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The impact of dietary phosphorus regimen on

muscle growth and quality

in pig differing in lean growth capacity

by

Mark John Bertram

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The impact of dietary phosphorus regimen on
muscle growth and quality
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Mark John Bertram

Major Professor: Tim S. Stahly

Iowa State University

An experiment was conducted to examine the impact of dietary phosphorus regimen

on body tissue deposition, the efficiency of utilization of dietary available phosphorus for

protein deposition, and on post-mortem exudative losses, water holding capacity and color

scores during retail storage of longissimuss muscle sections from pigs of two genetic strains

differing in lean growth capacity. Seven sets of six littermate barrows from each of a high and

moderate lean growth (LG) genotype were allotted within litterto one of six diets differing in

dietary available phosphorus (AP) concentration (.080, .110, .155, .222, .323, and .475%).

Pigs were allowed to consume their experimental diets ad-libitum. At 109 ± 4 kg pigs were

removed from feed at 7:00 am, transported 5 km, electrically stunned and killed by

exsanguination .25 to 1.5 hr post-transport. From 20 to 109 kg, high LG pigs accrued more

muscle daily, gained more weight per unit of feed, and produced carcasses with more

dissectable muscle and less fatty tissue than moderate LG pigs. Longissimus muscle (LM)

sections from high LG pigs had lower pH 45 minutes post-mortem, larger initial weights,

higher exudative losses from d 0 to d 3, lower water holding capacities (WHC), more

moisture and less fat, and were lighter in color on d 0, whereas redness and yellowness of the LM sections were unaffected by LG genotype. Longissimus muscle sections from high LG pigs had higher exudative loss early in the storage period and lower exudative loss later than LM sections from moderate LG pigs in both the fresh and post-thaw state. Longissimus muscle sections became lighter and less red in color as storage progressed, however the pattern of color change as storage progressed differed between genotypes in both the fresh and post-thaw states. As dietary AP concentration decreased, the rate of deposition of high protein, high phosphorus containing tissues (muscle, bone) decreased, whereas that of low protein, low phosphorus containing tissues (fatty tissue) increased. Pigs deposited 12 to 15 g of protein per g of dietary AP. Dietary AP concentration had minimal impact on LM quality traits. Thus, the phosphorus needs of pigs are determined largely by the animals deposition rates of proteinaceous tissues especially those rich in phosophorus.

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CHAPTER 1. GENERAL INTRODUCTION

Phosphorus is involved in a wide variety of biological functions. Approximately 80% of the phosphorus in the body is present in the skeleton with the remaining 20% being distributed in soft tissue in the form of high energy phosphate compounds, phospholipids, nucleic acids and phosphoproteins. Animals consuming inadequate levels of phosphorus have been reported to have decreased growth rate, efficiency of feed utilization and bone development (Cromwell, 1970; van Kempen et al., 1976; Jongbloed, 1987). Pigs and mice consuming deficient levels of phosphorus have been shown to retain less body nitrogen than those consuming adequate levels (Vipperman et al., 1974; Henry et al., 1979; Jongbloed, 1987).

Pigs have been reported to retain a relatively constant amount of phosphorus (35 to 39 g) per kilogram of body protein accrued (Jongbloed, 1987). On this basis, it would be hypothesized that animals with a greater genetic capacity for lean tissue growth would require greater intakes of phosphorus in order to attain their maximal accretion rate of proteinaceous tissues. However, the impact of differences in tissue growth capacities among genetic strains on dietary phosphorus needs has not been determined.

Phosphorus containing compounds (i.e., phospholipids, high energy phosphate compounds) play key roles in maintaining muscle membrane integrity, calcium movement in muscle and muscle energy charge, and thus may influence pork quality. The impact of dietary phosphorus regimen on pale, soft, and exudative (PSE) pork has not been extensively investigated.

An understanding of the role of lean growth genotype on phosphorus needs will allow phosphorus supplementation to be matched to the needs of pig. As pressure from environmental groups to minimize excretion of excess phosphorus intensifies it is important to provide adequate phosphorus to minimize the detrimental effects of phosphorus deficiency without over-supplementation which results in increased phosphorus excretion.

Dissertation Organization

The following dissertation is organized as a literature review followed by two papers which are in the style and form of the Journal of Animal Science, and will be submitted to the Journal of Animal Science, and finally a general summary of the whole dissertation. The review of the literature examines tissue growth and pork quality as it relates to the biological role of phosphorus in the body. The review will focus on the role that phosphorus plays in muscle growth and development and the response of muscle to limitations in dietary intake of phosphorus. The review is organized into two main sections. The first deals with the biology and physiology of muscle growth and meat quality and the second examines the biology of phosphorus metabolism and how this relates to animal growth in times of inadequate phosphorus intake. The research reported in the papers was conducted by Mark J. Bertram under the direction of Dr. Tim S. Stahly. Mark J. Bertram is the principal author of the paper and was principally involved in the design of the experiments, collection, analysis and interpretation of data as well as the writing of the papers.

CHAPTER 2. REVIEW OF LITERATURE

Overview of Muscle Growth and Quality

In the production of meat animals, the primary goal is the economical production of lean, high quality meat products. Consumer demands have changed dramatically over the last decade. Ease of preparation and consistent quality are musts if a product is to compete in today's marketplace. With the advent of nutritional labeling and increased competition from other protein foodstuffs, it is important to examine genetic, nutritional and environmental factors which influence muscle growth and quality in order to modify and control production practices to provide a product desired by the consumer.

Chemical and Anatomical Structure of Muscle

Skeletal muscle tissue in an anatomical sense is a contractile organ involved primarily in locomotion and its structure and composition is a result of this function. To begin the discussion of muscle it is important to clearly define muscle as it relates to animal agriculture and the production of meat products. In the most basic sense, muscle tissue is the collection of muscle cells along with small quantities of intramuscular fat and connective tissue. Chemically, muscle is 75% water, 19% protein, 2.5% lipid, 1.2% carbohydrate and 2.3 % other non-protein substances (Lawrie, 1985). The protein content can be broken down into myofibrillar, 11.5%; sarcoplasmic, 5.5%; and connective tissue and organelle, 2%. However, this composition is altered by gender, dietary regimen and health status (Lawrie, 1985). Muscle contains approximately .2% phosphorus which represents 17.2% of the total

phosphorus in the body, the highest concentration of phosphorus of any tissue in the body, except bone.

Anatomically, an individual muscle is composed of numerous muscle fibers which are the cellular unit of muscle. Fibers are elongated and multinucleated and range in size from 10 to 100 μm in diameter and 1 to 40 mm in length and are surrounded by a layer of connective tissue called the endomysium. Fibers are grouped into bundles and surrounded by a second connective tissue layer called the perimysium and finally groups of bundles are surrounded by a connective tissue layer which surrounds the whole muscle called the epimysium (Cassens, 1989). The sub-cellular structure of the muscle cell has been extensively reviewed by Swatland (1984) and Cassens (1989). Briefly, individual muscle fibers are surrounded by the cell membrane called the sarcolemma which lies immediately under the endomysium. Unlike many cell types, the nuclei of muscle cells are peripherally located just beneath the sarcolemma. The cytoplasm of muscle fibers, which is referred to as the sarcoplasm, is dominated by the actual contractile apparatus of muscle, the myofibrils. Several myofibrillar proteins have been identified and characterized with the predominant ones being actin, myosin, tropomysin, troponin, titan and nebulan. These proteins are organized into highly structured, three dimensional repeating units called sarcomeres, which are the contractile unit of muscle (Murray and Weber, 1974). The sarcomere is composed of two main filaments, the thick filament and the thin filaments and is devided into several zones and lines based on its appearance under a light microscope (Huxley, 1957). The predominant proteins of the thick filament is myosin, while thin filaments are composed mainly of actin, tropomysin and

troponin. Muscle also contains a highly developed intracellular membrane system, the

sarcoplasmic reticulum, which is responsible for regulation of cytoplasmic Ca^{2-} concentration,

the main regulator of contractile activity (Zot and Potter, 1987; Lytton and MacLennan,

1992). There are numerous invaginations in the sarcolemma called T-tubules which sit very

close to the sarcoplasmic reticulum and form structures called lateral cisternae. It is the

T-tubule system that allows propagation of nerve signals from the sarcolemma to the

sarcoplasmic reticulum. This signaling causes release of Ca^{2-} from the sarcoplasmic reticulum

and initiates contraction. Located between myofibrils is the power house of the cell, the

mitochondria (Cassens, 1989). Interspersed throughout the sarcoplasm are the glycolytic

proteins, myoglobin, creatine kinase and the intracellular energy stores of glycogen granules

and lipid droplets, however these sarcoplasmic components have been reported to be entirely

restricted from entry between myofibrils (Lawrie, 1985; Cassens, 1989). However, strong

evidence has been presented that the glycolytic enzymes are intimately associated with the

I-band region of the sarcomere, allowing rapid transfer of energy trapped in glycolysis to the

energy requiring enzymes involved in muscle contraction (Clarke et al, 1983).

Developmental Patterns of Tissue and Muscle Growth

Growth is the process through which environmental inputs are integrated into the body

and can consist of hyperplasia, an increase in cell number, and hypertrophy, an increase in cell

size (Brody, 1945). Animal growth follows a sigmoidal pattern relative to time (Clausen,

1953). An initial period of relatively slow growth is followed by a period of steep linear

increase in growth rate with a gradual slowing of growth as an animal approaches maturity.

Development refers to the qualitative aspect of growth as tissues and organ systems are formed or, in a more practical sense, how the composition of growth changes until an animal reaches maturity (Doornenbal, 1971) The composition of growth is highly dependent on developmental stage and the level of animal maturity (Richmond and Berg, 1971). Early, body growth is dominated by internal organ and digestive tract development, this is followed by a period where skeletal and muscle development predominate, and finally development of adipose tissue (McMeekan, 1940). It has been hypothesized that at given percentage of maturation, animals in a species will have the same body composition regardless of genotype (Taylor, 1980). However, genotypes with a higher potential for lean tissue growth have a larger mature weight. Thus, at a common market weight, high lean growth animals will generally possess carcasses that are less mature in their body composition, specifically, the carcasses will have more muscle and bone and less fatty tissue than lower lean growth animals at a similar body weight (Stahly, 1989).

The growth of individual muscles in the carcass have been reported to follow a specific developmental pattern consisting of anterior to posterior and ventral to dorsal order of development (McMeeken, 1940).

Tissue Growth and Regulation

The growth of an individual tissue is dependent on the growth and development of the cells which make up that tissue. Numerous nutritional, hormonal and enzymatic factors contribute to the growth and development of cells. The relative rate of growth of the various cell types determines body composition at a common market weight.

<u>Muscle Tissue</u>

Embryological Muscle Growth. Muscle tissue is composed of numerous multinucleated

myofibers which are further composed of myofibrils which are made up of the repeating unit

of the muscle cell, the sarcomere (Young, 1985). Muscle tissue arises from the lateral plate of

the mesoderm when pluripotiential stem cells differentiate to become presumptive myoblasts,

which in turn form myoblasts. Myoblasts then fuse and begin to synthesize muscle specific

proteins and form mutinucleated myotubes. Both presumptive myoblasts and myoblasts are

mitotic and capable of proliferation; however, upon fusion cells become non-mitotic and

proliferation ceases (Young, 1985). Fusion and cessation of mitosis of myoblasts essentially

are complete at or shortly after birth, thus, muscle fiber number is established at birth (Ontell

and Dunn, 1978). Growth and development of embryological muscle is regulated through the

sequential expression of a family of genes known as the myogenic regulatory genes (Miner

and Wold, 1991). These genes control the timing of differentiation and fusion and result in

orderly muscle development in the embryo. Gene expression is under the control of several

growth factors which include fibroblast growth factor and transforming growth factor type-β,

although the exact mechanism of control is unknown at this time (Olson et al., 1991).

Postnatal Muscle Growth. Postnatal muscle growth in pigs occurs through hypertrophy of

existing fibers by the addition of new myofibrills for diametric growth, and sarcomere units for

longitudinal growth. Additional nuclei can be added to muscle fibers as they grow through

addition of partially committed myogenic cells called satellite cells (Moss and Leblond, 1970).

Muscle protein accretion is a result of the combined effects of protein synthesis and degradation and occurs when the rate of synthesis outpaces the rate of degradation (Young, 1974). The relative importance ot the rate of protein synthesis versus the rate of protein breakdown in determining the rate of muscle protein accretion is an area of some debate. Maruyama et al. (1978) and Klasing and Jarrell (1985) demonstrated a negative relationship between fractional breakdown rate and fractional accretion rate, with no relationship between fraction synthesis rate and fractional accretion rate. However, Millward et al (1976) and Millward (1978) concluded that muscle protein mass was regulated primarily through alteration in protein synthesis in all except emergency conditions and that a rapid rate of muscle growth was associated with high rates of both protein synthesis and protein breakdown. These authors hypothesized that this may be due to a need for remodeling of the muscle cell during growth.

Muscle growth is controlled on a systemic basis primarily through the action of growth hormone, insulin, thyroxin and the insulin like growth factors with glucocorticoids coming into play in special cases. Numerous other growth factors also act on cells in a paracrine and/or autocrine basis. The effect of several hormones on protein synthesis, protein degradation and protein accretion is shown in Table 1.

Both insulin and growth hormone have been reported to be affected by dietary phosphorus regimen, thus a further discussion of the role of these hormones in muscle growth is warranted. Growth hormone is a protein hormone of approximately 191 amino acids which is secreted by the anterior pituitary under hypothalamic control (Stahly and Bark, 1991). The

response of muscle tissue to growth hormone is thought to be mediated in two ways: 1) an

indirect mechanism whereby growth hormone stimulates insulin-like growth factor (IGF)

production in the liver which in turn stimulates muscle growth and 2) a direct IGF dependent

mechanism whereby growth hormone stimulates muscle cells to produce IGF which acts in an

autocrine and/or paracrine manner to stimulate muscle growth (Beermann and DeVol, 1991).

Table 1. Hormonal effects on muscle protein synthesis, degradation and protein accretion
(Adapted from Navakofskin and McCusker; 1993, Young, 1980)

Hormone	Protein Synthesis	Protein Degradation	Protein Accretion
Insulin	++	--	+
Growth Hormone	++	?	++
IGFs	+	No effect	+
Catecholamines	+	-	+
Thyroid Hormone (to normal)	++	+	+
Thyroid Hormone (hyper)	+	++	-

Treatment of pigs with exogenous somatotropin has been reported to increase muscle

deposition rates by approximately 25 to 35%, in a dose dependent manner, compared to

untreated controls (Thiel et al., 1990). In addition, animals with higher lean growth potentials

such as high lean genetic strains and boars have been reported to have higher circulating

levels of growth hormone than lower lean growth animals and gilts (Bark, 1990).

Consequently, factors which reduce circulating levels of growth hormone would be expected to decrease muscle growth.

Insulin is a peptide hormone released from the β-cells of the pancreas, primarily in response to increased circulating levels of glucose. Insulin exerts a milieu of effects on muscle cells including increased glucose and amino acid uptake and increased RNA, DNA and protein synthesis (Young, 1980). Insulin exerts its effects by binding with a membrane receptor which stimulates a cascade of intracellular events, which in turn alter the activity of membrane transporter proteins and intracellular enzymes thus stimulating muscle protein accretion (Young, 1980). Decreased sensitivity of muscle cells to insulin would be expected to decrease glucose and amino acid uptake and protein synthesis.

Adipose Tissue

The primary hormones controlling adipose tissue growth and development are growth hormone, the catecholamines, and insulin. Elevated circulating levels of growth hormone and catecholamines generally depress adipose deposition, while insulin is generally anabolic. Increased circulating levels of growth hormone, either endogenous or exogenous, results in decreased carcass adipose tissue (Etherton et al., 1987). It appears that growth hormone results in decreased lipogenesis, with little or no change in lipolysis (Walton and Etherton, 1986; Davidson, 1987). Growth hormone administration to cultured adipocytes resulted in decreased activities of acetyl CoA carboxylase, fatty acid synthetase, malic enzyme, glycerol-3-phosphate dehydrogenase, and ATP citrate lyase, enzymes involved in lipogenesis (Bornstein et al., 1983; Schaffer, 1985). Catecholamines and their synthetic analogs, the

ß-adrenergic agonists, have been reported to depress adipose tissue deposition by decreasing pre-adipocyte proliferation and lipogenesis, and increasing lypolysis (Jones et al., 1988; Hausman et al., 1989). Insulin is primarily an anabolic hormone for adipose tissue. Obese strains of mice and pigs have been shown to have high circulating levels of insulin (Bray and York, 1971; Gregory et al., 1977; Mersmann et al., 1982). The binding of insulin to its membrane receptor resulted in increased glucose transport into adipocytes, increased activity of lipogenic enzymes and decreased activity of lipolytic hormones.

Bone Tissue

The main hormone which has been demonstrated to regulate bone growth, either directly or indirectly is growth hormone (Isaksson et al., 1987). Somatotropin has been reported to stimulate DNA synthesis in epiphyseal chondrocytes and promote differentiation of prechondrocytes (Madsen et al., 1983; Isaksson et al., 1987). Treatment of animals with exogenous somatotropin increased bone mass by 10 to 17% in a dose dependent manner (Theil et al., 1990). Decreasing circulating levels of growth hormones would therefore be expected to decrease bone growth and thus, phosphorus deposition in bone tissue.

Hormones have a multitude of actions which affect muscle, bone and adipose tissue growth. Changes in levels or activities of these hormones will result in changes in tissue deposition and body composition. The role that these hormones play in determining the response to dietary phosphorus regimen will be discussed in detail later in this review.

Muscle/Meat Quality

As we begin our discussion of muscle/meat quality it is important to understand the distinction between muscle and meat , what quality is and how muscle metabolism is regulated under normal circumstances to maintain meat quality. In general, meat is the metabolically inert substance that results from post-mortem changes in muscle and is a result of the bio-chemical and physiological processes which occur in muscle (Lawrie, 1985). Meat quality is determined by the functional and aesthetic characteristics of a muscle or group of muscles and can differ depending on their proposed food use of the meat , i.e. quality as defined in a fresh meat product can be different than quality defined in a product which will be processed and/or cured (Kauffman and Marsh, 1987). Quality is dependent on both composition and structure of the muscle. Composition is entirely the result of factors active during the life of the animal. Structure is a result of events that occur not only in life, but also in the early post-mortem period. In this discussion we will be concerned with quality as it relates to a fresh meat product and will deal largely with difference in meat color and water holding capacity. It is generally accepted that consumers desire pork which is reddish-pink in color, firm to the touch, and which does not have an over-abundance of water standing on the cut surface or in the storage tray. In addition, muscle water loss postmortem is important to packers who sell meat on a weight basis and has been reported to result in a loss of as much as $7 million annually to the packing industry (Miller,1989). Variations in color and in the ability of meat to bind and hold water are largely due to differences in the rate and magnitude of pH decline in the muscle post-mortem (Dutson, 1983).

The cellular functions of the muscle cell include but are not limited to protein synthesis and turnover and maintenance of ion gradients (i.e. Na^-/K^- and Ca^{2+}) and contraction. The currency of energy metabolism in muscle consists of high energy phosphate compounds such as adenosine triphosphate (ATP), adenosine diphosphate (ADP) and creatine phosphate (CP). ATP is the main source of energy for vital cellular functions. Adenosine triphosphate can be generated in the cell in one of two ways; 1) through anaerobic metabolism of glucose, in the cytoplasm, via glycolysis to lactic acid. and 2) through aerobic oxidation of glucose and/or fatty acids, in the mitochondria, via the Krebs cycle. It is believed that ATP is not free to diffuse throughout the cell, but rather, is highly compartmentalized in the cell to regions with high use of ATP (Bessman and Carpenter, 1985; Zeleznikar and Goldberg, 1991). Membrane bound creatine phosphokinase acts to transfer energy from ATP to creatine phosphate. Creatine phosphate then acts as a courier of energy from the mitochondria, to the various points of energy utilization (Bessman and Carpenter, 1985). In discussing meat quality, understanding anaerobic glycolysis is very important because the endproduct of the pathway is lactic acid. Buildup of lactic acid in the cell results in a decrease in pH. In the live animal, muscle pH is regulated and maintained at about 7.6 in rested muscle through the action of various buffering systems and through the Cori cycle (Lawrie, 1985). The Cori cycle involves transport of muscle lactate from muscle by the circulatory system to the liver, where it is metabolized releasing glucose back into the blood stream for use by the muscle (Pearson, 1989). The rate of glycolysis is largely controlled through regulation of the enzymes involved in the first committed step in the pathway, the conversion of fructose-6-phosphate to

fructose-1,6-diphate. When the local ratio of ATP/(ADP + P_i) is high, the enzyme is inactivated and glycolysis is shut down. As ATP is utilized to fuel cellular metabolism, it is replenished from CP and ADP through the action of creatine phosphokinase (ADP + CP $\leftrightarrow$ ATP + Creatine). When the supply of CP begins to decline or the rate of ATP utilization outpaces the activity of creatine phosphokinase, ADP begins to buildup and the ATP/(ADP + P_i) ratio increases and glycolysis is activated to replenish the energy stores of the cell. In post-mortem muscle, anaerobic glycolysis is the only pathway for energy metabolism and glucose from muscle glycogen the only fuel source, because circulation is shut off and oxygen and fuel delivery to the muscle cell cease. Thus, lactic acid begins to build up and pH declines. The rate and extent of post-mortem pH decline are dependent on the rate of muscle ATP utilization and the rate and extent of muscle glycogenolysis.

Muscle Water Holding Capacity

As discussed earlier, muscle contains approximately 75% water and the ability of the muscle to retain this water as it is converted to meat is one of the major determinants of meat quality. Myofibrillar proteins constitute over 50% of the protein of the muscle cell and occupy 85% of the volume of the fiber (Wismer-Pederson, 1987). Thus, due to their three dimensional structure, myofibrils play a large role in the binding of water in muscle. The water in muscle can be classified into one of three categories: 1) that which is tightly bound to other molecules, especially proteins; 2) that which is loosely associated with charged molecules; and 3) that which is in the extracellular space (Kaufman et al., 1986). Because water is a polar molecule, electrical charge plays a large role in it's behavior. The first two categories can be

sub-divided to form three layers around the protein molecule (Wismer-Pederson, 1987). The first of these is the primary hydration shell, this water is tightly bound to the charged amino acid end-groups of the primary muscle proteins actin and myosin, which contain a high proportion of acidic and basic amino acids. The water in this shell is tightly bound due to protein-charge-group/dipole interaction. The next layer is the secondary hydration shell in which dipole/dipole interaction of water is the major determinant of structure. As we move even further from the protein charge group, dipole-dipole interactions begin to break down and water becomes electrically unperturbed and is free to move about within the cell. In pre-rigor muscle, the majority of this "free" water is immobilized within the highly organized three dimensional myofibrillar matrix of the sarcomere and is therefore unable to escape as drip loss (Offer and Trinick, 1983). However the size of the myofibrillar matrix is highly dependent on pH and decreases substantially with the onset of rigor. The isoelectric point of the proteins in the myofibril averages about 5.0 to 5.1 (actin 4.7 and myosin 5.4). When the pH of the muscle is above or below this value there is a resultant charge associated with the proteins and the size of the myofibrillar matrix increases due to charge repulsion between the myofilament proteins. However, as the pH approaches the isoelectric point this charge repulsion decreases, myofilament space decreases and water must move into the sarcoplasm. This increase in water content in the sarcoplasm results in decreased osmolality and therefore decreased water holding (Offer and Trinick, 1983; Wismer-Pederson, 1987).

Rigor-mortis is the process in which permanent cross bridges of actin and myosin are formed due to depletion or cellular ATP levels (Lawrie, 1985). With the onset of rigor, the

myofibrillar volume shrinks and forces water out of the myofibril into the cytoplasm. This phenomenon is similar to that which occurs when muscle pH reaches the isoelectric point of the myofibrillar proteins, and water holding capacity decreases due to decreased myofibrillar space.

In addition to the effects of pH on myofibrillar space, the combination of high temperature and low pH can cause a decrease in water holding capacity. The decease is mainly the result of denaturation of myosin which decreases myofibrillar matrix size and decreases water immobilization (Offer and Knight, 1988). It appears that myosin denaturation is largely a result of the combination of high temperature and low pH, since the water holding capacity of normal muscle can be decreased simply by allowing it to go through rigor at a high temperature (Bendal and Wismer-Pedersen, 1962).

Meat Color

Color perception. In order to fully discuss factors which affect meat color it is important to first understand what color is. In a purely physical sense, color is an imbalance of visible radiant energy reaching the eye from a light source which is reflected by an object, in this case meat. However, it is the physiological transformation of light energy to chemical energy by the eye and the psychological processing of this chemical signal by the brain that allows perception of color by the consumer who then determines the acceptability of the product. Thus, perceived color is the brain's processed image of the various wavelengths of light that the eye receives. The perception of meat color can be influenced by several chemical (i.e. pigment) and physical (i.e. structure, moisture) aspects which are intrinsic components of

the meat. In addition, extrinsic environmental influences such as light sources and packaging color can influence the perceived color of meat. In an experimental setting, we try to limit extrinsic influences on meat color or most appropriately use some instrumental method to quantify color. However, mechanical methods of color assessment are only useful if they can be put into a scale which relates the value to some psychological perception of color by the consumer.

The main chemical factors associated with color in meat is the pigment myoglobin. Myoglobin can exist in several chemical states resulting from differences in the oxidation state of the Fe center and/or the state of the protein moiety and color varies depending on the chemical state (Lawrie, 1985; Fox, 1987). Physical differences in the structure of meat, such as the amount of moisture on the surface of the cut or the relative tightness of packing of the individual muscle fibers affect the amount and wavelengths of light that are reflected and therefore affect the energy of the light waves that the eye receives and the color that the mind perceives. Very tightly packed fibers result in more light being absorbed and perception of darker color, whereas fibers that are more loosely packed scatter more light and therefore appear lighter in color. Water on the cut surface results in more light scattering and therefore a perceived lighter color.

Color measurement. Color measurement of meat requires some detector of the energy of light reflected from a cut, this can either be the human eye or some electronic instrument. The American Meat Science association has developed extensive guidelines for measuring meat color (Hunt et al., 1991). Color has three definable dimensions, hue, value and chroma. Hue is

the color name and separates color families (i.e. red, blue, green etc.). Value is concerned with the relative lightness or darkness of the color. Chroma is the strength of color (i.e. strong or weak) (Hunt et al., 1991). Instrumental measurements of color utilize one of many color evaluation systems to convert hue, value and chroma to mathematical formulas which allows color to be plotted in a three dimensional solid with three coordinates. The relative amounts of each wavelength of light reflected are measured and, based on the formulas, plotted as a point in this solid. The most famous evaluation system is that of A.H. Munsell and was developed in 1905. This original system is the bases for other systems such as the CIE system and HunterLab system. The Hunter system has L, a, and b values. The L value can range from 0 (100% black) to 100 (100% white). The A values can be negative (green) or positive (red). And finally, b values can be negative (blue) or positive (yellow). These values give a relative measure of darkness and redness of the meat.

Etiology of Quality Defects

There are two main quality defects which are currently under extensive study and can affect pork muscle color and water holding capacity; pale, soft and exudative pork and dark, firm and dry pork. Understanding the metabolic basis of these defects in essential for their prevention.

Pale, Soft and Exudative Pork. Pale, soft and exudative pork, as the name implies, is a condition in which meat color is very light, texture is soft and water holding capacity is decreased. The cause of this disorder is a rapid, uncontrolled rate of post-mortem glycolysis which results in a rapid build-up of lactic acid and an abnormally low pH while muscle

temperature is still high (Offer and Knight, 1988). Low pH and warm temperature result in

precipitation of sarcoplasmic proteins and denaturation of myosin which results in pale color

and increased drip loss. As discussed earlier, glycolysis is regulated primarily by the ATP/ADP

+ P_i ratio. The uncontrolled rate of glycolysis that is observed in pale, soft and exudative pork

results from uncontrolled consumption of ATP by the cell. As the cell attempts to maintain

energy balance, the breakdown of glycogen increases which provides a large amount of

substrate for glycolysis. This leads to a large buildup of ADP and P_i which further activates

glycolysis.

Pale, soft and exudative pork can result from two main physiological irregularities; a

genetic defect in the ryanodine receptor also called the calcium channel protein of the

sarcoplasmic reticulum or excessive pre-slaughter stress. The genetic condition, referred to as

porcine stress syndrome or malignant hyperthermia, was first described by Topel et al. (1968)

and in the live animal consists of hypercontractility of muscles, hypermetabolism,

hyperthermia and acidosis. This disorder renders pigs extremely susceptible to stress or

exercise and results from hypersensitivity of the calcium channel protein to stimulators of

Ca^{2+} release and hyposensitivity to inhibitors of Ca^{2+} release (O'Brien , 1986). The defect

arises from a single base substitution (C for T) at base pair 1843 (Otsu et al., 1991) and is

inherited in a autosomal recessive manner (Christian, 1990). This disorder results in a buildup

of Ca^{2-} in the sarcoplasm of the muscle in response to the stress of transport, fighting and

stunning. This buildup of Ca^{2-} acts through troponin to activate actin-myosin crossbridge

formation and muscle contraction which utilizes ATP and decreases the local ATP/(ADP+ P_i)

ratio (Cheah and Cheah, 1976). As a result, glycogenolysis increases, glycolytic rate becomes

uncontrolled and pH drops to extremely low levels (5.5-5.8) within 45 minutes post-mortem,

while body temperature is still high (Christian and Rothschild, 1981). As discussed earlier, low

pH and high temperature result in myosin denaturation, movement of water into the

sarcoplasm and eventually out of the fiber which decreases water holding capacity. The low

pH also is responsible for the pale color of affected muscle. As water moves out of the fiber to

the intracellular space, fibers become less tightly packed and therefore scatter more light

(Lawrie, 1985). In addition, low pH causes increased oxidation of myoglobin to

metmyoglobin and decreases the natural red pigmentation of the muscle. A genetic blood test

has now been developed which allows for detection of pigs with this genetic defect, thus it is

possible to eliminate this gene from pig breeding. However, this will not eliminate the problem

of pale, soft and exudative pork. Excessive pre-slaughter stress in pigs without the ryanodine

receptor defect can result in a similar disorder of meat quality, through a slightly different

mechanism. It is thought that excessive stress causes the disorder primarily through the

somatomotor and sympathetic nervous systems (Gregory, 1990). When an animal is subjected

to stress there is a wide variety of hormonal and physiological responses of which release of

catecholamines is one of the most important. Binding of catecholamines to muscle cell surface

receptors can increase responsiveness of the cells to somatomotor nerve signal, increase

muscle contraction, increase glycogenolysis, increase glycolytic rate and ultimately result in

rapid pH decline. The low pH in turn results in decreased water holding capacity and pale

color.

Dark, Firm and Dry Pork. As the name implies, dark, firm and dry pork is very dark in color,

firm in texture and dry to the touch. The conditions results from a very high (>6.0) ultimate

pH as a result of low muscle glycogen reserves at death, usually due to stress or exercise that

occurs some period before slaughter. The timing of this stress is such that it allows the animal

to recover from the acidosis, but not time for glycogen stores to be replenished (Lawrie,

1985). The dark color results from myoglobin remaining in its native dark purple state due to

the abnormally high pH. The water holding capacity of this high pH meat is very good, thus,

little water comes to the cut surface resulting in a dry look. This condition results in increased

tenderness, due to the fact that the pH of the meat remains high and within the optimal range

of activity of the main tenderizing enzymes, the calpains (Robson et al., 1991). Although the

water holding capacity and tenderness of the meat is affected positively in this condition, the

dark color and decreased shelf life that results from the high pH make this condition

unattractive to the consumer and detrimental to the retailer. This condition is rare in pork in

comparison with pale, soft and exudative pork, however, it remains a problem in beef.

Physiology Of Phosphorus Metabolism

Phosphorus is an element that has a wide variety of biological functions in the growth

and development of animals. The most well researched role of phosphorus is as a component

of the hydroxyapatite crystal of bone. Skeletal development is extremely important in the

growth, development and longevity of swine. In muscle tissue, phosphorus is involved in some

form in nearly every metabolic reaction. It is a component of high energy phosphate

compounds, which are the currencies of energy exchange and phospholipids which enclose

and compartmentalize muscle cells. However, the roles of phosphorus in muscle tissue accretion remain relatively unexplored. A discussion of phosphorus homeostasis, and the cellular roles of phosphorus follows.

Phosphorus Homeostasis

Phosphorus retention in the body is very highly regulated in order to maintain phosphorus homeostasis. Homeostasis is regulated through independent regulation at three main sites; regulation of absorption from the intestine, regulation of excretion in the kidney, and regulation of mobilization/storage of labile body reserves, which are found predominately in the bone. It is these systems that enable an animal to maintain circulating inorganic phosphorus (P_i) levels in a fairly narrow range (3 to 5 mEq/l) through a variety of metabolic situations (Hannon et al., 1990). Circulating levels of P_i are the main controller of P_i partitioning in the body, so it is important that these levels are maintained at a constant level (Murer et al., 1991).

Intestinal Absorption

Dietary phosphorus can be provided either in the inorganic form or as one of several organophosphates such as phosphosugars, phosphorylated amino acids, phosphonucleotides, and phosphoinositols. The phosphate ions of all but the phosphoinositol compounds is cleaved by the enzyme alkaline phosphatase at the brush border membrane and absorbed as P_i (Wilkinson, 1976; Jongbloed, 1987). In plant material, the predominant form of phosphorus is as a component of inositol hexaphosphate (phytate) (Lolas et al., 1976). This form is largely unavailable to the animal, although there are low levels of a phytase enzyme in the brush

border membrane of the enterocyte which can cleave the phosphate ions from phytase to yield

P_i (Moore and Veum, 1983). The primary site of P_i absorption is in the proximal half the small

intestine (duodenum and jejunum). The mechanism of transport into the enterocyte in-vitro is

through passive diffusion and/or sodium gradient driven co-transport. However, at the pH of

the intestinal tract, the transmembrane electrical potential difference of the enterocyte prevents

diffusion of P_i at the concentrations which are commonly found in the intestinal tract

(Borowitz and Granrud, 1992). Thus, in-vivo, transport is almost entirely dependent on

Na^+/HPO_4^{2-} coupled co-transport at the ratio of one HPO_4^{2-} per two Na^+. Intra-enterocyte Na^-

concentration is maintained at a low level by a basolateral Na^+/K^+ antiport system (Borowitz

and Granrud, 1992). This allows movement of Na^+ from the lumen into the enterocyte down a

concentration gradient, thus, pulling in HPO_4^{2-} (Jongbloed, 1987). Absorbed HPO_4^{2-} moves

across the basolateral membrane and into circulation through passive and/or facilitated

diffusion.

Intestinal absorption of P_i is regulated through the action of

1,25-dihydroxycholecalciferol, the active form of vitamin D (Lee et al., 1986a, Bushinsky et

al., 1989). Vitamin D_3 or cholecalciferol can either be absorbed from the intestine or

synthesized from $\Delta^{5,7}$ cholesterol though the action of ultraviolet light in the skin (Jongbloed,

1987). Cholecalciferol is then converted by the liver enzyme 25-hydroxylase to

25-hydroxycholecalciferal. In a normal situation, 25-hydroxycholecalciferol is either stored or

converted by the kidney to 24,25-dihydroxycholecalciferol, the inactive form of vitamin D,

and excreted via the bile. However, under conditions of low plasma P_i or through the action of

parathyroid hormone, which is secreted in response to low circulating levels of calcium, 25-hydroxycholecalciferol is converted to 1,25-dihydroxycholecalciferol through the action of 25-hydroxycholecalciferol- 1α-hydroxylase in the kidney. The formation of 1,25-dihydroxycholecalciferol stimulates intestinal absorption of P_i by increasing the activity of the Na^+ dependent co-transporter (Lee et al., 1986a; 1986b). Quamme (1985) reported that dietary P_i restriction leads to an increase in the V_{max} of the co-transporter, with no effect on the K_m. The author speculated that this response was due to increases in the number of transporters in the enterocyte. Vitamin D has been shown to exert a direct effect at the nucleus which increases DNA translation and leads to increased protein synthesis. Actinomycin D, an inhibitor of protein synthesis, blocks the action of vitamin D on intestinal P_i transport. Vitamin D has been hypothesized to increase synthesis of the Na^+/HPO_4^{2-} co-transporter in the intestine. In addition to it's effect on phosphorus transport, vitamin D may also increase P_i absorption by increasing the activity of intestinal alkaline phosphatase and phytase activity, thus increasing the amount of P_i available for transport (Jongbloed, 1987). However, this seems unlikely since vitamin D deficiency had no effect on alkaline phosphatase or phytase activity in pigs (Fontaine et al., 1985) and P_i deficiency had no effect on the activities of these enzymes in mice (Moore and Veum, 1983).

In summary, phosphorus is absorbed in the form of P_i from the proximal half of the small intestine. The proportion of the total phosphorus that is absorbed is primarily a function of the amount of phosphorus that is in the form of phytate, which is indigestible by pigs.

Transport into the enterocyte occurs through a Na^-/HPO_4^{2-} dependent co-transporter and the activity of this transporter is regulated by 1,25-dihydroxycholecalciferol.

Renal Excretion

Excretion of P_i occurs almost exclusively through the filtering action of the kidney. As blood flows through the nepheron, nearly all P_i contained is filtered and the amount of phosphate excreted is solely a function tubule reabsorption (Dousa et al., 1980). The mechanism of phosphorus reabsorption has been extensively investigated and occurs through the action of a Na^+ dependent-HPO_4^{2-} co-transport at the ratio of 1 HPO_4^{2-} per 2 Na^+ in a manner similar to that occurring in the intestine (Dousa et al., 1980; Murer et al., 1991). This transporter is located in the brush border of the proximal tubule and is distributed mainly in the proximal convoluted and proximal straight segments of the nephron (Dennis et al., 1980).

Normally, approximately 80% of filtered P_i is reabsorbed by the proximal tubule (Murer et al., 1991). However, the level of reabsorption is highly regulated to maintain circulating P_i levels under a wide variety of physiological conditions. P_i reabsorption largely is controlled by circulating P_i levels, and the release of parathyroid hormone (Murer et al., 1991).

Serum P_i concentration, in the absence of increased parathyroid hormone levels, largely reflects dietary phosphorus intake. In animals with low phosphorus intakes, tubular reabsorption of P_i is increased, but when excess phosphorus is provided, reabsorption decreases (Murer et al., 1991). The increase in reabsorption is dependent on the action of vitamin D. Actinomycin D, an inhibitor of protein synthesis, blocks the increase in Na^+/HPO_4^{2-}

co-transport induced by vitamin D (Dousa et al., 1980). In addition, the K_m for Na^+/HPO_4^{2-} co-transport is not affected by P_i deprivation, while V_{max} is increased. Thus, it appears that the adaptive increase in P_i resorption is a result of an increased number of Na^+/HPO_4^{2-} co-transporter proteins (Murer et al., 1991). Additional support is provided by the fact that mRNA for the Na^+/PO_4^{2-} co-transporter is increased in phosphorus deficient rats (Werner et al., 1993).

Parathyroid hormone is a protein that is released from the parathyroid gland in response to low circulating levels of Ca^{2+}. In the kidney, parathyroid hormone results in greater reabsorption of Ca^{2+} and lower reabsorption of P_i, primarily through endocytosis and degradation of Na^+/HPO_4^{2-} co-transporter proteins and a resultant reduction in the V_{max} of Na^+/HPO_4^{2-} co-transport (Murer et al., 1991). Thus, when circulating levels of P_i are adequate but Ca^{2+} levels are low, parathyroid hormone acts to increase serum Ca^{2+} levels without affecting P_i levels. However, the effect of parathyroid hormone on renal P_i reabsorption can be overridden if circulating levels of both P_i and Ca^{2+} are low (Bern et al., 1988; Murer et al., 1991). The mechanism of this decrease in parathyroid hormone action is unknown.

The role of calcitonin in renal regulation of P_i excretion is an area of some confusion. Calcitonin is a peptide hormone produced in the thyroid gland. The main role of the hormone is to decrease osteoclastic release of Ca^{2+} and P_i from bone. It does appear that calcitonin decreases tubular reabsorption of P_i, however, the response is not as marked as the response to parathyroid hormone (Zalups and Knox, 1983; Yusufi et al., 1987; Lausson et al., 1990).

In summary, the kidney is the main regulator of P_i excretion. Excretion is regulated primarily through the action of circulating P_i levels and parathyroid hormone on the $Na^+HPO_4^{2-}$ co-transporter protein. Low circulating levels of P_i increase tubular reabsorption and decrease urinary excretion, while high circulating levels of P_i and parathyroid hormone decrease tubular reabsorption and increase urinary excretion.

Storage/Mobilization of Body Reserves

The only depot of phosphorus in the body that has been demonstrated to be under homeorhetic control is bone phosphorus. The primary function of bone is to provide structural integrity to the animal and to support the musculature of the animal. However, bone also plays a key role in Ca^{2+} and P_i homeostasis by acting as a highly regulated, readily mobilizable reservoir for these key minerals, at least in short term metabolic disturbances.

Bone in its simplest sense is inorganic salt crystals imbedded in organic fibers of collagen (Lovridge et al., 1992). The crystal is basically equivalent to hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$, with some addition of carbonate, citrate, fluoride, magnesium and sodium (Fleishch, 1980). Initial bone formation and crystallization in long bones occurs at the cartilaginous growth plate through the action of chondrocytes. After initial deposition of a core of mineralized cartilage, bone growth occurs through the action of osteoblasts which are members of the same fibroblast-like cell family as chondrocytes (Loveridge et al., 1992). Deposition of the apatite crystal is the result of complex physiochemical reactions that are basically non-enzymatic. The collagen framework or osteoid of the bone must first be laid down by chondrocytes at the growth plate or osteoblasts in growing bone (Ali, 1991). The

collagen is synthesized in the cell as insoluble protein monomers and exported to the matrix

where they aggregate to form insoluble collagen fibrils (Ali, 1991). After deposition of the

collagen framework, the chondrocyte or osteoblast stimulate local increases in P_i and Ca^{2+}

concentration, crystal nucleation and finally crystal growth in a manner that is as of yet not

well understood. The action of the chondrocyte and osteoblast in crystal formation and

growth appear to occur mainly through the action of extracelluar matrix vesicles which are

synthesized by the cells and move into the matrix. These vesicles appear to be involved in

formation of the initial mineralization foci, independent of the collagen fibril, and the crystals

then coalesce and grow to form fully calcified collagenous matrix (Ali, 1991). It is at the

stage of crystal growth that mineralization ceases when mineral intakes are inadequate,

resulting in the bone disorder osteomalacia. When mineral intakes are not adequate, the

collagenous matrix is normal, indicating normal osteoblastic activity, however, the matrix is

not crystallized due to inadequate quantities of Ca^{2+} or P_i (Ali, 1991).

After mineralization is complete, bone is by no means static. Turnover is constantly

occurring through the combined action of osteoblasts and the cells responsible for bone

resorption, osteoclasts (Price and Russell, 1991). The osteoclast is a product of fusion of

haematopoetic stem cells and when activated results in dissolution of the apatite crystal and

digestion of the organic matrix of bone. Bone resorption occurs at the junction of the

osteoclast and bone, in a portion of the cell called the clear zone. The clear zone is a

microenvironment established by the osteoclast which acts to dissolve the bone mineral crystal

(Teti et al., 1991). Dissolution of the mineral is a result of the action of cellular carbonic

anhydrase and a Na+/H+ ATPase protein which pumps protons into the clear zone and lowers

the pH in the microenvironment. After dissolution of the crystal, osteoclasts release lysosomal

proteinases which digest the organic matrix (Zaidi et al., 1993). Osteoblasts then move into

the areas cleared by the osteoclasts, reform the osteoid and initiate remineralization. This

constant remodeling allows for repair and rebuilding of the skeleton and is referred to as

coupling (Price and Russell, 1991).

When the rate of resorption outpaces the rate of formation, bone loss and skeletal

pathology occur. The rate of osteoclastic activity is highly regulated and responsive to

homeostatic changes in Ca^{2+} and P_i metabolism in the body. Although the osteoclast is the cell

responsible for bone resorption, the osteoblast is intimately involved in the process. In in-vitro

cell culture systems, bone resorption will not occur unless osteoblasts are present (Price and

Russell, 1991). The main regulation of osteoclastic bone resorption is via hormonal control by

parathyroid hormone, 1,25-dihydroxycholecalciferol, and calcitonin and also via

responsiveness to local Ca^{2+} concentrations. Of these regulatory factors, only

1,25-dihydroxycholecalciferol is responsive to circulating P_i levels (Zaidi et al., 1993). It has

been demonstrated that the osteoclasts have no receptors for parathyroid hormone or

1,25-dihydroxycholecalciferol, the main stimulators of bone resorption. It is believed that the

action of these hormones on bone resorption are mediated by osteoblasts which have

receptors and the signal is then passed to the osteoclasts through paracrine factors (Loveridge

et al., 1991; Price and Russell, 1991; Zaidi et al., 1993). The local regulation of osteoclasts by

osteoblasts is not well understood. Two mechanisms of regulation have been postulated;

regulation through a local signaling compound termed osteoclast stimulating factor, or through production of collagenase which digests the un-mineralized osteoid, exposing the mineralized matrix which is essential for osteoblast attack (Loveridge et al., 1992; Price and Russell, 1991; Zaidi et al., 1993). Calcitonin and high local concentrations of Ca^{2+} act directly on the osteoclast to decrease bone resorption (Zaidi et al., 1993). Both Calcitonin and high local levels of Ca^{2+} result in immediate retraction of the clear zone and a decrease in cell motility and bone resorptive activity.

Thus, several of the main regulators of bone resorption (parathyroid hormone, calcitonin and local Ca^{2+} concentrations are responsive exclusively to circulating Ca^{2+} levels. Only 1,25-dihydroxycholecalciferol is responsive to circulating levels of P_i.

Metabolic Roles of Phosphorus

Phosphorus is widely distributed in the body and is involved in numerous metabolic functions. Approximately 75-80% of the phosphorus in the body is contained in calcified tissue such as bone and teeth. The remaining 20-25% is widely distributed in the soft tissue of the body in a variety of forms. Behind bone, muscle contains the second highest concentration of phosphorus of any tissue in the body (Nielsen, 1973). Thus, phosphorus appears to be an important component in muscle growth. Following is a discussion of the metabolic roles of phosphorus in the body and potential biological impact of inadequate dietary intakes of phosphorus in mammals and birds.

Hydroxyappitite Crystal

The phosphorus present in bone is predominately in the form of hydroxyappitite, $Ca_{10}(PO_4)_6(OH)_2$, an inorganic salt crystal that is imbedded in the organic protein matrix of the bone (Fleisch, 1980). Phosphorus is a critical component of this crystal and providing a diet containing 33% of the required level of phosphorus results in a decrease in the rate of mineralization and matrix formation in rats as indicated by increased osteoid thickness and decreased incorporation of tetracycline into bone (Baylink et al., 1971). Tetracyline can be used as a tracer to determine bone development. In addition, bone resorption rate increased by as much as 122% compared with rats provided adequate dietary phosphorus. However, this increase in resorption and decrease in mineralization was unable to totally offset the decrease in phosphorus absorption as evidenced by moderate to severe hypophosphotemia in these animals. Without adequate dietary phosphorus, even if Ca is adequate, normal bone mineral deposition is unable to occur as evidenced by decreased bone breaking strength, decreased bone ash content and similar bone Ca/P ratios in animals fed inadequate phosphorus (van Kempen et al., 1976; Henry et al., 1979; Howe and Beecher, 1983; Schandler et al., 1991).

High Energy Phosphate Compounds

As previously discussed, the currencies of energy exchange in biologic systems are high energy phosphate compounds such as adenine nucleotides and creatine phosphate. These compounds trap energy liberated by the catabolism of nutrients in the cell and allow this energy to be used in useful metabolic processes. Proper energy charge is essential for maintenance of cellular ion gradients, synthesis of macromolecules and virtually all other

cellular processes. The majority of ATP synthesized in resting muscle cells occurs through oxidative phosphorylation via the electron transport chain of the mitochondria. In this system, electrons are transferred from NADH and $FADH_2$, obtained via glycolysis and the Krebs cycle, to various carrier proteins made up primarily of cytochromes. As these electrons move down the chain to the final electron acceptor (O_2), protons are pumped to the intramembrane space and a proton gradient is established. Thus, the energy released from biological fuels when they are catabolized via glycolysis and the Krebs cycle is converted to the potential energy of a proton gradient. As these protons are allowed to flow back into the mitochondria through a specialized intra-membrane protein called ATP synthase, the potential energy is captured through the synthesis of ATP from ADP and P_i. This energy is then transferred throughout the cell via the action of the creatine phosphate shuttle (Bessman and Carpenter, 1985). Thus, an adequate intra-mitochondrial P_i level is necessary for proper coupling of electron flow with ATP synthesis.

In muscle cells there are three main functions which utilize ATP; 1) maintenance of ion gradient, 2) contractile activity, and 3) synthesis of macromolecules (i.e. cellular enzymes, structural lipids and myofibrillar proteins). In general, available cellular energy is partitioned first towards maintaining cell homeostasis and intracellular ion gradients. After these needs are met, excess energy is then used for cellular functions and synthesis of macromolecules.

Muscle weakness, creatinureia and rhabdomylosis are common symptoms of phosphorus depletion and hypophosphatemia (Fuller et al., 1976). Several authors have reported that phosphorus depletion and hypophosphatemia result in decreased muscle Pi

concentration, decreased mitochondrial and total creatine phosphokinase activity and decreased mitochondrial oxygen consumption (Fuller et al., 1976; Hettleman et al., 1983; Brautbar and Massry, 1984). These data suggest that pathways for energy metabolism are impaired in moderate to severe phosphorus deficiency due to inactivation of mitochondrial enzymes. In addition, phosphorus deficiency also appears to result in decreased insulin sensitivity and decreased ability of muscle to take up and utilize glucose, a major substrate of energy metabolism (Davis et al., 1979; de Venanzi et al., 1987). Phosphorus deficiency in rats results in decreased muscle levels of glucose 6-phosphate and glycogen (Brautbar and Massry, 1984).

The decreased insulin sensitivity and decreased concentrations of glucose 6-phosphate and glycogen observed in muscle of animals consuming phosphorus deficient diets could potentially impact postmortem muscle quality. As discussed in the previous section on meat quality, muscle glycogen is the primary fuel for glycolysis in postmortem muscle which results in a buildup of lactic acid and decreased pH. Lower premortem levels of glycogen would result in less potential substrate for glycolysis and lower glycolytic potential. Thus, premortem phosphorus deficiency may result in high postmortem muscle pH and therefore, increased water holding capacity.

In summary, phosphorus is a major component of the currencies of energy exchange (adenine nucleotides and creatine phosphate). Phosphate depletion and hypophosphatemia result in anomalies of energy metabolism in skeletal muscle due to decreased activities of the enzymes of energy transfer and decreased availability and utilization of energy containing

substrates. These defects may result in profound effects on muscle growth and physiology in the live animal and postmortem meat quality, including decreased muscle growth, decreased protein deposition and high ultimate pH in postmortem muscle.

Phospholipids

Phospholipids are the main components of the membranes that enclose and compartmentalize cells. These membranes enable the cell to form micro-environments, maintain ion gradients for cellular regulation and action potential formation and allow for selective permeability of macromolecules and ions. Phospholipids are composed of a hydrophilic head connected by a phosphoester bond to long hydrophobic acyl groups. The amphipathic nature of phospholipids results in formation of a bilayer with a hydrophobic center region and hydrophilic exterior. This bilayer is essential for proper cell function. Phosphate forms the link between the hydrophobic diacylglyceride and the polar head group which can be one of several substances, most commonly choline and ethanolamines. Phospholipids are synthesized from glycerol 3-phosphate or dihydroxyacetone phosphate and two fatty acids to form 1,2 diacyl glycerol. This compound is then combined with a charged polar group such as CDP-ethanolamine or CDP-choline to form the final phospholipid. Thus, phosphorus is required for the synthesis of phospholipids as well as being an integral component .

Several lines of evidence point towards derangements of phospholipid synthesis and therefore membrane integrity in phosphate deficiency. In rats, phosphorus deficiency results in decreased muscle concentrations of the precursors of phospholipid synthesis, glycerol

3-phosphate and cytidine triphosphate (Brautbar and Massry, 1984). In addition, the total

concentration of phospholipids decreases and the ratio of individual phospholipids changes in

sarcoplasmic reticulum membranes and total muscle membranes (Kreusser et al., 1980;

Brautbar and Massry, 1984). The changes in the ratio of phospholipids in the membranes may

be mediated through the increase in levels of vitamin D resulting from phosphorus deficiency.

Vitamin D treatment of vitamin D deficient rats results in increased intestinal membrane

concentrations of phosphatidyl-choline while phospatidyl-ethanolamine remains constant

(Rasmussen et al., 1981). In addition, treatment of cultured myoblasts with vitamin D results

in increased concentrations of phosphatidyl-choline, mainly at the expense of decreased

phosphatidyl-ethanolamine (Bellido et al., 1987; Drittanti et al., 1988). Further evidence for

membrane derangement in phosphorus deficiency is provided by the observation that resting

membrane potential is decreased in the muscle of phosphate depleted dogs (Fuller et al.,

1976). This results mainly from increased intracellular concentrations of Na^+ and Cl^-.

The phospholipid environment of the cell membrane is also important in controlling the

function of numerous intra-membrane proteins (Knowles et al., 1976; Swoboda et al., 1978;

Bennett et al., 1980). Phosphorus deficiency may therefore result in decreased water holding

capacity due to derangements of the function of intra-membrane proteins which control

sarcoplasmic Ca^{2+} concentration as well as membrane integrity. As previously noted,

cytoplasmic Ca^{2+} concentration is the main controller of myofibrillar ATPase activity and

therefore regulates the rate of postmortem glycolysis. The level of cytoplasmic Ca^{2+} is

regulated by the sarcoplasmic reticulum (SR) through the combined action of the calcium

channel protein and the Ca^{2+} ATPase protein located in the SR membrane. In the presence of excess cytoplasmic Ca^{2+}, the Ca^{2+} ATPase is activated and pumps Ca^{2+} into the SR for storage (Bennet et al., 1980). Sarcoplasmic Reticular membrane phospholipid environment plays a large role in the Ca^{2+} pumping activity of the SR Ca^{2+} ATPase activity is reduced when the SR is manually delipidated. A minimum of 35 molecules of phospholipid per ATPase molecule appear to be necessary for Ca^{2+} ATPase Ca^{2+} pumping activity (Hesketh et al., 1976). If membrane phospholipid levels drop below 35 molecules per ATPase, it is hypothesize that the tertiary structure of the Ca^{2+} ATPase protein is disrupted and thus, Ca^{2+} pumping activity decreases. Because a phosphorus deficiency leads to decreased membrane phospholipid concentration in the SR and total muscle cell membranes, the Ca^{2+} pumping activity of SR may be adversely affected leading to a buildup of cytosolic Ca^{2+}, rapid postmortem glycolysis and development of the PSE condition.

In summary, phosphorus plays a major role in the synthesis and structure of phospholipids, the main component of cellular membranes which enclose and compartmentalize cells. Phosphorus deficiency results in decreased concentrations of substrates involved in phospholipid synthesis, decreased membrane phospholipid concentrations, and changes in the relative ratio of individual phospholipids in skeletal muscle. This appears to result in decreased membrane integrity and resting muscle membrane potential in phosphorus deficient animals. The phospholipid environment is very important to the proper functioning of one of the main regulators of cytoplasmic Ca^{2+} concentration, the sarcoplasmic Ca^{2+} ATPase protein. This derangement in membrane structure and function may result in

decreased water holding capacity in post-mortem muscle resulting from pre-mortem

phosphorus deficiency.

Other Functions

Phosphorus is also a component of other important compounds. The purine and

pyrimidine bases which make up DNA and RNA, the nucleic acids which carry the genetic

blueprint of the body and code for body proteins, are linked via phosphate bonds. Synthesis of

DNA and RNA is essential for cell proliferation and macromolecule synthesis. No information

is available on the impact of phosphorus deficiency on nucleic acid synthesis. However, it is

possible that cell proliferation and RNA synthesis may be limited in severe phosphorus

deficiency.

Phosphoproteins are widespread in the body. Protein phosphorylation plays a major

role in regulation of enzymatic and hormonal signal transduction pathways. The role of

phosphorylation in myofibrillar growth and assembly is currently unknown. The effect that

phosphate deficiency has on the amount of protein phosphorylation and what effect this may

have on growth and development is as of yet undetermined.

Role Of Phosphorus In Animal Growth

Impact of Phosphorus on Body and Tissue Growth

Muscle contains a large percentage of the phosphorus in the body. Low intakes of

phosphorus result in decreased growth rate and efficiency of feed conversion in growing and

finishing pigs (Cromwell et al., 1970; Cromwell et al., 1972; Thomas and Kornegay, 1981). In

addition, the percent lean cuts and loin eye area were decreased in pigs consuming diets

deficient in phosphorus (Cromwell et al., 1970; Cromwell et al., 1972). Nitrogen retention was increased linearly as phosphorus percentage of the diet increased from .25 to .75% in pigs consuming diets with .75% calcium (Vipperman et al., 1974). This decrease in nitrogen retention may result from decreased muscle deposition. In addition, pigs tend to retain a constant 37 to 39 grams of phosphorus per kilogram of protein accrued (Jongbloed, 1987). Howe and Beecher (1983) reported that muscle weight was decreased in growing rats consuming a diet containing .35% phosphorus compared with rats consuming a diet containing .8% phosphorus. As discussed earlier, phosphorus deficiency results in several derangements of energy metabolism, among them decreased creatine phosphokinase activity in muscle. Treating muscle tissue with a selective inhibitor of creatine phosphokinase, 2,4-dinitrofluorobenzene, results in decreased protein synthesis. This indicates that creatine phosphate may be the direct shuttle of high energy phosphate compounds from the mitochondria to areas of protein synthesis, similar to its role in shuttling energy to the myofibril for contraction. Thus, the decreased creatine phosphokinase activity observed in phosphorus deficiency may be responsible for the decreased muscle growth and nitrogen retention observed.

In summary, phosphorus is important in tissue deposition and muscle contains a large amount of phosphorus. Consuming diets deficient in phosphorus results in decreased growth rate efficiency of feed utilization, nitrogen retention, and muscle growth in growing rats and pigs.

Phosphorus Requirements

The pig's metabolic demand for phosphorus is determined by its needs for maintenance and growth. The needs for growth largely are determined by the types and amounts of phosphorus containing molecules which are deposited. This is a function of the types of tissues that are being deposited. As mentioned earlier, muscle, bone, viscera, and hair contain a relatively high concentration of phosphorus, whereas fat and skin contains minimal amounts (Nielsen, 1973).

The sum of the needs for growth and maintenance must be provided to the animal in a metabolically available form in order to maintain maximal growth rate. The amount of phosphorus which is metabolically available is determined by the efficiency of intestinal digestion and absorption and the efficiency of utilization of this phosphorus for maintenance and growth. These subjects have been extensively reviewed elsewhere by Jongbloed (1987).

The digestibility of phosphorus varies widely among feedstuffs. Inorganic forms of phosphorus tend to be very highly digestible, ranging from 80-100%, while the bioavailability of phosphorus in plant products is substantially lower, ranging from 15-50% (Jongbloed, 1987; NRC, 1988; Cromwell, 1990). A large portion of the phosphorus in plant sources is in the form of phytate which is poorly digested by pigs (Jongbloed, 1987). Phosphorus in wheat and other high fiber cereal grains is more digestible due to the presence of natural phytases, which break down the phytate phosphorus to a form which is digested by pigs (Cromwell et al., 1985). The digestibility of phytate phosphorus has also been reported to be increased by inclusion of bacterially derived phytase in the diet or by wet processing of cereal grains

(Jongbloed, 1987). Thus, the digestibility of phosphorus by pigs is highly dependent on the source of the phosphorus. Greater inclusion of cereal grains and plant protein sources, especially corn and soybean meal, will decrease phosphorus digestibility, whereas inclusion of inorganic phosphorus sources will increase digestibility.

As previously discussed, the retention of absorbed phosphorus is under renal control. When phosphorus intake and/or digestion is limited, circulating levels of 1,25-dihydroxycholecalciferol increase and excretion of phosphorus decreases. Thus, the metabolic efficiency of absorbed phosphorus usage is estimated to be very high (96 to 98%) in an animal when the amount of phosphorus absorbed is at or below the animals daily metabolic demand (Jongbloed, 1987).

The quantity of phosphorus needed daily by the pig for body maintenance is dependent on pig weight and the level of phosphorus intake. Jongbloed (1987) estimated that maintenance needs ranged from .005 to .010 g of P per kg of body weight in pigs consuming deficient and adequate levels of phosphorus, respectively. This appears to be due mainly to increased endogenous losses of phosphorus in pigs consuming high levels of phosphorus. Similar results were reported by Geuguen and Perez (1979).

The quantity of phosphorus required for maximum body growth is highly dependent on the composition of growth. Pigs have been reported to retain 35 to 39 g of phosphorus per kg of body protein accrued (Jongbloed, 1987). Composition of growth is dependent on stage of development, lean growth genotype and gender. As discussed earlier, as an animal matures, the relative rate of deposition of individual tissues changes. Neo-natal growth is

dominated by internal organ and digestive tract growth, followed by bone growth, muscle growth and finally fat growth. Thus, early in development (20 to 60 kg body weight) when the rate of bone and muscle growth is high, the daily need for phosphorus has been estimated to be high (.113 to.085 g per kg of body weight). As animal weight increases (60 to 110 kg) and fat deposition increases, this requirement decreases (.077 to .046 g per kg of body weight) (Jongbloed, 1987).

Factors such as gender and genotype, which affect the rate of lean tissue deposition also affect the metabolic demand for phosphorus. As the rate of lean tissue growth increases, the metabolic demand for phosphorus also increases (Jongbloed, 1987). It has been reported that boars require a 10 to 25% higher concentration of phosphorus than do barrows, with gilts being intermediate (Cromwell et al., 1970; Thomas and Kornegay, 1981; Jongbloed, 1987). Boars and gilts normally have a higher concentration of proteinaceous tissue at a given weight than do barrows. Thus, since pigs reportedly retain a relatively constant amount of phosphorus per kilogram of protein deposited, it would be expected that boars would have a higher metabolic demand for phosphorus (Jongbloed, 1987).

Lean growth genotype can also have a large impact on mature weight and thus, the composition of growth as pigs grow to market weight. This would be expected to have an impact on the metabolic demand for phosphorus. Indeed, Jongbloed (1987) estimated that as the lean growth potential of pigs increased from medium to high, the metabolic demand for phosphorus increased by approximately .2 to .3 g per kg of body weight gained.

In summary the metabolic demand for phosphorus is determined by the demand for maintenance and growth processes. The metabolic demand for body maintenance ranges form .005 to .010 g per kg of body weight. The demand for body growth is determined by the rate and composition of that growth, which is affected by genotype, gender and stage of pig development. On average, pigs retain 4.8 to 5.5 g of phosphorus per kg of body weight gain from 20 to 110 kg and improving lean growth genotype from moderate to high has been estimated to increase this value by .2 to .3 g per kg of body weight gain. Or expressed as retention per kg of protein accrued, pigs apparently retain 35 to 39 g of phosphorus per kg of body protein accrued. Efficiency of utilization of ingested phosphorus is a function of the efficiency of digestion and absorption and the efficiency of metabolic utilization of absorbed phosphorus. Efficiency of digestion is mainly a function of the amount of phosphorus which is supplied by plant sources, of which a majority is in the form of phytate. The bioavailability of plant sources of phosphorus range from 15% (corn) to 50% (wheat) with differences due to the amount of naturally occurring phytase activity. Inorganic sources of phosphorus have much higher bioavialabilities and range from 85% (deflorinated rock phosphate) to 100% (dicalcium phosphate). The metabolic efficiency of utilization of absorbed phosphorus is very high (98-99%) when an animal is at or below its requirement and decreases with increasing intake.

Assessment of Phosphorus Status

In determining an animals response to increasing dietary phosphorus it is important to have some measure of phosphorus status in the animal. Several invasive methods (i.e. bone

breaking strength, bone ash, bone biopsy) have been use extensively to determine the response of an animal to changes in dietary phosphorus. These methods do not allow repeated measures of the phosphorus status of the animal or they disturb the physiological state of the animal due to surgical procedures involved. Furthermore, these methods do not take into account the phosphorus status of tissues other than bone. Because of expected changes in phosphorus needs of an animal during development, a non-invasive method that could evaluate the physiological response of the whole growing animal to varying dietary phosphorus regimens over several stages of pig development would be valuable. Plasma phosphorus concentration has been used as an indicator of the phosphorus status of the animal, but circulating phosphorus levels are under extensive homeostatic control, as was discussed in preceding sections. Thus, it appears that plasma phosphorus is not a good indicator of phosphorus status.

Osteocalcin is a protein produced by osteoblasts which is released into the bloodstream under certain conditions (Price et al., 1980). In properly mineralized bone, osteocalcin is bound to the hydroxyapatite crystal. However, in bones with abnormal mineralization, osteocalcin is released into the blood. Radioimmunoassays for osteocalcin have been validated for human, rat, bovine and to a limited degree for porcine osteocalcin (Carter et al., 1995. Price, 1980) Serum osteocalcin levels have been reported to be elevated when phosphorus deficient diets are consumed by rats and pigs (Carter, 1995; Lian and Glimcher, 1984). In pigs serum osteocalcin concentrations appear to reflect phosphorus needs for bone

growth, more closely than those for overall body growth (Carter et al., 1995). Unfortunately, an antibody for porcine osteocalcin is not commercially available at this time.

Serum alkaline phosphatase activity has been demonstrated to decrease with increasing dietary phosphorus concentration and to plateau as phosphorus intake approaches the animals requirement (Boyd et al., 1983). Little is known about the role that alkaline phosphatase plays in skeletal development or phosphorus homeostasis. It is known that alkaline phosphatase is produced in a number of tissues, both skeletal and non skeletal (Boyd et al., 1983). In pigs, serum alkaline phosphatase was minimized by phosphorus levels which maximized body growth and efficiency of feed utilization while bone strength continued to increase as phosphorus was increased beyond this level (Carter et al., 1995). Thus, serum alkaline phosphatase may represent a good indicator of phosphorus status because of its relationship with phosphorus intake, its relationship with whole body phosphorus status and the fact that it can be easily measured at numerous growth stages in the pig.

Phosphorus Partitioning

In addition to utilizing the existing phosphorus reserves to maintain phosphorus homeostasis when dietary intake of phosphorus is limiting, the body can potentially adjust the rate of use of available phosphorus by decreasing the growth rate of high phosphorus containing tissues such as muscle and bone. The prioritization of use of metabolically available phosphorus is an area of some speculation. Historically, bone has been viewed as a large reservoir of Ca^{2-} and P_i that could be mobilized for support of soft tissue growth during periods of inadequate phosphorus intake. It does appear that when inadequate dietary

phosphorus is provided, bone development suffers at the expense of the phosphorus needs of other tissues (Henry et al., 1979; Schandler et al., 1991). As previously discussed, 20-25% of the phosphorus in the body is found in soft tissues, 17% of this in muscle, 3.5% in viscera, and only .8% in subcutaneous fat (Nielsen, 1973). Only about 1% of the calcium in the body is in soft tissues with 99% being found in calcified tissues. Thus, if the rate of bone growth and mineralization slows down to a greater extent than soft tissue growth, then the Ca/P ratio of the whole body should decrease. Feeding inadequate levels of dietary phosphorus has been reported to result in a decrease in Ca/P ratio of the whole body of approximately 10%, while the Ca/P ratio of bone remains constant, indicating that deposition of soft tissue phosphorus continues at the expense of bone development (Henry et al., 1979; Schandler et al., 1991). However, in the study by Henry et al. (1976) the weight of the animals provided the phosphorus deficient diets was 26% less than those consuming adequate diets. Rymarz et al. (1982) reported that as pig weight increased, Ca/P ratio of the carcass increased. Thus, based on this information, the change in Ca/P ratio in these studies may have resulted from differences in body weight.

In general, when metabolically available nutrient levels are below the animals requirement for maximal growth rate, tissues with a high rate of turnover are affected less detrimentally than slow turnover tissues. Visceral tissue tends to have a higher turnover rate than muscle, with bone being intermediate. Thus, visceral growth would be hypothesized to be reduced less than muscle or bone growth. If visceral tissue continues to be deposited, and muscle, and to a lesser extent, bone deposition decrease, this would result in a decrease in the

whole body Ca/P ratio. In addition, when adequate amounts of dietary calcium are provided, the ratio of Ca/P retained in the body has been reported to actually increase when low phosphorus levels are provided (Vipperman et al., 1974).

The length of time that bone mineral resorption can support growth of other tissues is largely unknown. Feeding low phosphorus diets to pigs has been reported to decrease circulating levels of P_i by 41% after only 8 days (Vipperman et al., 1974). If we assume that a 114 kg pig will have a total of 7 kg of bone (Richmond and Berg, 1971), and further assume that bone contains 3.13% phosphorus (Nielsen, 1973), bone would possess about 219 grams of phosphorus. An animal which was consuming an average of 2.4 kg of a diet containing .1% less available phosphorus than needed at each stage of development from 20 to 114 kg body weight would be deficient 240 grams of phosphorus. Thus, even if the animal were able to mobilize the entire amount of phosphorus stored in bone, it would still have a deficiency of approximately 21 grams of phosphorus if normal growth were maintained. To compensate for such a deficiency, reductions in growth of soft tissues, such as muscle, as well as bone would need to occur. Alterations in tissue growth patterns may be mediated directly by circulating phosphorus, or more likely, through hormonal alterations. Phosphorus deficiency in growing chicks has been shown to result in lower circulating levels of growth hormone and greater pituitary weights (Carew et al., 1984; Parmer et al., 1987). Growth hormone is one of the primary anabolic hormones of muscle and bone growth (Navakofskin and McCusker; Issaksson et al., 1987). A reduction in circulation growth hormone would be expected to result in increased deposition of fat and decreased deposition of muscle and bone. Since fat

contains a low level of phosphorus and muscle and bone a high level of phosphorus, decreased rates of muscle and bone deposition would allow metabolically available phosphorus to be used for other tissues such as viscera, which are more vital to maintaining life functions and have higher turnover rates.

Thus, at this time we are unable to fully ascertain how metabolically available phosphorus is partitioned when supply is limited. However, it is apparent that mobilization of bone reserves of phosphorus in times of dietary deficiency cannot fully offset the requirement for growth without alteration in the rate and composition of growth.

Summary/Objectives

Phosphorus is involved in a wide variety of biological functions. Muscle contains the second highest concentration of phosphorus of tissues in the body behind bone and pigs have been reported to retain a constant amount of phosphorus per kilogram of protein accrued. In addition, phosphorus is involved in a number of physiological processes (i.e. energy metabolism, membrane structure), that affect postmortem muscle quality. Historically bone has been viewed as a large, readily mobilizable depot of phosphorus that could sustain soft tissue deposition for long periods of inadequate dietary intakes. However, it appears that the length of time that bone depots can be mobilized to support soft tissue deposition may be somewhat limited. Despite the fact that phosphorus appears to be highly involved in muscle growth, the impact of varying phosphorus intake on muscle physiology and growth remains lightly investigated. It is therefore hypothesized that: 1) phosphorus deficiency will decrease muscle growth resulting in decreased carcass muscle and increase carcass fat; 2) phosphorus

response will be dependent on rate of lean tissue deposition as influenced by stage of pig

development and lean growth genotype and; 3) phosphorus deficiency will negatively affect

muscle quality due to changes in membrane integrity, functioning of the SR Ca^{2-} ATPase

protein and alteration in energy metabolism pathways. The studies presented in this

dissertation were designed to examine the impact that dietary phosphorus regimen has on

muscle growth and quality in pigs of differing lean growth genotypes.

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CHAPTER 3. IMPACT OF DIETARY PHOSPHORUS REGIMEN ON GROWTH OF PIGS DIFFERING IN LEAN GROWTH CAPACITY[1]

A paper to be published in the *Journal of Animal Science*

M.J. Bertram[2], T.S. Stahly[3,4], and R.C. Ewan[3]

Department of Animal Science, Iowa State University, Ames, 50011

Abstract

A study was conducted to examine the impact of dietary available phosphorus regimen on growth of pigs from two genetic strains differing in lean growth capacity. Seven sets of six littermate barrows from each of a high and moderate lean growth (LG) genotype were allotted within litter to one of six diets differing in dietary available phosphorus (AP) concentration (.080, .110, .155, .222, .323, and .475%). The muscle growth capacity of the high and moderate LG pigs from 20 to 109 kg body weight was estimated to be 375 and 330 g/d, respectively. Pigs were individually penned and were allowed to consume feed and water ad libitum. From 20 to 109 kg, high LG pigs accrued more muscle daily, gained more BW per unit of feed, and produced carcasses with more dissectable muscle and less fatty tissue than moderate LG pigs; whereas daily body weight and protein gains were similar between genotypes. Responses of pigs to dietary AP regimen were influenced by their capacity for proteinaceous tissue growth at various stages of growth. As dietary available phosphorus

[2]Present address, Continental Grain Company, Wayne Feed Division, 1230 Liberty Bank Lane Suite 120, Louisville, KY 40222.
[3]Department of Animal Science, 337 Kildee Hall, Iowa State University, Ames, IA 50011.
[4]To whom correspondences should be addressed.

(AP) concentration declined, the accretion rate of proteinaceous, high phosphorus tissues declined and fatty, low phosphorus containing tissues increased. Specifically, daily accretion rates of body weight, proteinaceous tissues of muscle, bone, and offal and body protein declined quadratically, but the magnitude of the decline in daily body weight and protein gain, and to a lesser extent daily muscle and offal gain, was greater in the high LG genotype. The amount of fatty tissue and ether extractable lipid accrued per unit of muscle and protein accrued, respectively, increased linearly in both genotypes as dietary AP concentrations were lowered. As the pig matured, dietary AP concentrations needed to maximize, gain/feed, and body protein gain decreased from .323 to .475 at BW of 25 kg to about .155% at BW of 97 kg. The growing pig was estimated to require about .067 to .077 g of dietary AP or equivalent amounts of bone P to support each gram of body protein deposited. When AP is limiting, muscle, bone and offal growth are depressed and dietary energy is partitioned toward fatty tissue accretion.

Introduction

Phosphorus is involved in a wide variety of biological functions. Approximately 75 percent of the phosphorus in the body is present in the skeleton, with the remaining 25 percent being distributed in soft tissue in the form of high-energy phosphate compounds, phospholipids, nucleic acids, and phosphoproteins. Muscle contains the second highest concentration of phosphorus of any tissue in the body and 18% of the total phosphorus in the body (Nielsen, 1973). Pigs consuming inadequate amounts of phosphorus have been reported to have decreased growth rate, efficiency of feed utilization and bone breaking strength

(Cromwell, 1970; van Kempen et al., 1976; Jongbloed, 1987). In addition, feeding deficient

levels of phosphorus has been reported to decrease nitrogen retention (Vipperman et al.,

1974; Jongbloed, 1987) and pigs have been reported to retain a constant 34 to 39 g of

phosphorus per kilogram of protein accrued in the body (Jongbloed, 1987). Consumption of

phosphorus deficient diets has been shown to be associated with lower pituitary weights and

circulating levels of growth hormone in chicks (Carew et al., 1985; Parmer et al., 1987). Thus,

it is hypothesized that feeding low levels of phosphorus will result in decreased muscle growth

with a resultant increase in fat deposition. As the genetic capacity for muscle growth

increases, it is expected that phosphorus required for maximal muscle accretion will increase.

The objective of this experiment was to quantify the impact of dietary available

phosphorus regimen on the rate, efficiency, and composition of growth in pigs with high and

moderate genetic capacities for lean tissue growth.

Materials and Methods

Seven sets of six littermate barrows from two each of two genetic strains of pigs

differing in their genetic capacity for lean growth (LG) (initial weight 20 ± 2 kg) were allotted

within litter to diets containing one of six dietary available phosphorus (AP) concentrations

(.080, .110, .155, .222, .323, and .475%). The muscle growth capacities of the pigs fed from

20 to 110 kg was approximately 375 and 330 g /d for the high and moderate LG genotypes,

respectively. Pigs in both genotypes were monitored and determined to be free of antibody

titers for the major antigens in swine populations (*mycoplasma hyopneumoniae*,

Actinobascillus pleuropneumonia, swine influenza virus, and transmissible gastroenteritis)

both at the initiation and conclusion of the trial.

Diets consisted of corn-soybean meal mixtures fortified with minerals, vitamins and an

antibiotic (Table 1). Monocalcium-dicalcium phosphate was used as the supplemental

phosphorus source. A single source of corn, soybean meal, and monocalcium-dicalcium

phosphate were used throughout the trial. The corn, soybean meal and moncalcium-dicalcium

phosphate contained an analyzed (AOAC, 1995) phosphorus content of .18, .67, and 18.6%,

respectively. The bioavailability of the phosphorus was assumed to be 16, 25, and 100%,

respectively, of the analyzed phosphorus content (NRC, 1988). Available phosphorus

concentrations were achieved by altering the ratios of corn starch, monocalcium-dicalcium

phosphate, and calcium carbonate. Diets were formulated so that the highest level of AP

would approach the estimated needs of the high LG genotype at a BW of 20 kg, and the

lowest level would be below the expected requirement of the moderate LG genotype at a BW

of 110 kg. Calcium was maintained at .85% in all diets. Based on analyzed amino acid

contents (ion exchange chromatography after acid hydrolysis as described by Gehrke et al.,

1985) and estimated digestibilities (NRC, 1988) of the the amino acids in each ingredient, the

diets contained 1.15% digestible lysine and digestible tryptophan, threonine, and

methionine-cystine levels were 21, 64 and 63 percent of the digestible lysine level,

respectively. Pigs were allowed to consume feed and water ad libitum.

Pigs were placed on test at an average BW of 20 ± 2 kg. Pigs were

individually-penned in fully slatted, .61 x 2.44 m pens in a temperature controlled building.

The average daily high and low ambient temperature in the building over the duration of the trial were 27 ± 5 °C and 22 ± 3 °C, respectively. Drip coolers were employed when the ambient temperature exceeded 27 °C.

Pig weight and feed consumption were recorded at 7 d intervals. Upon reaching 109 ± 4 kg, pigs were killed at the Iowa State University Meat Lab for subsequent evaluation of body tissue composition. On the morning of slaughter, pigs were stirred approximately 1.5 h before transport to encourage feed and water intake, weighed and gently loaded in groups of 3 to 8. Pigs were transported 5 km (20 ± 2 min) to the meat lab, re-weighed (post transport weight) and then electrically stunned (280 volts for 20 s) at 45 ± 19 min post transport, and killed by exsanguination 45 ± 10 s post-stun. The offal components of head, leaf fat, heart-lungs, liver, kidneys, gastrointestinal tract (with digesta) and jowl trim were removed, weighed individually and then frozen together at -20 °C for subsequent chemical analysis. At 45 min post-stun, hot carcass weights were recorded and the carcasses were chilled for 22 ± .5 hr at .5 °C. Standard carcass measurements of cold carcass weight, longissimus muscle area at the 10th rib, midline backfat at the first and last rib and last lumbar vertebrae, and tenth rib backfat 5 cm off the mid-line were collected. The right side of the cold carcass was separated into wholesale cuts of ham, loin, shoulder, belly, ribs, jowl, and feet and tail (NAMP, 1988) and the weight of each cut was recorded to the nearest gram. The cuts were then frozen at -18 °C and stored for later tissue separation. For tissue separation, wholesale cuts from each pig were thawed at 7 °C for approximately 38 h, individually weighed, physically dissected into tissue components of muscle, bone, skin, and fat, and each tissue component was weighed to

the nearest gram. Weight losses which occurred during storage and dissection procedures were assumed to be water. Weights of individual tissues were adjusted for water loss based on the percentage of total carcass water within each tissue (Nielsen, 1973) and the percentage of each tissue in a wholesale cut. The muscle from each cut was pooled, ground twice through a .64 cm die (Biro Model 75424852, 7.5 HP grinder, Marlehead, OH), and mixed thrice to ensure homogeneity. A 1000 g sub-sample was removed, reground through a .32 cm die, and stored at -20°C for subsequent chemical analysis.

Frozen visceral components were weighed, ground in the presence of dry ice through a 2.54 cm die (Buffalo Grinder Model 66BX, Buffalo, NY), mixed to ensure homogeneity, and reground through a .95 cm die. Two 500 g samples were collected and stored at -20°C for subsequent chemical analysis.

Muscle and visceral samples were weighed, freeze dried, allowed to air equilibrate, re-weighed and were then analyzed for Kjeldahl Nitrogen, ether extractable lipid and dry matter according to AOAC (1995) procedures. The chemical composition of the viscera was adjusted for the weight and composition of digesta assuming that digesta constitutes 1.175 % of body weight and that digesta contained 79.41% water, 7.47% protein and 3.94% ether extract (Gnaeger et al., 1963). The chemical composition of skin and bone and the amount and chemical composition of blood and hair were calculated based on the values of Nielsen (1973) and the chemical composition of fatty tissue was based on the values of Lonergan (1991).

A comparative slaughter technique was used to estimate empty body tissue and nutrient gain. Seven additional high LG pigs and five additional moderate LG pigs were slaughtered at 22.5 ± kg BW and processed in a manner similar to the experimental pigs in order to estimate initial body composition of the animals. The mean composition of the initial high and moderate LG pigs was then multiplied by the initial weight of the experimental pigs to calculate initial body composition.

Body nutrient composition also was estimated by a deuterium oxide dilution technique at 21 d intervals (Rudolph, 1984). Approximately .2 g of salinized D_2O (Sigma Chemical, St. Louis MO) per kg of body weight was infused via an ear vein, with exact amount infused determined by weighing the syringe before and after infusion to the nearest .001 g. Deuterium oxide was allowed to equilibrate for 2 h and a blood sample was collected into a heparinized tube, from the sub-orbital sinus. At subsequent infusion times, a blood sample was collected immediately prior to D_2O infusion for determination of residual D_2O concentration. Blood samples were frozen at -20° C and later, blood water was collected by vacuum sublimation and D_2O concentration was measured by infrared spectroscopy (Byers, 1979). D_2O space was assumed to be a single pool, and pool size was determined by dividing the amount of D_2O infused by equilibrated D_2O concentration after adjusting for residual D_2O concentration. Body water was calculated on the basis that D_2O space overestimates body water in growing/finishing pigs (Sheilds et al., 1983a; Ferrell and Cornelius, 1984). In the present study, the chemically determined body water space was .8202 ± .02 of the D_2O space at BW of 20 and 109 kg and the relationship was independent of pig genotype, dietary AP

concentration and body weight. Thus, body water was assumed to be 82.02% of D_2O space at each stage of development evaluated. Body protein was calculated based on the relationship that body water to protein ratio decreases linearly with increasing pig weight (Sheilds et al, 1983b). Based on the chemically analyzed body water to protein ratio determined in the present study in pigs at 20 and 109 kg BW, the body water to protein ratio of pigs at each pig weight was estimated as follows:

$$y = 4.1961 - .006538 \ x$$

Where y is body water to protein ratio, and x is pig weight in kg. Body ash was calculated by assuming that body protein to ash ratio was constant at 5.7 (Shields et al., 1983b). Body fat was calculated by the difference between pig empty BW and estimated body water, protein, and ash content.

Six pigs had to be removed from the study. One high LG pig consuming .080% dietary AP was lost due to sudden death. Two high LG pigs consuming .080% dietary AP had to be removed because of poor performance due to leg weakness. Two high LG pigs consuming .475 and .323% dietary AP and one moderate LG pig consuming .475% dietary AP were removed because of poor performance, specifically, their daily gains and gain/feed values were greater than 3 standard deviation units from the mean of their respective treatments for 2 or more evaluation periods.

Data were analyzed as a split plot design using the General Linear Model procedure of SAS (1988). Lean growth genotype was considered the whole-plot and dietary available phosphorus concentration the sub-plot. The effects of LG genotype was tested with the error

term of replicate within LG genotype and the effects of dietary AP and LG genotype by dietary AP were tested by the error term of replicate by dietary AP within LG genotype. Orthogonal, single degree of freedom contrasts were utilized to determine the nature of the response to dietary available phosphorus concentration. Least squares means are reported. Repeated measures analysis was utilized to examine the responses of pigs at 18 kg increments of body weight (25, 43, 61, 79, and 97 kg). The error term for testing repeated measures effects was replicate x day (genotype x dietary AP concentration). For the repeated measures analysis, raw means are reported.

Results

Genotype Effects, 20 to 109 kg BW. At the initiation of the study, the high LG pigs possessed a higher percentage of dissectable muscle and a lower percentage of dissectable fat and offal (Table 2). From 20 to 109 kg BW, the body growth rate of the high and moderate LG pigs was similar, however, the high LG pigs consumed less feed and gained more BW per unit of feed (Table 3). At 109 kg, high LG pigs had higher carcass yields and produced carcasses with larger longissimus muscle areas and lower tenth rib and average backfat depths compared with moderate LG pigs (Table 4). High LG pigs had lower liver, reproductive tract, empty gastrointestinal tract, leaf fat and head weights (Table 5). Carcasses from high LG pigs contained more dissectable muscle and less dissectable fat, skin and offal, but similar amounts of bone (Table 6). The dissected muscle and offal tissue of high LG pigs had a higher moisture and lower fat (ether extractable lipid) content; whereas, the phosphorus content of dissectable muscle was similar between genotypes (Table 7).

From 20 to 109 kg, high LG pigs gained more carcass muscle, and less fat, skin, and offal per day than moderate lean growth pigs; whereas, daily bone accretion was similar between genotypes (Table 8). Thus, the high LG pigs accrued lower amounts of fat, bone, skin, and offal per unit of muscle (Table 9). Similarly, the high LG pigs tended to deposit more body protein daily and less ether extractable lipid per unit of protein (Table 10). Futhermore, a lower proportion of the total accrued body protein in the high LG pigs was deposited in non-muscle tissue proteins (i.e. offal, skin, fat).

Available Phosphorus Effects, 20 to 109 kg BW. As dietary available phosphorus concentration declined, daily feed intake, BW gain and gain/feed ratio decreased quadratically; however, the magnitude of the decrease in BW gain was greater in the high versus the moderate LG genotype (Table 3). As dietary AP concentration declined, carcass yield tended to increase and measures of carcass muscling decreased and carcass fatty tissue content increased in both genotypes at 109 kg BW. Specifically, carcass longissimus muscle areas decreased linearly and backfat depths (tenth rib and average) increased linearly (Table 4). Heart-lung, and liver weights decreased and leaf fat weight increased linearly as dietary AP percentage decreased (Table 5). Dissectable carcass muscle and bone content also decreased linearly and dissectable fat content increased linearly as dietary AP concentration decreased, in both genotypes (Table 6). In addition, ether extractable lipid content of the dissected muscle and offal tissues increased quadratically, and moisture content decreased quadratically whereas the protein content of muscle and offal and phosphorus content of muscle were unaffected by dietary AP concentration (Table 7).

Daily gains of muscle, fat, bone, skin, and offal tissue from 20 to 109 kg body weight decreased quadratically with decreasing dietary AP concentration in both genotypes; although, the magnitude of the decrease in muscle and offal accretion tended to be greater (P<.16, P<.13, respectively) in the high LG genotype (Table 8). As dietary AP concentration decreased, the amount of dissectable fatty tissue deposited per unit of muscle increased linearly, whereas the amounts of bone, skin, and offal accrued relative to muscle accretion were unaffected (Table 9). Daily body protein, moisture and fat gains decreased quadratically as dietary AP concentration decreased, however the magnitude of the depression in protein gain was greater in the high LG genotype (Table 10). In addition, the amount of ether extractable lipid deposited per unit of protein increased linearly as dietary AP concentration decreased. The proportion of accrued body protein deposited in muscle and visceral tissues was not altered by decreasing dietary AP concentration (Table 11).

Stage of Growth Effects. Daily AP intakes, body weight gain, gain/feed, body protein gain, and body fat/protein gain during 21 d periods when the mean BW (± 9) of each pig during a period was 25, 43, 61, 79 and 97 kg are presented in Tables 12, 13, 14, 15, and 16, respectively. As average body weight increased from 25 to 97 kg, daily AP intakes increased, daily BW gains and protein gains changed quadratically, gain/feed decreased and body fat to protein gains increased; however, the pattern of the responses differed among genotypes. Specifically, daily AP intakes increased less and gain/feed declined more in the high LG pigs as BW increased. Daily body weight gain and daily protein gains peaked at 43 to 61 kg BW

and then declined in both genotypes, however, the magnitude of the decrease was greater in the high versus moderate LG genotype.

The response of pigs to the various dietary available phosphorus concentrations also differed as pig weight increased. A higher AP concentration was needed to maximize daily body weight gain and gain/feed at lighter versus heavier BW. A similar trend was observed for daily protein gain but the AP concentration by BW interaction was not significant.

Discussion

The biological demand for phosphorus is dependent on the deposition rate of phosphorus containing tissues and the rate of energy transfer associated with the release of P_i from high energy phosphate compounds (i.e. ATP). However, it is thought that the phosphorus released by ATP cleavage is readily re-incorporated into ATP via the creatine phosphate shuttle (Bessman and Carpenter, 1985). Thus, the phosphorus demand in pigs is hypothesized to be largely determined by the relative accretion rates of tissues, particularly phosphorus rich tissues. Proteinaceous tissues (i.e. muscle, bone, offal) contain moderate to high concentrations of phosphorus; whereas, fatty tissue contains a low phosphorus concentration (Nielsen, 1973). The phosphorus content of separable tissues in the 20 kg pig based on the average protein content and the phosphorus/protein ratio in individual porcine tissues are estimated to be about: .206% (19.7 x .0105) for muscle; 3.16% (19.9 x .159) for bone; .175% (12.5 x .014) for offal; .036% (5.5 x .0065) for fatty tissue; and .071% (26.3 x .0027) for skin (Nielsen, 1973). Muscle and bone growth are estimated to represent 21 to 32 and 63 to 71 %, respectively, of the daily phosphorus accrued in pigs and 55 to 65 and 10 to

15% of the daily body proteins accrued. Thus, it seems that factors such as the pigs' genetic capacity for lean tissue growth and stage of pig development which affect the rate and pattern of proteinaceous tissue deposition, especially those rich in phosphorus, will alter the pigs' dietary AP needs.

In the present study, the accretion rates of these phosphorus rich, proteinaceous tissues (muscle, bone, and offal) were depressed and that of low phosphorus fatty tissues were increased as dietary AP concentrations were lowered. Thus, based on these data, ingested energy is partitioned more toward the accretion of low phosphorus, fatty tissues and away from high phosphorus, proteinaceous tissues when dietary phosphorus intakes are insufficient to support maximum growth.

Because high lean growth genotypes normally have greater rates of muscle and bone accretion than moderate lean growth genotypes at a common body weight (Taylor, 1980), it was hypothesized that high LG pigs would have higher dietary AP needs and therefore their ability to accrue proteinaceous tissues would be more adversely affected by low dietary concentrations of AP. This relationship largely was observed. Specifically, daily gains of body weight and protein and to a lesser extent muscle and offal of high LG pigs were more detrimentally affected by low dietary AP concentrations than those of moderate LG pigs. The magnitude of this interaction may have been minimized to some extent by the fact that high LG pigs deposited a higher proportion of their accrued proteins in moderately (muscle) versus highly (bone) phosphorus rich tissues. The magnitude of the interaction also may have been minimized due to a sharp drop in growth rates which occurred in the high but not moderate

LG pigs during the last 20 to 30 kg of growth. The cause of this growth slump is not known. Neither the high or moderate LG pigs exhibited any clinical signs of disease when the change in performance occurred. Furthermore, both the high and moderate LG pigs were free of antibody titers for the prevalent pathogenic organisms of *mycoplasma hyopneumoniae*, *Actinobascillus pleuropneumonia*, transmissible gastroenteritis, and swine influenza virus, at the start and at the termination of the study, However, both daily body weight gains and gain/feed of the high LG pigs decreased by 10 to 15 % compared with earlier performance as weight increased from about 70 to 109 kg. This contributed to the lower than expected rates of protein deposition in the high LG pigs at the heavier (79 and 97 kg) body weights.

Because the accretion rates of proteinaceous tissues decline as the pig matures, it was hypothesized that dietary AP needs, expressed both as a percent of the diet and as grams/d, would decrease as pig body weight increased. In addition, because, the decline in body protein accretion in high LG pigs is generally less at heavier weights than moderate LG pigs (Taylor, 1980), it was hypothesized that the expected differences in dietary AP needs of the two genotypes would be greatest at the heavier weights. The high and moderate LG pigs needed dietary AP concentrations of .323 to . 475, .222 to .323, and .110 to .155% at 20 to 34 kg, 34 to 70 kg and 70 to 109 kg, respectively in the high and moderate LG genotypes in order to maximize daily BW gain, gain/feed ratio, and daily body protein gain and to minimize fat gain/protein gain ratio. This corresponds to daily AP intakes of 5 to 6, 6 to 7, and 5 to 6 g from 20 to 31 kg, 34 to 70 kg and 70 to 109 kg, respectively. Because of the higher feed intake of the moderate LG pigs, AP needs to maximize daily body weight gain, gain/feed ratio

and fat gain/protein gain ratio when expressed on a daily intake basis were similar between

genotypes despite the fact that high LG pigs tended to require a higher dietary concentration

of AP. Again, the lack of a statistically significant three-way interaction (LG genotype x

dietary AP concentration x average pig weight) likely may have been due to the fact that high

LG pigs deposited a higher proportion of total protein accrued in the muscle versus bone

compared with moderate LG pigs. In addition, the suspected, but unconfirmed, health

problem of the high LG pigs late in development may have prevented them from attaining

their genetic potential for protein accretion late in development and therefore, negated a

three-way interaction.

In order to more fully examine the impact of the pigs' capacity for body protein

accretion on dietary AP needs, dietary AP intake was regressed on body protein and fat

accretion rates. Examination of the efficiency of utilization of a nutrient for body nutrient

accretion requires that the response to increasing intakes of the dietary nutrient be linear.

Thus, only dietary AP concentrations of .080 to .222% for the high and .080 to .155% for

moderate LG pigs, respectively, were evaluated to ensure that dietary intakes were below the

pigs' daily needs. The regression analysis for 25 kg pigs yielded the following equations for the

high and moderate LG genotype, respectively:

$$y = -3.93 \ (\pm.74) + 47.44 \ (\pm5.97) \ x_1 + 4.101 \ (\pm1.84) \ x_2 \qquad r^2 = .81$$

$$y = -2.64 \ (\pm1.71) + 32.93 \ (\pm11.38) \ x_1 + 6.34 \ (\pm3.22) \ x_2 \qquad r^2 = .38$$

where y is daily AP intake in grams, x_1 is daily body protein gain in kilograms and x_2 is daily

ether extractable lipid gain in kilograms. The estimated amount of dietary AP (33 to 47 g)

needed to support 1 kg of body protein accretion matches closely the 34 to 39 g of P reported to be retained per unit of pody protein retained in the body by Jongbloed (1989). Based on these results, AP needs seem to be closely related to the rate of protein deposition and to a much lesser extent to fat deposition, based on the coefficient for fat deposition and due to the relatively low concentration of phosphorus in fat tissue compared with muscle tissue (Nielsen, 1973). However, these regressions do not take into account that the pigs likely are using some phosphorus from phosphorus rich body reserves (bone) to support deposition of other moderately phosphorus rich, proteinaceous tissues (muscle and offal). Thus the value of 33 to 47g of AP per kg of body protein accrued is likely an underestimation of the true dietary AP needs. Therefore, the daily body protein accretion rates of pigs in the current study were regressed on daily AP intakes of pigs weighing 25 ± 9 kg and receiving deficient phosphorus intakes. The regression analysis yielded the following equations for the high and moderate lean growth genotypes, respectively:

$$y = .085 \, (\pm .005) + .015 \, (\pm .002) \, x_1 \qquad r^2 = .75$$

$$y = .088 \, (\pm .014) + .013 \, (\pm .006) \, x_1 \qquad r^2 = .21$$

where y is daily body protein gain in kilograms and x_1 is daily available phosphorus intake in grams. Based on these equations, the high and moderate LG pigs receiving diets devoid of AP would be able to mobilize enough body phosphorus to allow the deposition of about 85 to 88 g of body protein daily. Each one gram increment of dietary AP intake would resulted in the deposition of an additional 13 to 15 g of body protein. On this basis, it is estimated that one gram of phosphorus derived from the diet or from body reserves will support the deposition of

13 to 15 g of body protein in the high and moderate LG genotypes. Consequently at 25 kg

BW, the high and moderate LG pigs consuming the lowest dietary AP regimen are estimated

to mobilize up to 5.67 (85 ÷15) and 6.78 (88 ÷ 13) g of body phosphorus for support of

proteinaceous tissue growth. However, the mobilization of body phosphorus stores alone

apparently was not sufficient to allow the 25 kg BW pigs to achieve their genetic capacities

for lean tissue growth. In the present study, the pigs initially (23 kg BW) contained about

2.65 kg of bone, which is estimated to contain 3.13 % phosphorus (Nielsen, 1973), or a total

of about 82.9 g of bone phosphorus. A mobilization of 5 to 7 g of bone phosphorus per day

represents 6 to 8 % of the pigs' total bone phosphorus reserves. Assuming 100% of an

animals bone phosphorus reserve could be mobilized, these reserves would be sufficient in the

high and moderate LG pigs recieving the lowest dietary AP concnetration to support this level

of growth for about 12 to 16 days before reductions in tissue growth would occur. Therefore,

it seems that pigs would not be able to maintain the rate of accretion of other high

phosphorus, proteinaceous tissues at the expense of bone for extended periods of time.

In conclusion, available dietary phosphorus needed to maximize proteinaceous tissue

accretion are closely related to rates of body protein deposition. When AP intake is limited,

the deposition of high phosphorus, proteinaceous tissues (muscle, bone, and offal) is slowed,

and the ingested energy normally used for accretion of proteineous tissues is partitioned

toward fat deposition. It seems that the growing pig needs about .067 to .077 g of dietary AP

to support each gram of body protein deposited in the high and moderate LG gentypes if bone

phosphorus stores are to be maintained. The pig seems to have the ability to utilize bone

phosphorus stores for support of the deposition of other proteinaceous tissues, however, the animal does not posses sufficient quantities to fully offset severe dietary shortfalls, especially early in development.

Implications

Because of the close relationship of dietary phosphorus intake with body protein deposition, determinations of dietary available phosphorus requirements in pigs should include consideration of the rate and type of body tissues deposited as influenced by genotype and stage of pig development. In addition, when determining the value of added dietary phosphorus it is important to consider the value of greater carcass leanness as well as the value of improvements in growth rate and efficiency of feed utilization.

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Table 1. Experimental diet composition.

Item	Dietary AP, %[a]					
	.080	.110	.155	0.22	.323	.475
Corn	66.41	66.41	66.41	66.41	66.41	66.41
Soybean meal, dehulled	29.13	29.13	29.13	29.13	29.13	29.13
Corn starch	1.00	.92	.81	.64	.39	-
Mono-dicalcium phosphate	.08	.24	.48	.84	1.38	2.20
Calcium carbonate	1.90	1.82	1.69	1.50	1.21	.77
L-lysine·HCL	.32	.32	.32	.32	.32	.32
D,L-methionine	.14	.14	.14	.14	.14	.14
L-tryptophan	.04	.04	.04	.04	.04	.04
L-threonine	.15	.15	.15	.15	.15	.15
Salt	.40	.40	.40	.40	.40	.40
Premix[b]	.43	.43	.43	.43	.43	.43

Table1. Cont'd

Item	Dietary AP, %[a]					
	.080	.110	.155	0.22	.323	.475
Analyzed composition						
Phosphorus, %	.33	.36	.40	.47	.57	.72
Calcium, %	.85	.85	.85	.85	.85	.85
Crude protein, %	16.10	16.10	16.10	16.10	16.10	16.10
Calculated composition						
ME, kcal/kg	3,197	3,195	3,191	3,185	3,176	3,163

[a]Single source of corn, soybean meal and monocalcium-dicalcium phosphate used in the trial. Available phosphorus (AP) levels were calculated based on NRC (1988) estimated bioavailablities of the analyzed phosphorus in each ingredient.
[b]Provided the following per kg of diet: Biotin, .03 mg; choline, 250 mg; folic acid, .17 mg; niacin, 28.5 mg; riboflavin, 6.2 mg; Pantothenic acid, 16.8; pyridoxine, .81 mg; thiamine, .55 mg; Vit E, 26 IU; Vit A, 2205 IU; Vit D_3 550 IU; menadione, 1.8 mg; Vit B_{12} .06 mg; Cu, 14 mg; Fe, 140 mg; Mn, 48 mg; Se, .3 mg; Zn, 120 mg; chlortetracycline, 110 mg.

Table 2. Body composition of pigs of high and moderate lean growth genotypes (G) at initiation of the trial.[a]

| Item | G | | CV | Probability < |
	High	Moderate		G
Pig weight, kg	22.30	23.09	8.7	.51
Whole body tissue content				
Muscle, %	39.05	34.39	4.6	.01
Fat, %	5.03	7.29	21.2	.01
Skin, %	5.53	5.80	15.1	.60
Bone, %	11.70	11.40	7.0	.55
Offal, %	26.44	28.89	6.3	.03
Dissected muscle chemical composition				
Moisture, %	73.34	72.72	1.4	.31
Protein, %	19.77	19.49	3.9	.54
Ether extract, %	6.07	6.03	20.9	.95
Offal chemical composition				
Moisture, %	71.57	69.48	1.8	.02
Protein, %	12.51	12.55	4.6	.90
Ether extract, %	5.43	7.76	21.3	.02

[a]Least squares means of 7 and 5 pigs for high and moderate LG genotypes, respectively.

Table 3. Impact of dietary available phosphorus (AP) regimen on feed intake and rate and efficiency of growth in pigs of high and moderate lean growth genotypes (G) from 20 to 109 kg[a]

| Item | G | Dietary AP, % | | | | | | CV | Probability < | | | |
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Initial weight, kg	High	20.1	20.1	20.0	19.9	19.8	19.9	10.2	.07	.88	.91	.99
	Moderate	21.4	21.1	21.2	22.1	21.1	21.2					
Final weight, kg	High	107.9	106.5	110.2	109.8	108.2	108.2	3.5	.40	.99	.32	.44
	Moderate	109.0	111.0	109.7	108.5	109.7	109.6					
Days, 20 to 109 kg	High	126	113	109	102	107	99	7.5	.13	.01	.01	.14
	Moderate	115	114	100	100	94	99					
Daily feed, kg	High	2.228	2.350	2.387	2.444	2.309	2.447	6.6	.01	.09	.02	.51
	Moderate	2.509	2.597	2.718	2.680	2.736	2.627					
Daily gain, kg	High	.702	.767	.838	.886	.834	.894	6.6	.23	.01	.01	.07
	Moderate	.769	.794	.884	.869	.945	.897					
Gain/feed	High	.316	.326	.350	.363	.361	.367	5.3	.01	.01	.01	.43
	Moderate	.306	.305	.326	.324	.346	.342					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

Table 4. Impact of dietary available phosphorus (AP) regimen on carcass measurements of pigs of high and moderate lean growth genotypes (G).[a]

Item	G	Dietary AP, %						CV	Probability <			
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Post-transport weight, kg	High	105.1	104.1	108.2	107.5	105.3	106.7	3.3	.54	.64	.16	.51
	Moderate	106.1	107.7	107.3	106.4	107.1	107.2					
Carcass yield, %	High	75.47	75.06	74.91	75.17	74.04	74.21	1.8	.01	.10	.76	.56
	Moderate	71.40	71.40	71.60	70.60	70.20	71.80					
Tenth rib fat, cm	High	2.98	2.92	2.74	2.78	2.25	2.41	17.4	.01	.01	.45	.92
	Moderate	4.15	3.85	3.79	3.84	3.60	3.27					
Average backfat, cm	High	2.93	2.81	2.60	2.75	2.52	2.52	11.5	.01	.01	.86	.53
	Moderate	3.77	3.79	3.93	3.87	3.60	3.33					
Longissimus area, cm^2	High	35.1	35.7	36.7	37.3	38.9	39.0	7.9	.57	.01	.28	.39
	Moderate	27.5	29.0	30.9	30.3	30.6	31.2					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

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Table 5. Impact of dietary available phosphorus (AP) regimen on offal component weights of high and moderate lean growth genotypes (G).[a]

| Item | G | Dietary AP, % | | | | | | CV | Probability < | | | |
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Heart and lung, g	High	1,535	1,493	1,580	1,523	1,595	1,758	11.7	.01	.04	.44	.34
	Moderate	1,370	1,407	1,448	1,400	1,516	1,386					
Liver, g	High	1,491	1,526	1,560	1,579	1,562	1,563	9.7	.01	.06	.27	.84
	Moderate	1,646	1,709	1,718	1,791	1,873	1,787					
Kidney, g	High	356	324	345	321	307	294	11.8	.43	.46	.74	.89
	Moderate	323	335	330	304	290	290					
Reproductive tract, g	High	287	226	235	247	249	236	25.9	.01	.39	.05	.37
	Moderate	338	374	281	304	292	322					
Empty GI tract, g	High	6,800	6,514	6,904	6,938	7,547	7,153	14.6	.01	.31	.21	.90
	Moderate	8,191	7,813	8,383	8,360	9,177	7,734					
Leaf fat, g	High	1,869	1,785	1,616	1,754	1,502	1,455	23.3	.04	.01	.31	.99
	Moderate	2,293	2,246	2,022	2,298	1,944	1,771					
Head, g	High	5,760	6,129	6,017	6,069	6,249	6,167	8.0	.01	.11	.24	.67
	Moderate	6,445	7,004	6,269	6,667	6,709	6,930					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

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Table 6. Impact of dietary available phosphorus (AP) regimen on dissectable empty body tissue content of pigs of high and moderate lean growth genotypes (G).[a]

Item	G	Dietary AP, %						CV	Probability <			
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Muscle, %	High	53.28	52.61	55.68	55.80	55.96	57.19	5.1	.01	.01	.27	.74
	Moderate	46.48	46.91	46.85	48.21	48.81	49.62					
Fat, %	High	30.77	31.02	27.30	27.89	26.44	25.68	10.5	.01	.01	.34	.60
	Moderate	36.93	35.67	36.55	35.08	32.90	31.63					
Bone, %	High	11.29	11.74	12.37	11.81	12.58	12.15	7.9	.57	.01	.28	.39
	Moderate	10.85	11.66	11.24	11.30	12.30	12.66					
Skin, %	High	4.55	4.62	4.63	4.46	4.92	4.58	13.2	.01	.24	.49	.87
	Moderate	5.77	5.84	5.45	5.50	6.06	6.11					
Offal, %	High	18.21	18.16	17.94	18.03	18.89	18.51	5.9	.01	.39	.69	.76
	Moderate	20.65	20.44	20.05	20.93	21.51	20.04					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

Table 7. Impact of dietary available phosphorus (AP) regimen on chemical composition of dissectable muscle of pigs of high and moderate lean growth genotypes (G).[a]

Item	G	Dietary AP, %						CV	Probability <			
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Dissectable muscle chemical composition												
Moisture, %	High	69.80	69.60	70.36	70.58	70.52	71.11	1.7	.01	.01	.03	.64
	Moderate	67.79	67.28	68.90	68.63	69.76	69.28					
Protein, %	High	20.09	20.28	20.75	20.58	20.50	20.43	2.7	.17	.61	.16	.45
	Moderate	20.08	20.26	20.10	19.93	20.16	19.83					
Ether extract, %	High	8.92	9.41	8.37	7.97	8.15	7.56	13.6	.01	.01	.01	.63
	Moderate	11.34	12.22	10.08	10.55	9.37	9.94					
Phosphorus, %	High	.18	.20	.19	.21	.20	.19	9.5	.18	.96	.26	.15
	Moderate	.19	.19	.18	.18	.18	.18					

Table 7. cont'd.

| Item | G | Dietary AP, % | | | | | | CV | Probability < | | | |
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Offal tissue chemical composition												
Moisture, %	High	55.89	56.75	59.04	56.72	59.96	58.54	6.4	.05	.02	.05	.95
	Moderate	52.62	54.31	56.33	52.86	56.99	57.69					
Protein, %	High	12.06	11.88	12.37	12.08	11.59	12.74	8.3	.07	.12	.31	.58
	Moderate	11.79	10.74	11.92	11.30	11.75	11.66					
Ether extract, %	High	27.17	26.55	24.08	25.40	21.93	23.22	3.8	.01	.01	.09	.97
	Moderate	30.62	30.40	28.04	31.26	26.20	26.53					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

Table 8. Impact of dietary available phosphorus (AP) regimen on daily tissue accretion in pigs of high and moderate lean growth genotypes (G).[a]

| Item | G | Dietary AP, % | | | | | | CV | Probability < | | | |
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Muscle, g/d	High	266	285	333	353	327	366	8.9	.01	.01	.01	.13
	Moderate	241	250	279	283	307	306					
Fat, g/d	High	181	201	191	205	179	190	13.2	.01	.66	.06	.35
	Moderate	226	225	258	245	243	225					
Bone, g/d	High	60	69	79	80	79	83	10.2	.74	.01	.01	.22
	Moderate	60	67	72	72	84	84					
Skin, g/d	High	19	21	24	23	25	24	19.3	.05	.01	.38	.72
	Moderate	27	28	28	29	35	34					
Offal, g/d	High	110	119	130	136	137	146	10.8	.01	.01	.01	.16
	Moderate	137	139	150	159	180	154					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

Table 9. Impact of dietary available phosphorus (AP) regimen on tissue accretion rates relative to muscle accretion of pigs of high and moderate lean growth genotypes (G).[a]

Item	G	.080	.110	.155	.222	.323	.475	CV	G	APL[b]	APQ[b]	G x AP
				Dietary AP, %						Probability <		
Amount of tissue accrued daily relative to accrued muscle												
Fat/muscle	High	.688	.713	.576	.586	.549	.528	17.8	.01	.01	.40	.58
	Moderate	.957	.899	.939	.869	.793	.741					
Bone/muscle	High	.228	.244	.241	.228	243	.229	9.5	.13	.38	.64	.79
	Moderate	.249	.269	.260	.255	.274	.274					
Skin/muscle	High	.073	.075	.071	.066	.076	.068	19.5	.01	.92	.29	.96
	Moderate	.112	.113	.102	.101	.112	.112					
Offal/muscle	High	.416	.423	.390	.391	.419	.399	13.2	.01	.30	.56	.84
	Moderate	.582	.565	.552	.568	.595	.514					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

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Table 10. Impact of dietary available phosphorus (AP) regimen on chemically determined nutrient accretion in pigs of high and moderate lean growth genotypes.[a]

| Item | G | Dietary AP, % | | | | | | CV | Probability < | | | |
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Moisture, g/d	High	311	336	390	407	394	429	8.6	.20	.01	.01	.17
	Moderate	308	319	360	359	411	392					
Protein, g/d	High	92	99	117	120	114	125	7.8	.15	.01	.01	.04
	Moderate	93	96	106	106	119	114					
Ether extract, g/d	High	230	255	248	267	232	249	11.3	.01	.82	.06	.50
	Moderate	289	292	321	322	315	296					
Ether extract gain/ protein gain	High	2.55	2.57	2.13	2.22	2.06	2.01	14.6	.01	.01	.14	.71
	Moderate	3.20	3.07	3.05	3.07	2.66	2.61					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

Table 11. Impact of dietary available phosphorus (AP) regimen on the proportion of total body protein gain contained in muscle and offal in pigs of high and moderate lean growth genotypes (G).[a]

Item	G	Dietary AP, %						CV	Probability <			
		.080	.110	.155	.222	.323	.475		G	APL[b]	APQ[b]	G x AP
Proportion of accrued body protein, %												
Muscle protein	High	58.43	58.36	60.02	60.78	60.10	60.03	4.6	.01	.38	.39	.82
	Moderate	52.67	53.73	53.31	53.91	52.62	53.68					
Offal protein	High	13.95	13.55	13.35	13.36	13.06	14.29	11.2	.01	.95	.90	.22
	Moderate	16.71	14.70	16.34	16.07	16.97	15.14					

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Linear (L) and quadratic (Q) effects of dietary AP concentration.

Table 12. Impact of dietary available phosphorus (AP) regimen and average body weight (ABW) on daily available phosphorus intakes (g) in pigs of high and moderate lean growth genotypes (G).[a]

| G | AP, % | ABW, kg[b] | | | | | CV | Probability < | | | | | | |
		25	43	61	79	97		G	AP	G x AP	ABW	G x ABW	AP x ABW	G x AP x ABW
High														
	.080	1.21	1.60	1.91	2.07	2.71	11.1	.01	.01	.08	.01	.06	.01	.02
	.110	1.66	2.32	2.74	2.94	3.04								
	.155	2.41	3.30	4.01	4.23	4.28								
	.222	3.69	4.97	5.88	6.12	6.28								
	.323	5.33	6.86	7.79	8.66	7.61								
	.475	7.98	10.20	12.60	12.67	13.03								
Moderate														
	.080	1.54	1.93	2.24	2.33	2.14								
	.110	1.98	2.55	2.90	3.35	3.42								
	.155	2.86	3.88	4.66	4.92	5.00								
	.222	4.16	5.48	6.21	6.98	6.49								
	.323	6.19	7.21	8.70	10.70	10.98								
	.475	9.50	11.19	12.97	14.19	14.56								

[a]Raw means are reported.
[b]Body weight of pigs during the 21 day period when average weight of the pig during the period was within 9 kg of the ABW classification.

Table 13. Impact of dietary available phosphorus (AP) regimen and average body weight (ABW) on daily body weight gain (g) in pigs of high and moderate lean growth genotypes (G).[a]

G	AP, %	ABW, kg[b] 25	43	61	79	97	CV	Probability < G	AP	G x AP	ABW	G x ABW	AP x ABW	G x AP x ABW
High														
	.080	643	786	759	667	835	10.0	.24	.01	.57	.01	.07	.01	.29
	.110	697	799	836	775	780								
	.155	743	885	910	799	808								
	.222	875	935	972	806	806								
	.323	886	916	903	923	650								
	.475	898	961	970	877	745								
Moderate														
	.080	712	846	835	821	776								
	.110	732	835	784	852	816								
	.155	767	903	981	884	911								
	.222	788	962	948	897	799								
	.323	881	907	982	1001	938								
	.475	935	936	1002	911	841								

[a]Raw means are reported.
[b]Body weight of pigs during the 21 day period when average weight of the pig during the period was within 9 kg of the ABW classification.

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Table 14. Impact of dietary available phosphorus (AP) regimen and average body weight (ABW) on gain/feed in pigs of high and moderate lean growth genotypes (G).[a]

| | | ABW, kg[b] | | | | | CV | | | | | Probability < | | | |
| | | | | | | | | G | AP | G x AP | ABW | G x ABW | AP x ABW | G x AP x ABW |
G	AP, %	25	43	61	79	97								
High														
	.080	.430	.396	.320	.254	.245	8.5	.01	.01	.85	.01	.01	.01	.87
	.110	.462	.379	.336	.292	.282								
	.155	.478	.416	.354	.293	.291								
	.222	.529	.419	.367	.291	.285								
	.323	.538	.431	.374	.344	.273								
	.475	.539	.453	.367	.330	.268								
Moderate														
	.080	.377	.351	.298	.282	.292								
	.110	.406	.359	.299	.279	.263								
	.155	.416	.362	.327	.280	.284								
	.222	.422	.389	.339	.286	.274								
	.323	.463	.411	.364	.302	.276								
	.475	.460	.400	.368	.305	.276								

[a]Raw means are reported.
[b]Body weight of pigs during the 21 day period when average weight of the pig during the period was within of the 9 kg ABW classification.

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Table 15. Impact of dietary available phosphorus (AP) regimen and average body weight (ABW) on estimated daily protein gain (g) in pigs of high and moderate lean growth genotypes (G).[a]

| G | AP, % | ABW, kg[b] | | | | | CV | Probability < | | | | | | |
		25	43	61	79	97		G	AP	G x AP	ABW	G x ABW	AP x ABW	G x AP x ABW
High														
	.080	98	112	91	94	90	16.1	.83	.01	.48	.01	.10	.30	.93
	.110	114	119	115	100	96								
	.155	122	135	125	110	107								
	.222	142	148	129	106	107								
	.323	149	132	131	124	87								
	.475	149	149	139	128	97								
Moderate														
	.080	107	117	104	102	92								
	.110	116	116	101	116	99								
	.155	118	127	133	96	106								
	.222	119	142	123	113	102								
	.323	135	136	148	135	132								
	.475	145	139	146	123	110								

[a]Potein gain estimated by D_2O dilution technique (Rudolph, 1984), raw means reported.
[b]Body weight of pigs during the 21 day period when average weight of the pig during the period was within 9 kg of the ABW classification.

Table 16. Impact of dietary available phosphorus (AP) regimen and average body weight (ABW) on estimated fat gain/protein gain in pigs of high and moderate lean growth genotypes (G).[a]

| G | AP, % | ABW, kg[b] | | | | | CV | Probability < | | | | | | |
		25	43	61	79	97		G	AP	G x AP	ABW	G x ABW	AP x ABW	G x AP x ABW
High														
	.080	1.393	2.082	3.841	2.760	4.913	44.2	.21	.09	.67	.01	.69	.87	.99
	.110	1.032	1.889	2.561	3.509	4.341								
	.155	.979	1.693	2.657	3.377	3.316								
	.222	1.083	1.447	3.038	2.985	3.502								
	.323	.875	2.069	2.107	2.939	3.901								
	.475	.945	1.575	2.356	2.475	3.562								
Moderate														
	.080	1.826	2.413	3.531	3.782	4.395								
	.110	1.238	2.289	3.089	2.931	4.087								
	.155	1.413	2.234	2.769	4.799	4.133								
	.222	1.550	1.890	3.143	3.508	3.747								
	.323	1.477	1.811	1.978	3.138	2.942								
	.475	1.398	1.906	2.100	2.859	3.297								

[a]Body protein and fat gain estimated by D_2O dilution technique (Rudolph, 1984), raw means reported.
[b]Body weight of pigs during the 21 day period when average weight of the pig during the period was within 9 kg of the ABW classification.

CHAPTER 4. THE IMPACT OF DIETARY PHOSPHORUS REGIMEN ON MUSCLE QUALITY IN PIGS OF HIGH AND MODERATE LEAN GROWTH GENOTYPES[1]

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M.J. Bertram[2], T.S. Stahly[3,4], and R.C. Ewan[3]
Department of Animal Science, Iowa State University, Ames, 50011

Abstract

Seven sets of 6 littermate barrows (initial BW of 20 kg) from each of a high and

moderate lean growth (LG) genotype were allotted within litter to one of 6 dietary

concentrations (.080, .110, .155, .222, .323, and .475%) of available phosphorus (AP). Pigs

were allowed to consume their experimental diets ad-libitum. At 109 ± 4 kg, pigs were

removed from feed at 7:00 am, transported 5 km, electrically stunned and killed by

exsanguination .25 to 1.5 hr post-transport. From 89 to 109 kg BW, high LG pigs consumed

less feed daily, grew slower but had similar gain/feed ratios compared with moderate LG pigs.

Longissimus muscle (LM) sections from high LG pigs had lower pH 45 minutes post mortem,

heavier initial weights, lower water holding capacities (WHC), more moisture and less

intramuscluar fat, and were lighter in color on d 0 of retail storage, whereas the degree of

redness and yellowness were similar between genotypes. During retail storage, exudative

losses from the LM sections in both the fresh and post-thaw states were initially (d 0 to 3)

higher in the high LG pigs and subsequently lower than that of moderate LG pigs. As storage

progressed from d 3 to 9, LM sections in both the fresh and post-thaw states became lighter

and less red in color, however, the magnitude of the changes differed between genotypes. As

dietary AP concentration declined initial weights and WHC of the LM sections were reduced.

But, during a 9 d retail storage period, the exudative losses and color scores of the LM

sections in both the fresh and post-thaw state were not altered by dietary AP regimen. Thus,

the LG genotype of pigs seems to alter quality traits of pork post-mortem and during retail

storage, whereas dietary AP regimens that limit muscle growth seem to have minimal impact.

Introduction

It is well known that animals consuming inadequate levels of phosphorus have

decreased growth rate, efficiency of feed utilization and bone development (Cromwell, 1970;

van Kempen et al., 1976; Jongbloed, 1987). A dietary phosphorus deficiency also has been

shown to depress carcass muscle accretion and increase carcass fat deposition in pigs

(Bertram et al., 1995). Furthermore, the water holding capacity of muscle postmortem may

be dependent on the animals dietary phosphorus regimen (Voon Foong, 1982). Phosphorus

containing compounds (i.e. phospholipids and high energy phosphate compounds) play key

roles in maintaining muscle membrane integrity, calcium movement in muscle and muscle

energy metabolism, and thus may influence pork quality. In rats, phosphorus deficiency has

been reported to decrease the concentration of phospholipids and change the ratio of

individual phospholipids in the sarcolemma and sarcoplasmic reticulum (Kreusser et al., 1980;

Brautbar and Massry, 1984). This may decrease the activity of the sarcoplasmic reticulum

Ca^{2+} induced Ca^{2+} ATPase protein (Hesketh et al., 1976), a major regulator of cytoplasmic

Ca^{2+} in muscle cells (Bennett et al., 1980). High levels of cytoplasmic Ca^{2+} have been reported

to increase postmortem muscle glycolytic rate, and decrease muscle pH and water holding

capacities (Cheah and Cheah, 1976). Thus, muscle from pigs consuming phosphorus

deficient diets may have decreased sarcoplasmic reticulum Ca^{2+} induced Ca^{2+} ATPase activity,

increased cytoplasmic Ca^{2+} concentrations and therefore, low water holding capacity,

increased lightness, and decreased redness as a result of the associated reduction in muscle

pH. Because pigs with a higher rate of lean tissue growth would be expected to have a higher

requirement for available phosphorus due to their greater capacity for lean tissue deposition

(Bertram et al., 1995), the magnitude of the reduction in water holding capacity with

decreasing available phosphorus concentrations would be hypothesized to be greater.

The objective of this experiment was to evaluate the influence of dietary phosphorus

regimen on water holding capacity, color and intramuscular fat and moisture content of the

longissimus muscle of pigs from high and moderate lean growth genotypes.

Materials and Methods

Seven sets of six littermate barrows from each of a high and moderate lean growth

(LG) genotype, were allotted within litter to diets containing one of six dietary available

phosphorus (AP) concentrations (.080, .110, .155, .222, .323, and .475%). The muscle

growth capacities of the high and moderate LG genotypes from 20 to 110 kg were

approximately 375 and 330 g /d, respectively. The pigs possessed negative antibody titers for

transmissible gastroenteritis, *mycoplasma hyopneumoniae, Actinobascillus pleuropneumonia,* and swine influenza virus both at the initiation and conclusion of the trial. Each pig was bled for to determine the halothane genotype of the animals (Fujii, 1991). However, a portion of the samples were destroyed during processing. Of the 17 high LG pigs and 33 moderate LG pigs in which assay results wer obtained, there were 1 and 3 halothane gene carriers, respectively.

Experimental diets consisted of corn-soybean meal mixtures fortified with minerals, vitamins and an antibiotic (Table 1). A single source of corn, dehulled soybean meal, and monocalcium-dicalcium phosphate were used throughout the trial and contained an analyzed phosphorus (P) content of .18%, .67%, 18.2%, respectively. The proportion of the analyzed P assumed to be bioavailable in corn, soybean meal and moncalcium-dicalcium phosphate was 16, 25, and 100%, respectively (NRC, 1988). Available phosphorus levels were achieved by altering the ratios of monocalcium-dicalcium phosphate, corn starch, and calcium carbonate. Diets were formulated so that the highest dietary concentration of AP would meet the estimated needs of the high LG genotype at 20 kg, and the lowest level would be below the estimated needs of the moderate LG genotype at market weight. Calcium was maintained at .85% in all diets. Crystalline amino acids were included in the diets to achieve a more ideal ratio of amino acids relative to lysine (NRC, 1988; Wang and Fuller, 1989). Diets contained 1.15% digestible lysine and digestible tryptophan, threonine, and methionine + cystine levels were 21, 64 and 63 percent of the digestible lysine level, respectively. Pigs were allowed to consume feed and water ad-libitum.

Pigs were individually-penned in fully slatted, .61 x 2.44 m pens in a temperature controlled building. The average daily high and low ambient temperature in the building over the duration of the trial were 27°C and 22°C, respectively, with a maximum and minimum of 32°C and 21°C, respectively. Drip coolers were employed when the ambient temperature exceeded 27°C. The trial was initiated in April and concluded in September.

Upon reaching 109 ± 4 kg, the pigs were killed for subsequent evaluation of muscle quality. On the morning of slaughter, pigs were stirred approximately 1.5 h before transport to stimulate feed intake, were then weighed, gently loaded in groups of 3 to 8 pigs and transported 5 km (20 ± 2 min) to the Iowa State University Meat Lab. At the Meat Lab, pigs were housed in groups of 3 to 4 in concrete floored pens and were allowed access to water. At 45 ± 19 min post-arrival, the pigs were electrically stunned with 280 V for 20 s, and then killed by exsanguination 45 ± 10 s post-stun. Immediately after exsanguination, a 10 g muscle sample was removed from the left longissimus muscle (LM) adjacent to the last rib, placed on ice and assessed for sarcoplasmic reticulum Ca^{2+} induced Ca^{2+} ATPase activity within 2 h, according to the procedure of Pierce and Dhalla (1981). Pigs were then placed in a scalding vat (Oskar Bauman, Stuttgart-Plienengen, Germany) maintained at 64°C for 5.5 min and dehaired. The head, gastrointestinal tract, internal organs, and leaf fat were removed from the carcass within 40 min of exsanguination. At 45 ± 3 min post-exsanguination, duplicate 1g samples were removed from the right LM adjacent to the last rib, homogenized in 1% iodoacetic acid in 150mM KCL for pH determination (Bendall, 1973) with a pH meter (Orion Reasearch, model 701A, Cambridge, MA). Carcasses were then chilled for 20 ±.5 hr at .55°C.

Then, five, 2.5 cm loin sections were removed from the left side of the carcass beginning at

the 10th rib and moving distally. In each loin section, the longissimus muscle (LM) was

removed with the perimysium intact, cleaned of adhering fat tissue and weighed to the nearest

gram. The first and second LM sections were utilized to examine fresh muscle exudative loss

and water holding capacity, respectively and the color of both LM sections was evaluated on

days 0, 3, 6 and 9 of retail storage. The storage conditions consisted of placing the LM

sections on 12 x 12 cm absorbent blotters in styrofoam trays, overwrapping the LM sections

with oxygen permeable film and storing the LM sections on a tray under ultraviolet lights in a

cooler maintained at 6°C with a mean air velocity of 61 m/min, light intensity of 5165 lx, and

relative humidity of 70%. LM sections were placed on a fresh absorbant blotter, re-wraped

using new oxygen permeable film, and returned to retail storage after each measurement.

Exudative loss was assessed by weighing the LM section to the nearest gram on each day of

measurement and examining the weight loss which occured during the 3 d storage period

expressed as a percentage of the initial section weight. Water holding capacity was assessed

utilizing a modified procedure of Grau and Hamm (1953) as presented by Wierbicki and

Deatherage (1958). Briefly, a .3 g core sample was collected from each LM section, placed on

a #2 Whatman filter paper (Whatman Laboratory division, Springfield Mill, Maidstone, Kent,

England), the sample and paper were then placed between 2 Plexiglas plates, and the plates

were pressed at 211 kg/cm^2 for 2 min with a Craver laboratory press (model 3437, Summit,

NJ). After pressing, the area of the meat and the total area (meat area + released water area)

were measured by the ISU image Analysis Facility using a Zeiss SEM-IPS image analysis

system (Zeiss-Kontron; IBAS version 2.00). Individual samples were placed on a copy stand, images were captured with a Sony DXC-3000A 3 CCD color video camera, interactively discriminated, and area was measured. The ratio of total area/meat area was calculated, with a lower ratio representing higher water holding capacity. Color scores were assessed by Hunter LAB analysis (Hunt et al., 1991) under F illuminate with a 10° standard observer using a Hunter Lab LabScan Spectrocolorimeter (Hunter Associates Laboratory, Reston, VA), with higher L values indicating lighter color, higher A values indicating greater redness, and higher B values indicating more yellowness.

The third and fourth LM sections were vacuum packaged and frozen at -20°C for assessment of post-thaw exudative losses and water holding capacity, respectively and color scores of both LM sections were evaluated. After 200 d of storage, frozen LM sections were thawed at 2°C for 12 h and weighed to determine exudative loss that occurred during frozen storage and the thaw process. Exudative loss, water holding capacity, and muscle color were assessed on d 0, 3, 6 and 9 of retail storage in the same manner as with fresh LM sections.

The fifth LM section was frozen in liquid nitrogen, vacuum packaged and stored at -20°C. The LM section was removed from the freezer and pulverized in liquid nitrogen using a Waring Blender (Waring Products Division, New Hartford, CT). Moisture and ether extractable lipid were analyzed according to AOAC procedures (1990). Protein was determined as N X 6.25 using a Kjeltec Analyzer (Tecator, Model 1028, Hoganas, Sweden).

Data were analyzed by analysis of variance utilizing the GLM procedure of SAS (1994) as a split plot design with lean growth genotype representing the whole-plot and

dietary AP level the sub-plot. LG genotype effects were tested with the error term of replicate within LG genotype, dietary AP and LG by AP effects were tested with the error term of replicate by dietary AP within LG genotype. Changes in measures of meat quality criteria over time were analyzed as a repeated measure within the split-plot design. Raw means are rep rted.

Results

Lean growth genotype effects. From 89 to 109 kg, high LG pigs grew more slowly, consumed less feed daily and had similar gain/feed ratios as moderate LG pigs (Table 4.2). At 109 kg BW, LM from the two genotypes had similar Ca^{2+} induced Ca^{2+} ATPase activities but pH of the LM 45 min postmortem was lower in the high LG genotype (Table 4.3).

LM sections of high LG pigs in both the fresh and post-thaw states had larger initial weights, higher exudative loss during the initial storage period (Table 4.4 and Table 4.7), lower water holding capacities (Table 4.5 and Table 4.8), less fat and more moisture (Table 4.6 and Table 4.9), and were lighter in color, but possessed similar degrees of redness and yellowness (Table 4.10 to 4.15) on day 0 compared with sections from moderate LG pigs. However the pattern of change in exudative losses, WHC, muscle moisture content, and color that occurred during retail storage of the LM sections during the fresh and post-thaw states differed between gentoypes. Exudative losses, expressed as a percentage of initial LM section weight, were initially greater in high LG pigs and subsequently were less compared with those of moderate LG genoytpe, resulting in genotype by storage period interactions. Water holding capacity, measured as the Hamm press ratio, in the post-thaw LM sections declined more in

the high LG sections as storage progressed. A similar, but non-significant trend was observed in the fresh LM sections. Longissimus muscle sections of high LG pigs initially (d 0) contained more moisture, but greater exudative loss early in storage resulted in LM moisture contents being similar between genotypes on d 3, 6 and 9, resulting in a genotype by day of storage interaction in both the fresh and post-thaw muscle. LM sections in both the fresh and post-thaw state became lighter in color as storage time progressed; however, the magnitude of the increase was greater in LM sections from moderate LG pigs. The degree of redness of fresh LM sections changed quadratically as storage progressed, increasing early, before decreasing late in the storage period. The magnitude of the reductions were greater in the later stage of storge for the high LG genotype. The degree of redness of the post-thaw LM sections decreased linearly as retail storage progressed, but again, the magnitude of the reduciton was greater in sections from high LG pigs.

Dietary AP effects. Dietary AP regimen did not influence body weight gains and gain/feed ratios of pigs from 89 to 109 kg BW. However, feed intake tended to decrease as dietary AP concentration decreased during the last 20 kg of pig growth. Postmortem muscle sarcoplasmic reticulum Ca^{2+} induced Ca^{2+} ATPase activity and 45 minute pH were unaffected by dietary AP concentration (Table 2). Weight of the LM sections in the fresh and post-thaw state increased as dietary AP concentration increased but exudative loss during the initial storage period, initial (d 0) muscle water holding capacity, moisture and fat content, and color scores did not differ among dietary treatments. The patterns of change in exudative loss, WHC and moisture content that occured in fresh LM sections as storage progressed were

similar among dietary treatments. However, exudative loss from freeze to thaw tended to

decrease with increasing phosphorus concentration in both LG genotypes, whereas exudative

loss was unaffected or tended to increase during subsequent retail storage, resulting in a

dietary AP concentration by day of storage interaction for post-thaw LM sections. Water

holding capacity of post-thaw LM sections also declined with increasing dietary phosphorus;

however, the magnitude of the decline became smaller as storage progressed, resulting in a

dietary AP concentration by day of storage interaction. The patterns of change in the

lightness, redness, and yellowness of LM sections as storage progressed were similar among

dietary treatments for LM sections in both the fresh and post-thaw states. The genotype by

dietary AP concentration interaction was not biologically important for any of the quality

traits of the LM sections that were evaluated..

Discussion

Meat quality measures are highly dependent on the postmortem pH of muscle (Bendall

and Swatland, 1988) which in turn is dependent on the fiber types in muscle (Guignot et al.,

1992) as well as other genteic (i.e. stress gene, Essen-Gustavsson et al., 1992) and

environmental factors (i.e. pre-slaughter handling, Nielsen, 1981)

Muscle of pigs which possess a high genetic capacity for muscle growth have been

reported to contain a higher proportion of white (fast twitch, glycolytic, type IIB) relative to

red (slow twitch, oxidative, type I) muscle fibers than pigs with lower capacities for muscle

growth (Ashmore, 1972; Rahelic and Puac, 1981). White fibers have higher glycolytic enzyme

activities and lower oxidative capacities, higher glycogen levels, and lower levels of myoglobin

(Seideman et al., 1984; Guignot et al., 1992). Thus, these fibers rely heavily on glucose and glycolysis for energy production. In the postmortem situation, when blood flow and oxygen delivery stop, the end-product of glycolysis is lactic acid. The buildup of lactic acid results in decreased pH which is associated with a lower water holding capacity and muscle redness score and a greater muscle lightness score (Bendall and Swatland, 1988). Differences in the relative proportion of white versus red fibers between the high and moderate LG genotypes (Ashmore, 1972; Rahelic and Puac, 1981) likely explain in part the lower muscle pH 45 minutes post-mortem in the high LG pigs. In the present study, muscle pH (expressed as H^- concentration) was highly correlated with both exudative loss from d 0 to 3 of storage ($r = .54$; $P < .01$) and muscle WHC on d 0 ($r = .44$; $P < .01$), d 3 ($r = .40$; $P < .01$), d 6 ($r = .49$; $P < .01$), and d 9 ($r = .51$; $P < .01$); consequently, it would be expected that the initial exudative loss would be higher and WHC would be lower in LM sections from high LG pigs. However, a large amount of the variation in both exudative loss and WHC was not accounted for by muscle pH. Thus it appears that other factors not measured have significant impact on WHC. Because of the high association of muscle water content with consumer acceptability of meat based on juiciness (Forrest et al., 1975; Cross et al., 1986; Schonfeldt et al., 1993), the LM sections from the high LG genotype may have greater consumer acceptability of juiciness during the initial stages of retail storage. Although the LM sectons from the high LG pigs lost more moisture during storage, their absolute moisture contents and potentially consumer acceptabily of juiciness on d 3, 6 and 9 of storage were similar between genotypes. However,

the lower fat content of the LM sections from the high LG genotype may negatively influence pork flavor and subsequent consumer acceptability (NPPC, 1995).

Meat color is related to the structure of meat and the level and chemical state of pigments in muscle, predominantly myoglobin (Seideman et al., 1984). A high degree of muscle lightness in muscle is mainly a result of an open structure of muscle fibrils causing the muscle to scatter more light and therefore to appear lighter in color (Seideman et al., 1984). In situations were pH is low and approaches the isoelectric point of muscle proteins (5.0 to 5.1), water moves out of the fiber to the intracellular space, fibers become less tightly packed and therefore scatter more light (Lawrie, 1985). This is also true when pH depression is very rapid and reaches extremely low levels (5.5-5.8) within 45 minutes post-mortem (Christian and Rothschild, 1981). In the present study, the pH of the LM from the high LG pigs reached this critical value, whereas that of the moderate LG pigs did not. These differences in pH explain, in part, the difference in L value between genotypes, because L value on day 0, 3, 6 and 9 of storage was correlated with pH (R^2 = .54, .53, .50, and .51; P< .01, .01, .01 and .01, respectively) and with exudative loss from d 0 to d 3 (R^2 = .78, .80, .77, and .78; P<.01, .01, .01, and .01, respectively). The pattern of the changes in the degree of muscle redness that occured as storage time progressed likely were related to muscle myoglobin. Because high LG animals tend to have a higher amount of white versus red muscle fibers (Ashmore, 1972; Rahelic and Puac, 1981), they would be expected to have a lower total concentration of myoglobin. In addition, low muscle pH has been reported to cause myoglobin to become more easily oxidized to metmyoglobin, which has low red color intensity, as storage progresses

(Seideman et al., 1984). This phenomena has been reported to result from one of two causes: a destabilization of the heme-globin linkage due to low pH, which allows greater exposure of the iron center of heme allowing myoglobin to be transformed directly to metmyoglobin without formation of oxymyoglobin, the bright red pigment of meat; or protonation of the oxygen in oxymyoglobin, which make electron transfer from the iron center energetically favorable and allows formation of metmyoglobin from oxymyoglobin (Livingson and Brown, 1981). In this study, because redness increased during the first 3 d of storage, it seems that oxymyoglobin was being formed and was then transformed to metmyoglobin. It has been reported that formation of oxymyoglobin, the bright red pigment of meat, does not begin to occur in pork in substantial amounts until after 24 hours of exposure to oxygen at 5°C (Millar et al., 1994). Thus, the increase in redness on d 3 versus d 0 is likely the result of increasing oxymyoglobin formation.

Pigs in the present study were initially placed on their experimental diets at 20 kg BW. From 20 to 109 kg BW, the body growth rates, efficiency of feed utilization and carcass muscle contents were depressed and carcass fat content increased as dietary AP concentrations were lowered from .475 to .080% (Bertram et al., 1995). Thus, it appears that the lower dietary AP concentrations used in this study were indeed below that needed to optimize pig body growth and proteinaceous tissue accretion, especially during early stages of pig development. However, the lower dietary AP concentrations (.080 and .110%) were only marginally deficient as pigs approached market weight (89 to 109 kg). The higher daily feed and thus phosphorus intake of the market weight pigs as well as their lower capacity for body

protein and phosphorus accretion, results in a lower dietary phosphorus requirement for the market weight pigs, when expressed as a percentage of the diet (Bertram et al., 1995). The absence of a substantial dietary AP deficiency at the time the pigs were marketed may have contributed to the minimal differences in LM exudative losses and color scores among dieary treatment groups. Since the LM pH was not consistently affected by increasing dietary AP concentration, the exudative losses and color scores of the LM sections, which are highly dependent on the rate and extent of postmortem glycolysis and pH depression (Offer and Knight, 1988), would not be expected to differ substantially among dietary treatments. The tendency for less exudative loss and greater water holding capacity in pigs consuming the low AP diets may have been associated with the marginally lower feed intakes of these pigs at market weight. Lower dietary energy intakes may have reduced muscle glycogen stores, a major fuel for postmortem glycolysis. Previous research has shown that feed restriction prior to slaughter results in greater pH 45 min and 24 h post-mortem, lower drip loss and higher Hunter L values (Eikelenboom, et al, 1991). The lack of statistically significant changes in exudative losses as dietary AP concentration decreased whereas WHC was significantly lowered, likely is due in part to the greater variability in exudative losses (CV 24.8%) versus that of water holding capacity (CV 12.9%).

It was hypothesized at the initiation of the trial that low AP intakes would result in decreased membrane phospholipid concentrations and therefore decreased membrane integrity. Thus, it was hypothesized that low dietary AP concentrations would result in greater exudative losses and lower WHC in LM sections which were frozen and then thawed due to

muscle membrane fracture resulting from ice crystal formation. Indeed, the amount of exudative loss that occured from freezing through the thawing of the LM sections were increased as dietary AP concentrations were lowered.

The influences of dietary AP regimen observed in this study were different than those observed by Voon Fong (1982). The depression in WHC observed by Voon Fong (1982) may be due to the fact that both low phosphorus and low calcium concentrations were fed. When phophorus concentration was lowered while calcium was maintained at dietary concentratins similar to those utilized in the present study, the increase in exudative losses observed as dietary AP concentration decreased was much less than if low dietary calcium concentrations were fed. In addition, feed intake of pigs utilized by Voon Fong (1982) averaged 39% less than the pigs utilized in this experiment, thus daily phosphorus intake was much lower in the previous study.

In conclusion, LG genotypes affects quality measures of LM section postmortem (WHC, water and fat content and color) and during retail storage (exudative losses, WHC, and color). Dietary AP regimens that limit muscle growth have minimal influence on quality measures of LM sections in the fresh and frozen states during retail storage.

Implications

Genetic selection programs directed at increasing lean gain must consider the impact of genetic make-up on pork quality in order to prevent quality deterioration. Although muscle growth and carcass muscle content are reduced dramatically by a dietary available phosphorus deficiency, meat quality characteristics in pigs are influenced minimally.

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Table 1. Experimental diet composition.

Item	Dietary available phosphorus, %[a]					
	.080	.110	.155	.222	.323	.475
Corn	66.41	66.41	66.41	66.41	66.41	66.41
Soybean meal, dehulled	29.13	29.13	29.13	29.13	29.13	29.13
Corn starch	1.00	.92	.81	.64	.39	-
Mono-dicalcium phosphate	.08	.24	.48	.84	1.38	2.20
L-lysine·HCL	.32	.32	.32	.32	.32	.32
Calcium carbonate	1.90	1.82	1.69	1.50	1.21	.77
D,L-methionine	.14	.14	.14	.14	.14	.14
L-tryptophan	.04	.04	.04	.04	.04	.04
L-threonine	.15	.15	.15	.15	.15	.15
Salt	.40	.40	.40	.40	.40	.40
Vitamin/trace mineral premix[b]	.43	.43	.43	.43	.43	.43

Table 1. Cont'd.

Item	Dietary available phosphorus, %[a]					
	.080	.110	.155	.222	.323	.475
Analyzed composition						
Phosphorus, %	.33	.36	.40	.47	.57	.72
Calcium, %	.85	.85	.85	.85	.85	.85
Crude protein, %	16.10	16.10	16.10	16.10	16.10	16.10
Calculated composition						
ME, Mcal/kg	3.20	3.20	3.19	3.19	3.18	3.16

[a]Single source of corn, soybean meal and monocalcium-dicalcium phosphate used in the trial. Available phosphorus (AP) levels were calculated based on NRC (1988) estimated bioavailabilities of the analyzed phosphorus in each ingredient.
[b]Provided the following per kg of diet: Biotin, .03 mg; choline, 250 mg; folic acid, .17 mg; niacin, 28.5 mg; riboflavin, 6.2 mg; Pantothenic acid, 16.8; pyridoxine, .81 mg; thiamine, .55 mg; Vit E, 26 IU; Vit A, 2205 IU; Vit D_3 550 IU; menadione, 1.8 mg; Vit B_{12} .06 mg; Cu, 14 mg; Fe, 140 mg; Mn, 48 mg; Se, .3 mg; Zn, 120 mg.

Table 2. Pig weight and feed intake, daily gain, and efficiency of feed utiliization during the last 20 kg of pig growth.[a]

| Item | LG Genotype | Dietary available phosphorus, % | | | | | | CV % | Probability < | | |
		.080	.110	.155	.222	.323	.475		G	AP	G x AP
Pig weight, kg											
Initial	High	20.1	20.1	20.0	19.9	19.8	19.9	10.2	.07	.95	.99
	Moderate	21.4	21.1	21.2	22.1	21.1	21.2				
Final	High	107.9	106.5	110.2	109.8	108.2	108.2	3.5	.40	.45	.99
	Moderate	109.0	111.0	109.7	108.5	109.7	109.6				
Growth and feed utilization											
Daily feed, kg	High	2.49	2.60	2.84	2.88	2.55	2.73	9.9	.01	.07	.14
	Moderate	2.89	3.17	3.20	3.13	3.38	3.04				
Daily gain, kg	High	.664	.721	.798	.811	.692	.794	14.7	.03	.16	.09
	Moderate	.769	.882	.889	.806	.962	.851				
Gain/feed	High	.273	.277	.280	.281	.270	.285	13.0	.80	.86	.81
	Moderate	.266	.279	.278	.256	.285	.285				

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.

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Table 3. Impact of lean growth genotype (G) and dietary available phosphorus (AP) concentration on muscle sarcoplasmic reticulum Ca^{2+} induced Ca^{2+} ATPase activity and 45 minute postmortem pH.[a]

Item	LG Genotype	Dietary available phosphorus, %						CV %	Probability <		
		.080	.110	.155	.222	.323	.475		G	AP	G x AP
ATPase Activity, mol Pi · mg protein^{-1}· min^{-1}											
	High	.313	.452	.396	.549	.457	.577	42.5	.55	.31	.59
	Moderate	.388	.410	.400	.458	.387	.429				
45 min pH[b]											
	High	6.07	5.95	5.92	6.11	5.93	5.96	67.6	.07	.59	.95
	Moderate	6.25	6.24	6.08	6.24	6.15	6.26				

[a]Least squares means of 7 sets of 6 littermate barrows from each genotype.
[b]Analyzed as H+ concentration.

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Table 4. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on fresh longissimus muscle exudative loss.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Initial longissimus muscle weight, g																
High	0		104	104	112	111	115	119	13.2	.01	.11	.99				
Moderate			85	86	96	96	96	96								
Exudative loss, % of day 0 weight																
High	0 to 3		6.73	7.94	8.28	7.66	7.21	8.59	36.3	.01	.72	.66	.01	.01	.68	.91
Moderate			6.03	4.55	5.48	4.37	4.38	5.53								
High	3 to 6		2.47	2.53	2.69	2.98	3.27	3.05								
Moderate			2.41	2.33	2.58	2.67	2.72	2.48								
High	6 to 9		1.18	1.79	2.00	1.93	1.93	1.69								
Moderate			2.11	2.03	1.84	1.85	1.91	2.48								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

Table 5. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on fresh longissimus muscle Hamm press ratio.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hamm press ratio, total area/meat area																
High	0		3.02	3.78	4.03	3.63	3.82	4.56	15.7	.05	.09	.66	.01	.59	.33	.92
Moderate			3.02	3.30	3.99	3.66	3.57	3.66								
High	3		3.76	3.59	3.82	3.43	4.44	4.02								
Moderate			3.34	3.31	3.46	3.31	3.45	3.63								
High	6		3.25	3.68	3.59	3.31	3.95	4.11								
Moderate			2.95	3.27	2.65	3.47	3.56	3.69								
High	9		3.44	3.57	3.57	3.14	3.90	3.97								
Moderate			3.07	3.04	3.13	3.42	3.41	3.04								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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Table 6. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on fresh longissimus muscle chemical composition.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Moisture, %																
	High	0	72.2	72.8	73.1	73.3	72.8	73.4	1.0	.10	.98	.89	.01	.01	.98	.90
	Moderate		71.9	71.6	72.0	71.2	71.5	72.0								
	High	3	70.2	70.4	70.7	71.0	70.6	70.9								
	Moderate		70.1	70.3	70.3	69.9	70.2	70.3								
	High	6	69.4	69.5	69.8	70.1	69.5	69.9								
	Moderate		69.3	69.5	69.5	69.1	69.3	69.5								
	High	9	69.0	68.9	69.1	68.4	68.7	69.3								
	Moderate		68.6	68.9	68.9	68.4	68.7	68.7								
Protein, %																
	High	0	24.1	23.6	24.1	24.0	24.2	24.1								
	Moderate		23.5	23.5	23.4	23.4	23.4	23.4								
Fat, %																
	High	0	3.7	3.6	2.9	2.7	3.1	2.7								
	Moderate		4.4	4.9	4.6	5.2	5.3	4.6								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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Table 7. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on post-thaw longissimus muscle weight and exudative loss.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Initial longissimus muscle wt, g																
	High	0	111	113	125	123	122	130	10.5	.01	.02	.84				
	Moderate		94	93	98	101	102	103								
Exudative loss, % of pre-freeze weight																
	High	Freeze to Thaw	6.54	6.06	4.70	5.67	5.04	4.55	24.8	.01	.61	.92	.01	.01	.46	.01
	Moderate		4.80	4.23	5.35	4.79	4.74	4.53								
	High	0 to 3	9.32	9.42	11.38	10.83	11.50	10.97								
	Moderate		9.85	9.93	9.39	9.69	9.44	9.79								
	High	3 to 6	2.21	2.01	2.57	1.81	2.46	2.23								
	Moderate		1.64	1.96	2.31	2.10	2.43	2.41								
	High	6 to 9	1.07	1.08	.96	1.06	.96	1.15								
	Moderate		.94	.93	1.18	1.08	1.20	1.17								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

Table 8. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration and day (D) of retail storage on post-thaw longissimus muscle Hamm press ratio.[a]

Item	G	D	.080	.110	.155	.222	.323	.475	CV	G	AP	G x AP	D	G x D	AP x D	G x AP x D
				Dietary available phosphorus, %								Probability <				
Hamm press ratio, total area/meat area																
	High	0	3.42	3.62	3.64	3.81	3.49	4.35	12.9	.01	.01	.30	.01	.01	.01	.02
	Moderate		3.06	3.07	3.34	3.78	3.87	3.95								
	High	3	3.52	3.59	3.41	3.41	3.69	4.27								
	Moderate		3.11	2.91	3.38	2.96	3.28	3.50								
	High	6	3.70	3.28	3.31	3.05	3.67	4.14								
	Moderate		2.86	3.00	3.08	3.04	2.59	3.05								
	High	9	3.42	2.72	3.05	2.87	3.65	3.98								
	Moderate		2.44	2.34	2.78	2.77	3.16	2.57								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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Table 9. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration and day (D) of retail storage post-thaw longissimus muscle moisture content.[a]

| Item | G | D | Dietary available phosphorus, % | | | | | | CV | Probability < | | | | | | |
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Moisture, %																
	High	0	72.2	71.0	71.8	71.7	71.3	72.1	2.8	.01	.66	.49	.01	.01	.69	.54
	Moderate		70.5	70.4	70.4	69.8	70.1	70.6								
	High	3	70.3	67.7	68.0	68.0	67.3	68.4								
	Moderate		67.1	66.9	67.1	66.8	66.8	67.2								
	High	6	67.0	66.9	67.0	67.3	66.3	67.5								
	Moderate		66.4	66.2	66.2	65.9	65.8	66.3								
	High	9	66.1	66.5	66.6	66.9	65.9	67.1								
	Moderate		66.0	65.8	65.7	65.5	65.3	65.3								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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Table10. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on fresh longissimus muscle Hunter L value.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hunter L value																
	High	0	50.70	51.07	51.89	50.78	51.14	53.84	1.7	.01	87	.97	.01	.01	.45	.85
	Moderate		45.95	47.13	48.72	46.97	46.93	47.35								
	High	3	52.17	53.91	54.47	53.14	53.74	55.52								
	Moderate		49.90	49.04	51.09	49.32	50.99	49.06								
	High	6	52.78	53.85	54.71	54.36	54.26	56.56								
	Moderate		50.19	50.30	51.46	50.01	51.50	51.04								
	High	9	53.70	54.88	55.02	54.61	55.18	56.57								
	Moderate		50.95	50.91	52.54	51.14	52.90	51.53								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

Table 11. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on fresh longissimus muscle Hunter A value.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hunter A value																
	High	0	5.24	5.81	5.81	4.86	5.29	5.33	14.0	.05	.53	.16	.01	.01	.45	.85
	Moderate		5.00	5.23	5.07	4.81	4.75	4.67								
	High	3	6.40	5.00	5.42	5.45	5.69	5.82								
	Moderate		6.60	6.78	5.74	6.28	5.90	6.01								
	High	6	5.42	4.39	4.76	4.82	5.18	5.04								
	Moderate		6.18	6.40	5.35	5.71	5.55	5.71								
	High	9	4.76	3.80	4.34	4.23	4.44	4.66								
	Moderate		5.74	5.85	4.96	5.44	5.12	5.20								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

Table 12. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on fresh longissimus muscle Hunter B value.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hunter B value																
	High	0	7.14	6.88	7.29	6.91	7.07	7.82	5.0	.13	.80	.90	.01	.16	.48	.96
	Moderate		6.53	7.69	6.96	6.54	6.91	6.89								
	High	3	8.13	7.92	8.13	7.91	7.83	8.20								
	Moderate		7.69	7.71	7.67	7.44	7.59	7.61								
	High	6	7.87	7.77	8.07	7.90	7.85	8.21								
	Moderate		7.81	7.90	7.66	7.44	7.72	7.68								
	High	9	8.00	7.67	7.98	7.76	7.71	8.16								
	Moderate		7.84	7.71	7.48	7.63	7.76	7.64								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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Table 13. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on post-thaw longissimus muscle Hunter L value.[a]

Item	G	D	Dietary available phosphorus, %						CV	Probability <						
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hunter L value																
	High	0	46.49	46.79	46.11	44.92	45.78	46.66	2.0	.23	.88	.95	.01	.01	.16	.13
	Moderate		44.01	43.89	45.81	44.28	45.11	44.26								
	High	3	49.56	49.42	48.29	47.65	48.74	49.51								
	Moderate		47.56	47.49	47.56	46.98	48.48	48.08								
	High	6	50.05	51.34	51.00	49.98	51.19	50.81								
	Moderate		48.20	48.78	48.89	47.25	48.30	47.99								
	High	9	50.88	52.25	51.39	50.71	51.35	51.45								
	Moderate		49.70	50.69	51.44	48.92	50.80	50.80								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

Table 14. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on post-thaw longissimus muscle Hunter A value.[a]

Item	G	D	Dietary available phosphorus, % .080	.110	.155	.222	.323	.475	CV	G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hunter A value																
High	0	4.99	5.27	5.10	4.89	4.96	5.36	11.8	.01	.18	.38	.01	.01	.86	47
Moderate		5.47	5.57	4.63	4.95	4.72	4.85								
High	3	5.15	4.59	4.62	4.44	4.43	4.86								
Moderate		5.34	5.31	4.76	5.28	5.05	4.96								
High	6	4.30	3.79	3.85	3.77	3.51	3.95								
Moderate		4.90	4.73	4.09	4.82	4.54	4.44								
High	9	3.99	2.80	2.93	3.07	2.82	3.11								
Moderate		4.17	4.37	3.93	4.53	4.25	4.21								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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Table 15. Impact of lean growth genotype (G), dietary available phosphorus (AP) concentration, and day (D) of retail storage on post-thaw longissimus muscle Hunter B value.[a]

| Item | G | D | Dietary available phosphorus, % | | | | | | CV | Probability < | | | | | | |
			.080	.110	.155	.222	.323	.475		G	AP	G x AP	D	G x D	AP x D	G x AP x D
Hunter B value																
High	0		8.07	7.58	7.40	7.19	7.31	7.58	4.4	.87	.84	.36	.01	.56	.10	.06
Moderate			7.01	7.32	7.77	7.19	7.67	7.65								
High	3		8.45	8.21	8.10	8.02	7.95	8.18								
Moderate			8.07	8.03	8.04	8.03	8.12	8.00								
High	6		9.01	8.41	8.22	8.01	8.01	8.32								
Moderate			8.09	8.23	8.25	8.45	8.57	8.51								
High	9		8.21	8.69	8.53	8.12	8.18	8.43								
Moderate			8.24	8.47	8.70	8.23	8.28	8.37								

[a]Raw means of 7 sets of six littermate barrows from each genotype.

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CHAPTER 5. GENERAL SUMMARY

An experiment was conducted to examine the impact of dietary available phosphorus regimen on the rate, efficiency and composition of growth, the efficiency of dietary available phosphorus use for body protein accretion and on post-mortem muscle exudative losses, water holding capacity, and color scores during retail storage of pigs from two genetic strains differing in lean growth capacity. Seven sets of six littermate barrows (initial weight 20 kg) from each of a high and moderate lean growth (LG) genotype were allotted to one of six diets differing in dietary available phosphorus (AP) concentration (.080, .110, .155, .222, .323, and .475%). The muscle growth capacity of the high and moderate LG pigs from 20 to 109 kg body weight was estimated to be 375 and 330 g/d, respectively. Pigs were individually penned and were allowed to consume feed and water ad libitum. Body weight and feed intake were collected at 7 d intervals and empty body composition was assessed at 21 d intervals utilizing a deuterium oxide dilution technique. Upon reaching 109 kg, the pigs were killed for examination of empty body tissue content and post-mortem longissimus muscle (LM) exudative losses, water holding capacity and color. From 20 to 109 kg, high LG pigs consumed less feed, had similar daily BW gains, and required less feed per unit of BW gain than moderate LG pigs. In addition, high LG pigs deposited more muscle and less fatty tissue daily, and produced carcasses with more muscle and less fatty tissue at 109 kg than moderate LG pigs.

As dietary AP concentration decreased, the rate and efficiency of BW gain declined quadratically; however, the magnitude of the decline was greater in the high LG genotype.

Daily gains of muscle, fat, bone, and offal tissue, and body protein, moisture and fat declined

quadratically as dietary AP concentration decreased; however, the magnitude of the decline in

protein gain was greater in the high LG pigs and that of muscle gain and offal gain tended to

be greater. The response to dietary AP concentration was dependent on stage of pig

development. Dietary AP concentrations needed to maximize BW gain, gain/feed ratio, and

body protein gain decreased from .475 to .110 to .155 as pig BW increased from 20 to 109 kg

in both genotypes. Phosphorus requirement seems to be dependent on the rate of muscle and

protein gain as influenced by lean growth genotype and stage of pig development. When

dietary phosphorus is limiting, the rate of high phosphorus containing, muscle growth is

depressed and low phosphorus containing, fat gain increased, possibly due to partitioning of

ingested energy towards fat gain when muscle growth is limited. Based on these data, pigs will

deposit approximately 12 to 15 g of body protein per g of dietary AP.

Longissimus muscle sections from high LG pigs had lower pH 45 minutes post

mortem, larger initial weights, more fat and water, lower water holding capacities and were

lighter in color on d 0, whereas the degree redness and yellowness did not differ between

genotypes. However, the pattern of change in exudative losses, WHC and color that occurred

in retail storage of the LM sections during the fresh and post-thaw states differed between

genotypes. Longissimus muscle sections from high LG pigs had higher exudative losses early

in the storage period and lower exudative losses later than LM sections from moderate LG

pigs in both the fresh and post-thaw state. Longissimus muscles sections became lighter in

color as storage progressed, but the magnitude of the increase was greater in the high LG

pigs. The degree of redness of the LM section increased to d 3 of storage and decreased thereafter in both genotypes, however; the magnitude of the decrease was greater in the high LG pigs.

Dietary AP regimen did not influence body weight gains and gain/feed ratios of pigs from 89 to 109 kg BW. However, feed intake tended to decrease as dietary AP concentration decreased during the last 20 kg of pig growth. Thus, it seems that the dietary AP concentrations utilized in this study were not substantially below the pigs' requirement as they approached market weight. Postmortem muscle sarcoplasmic reticulum Ca^{2+} induced Ca^{2+} ATPase activity and 45 minute pH were unaffected by dietary AP concentration, thus, dietary AP concentration had minimal effect on measures of meat quality.

Decreasing dietary AP concentration decreased the rate of proteinaceous tissue deposition and increased the rate of fatty tissue deposition relative to proteinaceous tissue, but had minimal effect on measures of muscle quality. It seems that a pig requires 1 g of dietary AP per 12 to 15 g of body protein deposited.