinal tract.

TABLE 1. PERCENTAGE COMPOSITION OF EXPERIMENTAL DIETS[a]

| | DCP[b] | | | DFP[b] | | |
|---|---|---|---|---|---|---|
| Ingredient | pH 5.5 | pH 6.0 | pH 6.7 | pH 5.5 | pH 6.0 | pH 6.7 |
| Trial 1 | | | | | | |
| Ground corn | 67.00 | 67.00 | 67.00 | 67.00 | 67.00 | 67.00 |
| SBM (44%) | 28.00 | 28.00 | 28.00 | 28.00 | 28.00 | 28.00 |
| DCP | 1.26 | 1.26 | 1.26 | - | - | - |
| DFP | - | - | - | 1.28 | 1.28 | 1.28 |
| $CaCO_3$ | - | .32 | .30 | - | .28 | - |
| Ca lactate[c] | 2.33 | .78 | - | 1.44 | - | - |
| Salt | .83 | .71 | .41 | .51 | .72 | .41 |
| NaOH | - | .09 | .30 | - | - | .21 |
| HCl | .08 | - | - | .27 | - | - |
| $CaCl_2$[d] | - | .29 | .67 | - | .29 | .67 |
| Na acetate | .02 | - | - | .34 | - | - |
| Vit/Se premix[e] | .25 | .25 | .25 | .25 | .25 | .25 |
| Trace min pmix[f] | .05 | .05 | .05 | .05 | .05 | .05 |
| L-lysine premix[g] | .06 | .06 | .06 | .06 | .06 | .06 |
| Dextrose[h] | .12 | 1.19 | 1.70 | .80 | 2.07 | 2.07 |
| Trial 2 | | | | | | |
| Ground corn | 65.73 | 65.73 | 65.73 | 65.73 | 65.73 | 65.73 |
| SBM (44%) | 28.22 | 28.22 | 28.22 | 28.22 | 28.22 | 28.22 |
| DCP | 1.24 | 1.24 | 1.24 | - | - | - |
| DFP | - | - | - | 1.28 | 1.28 | 1.28 |
| $CaCO_3$ | - | .45 | .84 | - | .49 | .49 |
| Ca lactate[c] | 2.46 | 1.15 | - | 1.41 | - | - |
| Salt | .08 | .40 | .40 | - | .40 | .40 |
| NaOH | - | - | .30 | - | - | .21 |
| HCl | .20 | - | - | .25 | - | - |
| Na acetate | 1.07 | .62 | - | 1.00 | .44 | - |
| KCl | .70 | .70 | .70 | .70 | .70 | .70 |
| Vit/Se premix[e] | .25 | .25 | .25 | .25 | .25 | .25 |
| Trace min pmix[f] | .05 | .05 | .05 | .05 | .05 | .05 |
| Dextrose[h] | - | 1.19 | 2.27 | 1.11 | 2.45 | 2.67 |

[a]All diets are calculated to contain 18% CP, 1.01% Lysine (.97% in Trial 2), .7% Ca, .6% P, .35% Na, .62% Cl, and .81% K (1.16% in Trial 2).
[b]DCP = dicalcium phosphate DFP = defluorinated phosphate
[c]Ca lactate contained 13% Ca.
[d]$CaCl_2$ as supplied contained 77% $CaCl_2$.
[e]Supplied the following (per kg of diet): 4,400 ICU vitamin A, 440 ICU vitamin D, 11 IU vitamin E, 4.4 riboflavin, 22 mg d-pantothenic acid, 22 mg niacin, 489.5 mg choline, .022 mg vitamin B12, .5 mg menadione, .44 mg d-biotin and .3mg Se.
[f]Supplied the following (mg/kg diet) Zn, 75; Fe, 68; Mn, 30; Cu, 8.75; I, 1.
[g]Contains 78% L-lysine.
[h]Dextrose was used to bring diets to 100%.

TABLE 2. ANALYZED COMPOSITION OF EXPERIMENTAL DIETS[a]

| | DCP[b] | | | DFP[b] | | |
|---|---|---|---|---|---|---|
| Ingredient | pH 5.5 | pH 6.0 | pH 6.7 | pH 5.5 | pH 6.0 | pH 6.7 |
| Trial 1 | | | | | | |
| Sodium | .21 | .23 | .21 | .25 | .24 | .24 |
| Potassium | .61 | .62 | .69 | .69 | .73 | .71 |
| Calcium | .99 | 1.05 | 1.04 | 1.10 | 1.06 | 1.13 |
| Magnesium | .12 | .12 | .13 | .13 | .13 | .13 |
| Chlorine | .49 | .57 | .53 | .54 | .59 | .55 |
| Phosphorus | .61 | .60 | .61 | .64 | .64 | .67 |
| Sulfur | .15 | .15 | .16 | .15 | .16 | .16 |
| pH | 5.38 | 5.76 | 6.40 | 5.50 | 6.16 | 6.55 |
| DM[c] | 88.8 | 90.0 | 89.0 | 89.0 | 90.3 | 89.8 |
| dUA[d] | 458.3 | 479.9 | 497.3 | 535.3 | 504.0 | 535.6 |
| dEB[e] | 109.4 | 97.8 | 118.3 | 132.9 | 124.7 | 130.9 |
| TV[f] | 57.6 | 41.2 | 28.4 | 54.0 | 30.4 | 19.6 |
| ATV[g] | 64.8 | 45.6 | 32.0 | 60.8 | 33.6 | 21.8 |
| Trial 2 | | | | | | |
| Sodium | .22 | .21 | .24 | .22 | .23 | .22 |
| Potassium | .77 | .92 | .97 | 1.01 | .77 | .94 |
| Calcium | .97 | .97 | .97 | .95 | 1.01 | 1.01 |
| Magnesium | .11 | .13 | .14 | .14 | .11 | .13 |
| Chlorine | .52 | .54 | .50 | .46 | .47 | .48 |
| Phosphorus | .58 | .58 | .58 | .63 | .64 | .63 |
| Sulfur | .13 | .16 | .16 | .16 | .13 | .15 |
| pH | 5.31 | 5.90 | 6.73 | 5.63 | 6.10 | 6.92 |
| DM | 88.7 | 89.2 | 89.4 | 88.9 | 89.0 | 89.8 |
| dUA | 492.8 | 528.3 | 573.6 | 560.2 | 511.8 | 561.6 |
| dEB | 146.0 | 174.4 | 211.0 | 224.3 | 164.0 | 200.8 |
| TV | 78.0 | 54.4 | 27.2 | 54.0 | 28.0 | 17.6 |
| ATV | 88.0 | 60.8 | 30.4 | 60.8 | 31.4 | 19.6 |

[a]Minerals are expressed as % of total diet.
[b]DCP - Dicalcium phosphate DFP - Defluorinated phosphate.
[c]DM - dry matter content in %.
[d]dUA - dietary undetermined anion = $(Na^+ + K^+ + Ca^{++} + Mg^{++}) - (Cl^- + P^- + S^-)$.
[e]dEB - dietary electrolyte balance = $(Na^+ + K^+ - Cl^-)$.
[f]TV - Titration value = (meq of NaOH/kg of diet to pH of 8) - (meq of HCl/kg of diet to pH of 4).
[g]ATV - adjusted titration value = TV/DM.

TABLE 3. THE EFFECT OF PHOSPHORUS SOURCE AND DIETARY pH ON THE PERFORMANCE OF WEANLING PIGS (COMBINED TRIALS)

| | DCP[b] | | | DFP[b] | | | | Dietary pH[c] | |
|---|---|---|---|---|---|---|---|---|---|
| | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | 5.4 v 6.0 | 6.0 v 6.7 |
| Daily gain, kg | | | | | | | | | |
| wk 1-3[d] | .274 | .233 | .198 | .247 | .259 | .210 | .014 | .29 | .01 |
| wk 4-6 | .549 | .556 | .523 | .533 | .549 | .525 | .017 | .53 | .11 |
| Overall[e] | .398 | .377 | .344 | .375 | .389 | .352 | .013 | .82 | .01 |
| Daily feed, kg | | | | | | | | | |
| wk 1-3[di] | .472 | .433 | .396 | .457 | .500 | .393 | .017 | .90 | .001 |
| wk 4-6[e] | 1.06 | 1.05 | .965 | 1.02 | 1.08 | .998 | .026 | .30 | .002 |
| Overall[d] | .736 | .711 | .654 | .711 | .764 | .667 | .019 | .50 | .001 |
| Feed to gain ratio | | | | | | | | | |
| wk 1-3[gj] | 1.75 | 1.90 | 2.18 | 1.87 | 1.98 | 1.92 | .097 | .20 | .30 |
| wk 4-6[h] | 1.92 | 1.89 | 1.84 | 1.92 | 1.98 | 1.90 | .032 | .70 | .06 |
| Overall[h] | 1.86 | 1.89 | 1.92 | 1.90 | 1.98 | 1.90 | .033 | .10 | .55 |
| Final weight[f] | 22.71 | 21.86 | 20.79 | 21.74 | 22.37 | 21.08 | .517 | .84 | .03 |

[a]Twelve pens of two pigs per pen per dietary treatment.
[b]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[c]Contrast probability levels.
[defg]dietary pH main effect ($P < .001$, $P < .01$, $P < .05$, $P < .1$).
[h]Phosphorus source main effect ($P < .1$).
[ij]Dietary pH times phosphorus source interaction ($P < .05$, $P < .10$).

TABLE 4. THE EFFECT OF PHOSPHORUS SOURCE AND DIETARY pH ON GASTROINTESTINAL pH, CHLORIDE ION CONCENTRATION AND TITRATION VALUES OF WEANLING PIGS[a] (COMBINED TRIALS)

| | DCP[b] | | | DFP[b] | | | | Dietary pHc | |
|---|---|---|---|---|---|---|---|---|---|
| | 5.4 | 6.0 | 6.7 | 5.4 | 6.0 | 6.7 | S.E. | 5.4 v 6.0 | 6.0 v 6.7 |
| **Stomach** | | | | | | | | | |
| TV[d] | 85.4 | 97.6 | 79.0 | 83.7 | 80.9 | 81.9 | .076 | .56 | .19 |
| DM[eg] | 38.8 | 38.8 | 38.3 | 37.7 | 37.5 | 37.1 | .550 | .85 | .41 |
| ATV[f] | 224.0 | 266.3 | 225.9 | 236.2 | 222.7 | 236.0 | 18.2 | .49 | .49 |
| **pH** | | | | | | | | | |
| stomach | 3.83 | 4.09 | 3.79 | 3.62 | 3.94 | 3.90 | .150 | .06 | .26 |
| jejunum | 5.72 | 5.76 | 5.63 | 5.67 | 5.72 | 5.77 | .067 | .52 | .56 |
| cecum | 5.71 | 5.69 | 5.63 | 5.69 | 5.66 | 5.64 | .039 | .50 | .37 |
| colon[h] | 6.51 | 6.51 | 6.48 | 6.42 | 6.30 | 6.38 | .055 | .28 | .70 |
| **Chloride** | | | | | | | | | |
| stomach[i] | .071 | .061 | .074 | .076 | .062 | .072 | .004 | .003 | .005 |
| jejunum | .099 | .103 | .101 | .101 | .104 | .106 | .003 | .42 | .97 |
| cecum | .014 | .011 | .012 | .013 | .012 | .014 | .001 | .16 | .28 |
| colon | .011 | .041 | .011 | .011 | .009 | .009 | .012 | .24 | .22 |

[a]Twelve pens of two pigs per pen per dietary treatment.
[b]DCP - Dicalcium phosphate, DFP - Defluorinated rock phosphate.
[c]Contrast probability levels.
[d]TV - Titration value = (meq HCl/ kg digesta to a pH of 2) subtracted from (meq NaOH/ kg digesta from pH of 2 to pH of 8).
[e]DM - dry matter.
[f]ATV - adjusted titration value (TV/DM).
[gh]Phosphorus source main effect ($P < .01$, $P < .001$).
[i]Dietary pH main effect ($P < .001$).

TABLE 5. THE EFFECT OF GENDER ON METACARPAL, METATARSAL AND HUMERUS MEASUREMENTS OF WEANLING PIGS[a] (COMBINED TRIALS)

| | GENDER | | | |
|---|---|---|---|---|
| | Barrows | Gilts | SE | $P^b$ |
| *Metacarpal* | | | | |
| Volume(ml) | 6.25 | 5.84 | .071 | .024 |
| sp gr[c] | 1.180 | 1.200 | .004 | .049 |
| Area($cm^2$) | .357 | .350 | .006 | .521 |
| Force(kg) | 80.0 | 86.8 | 1.54 | .030 |
| Stress[d] | 113.9 | 125.9 | 2.92 | .033 |
| *Metatarsal* | | | | |
| Volume(ml) | 8.05 | 7.65 | .081 | .059 |
| sp gr[c] | 1.170 | 1.179 | .002 | .057 |
| Area($cm^2$) | .395 | .381 | .006 | .313 |
| Force(kg) | 71.9 | 70.4 | 1.62 | .547 |
| Stress[d] | 92.2 | 94.8 | 2.63 | .564 |
| *Humerus* | | | | |
| Volume(ml) | 6.55 | 6.31 | .095 | .498 |
| sp gr[c] | 1.390 | 1.416 | .005 | .050 |

[a] All values adjusted for final body wt by covariance analysis.
[b] $P$ = Probability level.
[c] Specific gravity in g/ml.
[d] Stress is expressed in $kg/cm^2$.

TABLE 6. THE EFFECT OF PHOSPHORUS SOURCE AND DIETARY pH ON HUMERUS, METACARPAL AND METATARSAL MEASUREMENTS OF WEANLING PIGS[a] (COMBINED TRIALS)

| | DCP[b] | | | DFP[b] | | | | Dietary pH[c] | |
|---|---|---|---|---|---|---|---|---|---|
| Item | 5.4 | 6.0 | 6.7 | 5.4 | 6.0 | 6.7 | S.E. | 5.4 V 6.0 | 6.0 V 6.7 |
| *Metacarpal* | | | | | | | | | |
| Volume(ml) | 5.91 | 6.13 | 6.08 | 6.01 | 6.25 | 5.90 | .121 | .06 | .10 |
| sp gr[d] | 1.189 | 1.205 | 1.189 | 1.190 | 1.185 | 1.185 | .006 | .41 | .21 |
| Area($cm^2$) | .365 | .338 | .362 | .350 | .363 | .341 | .011 | .54 | .94 |
| Force(kg)[f] | 85.96 | 85.35 | 84.57 | 82.97 | 81.19 | 80.45 | 2.64 | .65 | .77 |
| Stress[e] | 119.9 | 128.2 | 118.1 | 121.0 | 112.0 | 120.6 | 4.99 | .94 | .88 |
| *Metatarsal* | | | | | | | | | |
| Volume(ml) | 7.66 | 7.86 | 7.92 | 7.87 | 8.08 | 7.70 | .138 | .15 | .27 |
| sp gr[dg] | 1.179 | 1.179 | 1.177 | 1.173 | 1.168 | 1.173 | .003 | .42 | .69 |
| Area($cm^2$) | .403 | .385 | .389 | .387 | .386 | .380 | .011 | .38 | .92 |
| Force(kg) | 68.18 | 73.47 | 71.56 | 69.52 | 72.50 | 72.09 | 2.80 | .14 | .68 |
| Stress[e] | 85.91 | 96.89 | 95.34 | 90.71 | 95.61 | 95.1 | 4.60 | .09 | .91 |
| *Humerus* | | | | | | | | | |
| Volume(ml) | 6.46 | 6.66 | 6.49 | 6.42 | 6.19 | 6.34 | .163 | .93 | .95 |
| sp gr[d] | 1.411 | 1.398 | 1.411 | 1.394 | 1.411 | 1.392 | .008 | .80 | .70 |

[a]Twelve pens of two pigs per pen per dietary treatment. All values adjusted for final body wt by covariance analysis.
[b]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate
[c]Contrast probability.
[d]Specific gravity in g/ml.
[e]Stress is expressed in $kg/cm^2$.
[fg]Phosphorus source main effect ($P < .1$, $P < .01$).

TABLE 7. THE EFFECT OF DIETARY pH AND PHOSPHORUS SOURCE ON CARCASS BACKFAT AND LOIN MUSCLE AREA IN WEANLING PIGS FED TWO PHOSPHORUS SOURCES[a] (COMBINED TRIALS)

| | DCP[b] | | | DFP[b] | | | | Trial | |
|---|---|---|---|---|---|---|---|---|---|
| | 5.4 | 6.0 | 6.7 | 5.4 | 6.0 | 6.7 | SE | 1 | 2 |
| Backfat (cm)[ef] | .884 | .900 | .846 | .851 | .855 | .857 | .043 | .757 | .935 |
| Loin area[cde] | 3.56 | 3.31 | 3.20 | 3.31 | 3.53 | 3.47 | .113 | 2.98 | 3.66 |

[a]Twelve pens of two pigs per pen per dietary treatment.
[b]DCP - Dicalcium phosphate, DFP - Defluorinated rock phosphate
[c]Loin muscle area is expressed in $cm^2$.
[de]Trial effect ($P < .001$, $P < .01$).
[f]Phosphorus source by dietary pH interaction ($P < .05$).

TABLE 8. PARTIAL CORRELATION COEFFICIENTS (r) FOR ULTRASONIC MEASUREMENTS ON THE EMPTY BODY AND CARCASS OF THE WEANLING PIG (TRIAL ONE)

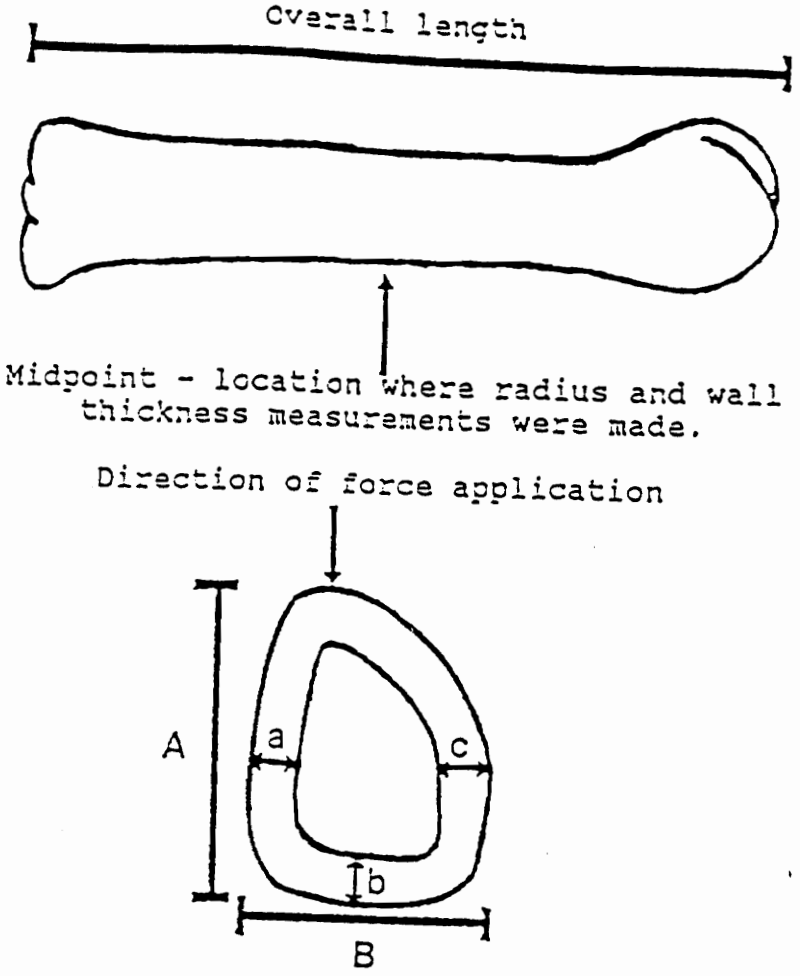
| | Backfat | Loin area | Fat1[a] | Loin1[b] | Fat2[c] | Fat3[d] |
|---|---|---|---|---|---|---|
| Backfat | | .422** | .503** | .353* | .386** | .430** |
| Loin area | | | .353* | .630** | .423** | .473** |
| Fat1[a] | | | | .317* | .365** | .513** |
| Loin1[b] | | | | | .336* | .372** |
| Fat2[c] | | | | | | .619** |
| Fat3[d] | | | | | | |

**$P < .01$.
*$P < .05$.
[a]Tenth rib fat ultrasound image made with 3.5 MHz probe.
[b]Loin muscle area ultrasound image made with 5.0 MHz probe.
[c]Tenth rib fat ultrasound image made with 7.5 MHz probe.
[d]Tenth rib fat ultrasound image made with 7.5 MHz probe.

TABLE 9. PARTIAL CORRELATION COEFFICIENTS FOR ULTRASONIC MEASUREMENTS IN WEANLING PIGS TAKEN ON THE LIVE ANIMAL AND CARCASS OF WEANLING PIG (TRIAL TWO)

| | Backfat | Loin area | Fat1[a] | Loin1[b] |
|---|---|---|---|---|
| Backfat | | .024 | .302* | .280* |
| Loin area | | | .076 | .510** |
| Fat1[a] | | | | .263* |
| Loin1[b] | | | | |

**$P < .01$.
*$P < .05$.
[a]Tenth rib fat ultrasound image made with 3.5 MHz probe.
[b]Loin muscle area ultrasound image made with 3.5 MHz probe.




A = Wide dimension
B = Narrow dimension

a, b, c = locations of wall thickness measurements

$$\text{Radius} = \frac{A + B}{2} = r \qquad \text{Wall Thickness} = \frac{a + b + c}{3} = t$$

$$\text{Area} = \left( \frac{\pi [r^2 - (r - t)^2]}{4} + (2rt - 3t^2) \right)$$

Figure 1. Determination of bone dimensions used in calculation of shear stress of the fourth metacarpals and metatarsals.

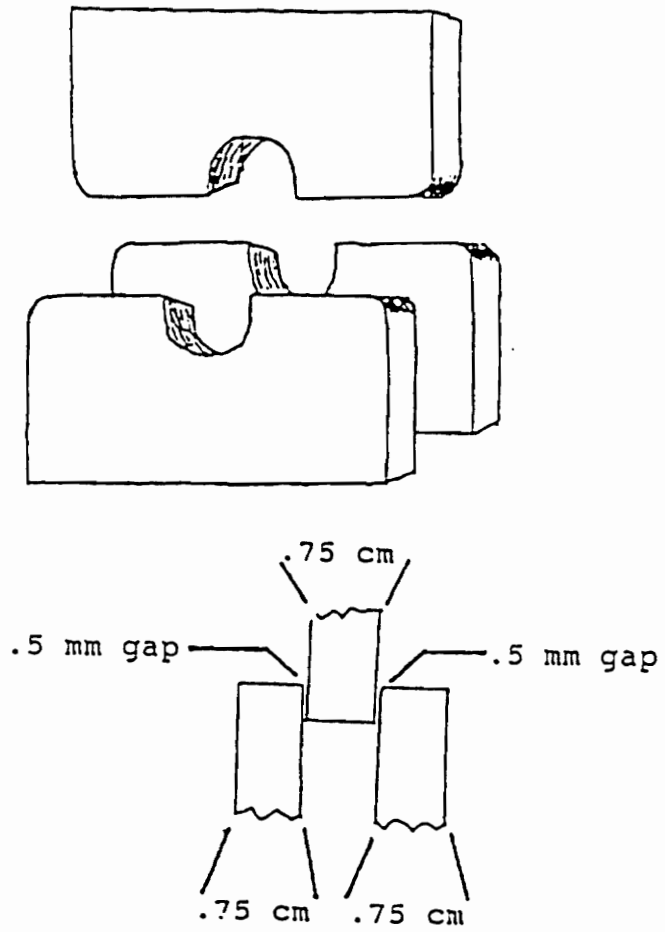
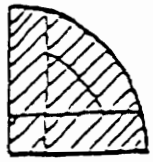



Figure 2. Diagram of shear fixtures used on Ingstrom Model 1123 Testing Machine for bone strength determination.

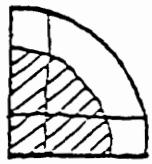
Area of entire quarter circle:

$$\frac{\pi * r^2}{4}$$

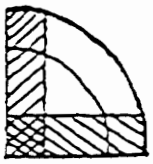
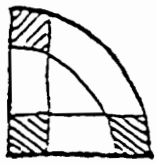
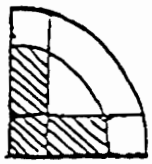


- Area of inner quarter circle:

$$\frac{\pi * (r - t)^2}{4}$$



+ Area of the "legs" minus the "corners":

$2 * r * t$  $-$ $3t^2$  $=$ $2rt - 3t^2$ 

"The area formula subtracts off the area of the marrow cavity from that of the entire quarter circle. This requires that the area of an inner quarter circle with radius (r - t) be subtracted off the area of the entire circle with radius r, then the area of the "legs" minus the "corners" be added back." (Combs, 1989)

Figure 3. Calculation of area for stress equations.

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APPENDIX TABLE 1. THE EFFECT OF DIETARY pH AND PHOSPHORUS SOURCE ON AVERAGE DAILY GAIN (kg) OF WEANLING PIGS[a] (COMBINED TRIALS)

| | DCP[b] | | | DFP[b] | | | | | Dietary pH[c] | |
|---|---|---|---|---|---|---|---|---|---|---|
| Week | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | $P$[d] | 5.4 v 6.0 | 6.0 v 6.7 |
| 1[e] | .168 | .124 | .098 | .156 | .169 | .105 | .015 | .186 | .32 | .005 |
| 2[g] | .284 | .214 | .222 | .243 | .268 | .203 | .017 | .021 | .20 | .11 |
| 3[g] | .368 | .360 | .274 | .343 | .341 | .321 | .022 | .214 | .82 | .02 |
| 4 | .456 | .511 | .486 | .494 | .507 | .446 | .026 | .337 | .20 | .11 |
| 5 | .606 | .568 | .536 | .540 | .576 | .539 | .020 | .127 | .96 | .09 |
| 6 | .650 | .639 | .632 | .619 | .593 | .707 | .039 | .245 | .64 | .18 |
| 1-2[e] | .226 | .169 | .160 | .200 | .219 | .154 | .014 | .025 | .18 | .01 |
| 1-3 | .274 | .233 | .198 | .247 | .259 | .210 | .014 | .150 | .29 | .01 |
| 1-4[f] | .320 | .304 | .271 | .310 | .322 | .270 | .014 | .583 | .88 | .004 |
| 1-5 | .377 | .356 | .323 | .355 | .373 | .323 | .014 | .367 | .91 | .004 |
| 1-6[h] | .410 | .397 | .372 | .384 | .410 | .371 | .015 | .413 | .66 | .04 |
| 4-6 | .549 | .556 | .523 | .533 | .549 | .529 | .017 | .090 | .53 | .106 |
| overall | .398 | .377 | .344 | .375 | .389 | .352 | .013 | .373 | .82 | .01 |

[a]Twelve pens of two pigs per pen per dietary treatment.
[b]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[c]Contrast probability levels.
[d]$P$ - Probability of differences due to treatments.
[efgh]diet pH main effect ($P < .001$, $P < .01$, $P < .05$, $P < .1$).

APPENDIX TABLE 2. THE EFFECT OF PHOSPHORUS SOURCE (BY TRIAL) ON AVERAGE DAILY GAIN (kg) OF WEANLING PIGS FED THREE DIETARY pH LEVELS

| Week | TRIAL 1 | | TRIAL 2 | | SE | P[b] |
|---|---|---|---|---|---|---|
| | DCP[a] | DFP[a] | DCP[a] | DFP[a] | | |
| 1 | .125 | .147 | .135 | .140 | .013 | .499 |
| 2[c] | .273 | .253 | .208 | .224 | .014 | .216 |
| 3 | .352 | .343 | .316 | .329 | .018 | .595 |
| 4 | .480 | .495 | .489 | .469 | .021 | .419 |
| 5 | .572 | .535 | .569 | .569 | .016 | .251 |
| 6 | .677 | .670 | .604 | .609 | .032 | .858 |
| 1-2 | .199 | .200 | .172 | .182 | .011 | .699 |
| 1-3 | .250 | .248 | .220 | .230 | .011 | .585 |
| 1-4 | .309 | .312 | .287 | .290 | .011 | .991 |
| 1-5 | .360 | .355 | .344 | .346 | .011 | .744 |
| 1-6[d] | .419 | .404 | .368 | .371 | .012 | .459 |
| 4-6 | .540 | .533 | .545 | .539 | .014 | .972 |
| overall | .378 | .373 | .368 | .371 | .011 | .746 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[b]*P* - Probability of trial by phosphorus source interaction.
[cd]Trial effect ($P < .1$, $P < .05$).

APPENDIX TABLE 3. THE EFFECT OF DIETARY pH (BY TRIAL) ON THE AVERAGE DAILY GAIN (kg) OF WEANLING PIGS FED TWO PHOSPHORUS SOURCES

| | TRIAL 1 | | | TRIAL 2 | | | | |
|---|---|---|---|---|---|---|---|---|
| Week | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | $P^a$ |
| 1 | .166 | .139 | .104 | .158 | .155 | .099 | .015 | .706 |
| 2[b] | .295 | .284 | .210 | .232 | .198 | .217 | .017 | .025 |
| 3 | .375 | .372 | .295 | .336 | .329 | .300 | .022 | .490 |
| 4 | .475 | .535 | .453 | .475 | .483 | .480 | .026 | .311 |
| 5 | .592 | .563 | .504 | .553 | .580 | .572 | .020 | .032 |
| 6 | .671 | .626 | .724 | .598 | .606 | .615 | .035 | .527 |
| 1-2 | .231 | .212 | .157 | .195 | .176 | .158 | .014 | .329 |
| 1-3 | .279 | .265 | .203 | .242 | .227 | .205 | .014 | .258 |
| 1-4 | .330 | .335 | .267 | .300 | .291 | .274 | .014 | .184 |
| 1-5 | .381 | .379 | .313 | .351 | .349 | .334 | .014 | .109 |
| 1-6[c] | .418 | .433 | .383 | .375 | .374 | .360 | .013 | .488 |
| 4-6 | .550 | .557 | .503 | .533 | .548 | .545 | .017 | .192 |
| overall | .398 | .393 | .336 | .375 | .374 | .360 | .014 | .155 |

[a]$P$ - Probability of trial by dietary pH interaction.
[bc]Trial effect($P < .05$, $P < .1$).

APPENDIX TABLE 4. THE EFFECT OF PHOSPHORUS SOURCE, DIETARY pH AND TRIAL ON AVERAGE DAILY GAIN (kg) IN WEANLING PIGS (COMBINED TRIALS)

| Week | Phosphorus Source[a] | | | | Dietary pH | | | | | Trial | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | DCP | DFP | S.E. | $P$[b] | 5.4 | 6.0 | 6.7 | S.E. | $P$[b] | 1 | 2 | S.E. | $P$[b] |
| 1 | .130 | .143 | .009 | .303 | .162 | .147 | .101 | .011 | .001 | .136 | .137 | .009 | .945 |
| 2[cd] | .240 | .238 | .011 | .884 | .264 | .241 | .213 | .013 | .018 | .263 | .216 | .011 | .062 |
| 3 | .334 | .335 | .012 | .964 | .355 | .350 | .298 | .015 | .022 | .347 | .322 | .012 | .534 |
| 4 | .484 | .482 | .015 | .923 | .475 | .509 | .466 | .019 | .236 | .487 | .479 | .015 | .795 |
| 5[d] | .570 | .552 | .012 | .261 | .573 | .572 | .538 | .015 | .143 | .553 | .569 | .012 | .571 |
| 6 | .637 | .613 | .028 | .995 | .595 | .617 | .662 | .034 | .399 | .644 | .606 | .024 | .253 |
| 1-2[c] | .185 | .190 | .008 | .628 | .213 | .194 | .157 | .010 | .001 | .199 | .178 | .008 | .354 |
| 1-3 | .235 | .239 | .008 | .728 | .261 | .246 | .204 | .010 | .001 | .249 | .225 | .008 | .291 |
| 1-4 | .298 | .301 | .008 | .827 | .315 | .313 | .271 | .010 | .003 | .311 | .289 | .008 | .314 |
| 1-5 | .351 | .350 | .008 | .341 | .366 | .364 | .323 | .010 | .001 | .358 | .345 | .008 | .961 |
| 1-6 | .392 | .386 | .008 | .702 | .393 | .401 | .373 | .010 | .095 | .409 | .369 | .008 | .033 |
| 4-6 | .543 | .536 | .010 | .651 | .541 | .552 | .524 | .012 | .548 | .536 | .542 | .010 | .817 |
| overall | .373 | .372 | .008 | .939 | .386 | .393 | .348 | .009 | .010 | .373 | .369 | .008 | .811 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate.
[b]$P$ - Probability of differences due to treatments.
[c]Phosphorus source by dietary pH interaction ($P < .05$).
[d]Trial by dietary pH interaction ($P < .05$).

APPENDIX TABLE 5. THE EFFECT OF DIETARY pH AND PHOSPHORUS SOURCE ON THE FEED INTAKE (kg) OF WEANLING PIGS[a] (COMBINED TRIALS)

| Week | DCP[b] | | | DFP[b] | | | SE | $P$[d] | Dietary pH[c] | |
|---|---|---|---|---|---|---|---|---|---|---|
| | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | | | 5.4 v 6.0 | 6.0 v 6.7 |
| 1 | .240 | .209 | .197 | .237 | .240 | .185 | .012 | .179 | .26 | .009 |
| 2 | .494 | .447 | .422 | .482 | .514 | .411 | .022 | .136 | .73 | .006 |
| 3[e] | .683 | .642 | .569 | .650 | .746 | .583 | .028 | .049 | .32 | .001 |
| 4 | .866 | .910 | .826 | .906 | .928 | .814 | .032 | .719 | .34 | .004 |
| 5 | 1.179 | 1.146 | 1.049 | 1.087 | 1.216 | 1.109 | .036 | .047 | .18 | .006 |
| 6 | 1.266 | 1.237 | 1.215 | 1.182 | 1.236 | 1.254 | .039 | .295 | .75 | .97 |
| 1-2 | .367 | .328 | .310 | .360 | .377 | .298 | .015 | .079 | .47 | .002 |
| 1-3 | .472 | .433 | .396 | .457 | .500 | .393 | .017 | .043 | .90 | .001 |
| 1-4 | .572 | .552 | .504 | .570 | .608 | .449 | .019 | .204 | .64 | .001 |
| 1-5 | .693 | .670 | .611 | .672 | .728 | .621 | .020 | .136 | .42 | .001 |
| 1-6 | .759 | .752 | .695 | .732 | .801 | .698 | .021 | .184 | .14 | .001 |
| 4-6 | 1.057 | 1.047 | .965 | 1.021 | 1.083 | .998 | .026 | .286 | .29 | .002 |
| overall | .737 | .711 | .654 | .711 | .764 | .667 | .019 | .117 | .46 | .001 |

[a]Twelve pens of two pigs per pen per dietary treatment.
[b]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[c]Contrast probability level.
[d]$P$ - Probability of differences due to treatment.
[e]Dietary pH main effect ($P < .001$, $P < .01$, $P < .05$).

APPENDIX TABLE 6. THE EFFECT OF PHOSPHORUS SOURCE (BY TRIAL) ON THE FEED INTAKE (kg) OF WEANLING PIGS FED THREE DIETARY PH LEVELS

| Week | TRIAL 1 DCP[a] | TRIAL 1 DFP[a] | TRIAL 2 DCP[a] | TRIAL 2 DFP[a] | SE | P[b] |
|---|---|---|---|---|---|---|
| 1 | .215 | .215 | .216 | .226 | .010 | .652 |
| 2 | .449 | .445 | .460 | .494 | .018 | .294 |
| 3[c] | .697 | .739 | .566 | .581 | .023 | .575 |
| 4 | .842 | .881 | .890 | .885 | .026 | .405 |
| 5 | 1.09 | 1.09 | 1.16 | 1.19 | .029 | .601 |
| 6 | 1.26 | 1.21 | 1.23 | 1.23 | .033 | .442 |
| 1-2 | .332 | .330 | .338 | .360 | .012 | .328 |
| 1-3 | .454 | .467 | .413 | .434 | .014 | .795 |
| 1-4 | .553 | .572 | .533 | .547 | .016 | .852 |
| 1-5 | .658 | .672 | .659 | .657 | .016 | .939 |
| 1-6 | .763 | .761 | .709 | .725 | .017 | .594 |
| 4-6 | .983 | .994 | 1.06 | 1.07 | .021 | .192 |
| overall | .692 | .704 | .709 | .725 | .154 | .892 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[b]*P* - Probability of trial by phosphorus source interaction.
[c]Trial effect ($P < .01$).

APPENDIX TABLE 7. THE EFFECT OF DIETARY pH (BY TRIAL) ON FEED INTAKE (kg) OF WEANLING PIGS FED TWO PHOSPHORUS SOURCES

| | TRIAL 1 | | | TRIAL 2 | | | | |
|---|---|---|---|---|---|---|---|---|
| Week | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | $P$[a] |
| 1 | .237 | .224 | .185 | .240 | .225 | .198 | .012 | .887 |
| 2 | .474 | .482 | .384 | .502 | .478 | .449 | .022 | .302 |
| 3[b] | .749 | .788 | .617 | .584 | .601 | .535 | .028 | .148 |
| 4 | .887 | .921 | .776 | .885 | .913 | .864 | .032 | .262 |
| 5 | 1.084 | 1.160 | 1.016 | 1.182 | 1.202 | 1.142 | .036 | .491 |
| 6 | 1.199 | 1.252 | 1.253 | 1.248 | 1.221 | 1.216 | .036 | .486 |
| 1-2 | .352 | .353 | .284 | .371 | .352 | .324 | .015 | .393 |
| 1-3 | .484 | .498 | .395 | .442 | .435 | .394 | .017 | .196 |
| 1-4 | .589 | .606 | .492 | .552 | .554 | .512 | .016 | .152 |
| 1-5 | .687 | .714 | .595 | .679 | .684 | .638 | .020 | .167 |
| 1-6 | .761 | .821 | .705 | .730 | .732 | .689 | .019 | .186 |
| 4-6 | 1.001 | 1.040 | .920 | 1.075 | 1.088 | 1.043 | .026 | .995 |
| overall | .718 | .743 | .632 | .730 | .732 | .689 | .019 | .192 |

[a]P - Probability of trial by dietary pH interaction.
[b]Trial effect ($P < .01$).

APPENDIX TABLE 8. THE EFFECT OF PHOSPHORUS SOURCE, DIETARY pH AND TRIAL ON DAILY FEED INTAKE (kg) IN WEANLING PIGS (COMBINED TRIALS)

| Week | Phosphorus Source[a] | | | | Dietary pH | | | | | Trial | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | DCP | DFP | S.E. | $P$[b] | 5.4 | 6.0 | 6.7 | S.E. | $P$[b] | 1 | 2 | S.E. | $P$[b] |
| 1 | .22 | .22 | .001 | .575 | .24 | .22 | .19 | .009 | .0011 | .22 | .22 | .007 | .642 |
| 2 | .45 | .47 | .013 | .411 | .49 | .48 | .42 | .016 | .004 | .45 | .48 | .013 | .328 |
| 3[d] | .63 | .66 | .016 | .211 | .67 | .69 | .58 | .020 | .0002 | .72 | .57 | .016 | .002 |
| 4 | .87 | .88 | .018 | .522 | .89 | .92 | .82 | .023 | .014 | .86 | .89 | .019 | .759 |
| 5[d] | 1.12 | 1.14 | .021 | .659 | 1.13 | 1.18 | 1.08 | .025 | .023 | 1.09 | 1.18 | .021 | .139 |
| 6 | 1.24 | 1.22 | .022 | .631 | 1.23 | 1.23 | 1.23 | .027 | .942 | 1.23 | 1.23 | .022 | .948 |
| 1-2[c] | .34 | .35 | .009 | .393 | .36 | .35 | .30 | .011 | .0004 | .33 | .35 | .009 | .354 |
| 1-3[d] | .43 | .45 | .010 | .249 | .46 | .47 | .39 | .013 | .0001 | .46 | .42 | .010 | .135 |
| 1-4 | .54 | .56 | .011 | .286 | .57 | .58 | .50 | .014 | .0002 | .56 | .54 | .011 | .527 |
| 1-5 | .66 | .67 | .012 | .341 | .68 | .70 | .62 | .014 | .0002 | .66 | .67 | .011 | .961 |
| 1-6 | .74 | .74 | .012 | .610 | .75 | .77 | .70 | .015 | .002 | .76 | .72 | .012 | .239 |
| 4-6 | 1.02 | 1.03 | .015 | .559 | 1.04 | 1.07 | .98 | .018 | .006 | .99 | 1.07 | .015 | .305 |
| Overall | .700 | .71 | .008 | .371 | .72 | .74 | .66 | .013 | .0003 | .70 | .72 | .011 | .716 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate.
[b]$P$ - Probability of differences due to treatment.
[c]Phosphorus source by dietary pH interaction ($P < .1$).
[d]Phosphorus source by dietary pH interaction ($P < .05$).

APPENDIX TABLE 9. THE EFFECT OF PHOSPHORUS SOURCE AND DIETARY pH ON THE FEED TO GAIN RATIO OF WEANLING PIGS[a] (COMBINED TRIALS)

| | DCP[b] | | | DFP[b] | | | | | Dietary pH[c] | |
|---|---|---|---|---|---|---|---|---|---|---|
| Week | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | $P$[d] | 5.4 v 6.0 | 6.0 v 6.7 |
| 1[g] | 1.49 | 1.25 | 2.48 | 1.61 | 1.45 | 1.89 | .480 | .666 | .68 | .09 |
| 2 | 1.81 | 2.22 | 2.41 | 2.04 | 2.02 | 2.07 | .241 | .475 | .41 | .63 |
| 3 | 1.92 | 1.86 | 2.32 | 1.96 | 2.37 | 1.93 | .159 | .025 | .28 | .95 |
| 4 | 1.94 | 1.78 | 1.70 | 1.85 | 1.86 | 1.88 | .082 | .242 | .37 | .74 |
| 5[f] | 1.97 | 2.03 | 1.95 | 2.03 | 2.12 | 2.06 | .054 | .886 | .15 | .19 |
| 6 | 1.99 | 2.04 | 1.94 | 2.01 | 2.18 | 1.89 | .110 | .644 | .36 | .08 |
| 1-2 | 1.66 | 2.06 | 2.18 | 1.86 | 1.78 | 1.98 | .132 | .162 | .22 | .22 |
| 1-3 | 1.74 | 1.90 | 2.18 | 1.87 | 1.98 | 1.92 | .097 | .099 | .18 | .29 |
| 1-4 | 1.79 | 1.83 | 1.90 | 1.85 | 1.91 | 1.88 | .045 | .503 | .28 | .69 |
| 1-5[e] | 1.85 | 1.89 | 1.92 | 1.90 | 1.97 | 1.89 | .034 | .707 | .13 | .85 |
| 1-6[g] | 1.86 | 1.90 | 1.88 | 1.91 | 1.97 | 1.89 | .034 | .728 | .15 | .11 |
| 4-6 | 1.92 | 1.89 | 1.84 | 1.92 | 1.98 | 1.90 | .032 | .308 | .70 | .06 |
| overall[f] | 1.86 | 1.89 | 1.92 | 1.90 | 1.98 | 1.90 | .033 | .237 | .10 | .55 |

[a]Twelve pens of two pigs per pen per dietary treatment.
[b]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[c]Contrast probability.
[d]$P$ - Probability of phosphorus source by dietary pH interaction.
[efg]Trial effect ($P < .01$, $P < .05$, $P < .1$).

APPENDIX TABLE 10. THE EFFECT OF PHOSPHORUS SOURCE (BY TRIAL) ON THE FEED TO GAIN RATIO OF WEANLING PIGS FED THREE DIETARY pH LEVELS.

| Week | TRIAL 1 | | TRIAL 2 | | SE | P[b] |
|---|---|---|---|---|---|---|
| | DCP[a] | DFP[a] | DCP[a] | DFP[a] | | |
| 1[e] | 1.13 | 1.52 | 2.37 | 1.79 | .390 | .215 |
| 2 | 2.01 | 1.84 | 2.29 | 2.25 | .198 | .730 |
| 3 | 2.23 | 2.36 | 1.85 | 1.82 | .131 | .730 |
| 4 | 1.79 | 1.81 | 1.83 | 1.92 | .067 | .558 |
| 5[d] | 1.92 | 2.04 | 2.05 | 2.11 | .044 | .491 |
| 6 | 1.88 | 1.88 | 2.11 | 2.17 | .090 | .681 |
| 1-2 | 1.93 | 1.71 | 2.02 | 2.05 | .108 | .247 |
| 1-3 | 1.98 | 1.93 | 1.91 | 1.91 | .080 | .790 |
| 1-4 | 1.81 | 1.85 | 1.87 | 1.91 | .037 | .997 |
| 1-5[c] | 1.84 | 1.90 | 1.93 | 1.97 | .031 | .836 |
| 1-6[e] | 1.82 | 1.89 | 1.93 | 1.96 | .028 | .543 |
| 4-6 | 1.81 | 1.87 | 1.95 | 2.00 | .026 | .958 |
| overall[d] | 1.85 | 1.89 | 1.93 | 1.96 | .027 | .863 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated phosphate.
[b]*P* - Probability of trial by phosphorus source interaction.
[cde]Trial effect ($P < .01$, $P < .05$, $P < .1$).

APPENDIX TABLE 11. THE EFFECT OF DIETARY pH (BY TRIAL) ON THE FEED TO GAIN RATIO OF WEANLING PIGS FED TWO PHOSPHORUS SOURCES.

| | TRIAL 1 | | | TRIAL 2 | | | | |
|---|---|---|---|---|---|---|---|---|
| Week | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | $P^a$ |
| 1[d] | 1.47 | 1.22 | 1.28 | 1.64 | 1.49 | 3.10 | .480 | .162 |
| 2 | 1.66 | 1.73 | 2.39 | 2.19 | 2.52 | 2.10 | .241 | .072 |
| 3 | 2.13 | 2.35 | 2.42 | 1.76 | 1.89 | 1.84 | .159 | .796 |
| 4 | 1.90 | 1.72 | 1.76 | 1.87 | 1.93 | 1.82 | .082 | .286 |
| 5[c] | 1.84 | 2.07 | 2.02 | 2.15 | 2.08 | 1.99 | .054 | .006 |
| 6 | 1.81 | 2.09 | 1.75 | 2.21 | 2.13 | 2.08 | .099 | .240 |
| 1-2 | 1.57 | 1.80 | 2.08 | 1.95 | 2.05 | 2.09 | .132 | .385 |
| 1-3 | 1.79 | 1.93 | 2.15 | 1.84 | 1.96 | 1.94 | .097 | .326 |
| 1-4 | 1.80 | 1.81 | 1.89 | 1.84 | 1.93 | 1.88 | .045 | .317 |
| 1-5[b] | 1.81 | 1.88 | 1.93 | 1.94 | 1.98 | 1.92 | .037 | .177 |
| 1-6[d] | 1.83 | 1.90 | 1.85 | 1.95 | 1.98 | 1.92 | .030 | .669 |
| 4-6 | 1.82 | 1.87 | 1.82 | 2.06 | 2.00 | 1.92 | .032 | .308 |
| overall[c] | 1.81 | 1.89 | 1.91 | 1.95 | 1.98 | 1.92 | .033 | .116 |

[a] $P$ - Probabilityof a trial by dietary pH interaction.
[bcd] Trial effect ($P < .01$. $P < .05$, $P < .1$).

APPENDIX TABLE 12. THE EFFECT OF PHOSPHORUS SOURCE, DIETARY pH AND TRIAL ON FEED TO GAIN RATIO IN WEANLING PIGS (COMBINED TRIALS)

| Week | Phosphorus Source[a] | | | | Dietary pH | | | | | Trial | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | DCP | DFP | S.E. | $P$[b] | 5.4 | 6.0 | 6.7 | S.E. | $P$[b] | 1 | 2 | S.E. | $P$[b] |
| 1 | 1.75 | 1.65 | .277 | .817 | 1.55 | 1.36 | 2.19 | .339 | .200 | 1.32 | 2.07 | .120 | .096 |
| 2[c] | 2.15 | 2.05 | .139 | .606 | 1.93 | 2.13 | 2.24 | .170 | .415 | 1.93 | 2.27 | .139 | .136 |
| 3[d] | 2.04 | 2.09 | .016 | .708 | 1.95 | 2.12 | 2.13 | .113 | .440 | 2.29 | 1.83 | .092 | .108 |
| 4 | 1.81 | 1.86 | .047 | .432 | 1.90 | 1.82 | 1.79 | .058 | .445 | 1.80 | 1.87 | .047 | .562 |
| 5[f] | 1.98 | 2.07 | .031 | .043 | 1.99 | 2.07 | 2.00 | .038 | .278 | 1.98 | 2.08 | .031 | .045 |
| 6 | 1.99 | 2.03 | .063 | .687 | 2.01 | 2.11 | 1.91 | .011 | .223 | 1.88 | 2.14 | .063 | .423 |
| 1-2 | 1.97 | 1.88 | .076 | .380 | 1.76 | 1.92 | 2.09 | .093 | .056 | 1.82 | 2.03 | .076 | .433 |
| 1-3[c] | 1.94 | 1.92 | .056 | .780 | 1.81 | 1.94 | 2.04 | .068 | .061 | 1.95 | 1.91 | .056 | .768 |
| 1-4 | 1.84 | 1.88 | .026 | .312 | 1.82 | 1.87 | 1.89 | .032 | .314 | 1.83 | 1.89 | .026 | .137 |
| 1-5 | 1.89 | 1.93 | .021 | .108 | 1.87 | 1.93 | 1.92 | .026 | .254 | 1.87 | 1.95 | .021 | .003 |
| 1-6 | 1.88 | 1.93 | .019 | .097 | 1.89 | 1.94 | 1.88 | .024 | .212 | 1.86 | 1.95 | .019 | .050 |
| 4-6 | 1.88 | 1.93 | .019 | .053 | 1.92 | 1.93 | 1.87 | .023 | .133 | 1.84 | 1.98 | .019 | .110 |
| overall | 1.89 | 1.93 | .019 | .370 | 1.88 | 1.93 | 1.91 | .023 | .249 | 1.87 | 1.95 | .019 | .035 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate.
[b]$P$ - Probability of difference due to treatment.
[c]Trial by dietary pH interaction ($P < .10$).
[d]Phosphorus source by dietary pH interaction ($P < .05$).
[e]Trial by dietary ph interaction ($P < .05$).

APPENDIX TABLE 13. THE EFFECT OF PHOSPHORUS SOURCE AND DIETARY pH ON GASTROINTESTINAL pH, CHLORIDE ION CONCENTRATION AND TITRATION VALUES OF WEANLING PIGS (COMBINED TRIALS)

| | Phosphorus Source[a] | | | | Dietary pH | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | DCP | DFP | S.E. | $P$[b] | 5.4 | 6.0 | 6.7 | S.E. | $P$[b] |
| Stomach | | | | | | | | | |
| TV[c] | 87.4 | 82.3 | 8.7 | .307 | 84.8 | 89.2 | 80.4 | 21.4 | .354 |
| DM[d] | 38.6 | 37.4 | .003 | .008 | 38.3 | 38.1 | 37.7 | .004 | .511 |
| ATV[e] | 226.4 | 219.8 | 52.4 | .632 | 221.4 | 224.7 | 213.3 | 64.0 | .673 |
| pH | | | | | | | | | |
| stomach | 3.90 | 3.82 | .087 | .508 | 3.72 | 4.01 | 3.84 | .106 | .156 |
| jejunum | 5.71 | 5.72 | .039 | .796 | 5.70 | 5.74 | 5.70 | .048 | .765 |
| cecum | 5.67 | 5.67 | .023 | .833 | 5.70 | 5.68 | 5.64 | .028 | .255 |
| colon | 6.50 | 6.37 | .032 | .001 | 6.47 | 6.41 | 6.43 | .039 | .553 |
| Chloride | | | | | | | | | |
| stomach | .067 | .070 | .002 | .667 | .074 | .061 | .073 | .003 | .002 |
| jejunum | .101 | .104 | .001 | .206 | .100 | .104 | .104 | .002 | .410 |
| cecum | .012 | .013 | .001 | .317 | .013 | .012 | .013 | .001 | .164 |
| colon | .021 | .010 | .007 | .233 | .011 | .025 | .011 | .008 | .369 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate.
[b]$P$ - Probability of differences due to treatment.
[c]TV - Titration value = (meq HCl/kg digesta to a pH of 2) subtracted from (meq NaOH/kg digesta from pH of 2 to pH of 8).
[d]DM - dry matter.
[e]ATV - adjusted titration value (TV/DM).

APPENDIX TABLE 14. THE EFFECT OF DIETARY pH (BY TRIAL) ON GASTROINTESTINAL pH, CHLORIDE ION CONCENTRATION AND TITRATION VALUES OF WEANLING PIGS FED TWO SOURCES

| | TRIAL 1 | | | TRIAL 2 | | | | |
|---|---|---|---|---|---|---|---|---|
| | 5.4 | 6.0 | 6.7 | 5.4 | 6.0 | 6.7 | S.E. | $P$[a] |
| Stomach | | | | | | | | |
| TV[b] | 88.2 | 95.4 | 86.4 | 80.8 | 83.0 | 74.4 | 6.12 | .903 |
| DM[c] | 33.3 | 33.0 | 32.7 | 43.7 | 43.3 | 42.6 | .527 | .948 |
| ATV[de] | 270.0 | 297.0 | 284.6 | 190.0 | 192.0 | 177.2 | 18.0 | .709 |
| pH | | | | | | | | |
| stomach | 3.73 | 4.09 | 4.01 | 3.72 | 3.94 | 3.68 | .149 | .568 |
| jejunum | 5.74 | 5.83 | 5.71 | 5.66 | 5.65 | 5.69 | .069 | .481 |
| cecum | 5.67 | 5.67 | 5.60 | 5.74 | 5.68 | 5.68 | .045 | .747 |
| colon | 6.51 | 6.36 | 6.46 | 6.43 | 6.45 | 6.40 | .055 | .233 |
| Chloride | | | | | | | | |
| stomach | .072 | .060 | .066 | .075 | .063 | .084 | .004 | .239 |
| jejunum | .094 | .096 | .096 | .109 | .111 | .112 | .003 | .974 |
| cecum | .012 | .011 | .012 | .014 | .013 | .014 | .001 | .936 |
| colon | .011 | .012 | .010 | .011 | .039 | .012 | .012 | .459 |

[a] $P$ - Probability of trial by pH interaction.
[b] TV - Titration value = (meq HCl/kg digesta to a pH of 2) subtracted from (meq NaOH/ kg digesta from pH of 2 to pH of 8).
[c] DM - dry matter.
[d] ATV - adjusted titration value (TV/DM).
[e] Trial effect ($P < .05$).

APPENDIX TABLE 15. THE EFFECT OF PHOSPHORUS SOURCE (BY TRIAL) ON GASTROINTESTINAL pH, CHLORIDE ION CONCENTRATION AND TITRATION VALUES OF WEANLING PIGS FED TWO PHOSPHORUS SOURCES

| Item | TRIAL 1 DCP[a] | TRIAL 1 DFP[a] | TRIAL 2 DCP[a] | TRIAL 2 DFP[a] | SE | P[b] |
|---|---|---|---|---|---|---|
| Stomach | | | | | | |
| TV[c] | 96.0 | 84.0 | 78.6 | 80.2 | 5.00 | .180 |
| DM[d] | 33.6 | 32.4 | 43.7 | 42.4 | .447 | .869 |
| ATV[ef] | 295.6 | 272.2 | 181.8 | 191.0 | 14.8 | .225 |
| pH | | | | | | |
| stomach | 3.87 | 4.01 | 3.93 | 3.63 | .122 | .076 |
| jejunum | 5.77 | 5.75 | 5.64 | 5.69 | .055 | .445 |
| cecum | 5.65 | 5.63 | 5.69 | 5.70 | .032 | .690 |
| colon | 6.50 | 6.39 | 6.50 | 6.35 | .045 | .552 |
| Chloride | | | | | | |
| stomach | .066 | .066 | .072 | .074 | .003 | .850 |
| jejunum | .093 | .097 | .110 | .111 | .002 | .502 |
| cecum | .011 | .012 | .014 | .014 | .001 | .456 |
| colon | .012 | .010 | .031 | .010 | .010 | .338 |

[a]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate.
[b]*P* - Probability of trial by phosphorus interaction.
[c]TV - Titration value = (meq HCl/kg digesta to a pH of 2) subtracted from (meq NaOH/kg digesta from pH of 2 to pH of 8).
[d]DM - dry matter.
[e]ATV - adjusted titration value (TV/DM).
[f]Trial effect ($P < .05$).

APPENDIX TABLE 16. THE EFFECT OF PHOSPHORUS SOURCE (BY TRIAL) ON METACARPAL, METATARSAL AND HUMERUS MEASUREMENTS OF WEANLING PIGS[a] FED TWO PHOPHORUS SOURCES

| Item | TRIAL 1 DCP[b] | TRIAL 1 DFP[b] | TRIAL 2 DCP[b] | TRIAL 2 DFP[b] | SE | P[c] |
|---|---|---|---|---|---|---|
| *Metacarpal* | | | | | | |
| Volume(ml) | 6.00 | 6.05 | 6.08 | 6.06 | .098 | .715 |
| sp gr[d] | 1.189 | 1.190 | 1.201 | 1.183 | .005 | .046 |
| Area($cm^2$) | .354 | .347 | .356 | .356 | .008 | .707 |
| Force(kg) | 83.57 | 82.85 | 87.02 | 80.23 | 2.16 | .161 |
| Stress[e] | 120.4 | 122.0 | 123.8 | 113.8 | 4.05 | .151 |
| *Metatarsal* | | | | | | |
| Volume(ml) | 7.79 | 7.86 | 7.84 | 7.91 | .112 | .986 |
| sp gr[d] | 1.178 | 1.173 | 1.179 | 1.169 | .002 | .263 |
| Area($cm^2$) | .374 | .383 | .411 | .385 | .009 | .051 |
| Force(kg) | 69.33 | 72.10 | 72.82 | 70.64 | 2.26 | .276 |
| Stress[e] | 94.2 | 94.5 | 91.2 | 93.8 | 3.77 | .771 |
| *Humerus* | | | | | | |
| Volume(ml)[f] | 4.92 | 4.96 | 8.15 | 7.68 | .134 | .063 |
| sp gr[dg] | 1.430 | 1.422 | 1.383 | 1.376 | .007 | .966 |

[a] All values adjusted for final body wt by covariance analysis.
[b]DCP - Dicalcium phosphate, DFP - Defluorinated rock phosphate.
[c]*P* - Probability of trial by phosphorus source interaction.
[d]Specific gravity in g/ml.
[e]Stress is expressed in $kg/cm^2$.
[fg]Trial effect ($P < .001$, $P < .01$).

APPENDIX TABLE 17. THE EFFECT OF DIETARY pH (BY TRIAL) ON METACARPAL, METATARSAL AND HUMERUS MEASUREMENTS OF WEANLING PIGS[a] FED TWO PHOSPHORUS SOURCES

| | TRIAL 1 | | | TRIAL 2 | | | | |
|---|---|---|---|---|---|---|---|---|
| | pH 5.4 | pH 6.0 | pH 6.7 | pH 5.4 | pH 6.0 | pH 6.7 | SE | $P^b$ |
| *Metacarpal* | | | | | | | | |
| Volume(ml) | 6.06 | 6.08 | 5.93 | 5.86 | 6.30 | 6.05 | .120 | .201 |
| sp gr[c] | 1.187 | 1.193 | 1.186 | 1.192 | 1.197 | 1.187 | .007 | .970 |
| Area($cm^2$) | .369 | .338 | .343 | .345 | .363 | .360 | .011 | .047 |
| Force(kg) | 84.60 | 83.91 | 81.11 | 84.33 | 82.63 | 83.91 | 2.62 | .723 |
| Stress[d] | 116.72 | 125.94 | 120.92 | 124.24 | 114.32 | 117.78 | 5.00 | .164 |
| *Metatarsal* | | | | | | | | |
| Volume(ml) | 7.80 | 7.96 | 7.71 | 7.73 | 7.98 | 7.91 | .138 | .598 |
| sp gr[c] | 1.175 | 1.176 | 1.175 | 1.177 | 1.171 | 1.174 | .003 | .557 |
| Area($cm^2$) | .387 | .371 | .377 | .402 | .400 | .391 | .011 | .745 |
| Force(kg) | 67.03 | 76.04 | 69.07 | 70.67 | 69,94 | 74.57 | 2.76 | .083 |
| Stress[d] | 87.47 | 101.89 | 93.77 | 89.15 | 90.61 | 97.73 | 4.65 | .205 |
| *Humerus* | | | | | | | | |
| Volume(ml)[e] | 4.95 | 5.00 | 4.87 | 7.93 | 7.85 | 7.96 | .164 | .767 |
| sp gr[cf] | 1.436 | 1.431 | 1.422 | 1.379 | 1.378 | 1.381 | .008 | .771 |

[a]All values adjusted for final body wt by covariance analysis.
[b]*P* - Probability of trial by dietary pH effect.
[c]Specific gravity in g/ml.
[d]Stress is expressed in $kg/cm^2$.
[ef]Trial effect ($P < .001$, $P < .01$)

APPENDIX TABLE 18. THE EFFECT OF PHOSPHORUS SOURCE, DIETARY pH AND GENDER ON HUMERUS, METACARPAL AND METATARSAL MEASUREMENTS OF WEANLING PIGS[a] (COMBINED TRIALS)

| | Phosphorus Source[b] | | | | Dietary pH | | | | | Gender | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | DCP | DFP | S.E. | $P$[c] | 5.4 | 6.0 | 6.7 | S.E. | $P$[c] | Male | Female | S.E. | $P$[c] |
| *Metacarpal* | | | | | | | | | | | | | |
| Volume(ml) | 6.04 | 6.05 | .069 | .892 | 5.96 | 6.19 | 5.99 | .086 | .126 | 6.24 | 5.85 | .070 | .035 |
| sp gr[d] | 1.194 | 1.186 | .004 | .143 | 1.189 | 1.195 | 1.186 | .005 | .441 | 1.180 | 1.200 | .004 | .047 |
| Area($cm^2$)[f] | .355 | .351 | .006 | .667 | .357 | .351 | .351 | .007 | .801 | .357 | .350 | .006 | .521 |
| Force(kg) | 85.3 | 81.5 | 1.52 | .084 | 84.5 | 83.3 | 82.5 | 1.88 | .763 | 79.9 | 86.9 | 1.52 | .030 |
| Stress[e] | 122.1 | 117.9 | 2.86 | .305 | 120.5 | 120.1 | 119.3 | 3.52 | .974 | 113.9 | 125.9 | 2.86 | .034 |
| *Metatarsal* | | | | | | | | | | | | | |
| Volume(ml) | 7.81 | 7.88 | .079 | .536 | 7.77 | 7.97 | 7.81 | .098 | .311 | 8.04 | 7.65 | .079 | .059 |
| sp gr[d] | 1.178 | 1.171 | .002 | .004 | 1.176 | 1.174 | 1.175 | .002 | .726 | 1.170 | 1.179 | .002 | .057 |
| Area($cm^2$) | .392 | .384 | .006 | .345 | .395 | .385 | .384 | .008 | .567 | .395 | .381 | .006 | .313 |
| Force(kg) | 71.1 | 71.4 | 1.60 | .897 | 68.8 | 73.0 | 71.8 | 1.97 | .316 | 71.9 | 70.5 | 1.60 | .547 |
| Stress[e] | 92.7 | 94.2 | 2.66 | .703 | 88.3 | 96.2 | 95.8 | 3.24 | .170 | 92.1 | 94.8 | 2.66 | .564 |
| *Humerus* | | | | | | | | | | | | | |
| Volume(ml)[g] | 6.54 | 6.32 | .095 | .107 | 6.45 | 6.43 | 6.41 | .116 | .988 | 6.54 | 6.31 | .094 | .498 |
| sp gr[dh] | 1.406 | 1.399 | .004 | .285 | 1.403 | 1.405 | 1.401 | .066 | .927 | 1.390 | 1.416 | .005 | .046 |

[a]All values adjusted for final body wt by covariance analysis.
[b]DCP - Dicalcium phosphate; DFP - Defluorinated rock phosphate.
[c]$P$ - Probability of differences due to treatment.
[d]Specific gravity in g/ml.
[e]Stress is expressed in $kg/cm^2$.
[f]Phosphorus source by dietary pH interaction ($P < .1$).
[gh]Trial effect ($P < .001$, $P < .01$)

APPENDIX TABLE 19. THE EFFECT OF PHOSPHORUS SOURCE AND DIETARY pH ON BACKFAT AND LOIN MUSCLE AREA OF WEANLING PIGS (COMBINED TRIALS)

| | Phosphorus Source[a] | | | | Dietary pH | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | DCP | DFP | S.E. | $P$[b] | 5.4 | 6.0 | 6.7 | S.E. | $P$[b] |
| Backfat (cm)[e] | .991 | .965 | .011 | .503 | .980 | .991 | .963 | .013 | .843 |
| Loin area[cdf] | 3.790 | 3.884 | .028 | .355 | 3.879 | 3.868 | 3.762 | .035 | .585 |

[a]DCP - Dicalcium phosphate, DFP - Defluorinated rock phosphate.
[b]$P$ - Probability of difference due to treatment.
[c]Loin area is expressed in $cm^2$.
[d]pH times phosphorus source interaction ($P < .05$).
[ef]Trial effect ($P < .01$, $P < .001$).
[f]Trial effect ($P < .001$)

# VITA

Mark Laroy Straw, is the second child of Roy L. Straw and Janet Roesener Straw. He was raised on a grain and livestock farm in east central Illinois and entered Berea College, Berea, Kentucky, in the fall of 1983. Among the honors received while a student at Berea were the Massey Award for labor achievement above and beyond the call of duty, the Danforth Creative Effort Award and the American Society of Animal Science Undergraduate Scholarship Award. He was elected president of the agriculture honorary Delta Tau Alpha and vice president of the student organization Agriculture Union. From 1985 to 1988 he was student manager of the Berea College Swine farm. He received his B.S. in Agriculture in May, 1988 and enrolled in Virginia Polytechnic Institute and State University in the fall. While at VPI & SU he was employed as a graduate research assistant.

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