THE EFFECT OF EARLY DIETARY AMINO ACID RESTRICTIONS ON SERUM METABOLITES IN PIGS SELECTED FOR LEAN GROWTH EFFICIENCY

Except where reference is made to the work of others, the work described in this thesis is my own or was done in collaboration with my advisory committee. This thesis does not include proprietary or classified information.

_____
Hazarath Reddy Mule

Certificate of Approval:

_____	_____
Werner G. Bergen Professor Animal Sciences	Lee I. Chiba, Chairperson Associate Professor Animal Sciences
_____	_____
Daryl L. Kuhlers Professor Animal Sciences	Lowell T. Frobish Emeritus Professor Animal Sciences

_____
Stephen L. McFarland
Dean
Graduate School

THE EFFECT OF EARLY DIETARY AMINO ACID RESTRICTIONS ON SERUM METABOLITES IN PIGS SELECTED FOR LEAN GROWTH EFFICIENCY

Hazarath Reddy Mule

A Thesis

Submitted to

the Graduate Faculty of

Auburn University

in Partial Fulfillment of the

Requirements for the

Degree of

Master of Science

Auburn University, Alabama

December 16, 2005

THE EFFECT OF EARLY DIETARY AMINO ACID RESTRICTIONS ON SERUM METABOLITES IN PIGS SELECTED FOR LEAN GROWTH EFFICIENCY

Hazarath Reddy Mule

_____
Hazarath Reddy Mule

_____
Date

VITA

Hazarath Reddy Mule, son of Rami Reddy Mule, and Subbalakshmi Mule, was born March 1, 1978 in Siddareddypalli, India, where he was raised with his sisters. In 1995, he graduated from Jawahar Navodaya Vidyalaya (High School) in Cuddapah, India. He attended Acharya N. G. Ranga Agricultural University in Tirupati, Andhra Pradesh, India and graduated with Bachelor of Veterinary Science Degree in November 2001. He entered the Graduate School at Auburn University under the guidance of Dr. Lee I. Chiba. Upon completion of his degree, he plans to work as a veterinarian.

THESIS ABSTRACT

THE EFFECT OF EARLY DIETARY AMINO ACID RESTRICTIONS ON SERUM METABOLITES IN PIGS SELECTED FOR LEAN GROWTH EFFICIENCY

Hazarath Reddy Mule

Master of Science, December 16, 2005
(B. V. Sc & A. H., A. N. G. R. Agricultural University, 2001)

100 Typed Pages

Directed by Dr. Lee I. Chiba

Thirty-two select line and 32 control line pigs (average, 20 kg) were used in each of the two experiments to assess the effect of dietary amino acid restrictions during the grower phase on serum metabolites. In Exp. 1, 16 pens with two gilts and 16 pens with two barrows per pen were assigned within the genetic line to grower diets (6.1 or 11.1 g lysine/kg) and finisher diets (6.1 or 8.9 g lysine/kg) in a 2 x 2 x 2 factorial arrangement of treatments. Similarly, 16 pens with two gilts and 16 pens with two barrows per pen were assigned within the genetic line to grower diets (5.0, 7.0, 9.0, or 11.0 g lysine/kg) in a 2 x 4 factorial arrangement of treatments, and pigs were offered common Finisher 1 and Finisher 2 diets in Exp. 2. Blood samples were collected at the end of the grower and finisher phases, in Exp. 1. Whereas, blood samples were collected at the beginning of the study, at the end of the grower and finisher phases, in Exp. 2. Serum concentrations of

albumin ($P = 0.001$) at the end of the grower phase and cholesterol ($P = 0.029$) and albumin ($P = 0.001$) at the end of the finisher phase were greater in pigs fed the high-amino acid grower diet than those fed the low-amino acid grower diet in Exp. 1. At the end of the finisher phase, triglyceride ($P = 0.029$), albumin ($P = 0.005$), and glucose ($P = 0.027$) concentrations were reduced in pigs fed the high-amino acid finisher diet compared with those fed the low-amino acid finisher diet. The select line pigs had higher concentrations of cholesterol ($P = 0.009$) at the end of the grower phase and triglyceride ($P = 0.036$) and albumin ($P = 0.016$) at the end of the finisher phase than the control line pigs. In Exp. 2, as the amino acid content of the grower diets increased, cholesterol was reduced (linear, $P = 0.005$; quadratic, $P = 0.026$; cubic, $P = 0.039$), but total protein (linear, P = 0.040) and albumin (linear, $P = 0.001$) concentrations were increased at the end of the grower phase. The select line pigs had greater concentrations of triglyceride ($P = 0.001$), total protein ($P = 0.041$), and glucose ($P = 0.005$) at the end of the grower phase and triglyceride ($P = 0.031$) and total protein ($P = 0.001$) at the end of the finisher phase than the control line pigs. Serum cholesterol was correlated negatively with lysine intake (r = -0.38; $P = 0.039$) during the grower phase and urea N (r = -0.39; $P = 0.032$) at the end of the grower phase in Exp. 1 and positively with ultrasound backfat (r = 0.78; $P = 0.001$) at the end of the grower phase in Exp. 2. The results indicated that the metabolite profile can be affected by both early dietary amino acid restrictions and the genotype. By understanding completely the effect of early dietary restrictions and genotypes on metabolite profile, we can contribute greatly to the development of optimum feeding strategies for successful and sustainable pig production.

ACKNOWLEDGEMENTS

The author would like to express his sincere gratitude and appreciation to his advisor, Dr. Lee I. Chiba, for his teaching, supervision, and guidance throughout the graduate program. Gratitude is expressed to Drs. Daryl L. Kuhlers, Lowell T. Frobish, and Werner G. Bergen for their willingness to serve on the advisory committee and support, encouragement, and constructive advice. The author would like to thank the faculty, staff, and graduate students of the Department of Animal Sciences for their support and inputs throughout the program.

Many thanks are extended to Elizabeth M. Whatley, College of Veterinary Medicine, for her technical support during the sample analysis. Sincere appreciation is extended to Dr. Elizabeth G. Welles for her support and guidance throughout the analysis of samples for metabolites. The author is grateful to Dr. Nada K. Nadarajah for providing assistance with the statistical data analyses. The author would like to thank his family for their continued support and encouragement throughout his graduate program.

Style Manual or Journal Used:	Journal of Animal Science
Computer Software Used:	Microsoft Office XP, Statistical Analysis System (SAS) Software Package for Windows v. 8.02.

TABLE OF CONTENTS

LIST OF TABLES

APPENDICES

I. INTRODUCTION

Pigs, like other species, may exhibit compensatory growth after a period of early dietary restrictions (Robinson, 1964; Wahlstrom and Libal, 1983; Crister et al., 1995). The nature and extent of compensatory response may depend on many factors, such as the type, severity, and duration of dietary restrictions, the age of the animal, and the nutritional management in the subsequent phase (Lawrence and Fowler, 1997; Chiba et al., 1999). In addition, many researchers have demonstrated the differences in growth performance, hormone and metabolite concentrations, and body composition in pigs because of genetic variations.

It is reasonable to assume that compensatory responses in growth performance are a reflection of changes in metabolism. This contention is supported by findings of several researchers (Campbell et al., 1983c; Prince et al., 1983; Valaja et al., 1992; Chiba et al., 2002) who reported that pigs subjected to a period of early dietary restrictions had better feed efficiency in the subsequent phase than those unrestricted pigs.

Selection of pigs for important traits over time has been an integral part of the survival and success of commercial pig production (Chiba et al., 2002). Partly because of those efforts, wide variations in the pigs's potential for growth and protein accretion continue to exist in today's pig industry. To maximize the economic efficiency, supplying essential nutrients as close as possible to meeting, but not exceeding, the requirements is advantageous (Chiba et al., 2000), which can also have a positive impact on the environment by reducing the excretion of unutilized nutrients. Such optimum feeding

strategies involve consideration of a multitude of factors, including the genetic variation in pigs. Pigs with distinct genotypes are likely to respond differently to the concentrations of nutrients or dietary manipulations simply because of metabolic and physiological alterations taking place in pigs selected for specific traits. Understanding fully the effect of dietary restrictions and genotype on growth performance, carcass traits, physiology, and metabolism is essential for developing environmentally friendly, optimum feeding strategies for successful and sustainable pig production.

II. LITERATURE REVIEW

Compensatory Growth in General

The phenomenon of compensatory growth has long been recognized as having the potential to have profound effects on the rate of growth and body composition of most species. Wilson and Osbourn (1960) mentioned that one of the first references on the subject was made by Rubner in 1908, who reported that beef steers subjected to early undernutrition recovered subsequently and reached "normal" mature weight and height at the same time as the contemporary group. Osborne and Mendel (1915) described how rats, which had been restricted growth during various stages of development, exhibited a greater rate of gain once the restriction was removed.

Bohman (1955) termed the faster rate of growth "compensatory growth." On the other hand, others (e.g., Yu and Robinson, 1992) feel that catch-up growth is a better term because the word "compensatory" implies the increased growth of a body part in compensation for the loss of the part or its function. Hence, depending on the author and their preference, these terms are used interchangeably, even though the term compensatory growth has been used for most of the investigations with farm animals.

Following the earlier report by Osborne and Mendel (1915), other researchers have demonstrated compensatory growth in a number of species, including beef cattle (e.g., Horton and Holmes, 1978; Rompala et al., 1985; Hayden et al., 1993), swine (e.g., McMeekan, 1940; Robinson, 1964; Zimmerman and Khajarern, 1973; Mersmann et al.,

1987; Kyriazakis et al., 1991, Chiba et al., 2002), and poultry (e.g., Wilson and Osbourn, 1960; Deaton et al., 1973; Zubair and Leeson, 1994).

Dietary Restrictions and Compensatory Growth in Pigs

As in other species, the concept of compensatory growth in pigs has been an area of research interest for many years. The effect of dietary restrictions during the suckling, starter, grower and even early finisher phases on subsequent and overall growth performance and carcass characteristics have been investigated under different experimental conditions.

Dietary Restrictions During the Suckling and Starter Phases. Campbell and Dunkin (1983a) indicated that the protein restriction between 1.8 and 15 kg BW reduced the animal's potential for subsequent protein accretion and growth, even though the extent of those effects seemed to be influenced by energy intake in the realimentation period. They concluded that feeding protein deficient diets during the early stages of growth would have detrimental effects on pigs because it would increase the time and feed required to reach a normal slaughter weight. In another study, the same protein restriction during early life reduced the DNA content of muscle and adipose tissues, indicating the adverse effects of the protein restriction on the development of those tissues (Campbell and Dunkin, 1983b). Those effects on DNA were still evident at 45 kg, but diminished when pigs reached 60 kg BW.

Chiba (1995) studied the effect of simple (corn-soybean meal) or complex (containing various special ingredients) starter diets on subsequent and overall growth and carcass traits. During the starter phase, pigs offered the complex starter diet grew

faster and more efficiently than those offered the simple starter diet. The starter diet, however, had no effect on weight gain of pigs in the subsequent phases, indicating that pigs offered the simple starter diet did not exhibit compensatory growth. The advantages of better early nutrition were maintained, as the time required to reach the slaughter weight from the beginning of the starter phase was 5.8 d less for pigs fed the complex diet than the ones fed the simple starter diet. They concluded that there was no compensatory growth in pigs fed the simple starter diet. Thus, the results indicated that formulating diets to promote optimum performance of starter pigs could be justified.

On the other hand, Kornegay (1974) reported that pigs grew faster when they were fed starter diets containing whey, but compensatory growth at latter stages removed the differences by the time the pigs reached market weight. Similarly, Campbell and Biden (1978) indicated that advantages of better nutrition during the starter phase disappeared by the time pigs reached 70 kg BW. Also, Valaja et al. (1992) reported that the restricted pigs grew faster and more efficiently in the realimentation period, eventhough the compensation was not complete.

The findings on the effect of dietary restrictions during the starter phase have been rather inconsistent. Thus, this may indicate that pigs can be offered a simple starter diet and(or) a diet slightly deficient in nutrients, without any adverse effects on overall growth rate or final carcass traits. However, there is some evidence that formulating diets to promote optimum performance during the starter phase can be justified because advantages can be maintained up to the market weight.

Dietary Restrictions During the Grower Phase. Prince et al. (1983) investigated the effect of short-term feed restrictions during the grower phase on pig performance. The short term feed restriction caused compensatory responses in growth rate in the subsequent phase, but it also resulted in improved efficiency during the realimentation phase. This indicates that the restriction resulted in a more efficient digestion, absorption, and(or) utilization of nutrients during the realimentation phase.

The amino acid restrictions during the grower phase and the amino acid content of the finisher diets on growth performance and carcass quality have been investigated (Chiba, 1994, 1995; Chiba et al., 1999, 2002). The results of earlier studies indicated that pigs fed the low-amino acid diet during the grower phase grew faster and more efficiently in the subsequent phase than those fed the high-amino acid diet (Chiba, 1994, 1995). Because of this, there was no difference in the overall growth performance or carcass quality of pigs fed the different grower diets, indicating pigs subjected to early dietary amino acid restrictions compensated completely. In the later studies, overall weight gain was reduced in pigs fed the low-amino acid grower diet, but those pigs had a similar lean accretion rate than pigs fed the grower diet high in amino acids (Chiba et al., 1999, 2002). The results indicated that compensatory response in the lean accretion may have taken place in restricted pigs. In addition, pigs fed the low-amino acid grower diet had improved feed efficiency in the subsequent phase compared with pigs fed the high-amino acid diet (Chiba et al., 2002).

In another study, Fabian et al. (2002) investigated the effect of dietary amino acid contents during the grower phase and pigs selected for lean growth efficiency on growth

performance, carcass traits, and meat quality. As the amino acid content of grower diets increased, pigs consumed less feed and more lysine and utilized feed more and lysine less efficiently for weight gain during the grower phase. Increasing the amino acid content of grower diets resulted in less ultrasound backfat and more serum urea nitrogen at the end of the grower phase. The grower diet, however, had no effect on the overall weight gain and feed efficiency, carcass traits, or meat quality scores. They indicated that pigs subjected to amino acid restrictions exhibited compensatory growth regardless of the genotype.

Based on these studies, it is very likely that pigs can be offered grower diets that are marginally deficient in amino acids without affecting overall growth performance or carcass traits. This may also improve overall efficiency of feed and(or) amino acid utilization.

Dietary Restrictions During the Early Finisher Phase. The effect of dietary energy densities and lysine:energy ratios during the grower to early finisher phases on growth performance and carcass characteristics of pigs was investigated by Smith et al. (1999). Increasing the dietary energy density and lysine:energy ratio improved growth rate and feed efficiency of growing pigs. However, pigs fed a low-energy diet or a diet with a low-lysine:energy ratio from 30 to 73 kg BW exhibited compensatory growth in the subsequent finisher phase. In addition, carcass characteristics were not affected by the energy density or lysine:energy ratios, indicating that pigs subjected to dietary restrictions during the grower to early finisher phases were able to compensate fully.

Protein Concentrations of Realimentation Diets. Crister et al. (1995) investigated the effect of dietary protein concentrations on compensatory growth. Pigs were fed one of five diets (13.1 to 18.4% CP) from approximately 40 to 100 kg BW, or fed a diet containing 14.8% CP at maintenance for 3 wk and then allowed ad libitum access to one of the five diets. Restricted pigs had lower feed intake and grew slower and less efficiently than the non-restricted pigs. During the realimentation phase, previously restricted pigs consumed more feed, and grew faster and more efficiently than the non-restricted pigs. Their results indicate that a compensatory growth response occurred during realimentation, but the effects of dietary protein concentration on growth rate and carcass measurements were similar in both unrestricted and restricted pigs. Thus, the results of their study showed no need for increased dietary protein concentration during realimentation. Likewise, other studies reported that there was no indication that pigs subjected to early dietary restrictions have different amino acid needs in the subsequent phase (Chiba, 1994, 1995; Chiba et al., 1999, 2002).

In summary, it seems that pigs subjected to dietary restrictions during the early stage of development may never fully compensate. However, it is most likely that once pigs pass the early, critical stages of development, the degree of compensation can become an issue. Grower diets that are marginally deficient in amino acids may not adversely affect overall growth performance and(or) carcass traits and may improve overall efficiency of amino acid utilization for weight gain and lean accretion. There was no indication that pigs subjected to dietary restriction have different amino acid needs in the subsequent phase.

Factors Affecting Compensatory Growth Response

Stage of Maturity of Animal at the Time of Restriction. Wilson and Osbourn (1960) stated that undernutrition during the earlier stages of growth is more detrimental to an animal than the restriction at a later stage. Consequently, the age at which an animal is subjected to undernutrition may be as important as the severity of undernutrition.

In beef cattle, most research involves feed restriction starting at around 240-270 kg, corresponding to the age of 7-10 months. However, to see the beneficial effects of compensatory growth, the age and weight ranges, at which the feed restriction should start with steers, has not been defined well. It would seem, however, the initial age or weight may not be critical in steers (Ryan et al., 1993).

In many pig studies, the protein restriction was implemented at about 15-25 kg body weight (Wahlstrom and Libal, 1983; Prince et al., 1983). However, Stamataris et al. (1985) restricted pigs to 300 grams of feed per day over the weight range of 6 to 12 kg live weight, and pigs were then offered feed ad libitum. The restricted pigs took 31.7 days to reach 12 kg while those offered ad libitum feed took only 12.6 days. Upon realimentation, the restricted pigs took 5.5 days less to reach 24 kg than did those offered ad libitum feed. So, pigs restricted between 6 to 12 kg live weights did not exhibit compensatory growth.

It would seem that dietary restrictions during the earlier stages of growth are more detrimental than at a later stage. However, the age and weight at which feed restrictions can be implemented has not been defined well to take advantages of compensatory growth.

Genotype and Sex of the Animal. Males and females may respond differently to dietary restrictions simply because of the relative rate, at which an animal matures, may affect the degree of recovery after a period of nutritional stress (Wilson and Osbourn, 1960). Kyriazakis et al. (1991) found no difference in the growth rate between male or female pigs previously fed a low-protein diet upon realimentation after sexual maturity. de Greef et al. (1992) found that two different strains of pigs responded similarly to the realimentation in terms of live weight, they had different fat to lean deposition ratios during the restriction and realimentation. This indicated that the partitioning of energy and other nutrients into protein and lipid tissues was different for the two strains of pigs, emphasizing the importance of designing feeding strategies for different genotypes to optimize carcass quality (de Greef et al., 1992). On the otherhand, Hogberg and Zimmerman (1978) found that, unlike an obese strain, a lean strain of pig exhibited little growth compensation possibly because the protein restriction they used was too severe for leaner pigs. However, the two strains of pigs used by Hogberg and Zimmerman (1978) differed considerably more than those used by de Greef et al. (1992), indicating the importance of the degree of divergence in genetic differences. Chiba et al. (1999) also reported that the two strains of pigs with slight genetic differences responded similarly to early dietary restrictions.

Duration and Severity of Dietary Restrictions. During a period of dietary restriction, animals may be fed at below the maintenance energy requirements to see the compensatory growth. Wilson and Osbourn (1960) reported that the more severe the restriction, the greater the initial rate of gain immediately after the realimentation.

Prince et al. (1983) restricted pigs to 70 or 85% of ad libitum feed intake for either 2 or 4 weeks. Those restricted to 70% of ad libitum intake for 4 weeks were unable to fully compensate, indicating that the restriction was either too severe and(or) too long. But, upon realimentation, the pigs restricted to 70% of ad libitum for only 2 weeks showed average daily gain and feed to gain ratio similar to those of the control and the 85% restricted groups.

Pond and Mersmann (1990) restricted the feed intake of weanling pigs such that they lost weight over a 21-day period. In this trial, energy intake was restricted to see if a compensatory response could be obtained because their earlier work (Pond et al., 1980) has shown that early protein restriction had little effect on response to realimentation. There was no evidence of compensatory growth in terms of weight gain during the realimentation phase. However, they saw compensatory responses in the liver and kidney weights and backfat depth. The degree of restriction used in their study was, perhaps, too severe, for the pigs to recover. As stated by Mersmann et al. (1987), the magnitude of increase in the growth rate following the feed restriction may well be affected by the physiological changes imposed by the weight loss during the restriction.

Feed Intake During Realimentation. The amount of feed consumed during the realimentation may have an effect on compensatory growth (Ryan et al., 1993). However, cattle and swine have shown varying results with respect to the amount of feed consumed during the realimentation. Ryan et al. (1993) found that steers that were previously restricted had a greater feed intake for approximately 140 days during the first part of the realimentation than non-restricted control animals, and the compensatory response could

be explained entirely on the basis of higher feed intake. Thus, Ryan et al. (1993) postulated that the greater feed intake as the main mechanism responsible for a long term compensatory growth, supporting the conclusions of Graham and Searle (1975). However, other workers (Coleman and Evans, 1986; Wright and Russel, 1991) have found that a period of feed restriction has resulted in a decrease in the total amount of feed required to reach a given weight in beef cattle.

Wahlstrom and Libal (1983) and Pond and Mersmann (1990) found no evidence of increased feed intake in previously restricted pigs fed ad libitum during the realimentation. Similar results were reported by de Greef et al. (1992) who found that pigs fed low-protein diets had reduced feed intakes during the restriction period, which carried over into the realimentation phase. The pigs, however, demonstrated compensatory growth responses, even though the compensation was not complete (de Greef et al., 1992). On the otherhand, the increase in feed intake and daily gain after a period of early feed restriction has been reported by other workers (Owen et al., 1971; Donker et al., 1986). Bikker et al. (1996) demonstrated that the feed intake was similar for the restricted and non-restricted gilts during the realimentation.

In summary, it seems that dietary restrictions during the earlier stages of growth are more detrimental than later stages of growth. However, the age and weight at which feed restrictions can be implemented are not defined well. Pigs with differences in genotypes would respond similarly to dietary restrictions.

Possible Mechanisms of Compensatory Growth

The mechanisms underlying compensatory growth are not clear. Decreased maintenance costs, increased feed intake, increased efficiency of growth, and hormonal changes during the realimentation phase have been suggested as possible mechanisms. Obviously, the increased growth rates observed in the animal exhibiting compensatory growth must be the result of an increased quantity of "available" nutrients to that animal.

Reduced Maintenance Energy Requirement. The reduction in maintenance energy during the restriction phase and in the subsequent phase may allow for more energy for growth upon realimentation, thus contributing to the compensatory growth response. Yambayamba et al. (1996) measured heat production (HP) and noted a decline in HP during the restriction in steers. Following the realimentation, HP remained lower in restricted steers than the controls during the first two weeks of the post-realimentation, but it slowly rose to levels similar to the control steers by five weeks of the post-realimentation. Therefore, the reduced HP has been implicated as being partially responsible for the compensatory growth observed in the steers. Considering that the higher rates of gain were seen up to 14 weeks following the realimentation (Yambayamba et al., 1996), there had to be other mechanisms responsible for the compensatory growth. Zubair and Leeson (1994) observed the reduced metabolic rates during the dietary restriction in broilers. However, measurements taken five days after re-feeding showed no differences in metabolic rates, leading them to conclude that other mechanisms must be involved. Thus, the reduced maintenance costs seemed to be only responsible for a short term compensation.

Increased Feed Intake. Many researchers have implicated increased feed intake as the main mechanism responsible for compensatory growth. Yambayamba et al. (1996) did not observe an increase in feed intake in restricted steers compared with the control steers at the same age. However, Yambayamba et al. (1996) stated that, if feed intake had been measured as a percent of body weight, differences in feed intake would have been observed. In cattle, the increase in feed intake in restricted-realimented animals has been 5 to 17 percent compared with full-fed control animals. On the otherhand, Wyllie et al. (1969) and de Greef et al. (1992) observed a decreased feed intake during realimentation after a period of protein restriction in pigs. However, pigs exhibited compensatory growth responses despite decreased feed intake, even though the compensation was not complete.

Increased Efficiency of Growth. An alteration in protein and lipid deposition seems to be another mechanism involved in compensatory growth. The increased efficiency of protein deposition and concomitant water deposition in restricted animal results in more lean tissue deposited than fatty tissue deposited. This higher rate of lean deposition during the realimentation in animals subjected to the earlier restrictions would have a significant impact on the overall growth rate. de Greef et al. (1992) observed a higher fatty tissue deposition rate than the lean tissue deposition rate in protein restricted pigs during the restriction phase. Upon realimentation, lean tissue deposition rate was higher in the restricted group, and no difference was observed in the lipid deposition rate between the two groups. de Greef et al. (1992) concluded that the greater the severity of

the protein restriction, the greater the effect of the increased lean tissue deposition rate have on compensatory growth responses.

Hormonal Changes. The actions of various hormones are probably an integral part of the compensatory response (Blum et al., 1985; Breier et al., 1986). However, the role of various hormones in compensatory growth still remains somewhat obscure because of lack of research on this area. The effect of growth hormone (GH), insulin-like growth factor-I (IGF-I), and insulin during the feed restriction and realimentation has undergone the most intensive research to date (Atinmo et al., 1976; Yambayamba et al., 1996). The experimental results of Yambayamba et al. (1996) showed that inspite of elevated plasma GH concentration during the feed restriction, the IGF-I concentration was low due to insensitivity of hepatic and extra hepatic tissues. This uncoupling of the GH-IGF-I axis seems to be important in compensatory growth as it is involved in protein synthesis and growth of the animals.

Effect of Dietary Restrictions and Genotypes on Blood Hormones

Hormone regulation plays an integral role in growth and development. Therefore, it is safe to assume that hormones can drive the metabolic activity and function of the body during the restriction and realimentation. Some hormones like GH and IGF-I, insulin, and leptin are involved in the protein, glucose and(or) lipid metabolism. So, these hormones control growth and affect the metabolite concentrations in the body.

Growth Hormone and Insulin like Growth Factor-I in General. Metabolic hormones such as GH and IGF-I, as well as insulin, have been shown to be involved in growth. Mean plasma GH concentration is elevated, but IGF-I concentration is reduced

during chronic feed restriction in cattle (Breier et al., 1986). When feed-restricted cattle are realimented, there is a reversal in the GH and IGF-I concentrations, i.e., GH decreases as IGF-I increases (Blum et al., 1985; Ellenberger et al., 1989). This reversal may affect the utilization of nutrients for growth.

Growth Hormone, Insulin like Growth Factor-I and Genotypes. McCusker et al. (1984) reported that plasma GH increased with fasting in both obese and lean pigs. But, it has been reported that GH concentration was lower in obese pigs than in lean pigs (Wangsness et al., 1981). A higher somatostatin response to feeding in obese pigs could be responsible for the lower basal GH concentration during normal feeding in obese pigs. The greater concentration of IGF-I was found in a line of swine simultaneously selected for increased growth rate and decreased backfat thickness than a line of pigs selected in the opposite direction (Lund-Larsen and Bakke, 1975).

Growth Hormone, Insulin like Growth Factor-I and Nutritional Status. The IGF-I is a peptide hormone and known as a protein synthesis factor. The IGF-I partly mediates the growth, promoting actions of GH when the nutritional status is adequate (Buonomo et al., 1987). Under normal physiological conditions, the major regulatory factor influencing the synthesis and secretion of IGF-I in swine is GH. For example, GH administration is associated with an elevation of circulating concentrations of IGF-I in swine (Buonomo et al., 1987). Although GH is a primary stimulus for the IGF-I synthesis, other factors such as nutrient availability and other hormone status are important modulators of IGF-I concentration. Nutritional status of an animal influences IGF-I concentration, i.e., fasting

or feeding a diet deficient in energy or protein is associated with reduced plasma concentration of IGF-I.

Restrictive feeding during the grower phase decreased serum concentration of IGF-I in pigs (Deng YueLin and Oksbjerg, 2002). The depression in circulating IGF-I concentration following the nutritional deprivation seems to be due to a reduction in the synthesis and(or) secretion of IGF-I (Emler and Schalch, 1987). Fasting has been reported to reduce IGF-I mRNA concentrations in hepatic and in most extra hepatic tissues, but fasting does not alter the half-life of circulating IGF-I. The dietary restriction alters the GH dependency of IGF-I and induces a state of growth hormone resistance in swine (Burleigh et al., 1987). Similarly, maintenance of normal circulating concentration of IGF-I is dependent on factors other than GH when nutrient supply is limiting (Buonomo and Baile, 1991). Increasing the degree of feed restriction can cause progressive growth retardation, and the degree of reduction in the plasma IGF-I concentration seemed to be related to the magnitude of feed restriction (Leili et al., 1997). A tendency for the greater decrease in IGF-I concentration seemed to be associated with greater feed restriction (Leili et al., 1997).

In summary, the GH and IGF-I axis seems to be uncoupled, the concentration of GH increased, and concentration of IGF-I decreased during the feed restriction. The concentration of IGF-I was found to be greater in a line of swine selected for increased growth rate and decreased backfat thickness. The IGF-I concentration during the feed restriction seemed to be dependent on the degree of feed restriction.

Insulin in General. One of the major roles of insulin is to increase peripheral uptake and utilization of glucose. In particular, insulin plays an important role in lipid metabolism, stimulating lipogenesis by transport of glucose into cells, and by activating pyruvate dehydrogenase and acetyl-coA carboxylase and inhibiting lipolysis.

Insulin and Genotypes. Plasma insulin concentration has been shown to be similar in young Ossabaw and Yorkshire pigs after overnight fasting despite the greater degree of adiposity in the Ossabaw strain (Cote and Wangsness, 1978). But, Mersmann et al. (1982) indicated that plasma insulin concentration is lower in lean than in obese pigs, and plasma insulin concentration has been shown to be positively related to carcass fatness in pigs (Wood et al., 1977).

Insulin and Nutritional Status. The postprandial insulin concentration was lower in the restricted pigs than in control pigs (McNeel et al., 2000). Buonomo and Baile (1991) observed that the plasma insulin concentration was reduced within 24 h after removal of the feed. The effects of feed restriction on pre- and postprandial concentrations of insulin secretion probably depend on the degree of undernutrition. For example, when the feed allowance is lower than the maintenance requirement, body reserves can be mobilized and not enough insulin to inhibit lipolysis, therefore, concentrations of free fatty acids are increased and increased protein catabolism resulting in increased urea concentrations (Armstrong and Britt, 1987). Insulin is implicated in postprandial protein synthesis by increased uptake of amino acids and inhibiting protein catabolism (Preedy and Garlick, 1986). Although insulin would be expected to have a major endocrine influence on lipid metabolism, there is little evidence that an alteration

in insulin concentration or carbohydrate metabolism is playing a major role in the development of obesity in pigs (Mersmann, 1991).

The carcass fatness seems to be positively related to insulin concentration, so the lean pigs have a lower concentration of insulin than the obese pigs. Insulin concentration fluctuates with the nutritional status of the animal and it seems to be dependent on the degree of undernutrition.

Leptin in General. Leptin, a recently discovered protein synthesized and secreted by the adipose tissue, may act as a circulating signal of nutritional status affecting feed intake and energy metabolism (Houseknecht et al., 1998; Barb, 1999). The expression and secretion of this hormone may be related to the mass of body fat and the size of adipocytes (Houseknecht et al., 1998).

Leptin and Genotypes. Ramsay et al. (1998) demonstrated a higher leptin mRNA concentration in adipose tissues from obese than lean pigs. Plasma leptin concentration was greater in obese than in contemporary crossbred pigs. Leptin mRNA concentration was elevated in adipose tissues from Zucker obese rats compared to lean controls (Rayner et al., 1997). Thus, in pigs and rats, many of adipocyte transcripts involved with controlling adipocyte differentiation and function are elevated in adipose tissues from obese compared to lean animals (McNeel et al., 2000).

Leptin and Nutritional Status. It has been demonstrated that administration of leptin can suppress feed intake in pigs (Barb et al., 1998). On the other hand, Cameron et al. (2000) reported that serum leptin was more highly correlated with fat deposition than feed intake, indicating that the response in serum leptin in pigs selected for high or low

daily feed intake was primarily due to increased fat deposition. The leptin concentration has been shown to be positively correlated with the carcass backfat thickness (Ramsay et al., 1998; Cameron et al., 2000; Fabian et al., 2003).

A higher leptin concentration in obese pigs compared with lean pigs and a positive correlation with fatness show that leptin is a good indicator of fatness in pigs. It seems that leptin is an indicator of nutritional status of the body and controls feed intake and energy metabolism.

Effect of Dietary Restrictions and Genotypes on Blood Metabolites

Blood metabolite concentrations fluctuate with the nutritional status, and attempts have been made to use these fluctuations as indicators of the adequacy of feeding management. The utilization of absorbed nutrients is under control of the endocrine system. Variations observed in postprandial concentrations of metabolites reflect some of the metabolic changes that an animal undergoes following feeding.

Cholesterol in General. Cholesterol is a soft, waxy substance found among the lipids (fats) in the blood stream and in all body cells. It's an important part of the body because it is used to form cell membranes, some hormones, and is needed for other functions. The pig is accepted as an appropriate animal model in which to assess the relationship between plasma cholesterol concentration and coronary artery disease in humans (Pond et al., 1986b). The heritability (h^2 = 0 .25 to 0.45) of plasma cholesterol concentration is high in pigs (Pond et al., 1986b). High-fat, high-cholesterol diets can produce atherosclerosis in the coronary arteries and aorta of some genetic lines of swine (Pond et al., 1986a).

Cholesterol and Genotypes. Pond et al. (1985) investigated the effect of obesity on plasma lipid and aortic responses to a diet low in fat and cholesterol and the other with added beef tallow and dried egg yolk in swine. They observed rise in plasma cholesterol in lean and obese pigs regardless of diets. Obese pigs had lower plasma cholesterol than lean pigs initially, but they observed the opposite effect at 6 months of age. They suggested that the genetically controlled obesity is not associated with increased plasma cholesterol compared with lean pigs. The plasma cholesterol was increased by low dietary protein and high dietary fat and cholesterol in genetically lean and obese pigs, but genetically obese pigs showed a smaller percentage rise than lean pigs in plasma cholesterol when fed low protein diets (Pond et al., 1986a). McNeel et al. (2000) reported that plasma cholesterol concentration was greater in obese than in lean pigs and no difference in pigs fed ad libitum or 50% of ad libitum intake.

Pond et al. (1992) used female pigs selected for three generations for high and low-serum cholesterol at 56 d of age to test the hypothesis that the two populations would respond differently to a high-fat, high-cholesterol diet and a low-fat, low-cholesterol diet. Initial serum total cholesterol concentration was higher in high-cholesterol pigs than in low-cholesterol pigs, and there was no effect of genetic background on high-density lipoprotein-cholesterol concentration. The serum cholesterol concentration was high in pigs fed the high-fat, high-cholesterol diet regardless of pig type. Thus, the data did not support the hypothesis that growing pigs selected for high serum cholesterol may respond differently to dietary manipulations compared with those selected for low serum cholesterol.

Cholesterol and Dietary Restrictions. Friesen et al. (1995) used high-lean growth gilts to determine the apparent digestible lysine requirement. They observed that total plasma cholesterol decreased and then increased as digestible lysine increased from 0.44 to 0.94% at day 14, but no difference was observed at day 28. They indicated that the increased dietary lysine did not influence either plasma or tissue cholesterol content.

The plasma cholesterol concentration in obese pigs is higher than lean pigs. The genetically controlled obesity is not associated with increased plasma cholesterol concentration. The cholesterol concentration seems to be unaffected by the lysine concentration of the diet.

Triglycerides in General. Triglycerides are the chemical form in which most fat exists in food as well as in the body. They're also present in blood and, in association with cholesterol, form the plasma lipids. Triglycerides in plasma are derived from fats eaten in foods or made in the body from other energy sources like carbohydrates. Calories ingested in a meal and not used immediately by tissues are converted to triglycerides and transported to fat cells to be stored. Hormones regulate the release of triglycerides from the fat tissue so that the animal can meet the body's needs for energy between meals.

Triglycerides and Genotypes. Fabian et al. (2003) investigated the effect of genotype on the serum profile in pigs and reported that there were some differences in serum triglyceride concentrations between pigs selected for lean growth efficiency and their contemporary control line pigs. They indicated that these differences might be due to the selection pressure for lean growth efficiency. It has been reported, however, that

genetically controlled obesity per se (i.e., obese line of pigs) is not necessarily associated with the increased plasma triglycerides compared with lean pigs (Pond et al., 1985).

The fatty acid and the triglyceride synthesis rates are greater in the adipose tissue obtained from obese than in that from lean pigs (Steele et al., 1974; Rule et al., 1989). Lipoprotein lipase cleaves fatty acids from circulating triglycerides to allow fatty acids to enter the adipocyte, and it has been reported that the lipoprotein lipase activity was greater in the adipose tissue from obese than in that from lean pigs (McNamara and Martin, 1982). The triglyceride concentration was higher for the high-total plasma cholesterol line pigs than the low-plasma total cholesterol line of pigs selected for seven generations (Pond et al., 1997). Yambayamba et al. (1996) observed that the concentration of NEFA increased as the feed restriction progressed, indicating a more negative energy balance in those restricted animals, and therefore the increased need to mobilize body lipids.

Triglycerides and Dietary Restrictions. Friesen et al. (1995) observed that plasma triglyceride concentration increased at day 14 as digestible lysine increased. However, it decreased at day 28, indicating that the dietary lysine seems to have some effect on the plasma triglyceride content.

During the feed restriction, glucose concentration is decreased and lipids are mobilized and used as a source of energy. Therefore, feed restriction increases the concentration of free fatty acids. The triglyceride concentration seems to be closely associated with cholesterol and high for a line of pigs selected for high total plasma

cholesterol concentration. It seems that the dietary amino acid content has no effect on the plasma triglyceride concentration.

Total Protein and Albumin in General. Total protein can be reflection of the nutritional status of animal. Serum proteins are grossly separated into albumin and globulins, i.e., total protein equals albumin plus globulin. Albumin is the protein of highest concentration in the serum. Albumin is a carrier of many small molecules, but is also of prime importance in maintaining the osmotic pressure of the blood. Serum total protein and albumin can be sensitive indicators of dietary protein status (Lowrey et al., 1962).

Total Protein and Albumin and Genotypes. McNeel et al. (2000) found higher plasma protein and albumin concentrations in obese than lean pigs. However, there were no differences in plasma protein and albumin concentrations in pigs given ad libitum feed or 50% of ad libitum for 5 wk.

Total Protein and Albumin and Dietary Restrictions. Protein restriction, but not energy restriction, can reduce serum total protein and albumin in early post weaning life of the pig (Atinmo et al., 1976). Pond et al. (1980) investigated the effect of early protein deficiency on, among others, serum total protein and albumin in the lean or obese line of pigs. They found that the serum total protein concentration was reduced by feeding the low-protein diet for 8 weeks in both genetic lines, but serum albumin was reduced more in lean than in obese line pigs. Serum albumin concentrations, but not serum total protein concentrations, returned to normal in both lean and obese line pigs after 4 weeks of

realimentation. They suggested that genetically obese pigs are less severely affected by the dietary protein restriction than lean pigs during the early post weaning period.

Pond et al. (1986a) investigated the interactive effect of high-fat and high-cholesterol diet with low- and high-dietary protein contents in genetically lean and obese pigs. They noted that plasma total protein was greater in obese than in lean pigs and greater in pigs fed high-protein than in those fed low-protein diet. The trends observed for plasma albumin were similar to that of plasma protein, i.e., obese pigs had higher albumin concentration than lean pigs and pigs fed a high-protein diet had a higher albumin concentration than those fed a low-protein diet. Fabian et al. (2004) observed a similar result in serum total protein concentration with grower-finisher pigs.

The total protein and albumin concentrations are reflection of the protein status of the animal. The serum total protein and albumin concentrations seem to depend on the protein content of the diet. During the early post weaning period, genetically obese pigs seems to be less severely affected by the dietary protein restriction than lean pigs, as daily feed consumption and feed/gain during depletion were not affected in obese pigs.

Glucose in General. Glucose, a simple sugar, is one of the most important carbohydrates and is used as a major source of energy. Most dietary carbohydrates eventually end up as glucose in the blood. Excess glucose is converted to lipids and some to glycogen for storage by the liver and skeletal muscles after meals. Glycogen is gradually broken down to glucose and released into the blood by the liver between meals. The major hormone regulating glucose concentration in the body is insulin.

Glucose and Genotypes. Cote et al. (1982) showed that Ossabaw, obese type, pigs exhibited a lower glucose concentration when compared with Yorkshire pigs. Plasma glucose concentrations in lean and obese pigs were influenced by the timing of food removal. Immediately after feed removal, insulin concentration was increased in obese pigs but glucose concentration was similar to those in lean pigs. At the end of the 24 h fasting period, insulin concentrations were similar, but glucose was lower in obese pigs than in lean pigs (Cote et al., 1982). Fabian et al. (2003) reported that the select line pigs had a higher glucose concentration than the control line pigs. McNeel et al. (2000) found that fasting plasma glucose concentration between the two genetic groups was not different, but plasma glucose concentration was lower in pigs fed 50% of ad libitum than in pigs had ad libitum access to feed regardless of the genotype (i.e., obese and lean lines of pigs).

Glucose and Dietary Restrictions. In swine, a little variation in plasma glucose concentration was detected during 48 h of feed deprivation (Baetz and Mengeling, 1971). Glucose concentration decreased in fasted animals prior to changes in insulin secretion (Barb et al., 2001). Stability of glucose concentration despite the feed restriction could be partly explained by decreased insulin and IGF-I concentrations, which may have lowered tissue mobilization of glucose and amino acids (Ouellet et al., 2001). Fabian et al. (2004) observed that pigs fed the low-amino acid diet had a higher glucose concentration than the pigs fed the high-amino acid diet. They suggested that this is due to the high carbohydrate concentration of the low-amino acid diet and(or) decrease in insulin concentration associated with the protein restriction (Atinmo et al., 1976).

Gomez et al. (2002) conducted an experiment to evaluate, among others, plasma metabolites of pigs fed corn-soybean meal diet, low-protein diet supplemented with appropriate amino acids, and corn-soybean meal diet supplemented with amino acids. They found that plasma glucose concentration was lower in pigs fed the corn-soybean meal diet supplemented with amino acids than in pigs fed the low-protein diet supplemented with appropriate amino acids.

There seem to be some differences in glucose concentrations of lean and obese pigs. The glucose concentrations highly fluctuate with the nutritional status of the animal, and depend on the degree of feed restriction. The high carbohydrate content of the low protein diet may result in high glucose concentrations.

Summary

As in other species, the concept of compensatory growth in pigs has been an area of research interest for many years. The effect of the dietary restriction during the suckling, starter, grower and even early finishing phases on the subsequent and overall growth performance, carcass characteristics, and hormone and metabolite profiles have been investigated under different experimental conditions. Some researchers described the factors affecting compensatory growth and possible mechanisms responsible for the compensatory growth.

In general, nutritional restrictions during the very early, critical stages of development may result in no or partial compensation. The findings on the effect of dietary restrictions during suckling and starter phases indicate that pigs offered simple starter diet resulted in no compensatory growth. So, offering a complex diet with various

ingredients would be beneficial during the early stages. On the other hand, pigs during the grower phase can be offered diets that are marginally deficient in amino acids without adversely affecting overall growth performance.

Compensatory growth can be affected by the age of animals at the time of restriction. The undernutrition in the earlier stages of growth is more detrimental than at a later stage. Pigs with different genotypes may respond similarly to dietary restrictions. Compensatory growth depends on the degree of early feed restriction and feed intake during realimentation. Although the mechanisms underlying compensatory growth are not clear, the decreased maintenance costs, increased feed intake, increased efficiency of growth and hormonal changes during the realimentation phase have been suggested as possible mechanisms.

The effect of GH, IGF-I, and insulin during the restriction and realimentation has undergone the most intensive research. The concentration of GH is increased and IGF-I decreased during the feed restriction. The concentration of IGF-I was found to be greater in a line of pigs selected for increased growth rate and decreased backfat thickness. The insulin concentration is highly variable and depends on the degree of feed restriction. Leptin can be an indicator of nutritional status of body and controls feed intake and energy metabolism, and a higher leptin concentration has been found in obese pigs than lean pigs.

Blood metabolite concentrations fluctuate with the nutritional status, and attempts have been made to use these fluctuations as indicators of the adequacy of feeding management. The plasma cholesterol concentration increased with the low protein

content of diets, and higher cholesterol concentrations were found in obese pigs than lean pigs. The serum total protein and albumin concentrations depend on the protein content of diet. Glucose concentration depends on the degree of feed restriction, and there seems to be some differences in the glucose concentration between lean and obese pigs.

Pigs with distinct genotypes are likely to respond differently to the concentrations of nutrients or dietary manipulations simply because of metabolic and physiological alterations taking place in pigs selected for specific traits. Understanding fully the effect of dietary restrictions and genotype on growth performance, physiology, and metabolism is essential in developing environmentally friendly, optimum feeding strategies for successful and sustainable pig production.

III. THE EFFECT OF EARLY DIETARY AMINO ACID RESTRICTIONS ON SERUM METABOLITES IN PIGS SELECTED FOR LEAN GROWTH EFFICIENCY

Running head: Dietary restrictions and serum metabolites

Effect of early dietary amino acid restrictions on serum metabolites in pigs selected for lean growth efficiency[1]

H. R. Mule, L. I. Chiba[2], J. Fabian[3], D. L. Kuhlers, S. B. Jungst[4], L. T. Frobish, K. Nadarajah, W. G. Bergen, and E. G. Welles[5]

Department of Animal Sciences, Auburn University

Auburn University, AL 36849-5415

[1]The technical assistance of B. Anderson, B. Brown, M. Carroll, S. Dale, C. Dowdell, R. Johnson, C. M. Lin, and C. Morgan is gratefully acknowledged.

[2]Correspondence: 303C Ann S. Upchurch Hall (phone: 334-844-1560; fax: 334-844-1519; E-mail: chibale@auburn.edu)

[3]Present address: Babolna Feed, Ltd., P. O. Box 16, 2942 Nagyigmand, Hungary.

[4]Present address: PIC USA, Franklin, KY.

[5]Department of Pathobiology, College of Veterinary Medicine, Auburn University.

ABSTRACT: Thirty-two select line and 32 control line pigs (average, 20 kg) were used in each of the two experiments to assess the effect of dietary amino acid restrictions during the grower phase and amino acid content of realimentation diets on serum metabolites. In Exp. 1, 16 pens with two gilts and 16 pens with two barrows per pen were assigned within the genetic line to grower diets (6.1 or 11.1 g lysine/kg) and finisher diets (6.1 or 8.9 g lysine/kg) in a 2 x 2 x 2 factorial arrangement of treatments. Similarly, 16 pens with two gilts and 16 pens with two barrows per pen were assigned within the genetic line to grower diets (5.0, 7.0, 9.0, or 11.0 g lysine/kg) in a 2 x 4 factorial arrangement of treatments, and pigs were offered common Finisher 1 and Finisher 2 diets in Exp. 2. Blood samples were collected at the end of the grower and finisher phases in Exp. 1 and 2, and the samples were also collected at the beginning of the study in Exp. 2. Serum concentrations of albumin ($P = 0.001$) at the end of the grower phase and cholesterol ($P = 0.029$) and albumin ($P = 0.001$) at the end of the finisher phase were greater in pigs fed the high-amino acid grower diet than those fed the low-amino acid grower diet in Exp. 1. At the end of the finisher phase, triglyceride ($P = 0.029$), albumin ($P = 0.005$), and glucose ($P = 0.027$) concentrations were reduced in pigs fed the high-amino acid finisher diet compared with those fed the low-amino acid finisher diet. The select line pigs had higher concentration of cholesterol ($P = 0.009$) at the end of the grower phase and triglyceride ($P = 0.036$) and albumin ($P = 0.016$) at the end of the finisher phase than the control line pigs. In Exp. 2, as the amino acid content of the grower diets increased, cholesterol was reduced (linear, $P = 0.005$; quadratic, $P = 0.026$; cubic, $P = 0.039$), but total protein (linear, $P = 0.040$; quadratic, $P = 0.093$) and albumin

(linear, $P = 0.001$) concentrations were increased at the end of the grower phase. The select line pigs had greater concentrations of triglyceride ($P = 0.001$), total protein ($P = 0.041$), and glucose ($P = 0.005$) at the end of the grower phase and triglyceride ($P = 0.031$) at the end of the finisher phase, and tended to have a higher serum albumin concentration ($P = 0.059$) at the end of the finisher phase than the control line pigs. Similarly, the select line pigs seemed to have a greater total protein concentration at the end of the finisher phase, even though there was a trend for grower diet x genotype interaction ($P = 0.095$). Serum cholesterol was correlated negatively with lysine intake (r = -0.38; $P = 0.039$) during the grower phase and urea N (r = -0.39; $P = 0.032$) at the end of the grower phase in Exp. 1 and positively with ultrasound backfat (r = 0.78; $P = 0.001$) at the end of the grower phase in Exp. 2. The results indicated that the metabolite profile can be affected by both the genotype and early dietary amino acid restrictions.

Key words: Amino Acid Restrictions, Genotypes, Pigs, Serum Metabolites

Introduction

Pigs, like other species, may exhibit compensatory growth after a period of early dietary restrictions (Robinson, 1964; Wahlstrom and Libal, 1983; Crister et al., 1995). The nature and extent of compensatory response may depend on many factors, such as the type, severity, and duration of dietary restrictions, the age of the animal, and the nutritional management in the subsequent phase (Lawrence and Fowler, 1997; Chiba et al., 1999). It is reasonable to assume that compensatory responses in growth performance are a reflection of changes in metabolism. This contention is supported by findings of several researchers (Prince et al., 1983; Valaja et al., 1992; Chiba et al., 2002) who

reported that pigs subjected to a period of early dietary restrictions had better feed efficiency in the subsequent phase than unrestricted pigs.

Wide variations in the potential for growth and protein accretion exist in today's pig industry, perhaps because of the selection effort over the years. It is possible that the nature and extent of the compensatory response in growth performance and body composition can be affected by genetic variations in addition to aforementioned factors. Pigs with different genotypes show differences in blood concentrations of metabolites (Pond et al., 1981, 1988, 1997) and hormones (Buonomo and Klindt, 1993), indicating the metabolic and physiological changes taking place in pigs selected for specific traits. However, the effect of dietary manipulations or genetic background on serum metabolites and hormones has been rather inconsistent (e.g., Pond et al., 1992; Fabian et al., 2003).

To maximize the efficiency of pig production, it is important to understand fully the effect of dietary manipulations and genotype on growth performance, physiology, and metabolism. The present research was conducted to investigate the effect of the degree of dietary amino acid restrictions during the grower phase and the amino acid content of realimentation diets on serum metabolites in pigs with distinct genotypes.

Experimental Procedures

This research consisted of two experiments, and general features, such as animals, housing, management practices and statistical methods, have been described in detail previously (Chiba et al., 2002; Fabian et al., 2002). Pigs were obtained from a line of Duroc pigs established at Auburn University by six generations of index selection for

improved lean growth efficiency and a contemporary line of pigs selected randomly (Kuhlers et al., 2003).

Animals and Facilities

General. In both experiments, 32 pigs selected for lean growth efficiency and 32 pigs selected randomly were used. Pigs weighing approximately 20 kg were assigned to 16 pens with two gilts or two castrated males per pen. Pigs were housed in pens with solid concrete floors in two open-front nursery and grower-finisher buildings (8.9 and 11.9 m^2/pen, respectively) in Exp. 1 and grower-finisher building in Exp. 2. Blood samples were collected from each pig at the end of the grower phase and before slaughter in Exp. 1, and the start of the study, the end of the grower phase, and before slaughter in Exp. 2. Blood samples were collected via vena cava puncture using a sterile needle and an evacuated tube in the afternoon (1500 to 1600) in Exp. 1 and in the morning (1000 to 1200) in Exp. 2. Serum was separated by centrifugation, and an aliquot was stored at −20°C until analyzed for metabolites. Pigs were allowed ad libitum access to feed and water. The protocol for animal care was approved by the university's Institutional Animal Care and Use Committee.

Experiment 1. Sixteen pens of gilts and 16 pens of castrated males were randomly assigned within the genetic line to grower and finisher diets in a 2 x 2 x 2 factorial arrangement of treatments. Pigs were offered one of the two grower diets from 19.6 ± 1.4 to 50.5 ± 2.4 kg BW, and finisher diets from 50.5 ± 2.4 to 112.7 ± 3.4 kg BW. Pigs were slaughtered at an average pen weight of 112.7 ± 3.4 kg BW.

Experiment 2. In each of the two trials, eight gilt and eight castrated male pens were randomly assigned within the genetic line to one of the four grower diets in a 2 x 4 factorial arrangement of treatments. After the grower phase, all pigs were fed common Finisher 1 and Finisher 2 diets. The grower diets were fed from 20.7 ± 2.0 to 50.2 ± 2.1 kg BW, the Finisher 1 diet from 50.2 ± 2.1 to 80.5 ± 2.4 kg BW, and the Finisher 2 diet from 80.5 ± 2.4 to 108.2 ± 3.6 kg BW. Pigs were slaughtered at an average pen weight of 108.2 ± 3.6 kg BW.

Experimental Diets

In both experiments, diets with different amino acid contents were used to create differences in growth performance and body composition during the grower phase. The two grower diets used in Exp. 1 were designed to be either marginally deficient (6.1 g lysine/kg; 80% of the 1988 NRC recommendation) or adequate in lysine (11.1 g lysine/kg; Chiba et al., 1991a,b; Table 1). The two finisher diets were designed to contain either the lysine content equal to that recommended by the NRC (1988; 6.1 g lysine/kg) or 80% (8.9 g lysine/kg) of the grower diet containing 11.1 g lysine/kg. In Exp. 2, the grower diets were formulated to contain 5.0, 7.0, 9.0, or 11.0 g lysine/kg (Table 2). Common Finisher 1 and Finisher 2 diets were formulated to meet the total lysine requirements (NRC, 1998).

Corn and soybean meal were used as sources of energy and amino acids to formulate practical diets, and an effort was not made to maintain a constant amino acid balance. The proportions of indispensable amino acids relative to lysine were, however, above the ideal protein (ARC, 1981) or balanced protein (NRC, 1998) and the DE content

was relatively similar (14.4 to 14.6 MJ/kg) for all grower and finisher diets. Minerals and vitamins were provided in amounts calculated to meet or exceed the NRC (1988 and 1998 for Exp. 1 and 2, respectively) recommendations. Feed samples collected at each mixing were pooled, and subsamples were analyzed for crude protein (AOAC, 1984 and 1990 for Exp. 1 and 2, respectively) and amino acids (Chiba et al., 1991a; Chiba, 1994).

Chemical Analysis of Blood Samples

Serum samples were analyzed for cholesterol, triglycerides, total protein, albumin, and glucose at the Clinical Pathology Laboratory, College of Veterinary Medicine, Auburn University. These metabolites were analyzed by using Boehringer Mannheim/Hitachi 911 Automatic Analyzer (Boehringer Mannheim Corporation, Indianapolis, IN). The serum samples were thawed to room temperature, and 1 mL of each sample in a cuvette was placed in the automatic analyzer. The data were generated by the automatic data reduction program of the analyzer.

In brief (Diagnostic Chemicals Ltd., Oxford, CT), cholesterol esters present in serum were hydrolyzed quantitatively into free cholesterol and fatty acids by microbial cholesterol esterase, and free cholesterol concentration was measured photometrically. Triglycerides were hydrolyzed by microbial lipases to yield glycerol and free fatty acids. Glycerol was phosphorylated, and then oxidized to dihydroxyacetone phosphate, and concentration was measured photometrically. The protein was reacted with copper in the biuret reagent at alkaline pH, which resulted in the formation of a colored complex. The colored complex was subsequently measured photometrically. Albumin was reacted with bromocresol green to form a blue-green complex, which was determined photometrically.

Glucose was phosphorylated by hexokinase to form glucose-6-phosphate and adenosine diphosphate. The glucose-6-phosphate was then oxidized, producing NADH, which was measured photometrically.

Statistical Analysis

Data were subjected to the statistical analysis using the GLM procedure of SAS (SAS Inst. Inc., Carry, NC). Orthogonal polynomials were used to asses the effect of the amino acid content of grower diets in Exp. 2. The pen was considered as the experimental unit. The initial analysis of the data in Exp. 2 indicated that the interactions between experimental factors and trial were not important sources of variation; thus, the two data sets were combined. In addition to the genetic line and dietary treatments, sex, building (Exp. 1) and(or) trial (Exp. 2), and all appropriate interaction terms were included in the statistical models initially, and those interactions that did not reach at least a statistically significant trend (i.e., $P > 0.10$) were deleted from the final models. The initial and final weights were included in the model as covariates for the blood metabolite data in Exp. 1, whereas the initial values were used as covariates in Exp. 2. To describe the relationship between serum metabolites and growth performance or carcass quality traits (Chiba et al., 2002; Fabian et al., 2002), residual correlation coefficients were estimated after fitting the model using the MANOVA option of the GLM procedure. The model included the effect of the diet, genotype, sex, and genotype x sex interaction.

Results

Experiment 1

Grower Phase. There were no interactions between grower diets and genotype on serum cholesterol, triglycerides, total protein, albumin, and glucose concentrations at the end of the grower phase (Table 3). Pigs fed the high-amino acid grower diet had a high serum albumin ($P = 0.001$) concentration than pigs fed the low-amino acid grower diet. The control line pigs had less concentration of serum cholesterol ($P = 0.009$) than the select line pigs.

Finisher Phase. There were no two or three-way interactions in any of the response criteria, except trend for a grower diet x finisher diet interaction ($P = 0.089$) in the total protein concentration at the end of the finisher phase (Table 4). The total protein concentration in pigs fed the low-amino acid grower diet seemed to increase as the amino acid content of the finisher diet increased, whereas the finisher diet seemed to have no effect on total protein in pigs fed the high-amino acid grower diet. Increased serum cholesterol ($P = 0.029$) and albumin ($P = 0.001$) concentrations were observed in pigs fed with high-amino acid grower diet. Pigs fed the high-amino acid finisher diet had lower serum triglyceride ($P = 0.029$), albumin ($P = 0.005$), and glucose ($P = 0.027$) concentrations than those fed the low-amino acid diet. Serum triglyceride ($P = 0.036$) and albumin ($P = 0.016$) concentrations were lower in the control line pigs compared with the select line pigs.

Experiment 2

Grower Phase. There were no grower diet x genotype interactions in any of the response criteria (Table 5). The increase in the amino acid content of the grower diet resulted in a decrease in cholesterol concentration (linear, $P = 0.005$; quadratic, $P = 0.026$; cubic, $P = 0.039$) at the end of the grower phase. As the amino acid content increased from 5.0 to 7.0 g lysine/kg, serum total protein increased, but it remained constant with further increases (linear, $P = 0.040$; quadratic, $P = 0.093$). The increased amino acid content of the grower diets resulted in an increased serum albumin concentration (linear, $P = 0.001$). The control line pigs had less serum triglyceride ($P = 0.001$), total protein ($P = 0.041$), and glucose ($P = 0.005$) concentrations at the end of the grower phase than the select line pigs.

Finisher Phase. There were no grower diet x genotype interactions, except a trend ($P = 0.095$) in the serum total protein concentration at the end of the finisher phase (Table 6). As the amino acid content of grower diets increased from 5.0 to 7.0 g lysine/kg, the total protein concentration increased in the control line pigs, but it decreased slightly in the select line pigs, thus resulting in the interaction. The control line pigs had less serum triglyceride ($P = 0.031$), and a trend for less albumin ($P = 0.059$) concentrations than the select line pigs.

Correlation Analysis

The least square means of growth performance, carcass data, ultrasound backfat, and blood urea nitrogen concentrations are presented in Tables 7 and 8 for Exp. 1 and 2, respectively. Those and metabolite data sets were used for the correlation analysis.

Experiment 1. The cholesterol concentration at the end of the grower phase was negatively correlated with the lysine intake during the grower phase (r = -0.38; *P* = 0.039) and blood urea nitrogen concentration (r = -0.39; *P* = 0.032) at the end of the grower phase (Table 9). The albumin concentration at the end of the grower phase was positively correlated with lysine intake (r = 0.61; *P* = 0.004), average daily gain (r = 0.49; *P* = 0.006) and gain:feed (r = 0.54; *P* = 0.002) during the grower phase, and blood urea nitrogen concentration at the end of the grower phase (r = 0.44; *P* = 0.016).

The triglyceride concentration at the end of the finisher phase was negatively correlated with carcass longissimus muscle area (r = -0.40; *P* = 0.029) at the end of the finisher phase and tended to be negatively correlated with lysine intake (r = -0.35; *P* = 0.062) during the finisher phase (Table 10). There was a trend for a positive correlation between total protein and blood urea nitrogen concentrations (r = 0.33; *P* = 0.078) at the end of the finisher phase. The albumin concentration tended to be positively correlated with feed intake during the finisher phase (r = 0.34; *P* = 0.063). The glucose concentration was negatively correlated with lysine intake during the finisher phase (r = -0.42; *P* = 0.020), and tended to be correlated negatively with carcass backfat (r = -0.34; *P* = 0.063) and blood urea nitrogen concentration (r = -0.32; *P* = 0.081) at the end of the finisher phase.

Experiment 2. The cholesterol concentration at the end of the grower phase was correlated positively with feed intake during the grower phase (r = 0.44; *P* = 0.016) and ultrasound backfat at the end of the grower phase (r = 0.78; *P* = 0.001), and there was a trend for a negative correlation with gain:feed (r = -0.35; *P* = 0.057) during the grower

phase (Table 11). The total protein concentration was correlated positively with gain:feed (r = 0.43; *P* = 0.018) during the grower phase. The albumin concentration was correlated positively with lysine intake (r = 0.55; *P* = 0.001), average daily gain (r = 0.47; *P* = 0.010), and gain:feed (r = 0.41; *P* = 0.024) during the grower phase, and blood urea nitrogen concentration (r = 0.43; *P* = 0.019) at the end of the grower phase. The glucose concentration tended to be correlated positively with average daily gain (r = 0.34; *P* = 0.065) and negatively with lysine intake (r = -0.33; *P* = 0.079) during the grower phase.

There was a trend for positive correlation between cholesterol concentration at the end of the finisher phase and carcass backfat (r = 0.34; *P* = 0.062) at the end of the finisher phase (Table 12). The glucose concentration was negatively correlated with carcass backfat (r = -0.39; *P* = 0.033).

Discussion

Pigs with distinct genotypes are likely to have metabolic and physiological differences, and these differences can be reflected in blood metabolite concentrations. Mersmann et al. (1982) reported in their review that obese pigs do not seem to be hyperglycemic, hypertriglyceridemic, or hypercholesterolemic. Similarly, serum glucose and triglyceride concentrations have been reported to be lower in obese pigs compared with lean and contemporary pigs (Pond et al., 1981). In the present research, the control line pigs had a lower concentration of cholesterol at the end of the grower phase in Exp. 1, and lower concentrations of triglycerides at the end of the finisher phase in Exp. 1, and at the end of the grower and finisher phases in Exp. 2, and lower concentration of glucose at the end of the grower phase in Exp. 2. These results indicate that randomly selected

control line pigs are not hypercholesterolemic, hypertriglyceridemic, and hyperglycemic, which are in agreement with Mersmann et al. (1982).

The select line pigs had a higher albumin concentration at the end of the finisher phase in Exp. 1 and tended to have a higher serum albumin at the end of the finisher phase in Exp. 2, and also had a higher total protein concentration at the end of the grower phase in Exp. 2. Similarly, the select line pigs had a higher total protein concentration at the end of the finisher phase in Exp. 2, even though both lines of pigs were fed common diets during the finisher phase. It has been reported that the select line pigs may utilize amino acids more efficiently for lean accretion than the control line pigs (Fabian et al., 2002), implying that pigs with the greater efficiency of amino acid utilization may have a higher total protein concentration. Thus, the higher total protein concentration, along with the lower serum urea nitrogen concentrations (Chiba et al., 2002; Fabian et al., 2002), observed in the select line pigs may be a further indication that these pigs utilized amino acids more efficiently for growth and protein accretion compared with the control line pigs. In addition, the select line pigs had less carcass backfat in Exp. 1 and 2, larger longissimus muscle area in Exp. 1 (Table 7 and 8), indicating that the select line pigs may had a more efficient lean growth than the control line of pigs.

In Exp. 2, the cholesterol concentration at the end of the grower phase decreased as the amino acid content of grower diets increased. These results are in agreement with Pond et al. (1986) who reported that dietary protein restriction has a hypercholesterolemic effect on growing pigs. The negative correlation between cholesterol and lysine intake during the grower phase and blood urea nitrogen

concentration at the end of the grower phase observed in Exp. 1 may support these findings. The mechanisms are not clear, but those effects may be due to the changes in lipoprotein composition and(or) transport that are taking place in lipid metabolism during the protein restriction as suggested by Pond et al. (1986).

Pigs fed the high-amino acid finisher diet in Exp. 1 had a higher serum total protein concentration at the end of the finisher phase than those fed the low-amino acid diet. In Exp. 2, as the amino acid content of diets increased, serum total protein concentration at the end of the grower phase increased. These results are in agreement with the earlier reports that the dietary protein restriction can reduce serum total protein concentration in pigs (Pond et al., 1980; Atinmo et al., 1976).

Lowrey et al. (1962) proposed that the total protein concentration can be used as an indicator of the adequacy of dietary protein content. In the present research, although serum total protein concentration at the end of the grower phase increased initially as the dietary amino acid content increased from 5.0 to 7.0 g lysine/kg in Exp. 2, it remained constant with further increases. On the other hand, growth performance of pigs increased linearly and ultrasound backfat decreased linearly with the increase in the amino acid content of grower diets (Fabian et al., 2002). This may indicate that it may not be appropriate to use the total protein concentration alone as an indicator of the amino acid requirement.

Serum albumin is considered to be a more sensitive indicator of protein nutrition (Lowrey et al., 1962). Pigs fed the high-amino acid diet had a high serum albumin concentration at the end of the grower and finisher phases in Exp. 1. Similarly, as the

dietary lysine content increased, the albumin concentration at the end of the grower phase increased in Exp. 2. These results are also in agreement with the earlier studies that the protein restriction can reduce the serum albumin concentration in pigs (Pond et al., 1980; Atinmo et al., 1976). At the end of the finisher phase, serum albumin concentration in pigs fed the high-amino acid diet decreased in Exp. 1, which was not expected.

There was a trend for an increased serum total protein concentration at the end of the finisher phase as the dietary amino acid increased in Exp. 2. Similarly, serum albumin concentration at the end of the finisher phase seemed to increase as the amino acid content of the grower diets increased. The linear effect of the grower diet on these metabolites was rather obscure, but nevertheless, it is clear that the grower diet had carryover effects on serum total protein and albumin concentrations simply because pigs were fed common diets during the finisher phase.

As mentioned before, serum total protein, and perhaps albumin too might be associated with the efficiency of amino acid utilization. It has been reported that pigs subjected to early dietary amino acid restrictions can reduce nitrogen excretion during the restriction and in the subsequent phases and tended to exhibit compensatory nitrogen retention during the realimentation phase (Fabian et al., 2004). And, Fabian et al. (2004) suggested that the compensatory nitrogen retention might be responsible for compensatory growth responses in terms of both growth performance and lean growth (e.g., Chiba, 1994, 1995; Fabian et al., 2002), the efficiency of feed or nutrient utilization (e.g., Valaja et al., 1992; Chiba et al., 2002; Fabian et al., 2002), or lean growth (e.g., Chiba et al., 1999, 2002). Thus, it is expected that those pigs subjected to early dietary

restrictions to show some indication of the positive protein metabolism such as a greater serum total protein and(or) albumin concentration. Findings on serum total protein and(or) albumin in the present research are unexpected, and therefore they do not reflect the protein status of pigs exhibiting compensatory growth.

The increased glucose concentration at the end of the finisher phase in pigs fed the low-amino acid finisher diet in Exp. 1 can be explained by the higher carbohydrate content of the low-amino acid diet (Fabian et al., 2004). This may be also due to a decrease in insulin concentration or activity associated with the protein restriction (Atinmo et al., 1976). It is rather difficult to explain the trend in interaction between grower diet and finisher diet in serum total protein concentration at the end of the finisher phase in Exp. 1, and the interaction between the genotype and grower diet in total protein observed at the end of the finisher phase in Exp. 2.

In summary, pigs fed diets low in amino acids had high concentrations of cholesterol in the present research. Serum cholesterol was correlated negatively with lysine intake and blood urea nitrogen and positively with ultrasound backfat. The results also indicated that the serum total protein and albumin concentrations can be a reflection of the amino acid content of the diet. The status of serum total protein may be an indication of the efficiency of amino acid utilization, and this might be a further indication that the pigs selected for lean growth efficiency may have utilized amino acids more efficiently for growth and protein accretion.

Implications

The results indicated that the metabolite profile can be affected by both the genotype and early dietary amino acid restrictions. Wide variations in the potential of pigs for growth and protein retention continue to exist in the today's pig industry. Pigs with different genotypes may respond differently to dietary manipulations, and it is important to understand better the effect of genotype and dietary restrictions on serum metabolites in satisfying their nutritional needs. The results of the present research may contribute to developing environmentally friendly, optimal feeding strategies for successful and sustainable pig production.

Literature Cited

AOAC. 1984. Official Methods of Analysis. 14th ed. Assoc. Offic. Anal. Chem., Washington, DC.

AOAC. 1990. Official Methods of Analysis. 15th ed. Assoc. Offic. Anal. Chem., Arlington, VA.

ARC. 1981. The Nutrient Requirements of Pigs. Commonwealth Agriculture Bureaux, Slough.

Atinmo, T., C. Baldijao, W. G. Pond, and R. H. Barnes. 1976. Plasma insulin levels in weaned pigs fed protein or energy restricted diets. J. Nutr. 106:1654-1658.

Buonomo, F. C., and J. Klindt. 1993. Ontogeny of growth hormone (GH), insulin like growth factors (IGF-I and IGF-II) and IGF binding protein-2 (IGFBP-2) in genetically lean and obese swine. Domest. Anim. Endocrinol. 10:257-265.

Chiba, L. I. 1994. Effects of dietary amino acid content between 20 and 50 and 100 kg live weight on the subsequent and overall growth performance of pigs. Livest. Prod. Sci. 39:213-221.

Chiba, L. I. 1995. Effects of nutritional history on the subsequent and overall growth performance and carcass traits of pigs. Livest. Prod. Sci. 41:151-161.

Chiba, L. I., H. W. Ivey, K. A. Cummins, and B. E. Gamble. 1999. Growth performance and carcass traits of pigs subjected to marginal dietary restrictions during the grower phase. J. Anim. Sci. 77:1769-1776.

Chiba, L. I., D. L. Kuhlers, L. T. Frobish, S. B. Jungst, E. J. Huff-Lonergan, S. M. Lonergan, and K. A. Cummins. 2002. Effect of dietary restrictions on growth performance and carcass quality of pigs selected for lean growth efficiency. Livest. Prod. Sci. 74:93-102.

Chiba, L. I., A. J. Lewis, and E. R. Peo. Jr. 1991a. Amino acid and energy interrelationships in pigs weighing 20 to 50 kilograms: I. Rate and efficiency of weight gain. J. Anim. Sci. 69:694-707.

Chiba, L. I., A. J. Lewis, and E. R. Peo. Jr. 1991b. Amino acid and energy interrelationships in pigs weighing 20 to 50 kilograms: II. Rate and efficiency of protein and fat deposition. J. Anim. Sci. 69:708-718.

Critser, D. J., P. S. Miller, and A. J. Lewis. 1995. The effects of dietary protein concentration on compensatory growth in barrows and gilts. J. Anim. Sci. 73:3376-3383.

Fabian, J., L. I. Chiba, L. T. Frobish, W. H. McElhenney, D. L. Kuhlers, and K. Nadarajah. 2004. Compensatory growth and nitrogen balance in grower-finisher pigs. J. Anim. Sci. 82:2579-2587.

Fabian, J., L. I. Chiba, D. L. Kuhlers, L. T. Frobish, K. Nadarajah, C. R. Kerth, W. H. McElhenney, and A. J. Lewis. 2002. Degree of amino acid restrictions during the grower phase and compensatory growth in pigs selected for lean growth efficiency. J. Anim. Sci. 80:2610-2618.

Fabian, J., L. I. Chiba, D. L. Kuhlers, L. T. Frobish, K. Nadarajah, and W. H. McElhenney. 2003. Growth performance, dry matter and nitrogen digestibilities, serum profile, and carcass and meat quality of pigs with distinct genotypes. J. Anim. Sci. 81:1142-1149.

Kuhlers, D. L., K. Nadarajah, S. B. Jungst, B. L. Anderson, and B. E. Gamble. 2003. Genetic selection for lean feed conversion in a closed line of Duroc pigs. Livest. Prod. Sci. 84:75-82.

Lawrence, T. L. J., and V. R. Fowler. 1997. Growth of Farm Animals. CAB International, Wallingford.

Lowrey, R. S., W. G. Pond, R. H. Barnes, L. Krook, and J. K. Loosli. 1962. Influence of caloric level and protein deficiency in the young pig. J. Nutr. 78:245-252.

Mersmann, H. J., W. G. Pond, and J. T. Yen. 1982. Plasma glucose, insulin and lipids during growth of genetically lean and obese swine. Growth 46:189-198.

NRC. 1988. Nutrient requirements of swine. 9th rev. ed. Natl. Acad. Press, Washington, DC.

NRC. 1998. Nutrient Requirements of Swine. 10th ed. Natl. Acad. Press, Washington, DC.

Pond, W. G., W. Insull, H. J. Mersmann, W. W. Wong, K. B. Harris, H. R. Cross, E. O. Smith, J. P. Heath, and L. G. Komuves. 1992. Effect of dietary fat and cholesterol level on growing pigs selected for three generations for high or low serum cholesterol at age 56 days. J. Anim. Sci. 70:2462-2470.

Pond, W. G., H. G. Jung, and V. H. Varel. 1988. Effect of dietary fiber on young adult genetically lean, obese and contemporary pigs: bodyweight, carcass measurements, organ weights and digesta content. J. Anim. Sci. 66:699-706.

Pond, W.G., H. J. Mersmann, and L. D. Young. 1986. Heritability of plasma cholesterol and triglyceride concentration in swine. Proc. Soc. Exp. Biol. Med. 182:221-224.

Pond, W. G., D. R. Su, and H. J. Mersmann. 1997. Divergent concentrations of plasma metabolites in swine selected for seven generations for high or low plasma total cholesterol. J. Anim. Sci. 75:311-316.

Pond, W. G., J. T. Yen, and R. N. Lindvall. 1980. Early protein deficiency: Effects on later growth and carcass composition of lean or obese swine. J. Nutr. 110:2506-2513.

Pond, W. G., J. T. Yen, R. N. Lindvall, and D. Hill. 1981. Dietary alfalfa meal for genetically obese and lean growing pigs: Effect on body weight gain and on carcass and gastrointestinal tract measurements and blood metabolites. J. Anim. Sci. 51:367-373.

Prince, T. J., S. B. Jungst, and D. L. Kuhlers. 1983. Compensatory responses to short-term feed restriction during the growing period in swine. J. Anim. Sci. 56:846-852.

Robinson, D. W. 1964. The plane of nutrition and compensatory growth in pigs. Anim. Prod. 6:227-236.

Valaja, J., T. Alaviuhkola, K. Suomi, and I. Immonen. 1992. Compensatory growth after feed restriction during the rearing period in pigs. Agric. Sci. Finland. 1:15-20.

Wahlstrom, R. C., and G. W. Libal. 1983. Compensatory responses of swine following protein insufficiency in grower diets. J. Anim. Sci. 56:118-124.

Table 1. Composition of grower and finisher diets (Experiment 1)[a]

	g Lysine/kg			
	Grower		Finisher	
Item	6.1	11.1	6.1	8.9
Ingredients, g/kg				
Corn	841.8	668.1	841.8	745.8
Soybean meal (48% CP)	126.4	302.6	126.4	223.8
Dicalcium phosphate	18.0	14.6	18.0	16.1
Limestone	7.8	8.7	7.8	8.3
Salt	3.5	3.5	3.5	3.5
Trace mineral-vitamin mix[b]	2.5	2.5	2.5	2.5
Calculated composition				
DE, MJ/kg	14.4	14.5	14.4	14.5
CP, g/kg	133	204	133	172
Lysine, g/kg	6.1	11.1	6.1	8.9
Ca, g/kg	7.5	7.5	7.5	7.5
P, g/kg	6.5	6.5	6.5	6.5
Analyzed composition, g/kg[c]				
CP	139	202	137	178
Lysine	6.3	10.9	6.4	9.1
Threonine	4.6	6.9	4.6	6.2
Isoleucine	4.8	7.6	4.8	6.6
Valine	5.7	8.4	5.7	7.5
Histidine	3.2	4.9	3.3	4.5

[a]Grower diets were offered from 19.6 ± 1.4 kg to 50.5 ± 2.4 kg, and then finisher diets were offered until slaughter at an average pen weight of 112.7 ± 3.4 kg.

[b]Provided the following (unit/kg diet): Zn (zinc oxide), 80 mg; Fe (ferrous sulfate), 80 mg; Mn (manganous oxide), 40 mg; Cu (copper chloride), 10 mg; I (ethylenediamine dihydroiodide), 1 mg; Co (cobalt carbonate), 0.4 mg; Se (sodium selenite), 0.3 mg; vitamin A, 5,500 IU; vitamin D_3, 1,760 IU; vitamin E, 16.5 IU; menadione dimethylpyrimidinol bisulfite, 2.2 mg; riboflavin, 4.4 mg; d-pantothenic acid, 17.6 mg; niacin, 35.2 mg; vitamin B_{12}, 27.5 μg; choline, 95 mg.

[c]No analysis for sulfur amino acids and tryptophan.

Table 2. Composition of grower and finisher diets (Experiment 2)[a]

Item	Grower, g lysine/kg				Finisher 1	Finisher 2
	5.0	7.0	9.0	11.0		
Ingredient, g/kg						
Corn	882.2	811.0	739.8	668.6	797.2	853.1
Soybean meal (48% CP)	89.6	162.0	234.4	306.7	179.7	125.2
Dicalcium phosphate	15.7	14.1	12.5	10.9	11.0	9.5
Limestone	7.0	7.4	7.8	8.3	6.6	6.7
Salt	3.5	3.5	3.5	3.5	3.5	3.5
Mineral-vitamin premix[b]	2.0	2.0	2.0	2.0	2.0	2.0
Calculated composition						
DE, MJ/kg	14.4	14.5	14.5	14.6	14.5	14.5
CP, g/kg	115.8	144.2	172.7	201.2	151.5	130.5
Lysine, g/kg	5.0	7.0	9.0	11.0	7.5	6.0
Ca, g/kg	7.0	7.0	7.0	7.0	6.0	5.5
P, g/kg	6.0	6.0	6.0	6.0	5.5	5.0
Analyzed composition, g/kg[c]						
CP	125.5	153.0	172.7	205.2	153.2	133.3
Lysine	5.2	6.9	8.7	10.6	7.3	5.7
Threonine	4.5	5.5	6.7	8.0	5.8	5.3
Isoleucine	4.1	5.5	6.4	7.9	5.5	4.5
Valine	4.8	6.1	7.3	8.7	6.5	5.4
Histidine	3.0	3.8	4.4	5.2	4.0	3.4

[a]Grower diets were formulated to contain 5.0, 7.0, 9.0 or 11.0 g lysine/kg, whereas Finisher 1 and 2 diets were formulated to contain 7.5 and 6.0 g lysine/kg, respectively. Grower diets were offered from 20.7 ± 2.0 to 50.2 ± 2.1 kg, Finisher 1 diet from 50.2 ± 2.1 to 80.5 ± 2.4 kg, and Finisher 2 diet from 80.5 ± 2.4 to 108.2 ± 3.6 kg average pen weight.

[b]Provided the following (unit/kg diet): Zn (zinc oxide), 90 mg; Fe (ferrous sulfate), 80 mg; Mn (manganous oxide), 32 mg; Cu (copper chloride), 10 mg; I (ethylenediamine dihydroiodide), 0.4 mg; Se (sodium selenite), 0.3 mg; vitamin A, 5,514 IU; vitamin D_3, 1,103 IU; vitamin E, 24 IU; menadione sodium bisulfite complex, 2 mg; vitamin B_{12}, 26 μg; riboflavin, 4 mg; pantothenic acid, 18 mg; niacin, 26 mg; choline, 66 mg.

[c]No analysis for sulfur amino acids or tryptophan.

Table 3. Effect of the dietary amino acid content and genotype on serum metabolites at the end of the grower phase (Experiment 1)[a]

Item	Cholesterol, mg/dL	Triglycerides, mg/dL	Total protein, g/dL	Albumin, g/dL	Glucose, mg/dL
Grower diet, g lysine/kg					
6.1	108.3	47.6	7.3	4.4	88.5
11.1	101.8	53.5	7.4	4.9	88.4
Genotype					
Control	99.3	46.7	7.3	4.6	85.5
Select	110.7	54.5	7.5	4.7	91.4
P-value					
Grower diet	0.126	0.256	0.452	0.001	0.986
Genotype	0.009	0.129	0.181	0.470	0.145
Grower diet x genotype	0.951	0.322	0.196	0.425	0.307
SEM[b]					
Grower diet	2.9	3.5	0.1	0.1	2.8
Genotype	2.9	3.5	0.1	0.1	2.8

[a]Least squares means based on 16 pens containing two gilts or two castrated males; used the initial (19.6 ± 1.4 kg) and final (50.5 ± 2.4 kg) weights as covariates.

[b]Pooled SEM.

Table 4. Effect of the dietary amino acid content and genotype on serum metabolites at the end of the finisher phase (Experiment 1)[a]

Item	Cholesterol, mg/dL	Triglycerides, mg/dL	Total protein, g/dL	Albumin, g/dL	Glucose, mg/dL
Grower diet, g lysine/kg					
6.1	91.3	47.5	7.8	4.9	72.1
11.1	98.3	46.3	7.9	5.2	71.5
Finisher diet, g lysine/kg					
6.1	95.9	51.3	7.8	5.2	73.6
8.9	93.7	42.5	8.0	4.9	70.0
Genotype					
Control	93.2	42.7	7.9	5.0	70.7
Select	96.5	51.1	7.8	5.2	72.9
P-value					
Grower	0.029	0.755	0.184	0.001	0.707
Finisher	0.447	0.029	0.061	0.005	0.027
Genotype	0.270	0.036	0.268	0.016	0.132
Grower x finisher[b]	0.876	0.328	0.089	0.966	0.435
Grower x genotype	0.788	0.556	0.865	0.150	0.759
Finisher x genotype	0.276	0.269	0.450	0.647	0.433
Grower x finisher x genotype	0.275	0.404	0.967	0.174	0.104
SEM[c]					
Grower diet	1.0	2.7	0.1	0.1	1.0
Finisher diet	1.0	2.6	0.1	0.1	1.0
Genotype	1.0	2.6	0.1	0.1	1.0

[a]Least squares means based on 16 pens containing two gilts or two castrated males; used the initial (50.5 ± 2.4 kg) and final (112.7 ± 3.4 kg) weights as covariates.

[b]Total protein = 7.6, 8.1, 8.0, and 7.9 g/dL in pigs fed 6.1 and 6.1, 6.1 and 8.9, 11.1 and 6.1, and 11.1 and 8.9 g lysine/kg combinations during the grower and finisher phases, respectively.

[c]Pooled SEM.

Table 5. Effect of the dietary amino acid content and genotype on serum metabolites at the end of the grower phase (Experiment 2)[a]

Item	Cholesterol, mg/dL	Triglycerides, mg/dL	Total protein, g/dL	Albumin, g/dL	Glucose, mg/dL
Grower diet, g lysine/kg					
5.0	86.2	56.5	6.4	3.3	83.1
7.0	80.9	48.4	6.9	3.8	84.7
9.0	68.3	47.5	6.9	4.0	79.3
11.0	76.7	55.7	6.9	4.2	87.2
Genotype					
Control	76.1	40.2	6.6	3.8	78.3
Select	80.0	63.9	6.9	3.9	88.9
P-value					
Grower diet					
Linear	0.005	0.878	0.040	0.001	0.610
Quadratic	0.026	0.125	0.093	0.157	0.277
Cubic	0.039	0.932	0.170	0.984	0.129
Genotype	0.232	0.001	0.041	0.393	0.005
Grower diet x genotype	0.644	0.734	0.484	0.473	0.175
SEM[b]					
Grower diet	2.6	5.1	0.2	0.1	2.8
Genotype	2.0	4.2	0.1	0.1	2.2

[a]Least squares means were based on eight pens per grower diet or 16 pens per genotype; used the initial value as a covariate.

[b]Pooled SEM.

Table 6. Effect of the dietary amino acid content and genotype on serum metabolites at the end of the finisher phase (Experiment 2)[a]

Item	Cholesterol, mg/dL	Triglycerides, mg/dL	Total protein, g/dL	Albumin, g/dL	Glucose, mg/dL
Grower diet, g lysine/kg					
5.0	96.4	69.1	7.2	4.7	73.9
7.0	102.5	59.1	7.4	4.8	70.1
9.0	102.2	53.3	7.4	4.9	73.1
11.0	96.9	58.8	7.4	4.9	71.7
Genotype					
Control	101.8	52.5	6.9	4.8	72.4
Select	97.1	67.6	7.7	4.9	72.1
P-value					
Grower diet					
Linear	0.944	0.111	0.053	0.102	0.714
Quadratic	0.160	0.137	0.182	0.580	0.579
Cubic	0.936	0.754	0.705	0.468	0.277
Genotype	0.322	0.031	0.001	0.059	0.908
Grower diet x genotype[b]	0.867	0.486	0.095	0.232	0.186
SEM[c]					
Grower diet	3.7	5.0	0.1	0.1	2.2
Genotype	2.9	4.1	0.1	0.1	1.7

[a]Least squares means were based on eight pens per grower diet or 16 pens per genotype; used the initial value as a covariate.

[b]Total protein = 6.7, 7.2, 7.5, and 7.0 g/dL in the control line pigs and 7.7, 7.6, 7.8, and 7.8 g/dL in the select line pigs fed diets containing 5.0, 7.0, 9.0, and 11.0 g lysine/kg, respectively.

[c]Pooled SEM.

Table 7. Least square means of growth performance during grower (19.6 ± 1.4 to 50.5 ± 2.4 kg BW) or finisher (50.5 ± 2.4 to 112.7 ± 3.4 kg BW) phase, and ultrasound measurements of backfat, blood urea nitrogen, and carcass data at the end of the grower or finisher phase (Experiment 1)[a,b,c]

Item	ADFI, g/d	DLysI[d], g/d	ADG, g/d	G:F, g/kg	BUN, mg/dL	BF, mm	LMA, cm^2
Grower Phase:							
Grower diet, g lysine/kg							
6.1	2,027	12.2	681	337	9.5	17.7	-
11.1	1,889	20.9	748	396	16.4	14.6	-
Genotype							
Control	1,924	16.4	710	370	13.9	17.9	-
Select	1,992	16.8	719	362	12.0	14.4	-
P-value[e]							
Grower diet	0.001	0.001	0.009	0.001	0.001	0.001	-
Genotype	-	-	-	-	0.029	0.001	-
SEM[f]	28.0	0.2	17.0	7.0	0.5	0.5	-
Finisher Phase:							
Grower diet, g lysine/kg							
6.1	3,052	22.5	804	262	14.1	26.5	30.9
11.1	3,288	24.3	802	245	15.0	27.2	29.8
Finisher diet, g lysine/kg							
6.1	3,244	19.6	815	251	12.2	27.1	30.3
8.9	3,095	27.3	792	256	16.9	26.6	30.5
Genotype							
Control	3,186	23.5	792	250	15.2	31.5	29.2
Select	3,154	23.4	814	257	13.9	22.2	31.6
P-value[e]							
Grower diet	0.001	0.001	-	0.015	-	-	-
Finisher diet	0.016	0.001	-	-	0.001	-	-
Genotype	-	-	-	-	-	0.001	0.018
SEM[f]	44.2	0.3	19.0	4.0	0.4	0.9	0.5

[a] DLysI, daily lysine intake; BF, ultrasound and carcass backfat at the end of the grower and finisher phases, respectively; BUN, blood urea nitrogen; LMA, carcass longissimus muscle area.

[b] Least squares means based on 16 pens containing two gilts or two castrated males.

[c] Adapted from Chiba et al. (2002).

[d] Not reported by Chiba et al. (2002).

[e] Reported *P*-values ≤ 0.05

[f] Pooled SEM.

Table 8. Least square means of growth performance during grower (20.7 ± 2.0 to 50.2 ± 2.1 kg BW) or finisher (80.5 ± 2.4 to 108.2 ± 3.6 kg BW) phase, and ultrasound measurements of backfat, blood urea nitrogen, and carcass data at the end of the grower or finisher phase (Experiment 2)[a,b,c]

Item	ADFI, g/d	DLysI, g/d	ADG, g/d	G:F, g/kg	BUN, mg/dL	BF, mm	LMA, cm^2
Grower Phase:							
Grower diet, g lysine/kg							
5.0	2,176	11.4	642	295	11.5	17.8	-
7.0	2,059	14.2	701	341	11.1	15.6	-
9.0	1,827	15.9	705	385	12.6	12.1	-
11.0	1,722	18.3	730	427	16.7	12.1	-
Genotype							
Control	1,909	14.6	669	357	14.0	16.4	-
Select	1,983	15.4	720	368	11.9	12.4	-
P-Value[d]							
Grower diet							
Linear	0.001	0.001	0.027	0.001	0.003	0.001	-
Quadratic	-	-	-	-	0.010	-	-
Cubic	-	-	-	-	-	-	-
Genotype	-	-	0.039	-	0.014	0.001	-
SEM[e]							
Grower Diet	80.0	0.7	24.4	11.5	0.8	0.9	-
Genotype	50.0	0.4	16.0	7.5	0.5	0.6	-
Finisher Phase:							
Grower diet, g lysine/kg							
5.0	3,124	17.8	816	261	13.1	27.9	29.4
7.0	3,221	18.3	772	541	13.5	28.5	30.7
9.0	3,233	18.4	759	237	15.3	28.1	30.0
11.0	3,206	18.2	737	231	13.7	28.3	31.3
Genotype							
Control	3,137	17.8	741	238	14.8	33.4	29.2
Select	3,255	18.5	801	248	13.0	23.0	31.5
P-Value[d]							
Grower diet							
Linear	-	-	0.066	0.029	-	-	-
Quadratic	-	-	-	-	-	0.099	-
Cubic	-	-	-	-	-	-	-
Genotype	-	-	-	0.036	-	-	0.093
SEM[e]							
Grower diet	80.0	0.5	28.5	8.8	1.1	2.2	1.3
Genotype	50.0	0.3	20.0	6.5	0.8	1.5	0.9

[a] DLysI, daily lysine intake; BF, ultrasound and carcass backfat at the end of the grower and finisher phases, respectively; LMA, carcass longissimus muscle area; BUN, blood urea nitrogen.

[b] Least squares means were based on eight pens per grower diet or 16 pens per genotype.

[c] Adapted from Fabian et al. (2002).

[d] Reported *P*-values ≤ 0.10

[e] Pooled SEM.

Table 9. Correlation coefficients (r) of error residuals between serum metabolite concentrations at the end of the grower phase and growth performance, ultrasound backfat, and blood urea nitrogen (Experiment 1)[a]

	Cholesterol		Triglycerides		Total protein		Albumin		Glucose	
Item	r	*P*	r	*P*	r	*P*	r	*P*	r	*P*
GFI, g/d	0.07	0.705	-0.06	0.733	0.26	0.160	-0.07	0.698	0.18	0.326
GLysI, g/d	-0.38	0.039	0.22	0.238	0.12	0.510	0.61	0.004	0.16	0.399
GADG, g/d	-0.18	0.338	0.04	0.816	0.20	0.276	0.49	0.006	0.24	0.201
GG:F, g/kg	-0.23	0.213	0.13	0.486	-0.03	0.848	0.54	0.002	0.09	0.632
UBF50, mm	0.11	0.535	-0.04	0.830	0.01	0.970	-0.19	0.306	0.21	0.254
BUN50, mg/dL	-0.39	0.032	0.27	0.144	0.10	0.587	0.44	0.016	0.10	0.593

[a]GFI, average daily feed intake, GLysI, lysine intake, GADG, average daily gain, and GG:F, gain:feed during the grower phase; UBF50, ultrasound back fat, and BUN50, blood urea nitrogen at the end of the grower phase.

Table 10. Correlation coefficients (r) of error residuals between serum metabolite concentrations at the end of the finisher phase and growth performance, ultrasound backfat, blood urea nitrogen, and carcass characteristics (Experiment 1)[a]

	Cholesterol		Triglycerides		Total protein		Albumin		Glucose	
Item	r	*P*	r	*P*	r	*P*	r	*P*	r	*P*
FFI, g/d	0.25	0.179	-0.01	0.621	0.06	0.731	0.34	0.063	-0.07	0.709
FLysI, g/d	0.01	0.932	-0.35	0.062	0.30	0.107	-0.26	0.157	-0.42	0.020
FADG, g/d	0.02	0.917	-0.09	0.609	-0.11	0.570	0.17	0.368	-0.16	0.383
FG:F, g/kg	-0.16	0.392	-0.10	0.603	-0.11	0.543	-0.06	0.751	-0.09	0.617
CBF, mm	0.21	0.252	0.29	0.114	-0.19	0.298	0.14	0.452	-0.34	0.063
LMA, cm^2	-0.03	0.871	-0.40	0.029	-0.16	0.401	-0.22	0.243	0.30	0.108
BUN100, mg/dL	0.01	0.964	-0.15	0.409	0.33	0.078	-0.10	0.578	-0.32	0.081

[a]FFI, average daily feed intake, FLysI, lysine intake, FADG, average daily gain, FG:F, and gain:feed during the finisher phase; CBF, carcass backfat at 10^{th} rib, LMA, carcass longissimus muscle area, and BUN100, blood urea nitrogen at the end of the finisher phase.

Table 11. Correlation coefficients (r) of error residuals between serum metabolite concentrations at the end of the grower phase and growth performance, ultrasound backfat, and blood urea nitrogen (Experiment 2)[a]

	Cholesterol		Triglycerides		Total protein		Albumin		Glucose	
Item	r	*P*	r	*P*	r	*P*	r	*P*	r	*P*
GFI, g/d	0.44	0.016	0.05	0.787	-0.25	0.179	-0.21	0.272	0.09	0.630
GLysI, g/d	-0.11	0.535	-0.08	0.674	0.24	0.197	0.55	0.001	-0.33	0.079
GADG, g/d	-0.08	0.659	-0.26	0.162	0.25	0.188	0.47	0.010	0.34	0.065
GG:F, g/kg	-0.35	0.057	-0.02	0.892	0.43	0.018	0.41	0.024	0.08	0.665
UBF50, mm	0.78	0.001	0.30	0.108	-0.01	0.939	0.01	0.926	0.24	0.203
BUN50, mg/dL	-0.01	0.969	0.02	0.896	0.28	0.135	0.43	0.019	-0.24	0.208

[a]GFI, average daily feed intake, GLysI, lysine intake, GADG, average daily gain, and GG:F, gain:feed during the grower phase; UBF50, ultrasound back fat, and BUN50, blood urea nitrogen at the end of the grower phase.

Table 12. Correlation coefficients (r) of error residuals between serum metabolite concentrations at the end of the finisher phase and growth performance, ultrasound backfat, blood urea nitrogen, and carcass characteristics (Experiment 2)[a]

	Cholesterol		Triglycerides		Total protein		Albumin		Glucose	
Item	r	*P*	r	*P*	r	*P*	r	*P*	r	*P*
FFI, g/d	0.13	0.493	0.00	0.992	-0.12	0.523	0.17	0.359	-0.29	0.126
FLysI, g/d	-0.11	0.546	-0.09	0.640	-0.22	0.250	0.17	0.353	-0.20	0.285
FADG, g/d	-0.01	0.959	0.04	0.823	-0.03	0.857	0.04	0.830	-0.01	0.970
FG:F, g/kg	-0.05	0.768	0.01	0.924	0.13	0.486	-0.14	0.455	0.00	0.981
CBF, mm	0.34	0.062	-0.30	0.112	0.01	0.949	-0.16	0.400	-0.39	0.033
LMA, cm^2	0.08	0.666	0.24	0.197	-0.01	0.932	-0.03	0.865	0.17	0.362
BUN100, mg/dL	0.11	0.538	0.17	0.350	-0.03	0.855	0.22	0.231	0.04	0.804

[a]FFI, average daily feed intake, FLysI, lysine intake, FADG, average daily gain, and FG:F, gain:feed during the finisher 2 phase; CBF, carcass back fat, LMA, carcass longissimus muscle area, and BUN100, blood urea nitrogen at the end of the finisher 2 phase.

IV. SUMMARY AND CONCLUSIONS

In this research, two experiments were conducted to investigate the effect of dietary amino acid restrictions during the grower phase on blood metabolites in pigs with distinct genotypes. In Exp.1, pigs selected for lean growth efficiency and pigs selected randomly were fed two grower diets (6.1 and 11.1 g lysine/kg) and two finisher diets (6.1 and 8.9 g lysine/kg) in a 2 x 2 x 2 factorial arrangement of treatments. Pigs fed the high-amino acid grower diet had a higher albumin concentration at the end of the grower phase, and higher cholesterol and albumin concentrations at the end of the finisher phase than those fed the low-amino acid diet. Pigs fed the high-amino acid finisher diet had high albumin and low triglyceride and glucose concentrations at the end of the finisher phase, and tended to have a higher total protein concentration than those fed the low-amino acid finisher diet. The select line pigs had a higher cholesterol concentration at the end of the grower phase and higher triglyceride and albumin concentrations at the end of the finisher phase than the control line pigs.

In Exp. 2, pigs selected for lean growth efficiency and pigs selected randomly were fed grower diets (5.0, 7.0, 9.0, or 11.0 g lysine/kg) in a 2 x 4 factorial arrangement of treatments, and then offered common Finisher 1 and 2 diets. Pigs fed diets high in amino acids had decreasing concentrations of cholesterol and increasing concentrations of total protein and albumin at the end of the grower phase, and tended to

have increasing concentrations of total protein at the end of the finisher phase. The select line pigs had higher concentrations of triglycerides, total protein and glucose during the grower phase, and triglyceride, total protein and albumin concentrations during the finisher phase than the control line pigs.

The results indicated that the metabolite profile can be affected by both the early dietary amino acid restrictions and genotype. Wide variations in the potential of pigs for growth and protein retention continue to exist in the today's pig industry. Pigs with different genotypes may respond differently to dietary manipulations, and it is important to better understand the effect of genotype and dietary restrictions on serum metabolites to satisfy the pig's nutritional needs. The results of the present research may contribute to developing environmentally friendly, optimal feeding strategies for successful and sustainable pig production.

V. CUMULATIVE BIBLIOGRAPHY

AOAC. 1984. Official Methods of Analysis. 14th ed. Assoc. Offic. Anal. Chem., Washington, DC.

AOAC. 1990. Official Methods of Analysis. 15th ed. Assoc. Offic. Anal. Chem., Arlington, VA.

ARC. 1981. The Nutrient Requirements of Pigs. Commonwealth Agriculture Bureaux, Slough.

Armstrong, J. D., and J. H. Britt. 1987. Nutritionally-induced anestrus in gilts: Metabolic and endocrine changes associated with cessation and resumption of estrous cycles. J. Anim. Sci. 65:508-523.

Atinmo, T., C. Baldijao, W. G. Pond, and R. H. Barnes. 1976. Plasma insulin levels in weaned pigs fed protein or energy restricted diets. J. Nutr. 106:1654-1658.

Baetz, A. L., and W. L. Mengeling. 1971. Blood constituent changes in fasted swine. Am. J. Vet. Res. 32:1491-1498.

Barb, C. R. 1999. The brain-pituitary-adipocyte axis: Role of leptin in modulating neuroendocrine function. J. Anim. Sci. 77:1249-1257.

Barb, C. R., J. B. Barrett, R. R. Kraeling, and G. B. Rampacek. 2001. Serum leptin concentrations, leutinizing hormone and growth hormone secretion during feed and metabolic fuel restriction in the prepubertal gilt. Domest. Anim. Endocrinol. 20:47-63.

Barb, C., R. X. Yan, M. J. Azain, R. R. Kraeling, G. B. Rampacek, and T. G. Ramsay. 1998. Recombinant porcine leptin reduces feed intake and stimulates growth hormone secretion in swine. Domest. Anim. Endocrinol. 15:77-86.

Bikker, P., M. W. A. Verstegen, B. Kemp, and M.W. Bosch. 1996. Performance and body composition of finishing gilts (45 to 85 kilograms) as affected by energy intake and nutrition in earlier life: I. Growth of body and body components. J. Anim. Sci. 74:806-816.

Blum, J. W., W. Schnyder, P. L. Kunz, A. K. Blom, H. Bickel, and A. Schurch. 1985. Reduced and compensatory growth: Endocrine and metabolic changes during feed restriction and refeeding in steers. J. Nutr. 115:417-424.

Bohman, V. R. 1955. Compensatory growth of beef cattle: The effect of hay maturity. J. Anim. Sci. 14:249-255.

Breier, B. H., J. J. Bass, J. H. Butler, and P. D. Gluckman. 1986. The somatotrophic axis in young steers: Influence of nutritional status on pulsatile release of growth hormone and circulating concentrations of insulin-like growth factor-I. J. Endocrinol. 111:209-215.

Buonomo, F. C., and C. A. Baile. 1991. Influence of nutritional deprivation on insulin like growth factor-I, somatotropin, and metabolic hormones in swine. J. Anim. Sci. 69:755-760.

Buonomo, F. C., C. A. Baile, D. R. Campion, and T. J. Lauterio. 1987. Determination of insulin like growth factor-I (IGF-I) and IGF binding protein levels in swine. Anim. Endocrinol. 4:23-30.

Buonomo, F. C., and J. Klindt. 1993. Ontogeny of growth hormone (GH), insulin like growth factors (IGF-I and IGF-II) and IGF binding protein-2 (IGFBP-2) in genetically lean and obese swine. Domest. Anim. Endocrinol. 10:257-265.

Burleigh, B. D., S. R. Davis, P. D. Gluckman, H. V. Henderson, and S. C. Hodgkinson. 1987. Metabolic clearance rate of insulin like growth factor-I in fed and starved sheep. J. Endocrinol. 115:233-239.

Cameron, N. D., J. C. Penman, and E. McCullough. 2000. Serum leptin concentration in pigs selected for high or low daily food intake. Genet. Res. 75:209–213.

Campbell, R. G., and R. S. Biden. 1978. The effect of protein nutrition between 5.5 and 20 kg live weight on the subsequent performance and carcass quality of pigs. Anim. Prod. 27:223-228.

Campbell, R. G., and A. C. Dunkin. 1983a. The influence of protein nutrition in early life on growth and development of the pig. 1. Effects on growth performance and body composition. Br. J. Nutr. 50:605-617.

Campbell, R. G., and A. C. Dunkin. 1983b. The influence of protein nutrition in early life on growth and development of the pig. 2. Effects on the cellularity of muscle and subcutaneous adipose tissue. Br. J. Nutr. 50:618-626.

Campbell, R. G., M. R. Taverner, and D. M. Curic. 1983c. Effects of feeding level from 20 to 45 kg on the performance and carcass composition of pigs grown to 90 kg live weight. Livest. Prod. Sci. 10:265-272.

Chiba, L. I. 1994. Effects of dietary amino acid content between 20 and 50 and 100 kg live weight on the subsequent and overall growth performance of pigs. Livest. Prod. Sci. 39:213-221.

Chiba, L. I. 1995. Effects of nutritional history on the subsequent and overall growth performance and carcass traits of pigs. Livest. Prod. Sci. 41:151-161.

Chiba, L. I. 2000. Feeding systems for pigs. Pages 181-209 in M. K. Theodorou, and J. France, Feeding Systems and Feed Evaluation Models. CABI Publishing, Wallingford.

Chiba, L. I., H. W. Ivey, K. A. Cummins, and B. E. Gamble. 1999. Growth performance and carcass traits of pigs subjected to marginal dietary restrictions during the grower phase. J. Anim. Sci. 77:1769-1776.

Chiba, L. I., D. L. Kuhlers, L. T. Frobish, S. B. Jungst, E. J. Huff-Lonergan, S. M. Lonergan, and K. A. Cummins. 2002. Effect of dietary restrictions on growth performance and carcass quality of pigs selected for lean growth efficiency. Livest. Prod. Sci. 74:93-102.

Chiba, L. I., A. J. Lewis, and E. R. Peo. Jr. 1991a. Amino acid and energy interrelationships in pigs weighing 20 to 50 kilograms: I. Rate and efficiency of weight gain. J. Anim. Sci. 69:694-707.

Chiba, L. I., A. J. Lewis, and E. R. Peo. Jr. 1991b. Amino acid and energy interrelationships in pigs weighing 20 to 50 kilograms: II. Rate and efficiency of protein and fat deposition. J. Anim. Sci. 69:708-718.

Coleman, S. W., and B. C. Evans. 1986. Effect of nutrition, age and size on compensatory growth in two breeds of steers. J. Anim. Sci. 63:1968-1982.

Cote, P. J., and P. J. Wangsness. 1978. Rate, composition and efficiency of growth in lean and obese pigs. J. Anim. Sci. 47:441-449.

Cote, P. J., P. J. Wangsness, H. Varela-Alvarez, L. C. Griel, Jr., and J. F. Kavanaugh. 1982. Glucose turnover in fast-growing, lean and in slow-growing, obese swine. J. Anim. Sci. 54:89-94.

Critser, D. J., P. S. Miller, and A. J. Lewis. 1995. The effects of dietary protein concentration on compensatory growth in barrows and gilts. J. Anim. Sci. 73:3376-3383.

Deaton, J. W., F. N. Reece, L. F. Kubena, B. D. Colt, and J. D. May. 1973. The ability of the broiler to compensate for early growth depression. Poult. Sci. 52:262-265.

de Greef, K. H., B. Kemp, and M. W. A. Verstegen. 1992. Performance and body composition of fattening pigs of two strains during protein deficiency and subsequent realimentation. Livest. Prod. Sci. 30:141-153.

Deng YueLin, and N. Oksbjerg. 2002. The effect of different feeding levels on performance and serum insulin-like growth factor-I of growing-finishing pigs. J. South China Agric. Univ. 23:64-67.

Donker, R. A., L. A. denHartog, E. W. Brascamp, J. W. M. Merks, G. J. Noordewier, and G. A. J. Buiting. 1986. Restriction of feed intake to optimize overall performance and composition of pigs. Livest. Prod. Sci. 15:353-365.

Ellenberger, M. A., D. E. Johnson, G. E. Cartsens, K. L. Hossner, M. D. Holland, T. M. Nett, and C. F. Nockels. 1989. Endocrine and metabolic changes during altered growth rates in beef cattle. J. Anim. Sci. 67:1446-1454.

Emler, C. A., and D. S. Schalch. 1987. Nutritionally induced changes in hepatic insulin-like growth factor-I (IGF-I) gene expression in rats. Endocrinology. 120:832-840.

Fabian, J., L. I. Chiba, L. T. Frobish, W. H. McElhenney, D. L. Kuhlers, and K. Nadarajah. 2004. Compensatory growth and nitrogen balance in grower-finisher pigs. J. Anim. Sci. 82:2579-2587.

Fabian, J., L. I. Chiba, D. L. Kuhlers, L. T. Frobish, K. Nadarajah, C. R. Kerth, W. H. McElhenney, and A. J. Lewis. 2002. Degree of amino acid restrictions during the grower phase and compensatory growth in pigs selected for lean growth efficiency. J. Anim. Sci. 80:2610-2618.

Fabian, J., L. I. Chiba, D. L. Kuhlers, L. T. Frobish, K. Nadarajah, and W. H. McElhenney. 2003. Growth performance, dry matter and nitrogen digestibilities, serum profile, and carcass and meat quality of pigs with distinct genotypes. J. Anim. Sci. 81:1142-1149.

Friesen, K. G., J. L. Nelssen, R. D. Goodband, M. D. Tokach, J. A. Unruh, D. H. Kropf, and B. J. Kerr. 1995. The effect of dietary lysine on growth, carcass composition, and lipid metabolism in high-lean growth gilts fed from 72 to 136 kilograms. J. Anim. Sci. 73:3392-3401.

Gomez, R. S., A. J. Lewis, P. S. Miller, and H. Y. Chen. 2002. Growth performance, diet apparent digestibility, and plasma metabolite concentrations of barrows fed corn-

soybean meal diets or low-protein, amino acid-supplemented diets at different feeding level. J. Anim. Sci. 80:644-653.

Graham, N. M., and T. W. Searle. 1975. Studies in weaner sheep during and after a period of weight stasis. I. Energy and nitrogen utilization. Aust. J. Agric. Res. 26:135-141.

Hayden, J. M., J. E. Williams, and R. J. Collier. 1993. Plasma growth hormone, insulin-like growth factor, insulin, and thyroid hormone association with body protein and fat accretion in steers undergoing compensatory gain after dietary energy restriction. J. Anim. Sci. 71:3327-3338.

Hogberg, M. G., and D. R. Zimmerman. 1978. Compensatory responses to dietary protein, length of starter period and strain of pig. J. Anim. Sci. 47:893-899.

Horton, G. M. J., and W. Holmes. 1978. Compensatory growth by beef cattle at grassland or on an alfalfa-based diet. J. Anim. Sci. 46:297-303.

Houseknecht, K. L., C. A. Baile, R. L. Matteri, and M. E. Spurlock. 1998. The biology of leptin: A review. J. Anim. Sci. 76:1405–1420.

Kornegay, E. T., H. R. Thomas, and C. Y. Kramer. 1974. Evaluation of protein levels and milk products for pig starter diets. J. Anim. Sci. 39:527-535.

Kuhlers, D. L., K. Nadarajah, S. B. Jungst, B. L. Anderson, and B. E. Gamble. 2003. Genetic selection for lean feed conversion in a closed line of Duroc pigs. Livest. Prod. Sci. 84:75-82.

Kyriazakis, I., C. Stamataris, G. C. Emmans, and C. T. Whittemore. 1991. The effects of food protein content on the performance of pigs previously given foods with low or moderate protein content. Anim. Prod. 52:165-172.

Lawrence, T. L. J., and V. R. Fowler. 1997. Growth of Farm Animals. CAB International, Wallingford.

Leili, S., F. C. Buonomo, and C. G. Scanes. 1997. The effects of dietary restriction on insulin-like growth factor-I (IGF-I) and II, and IGF-binding protein in chickens. Proc. Soc. Exp. Biol. Med. 216:104-111.

Lowrey, R. S., W. G. Pond, R. H. Barnes, L. Krook, and J. K. Loosli. 1962. Influence of caloric level and protein deficiency in the young pig. J. Nutr. 78:245-252.

Lund-Larsen, T. R., and H. Bakke. 1975. Growth hormone and somatomedin activities in lines of pigs selected for rate of gain and thickness of backfat. Acta Agric. Scand. 25:231-239.

McCusker, R. H., P. J. Wangsness, L. C. Griel, Jr., and J. F. Kavanaugh. 1984. Effects of feeding, fasting and refeeding on growth hormone and insulin in obese pigs. Physiol. Behavior 35:383-388.

McMeekan, C. P. 1940. Growth and development in the pig, with special reference to carcass quality characters. III. Effect of plane of nutrition on the form and composition of the bacon pig. J. Agr. Sci. (Camb.) 30:511-516.

McNamara, J. P., and R. J. Martin. 1982. Muscle and adipose tissue lipoprotein lipase in fetal and neonatal swine as affected by genetic selection for high or low backfat. J. Anim. Sci. 55:1057–1061.

McNeel, R. L., S. T. Ding, E. O'Brian Smith, and H. J. Mersmann. 2000. Effect of feed restriction on adipose tissue transcript concentrations in genetically lean and obese pigs. J. Anim. Sci. 78:934-942.

Mersmann, H. J. 1991. Characteristics of obese and lean swine. Pages 75-89 in E. R. Miller, D. E. Ullrey, and A. J. Lewis, Swine Nutrition. Butterworth-Heinemann, Boston.

Mersmann, H. J., M. D. MacNeil, S. C. Seidemann, and W. G. Pond. 1987. Compensatory growth in finishing pigs after feed restriction. J. Anim. Sci. 64:752-764.

Mersmann, H. J., W. G. Pond, and J. T. Yen. 1982. Plasma glucose, insulin and lipids during growth of genetically lean and obese swine. Growth 46:189-198.

NRC. 1988. Nutrient requirements of swine. 9th rev. ed. Natl. Acad. Press, Washington, DC.

NRC. 1998. Nutrient Requirements of Swine. 10th ed. Natl. Acad. Press, Washington, DC.

Osborne, T. B., and L. B. Mendel. 1915. The resumption of growth after long continued failure to grow. J. Biol. Chem. 23:439-443.

Ouellet, D. R., J. R. Seoane, J. F. Bernier, and H. Lapierre. 2001. Effect of feed restriction on plasma concentration of hormones and metabolites in steers fed grass silage. Can. J. Anim. Sci. 81:553-561.

Owen, J. B., W. F. Ridgman, and D. Wyllie. 1971. The effect of food restriction on subsequent voluntary intake of pigs. Anim. Prod. 13:537-543.

Pond, W. G., W. Insull, H. J. Mersmann, W. W. Wong, K. B. Harris, H. R. Cross, E. O. Smith, J. P. Heath, and L. G. Komuves. 1992. Effect of dietary fat and cholesterol level on growing pigs selected for three generations for high or low serum cholesterol at age 56 days. J. Anim. Sci. 70:2462-2470.

Pond, W. G., Jong-Tseng Yen, H. J. Mersmann, and W. M. Haschek. 1986a. Comparitive effects of dietary protein and cholesterol-fat content on genetically lean and obese pigs. J. Nutr. 116:1116-1124.

Pond, W. G., H. G. Jung, and V. H. Varel. 1988. Effect of dietary fiber on young adult genetically lean, obese and contemporary pigs: bodyweight, carcass measurements, organ weights and digesta content. J. Anim. Sci. 66:699-706.

Pond, W. G., and H. J. Mersmann. 1990. Differential compensatory growth in swine following control of feed intake by a high-alfalfa diet fed ad libitum or by limited feed. J. Anim. Sci. 68:352-362.

Pond, W. G., H. J. Mersmann, and J. T. Yen. 1985. Effect of obesity per se on plasma lipid and aortic responses to diet in swine. Proc. Soc. Exp. Bio. Med. 179:90-95.

Pond, W.G., H. J. Mersmann, and L. D. Young. 1986b. Heritability of plasma cholesterol and triglyceride concentration in swine. Proc. Soc. Exp. Bio. Med. 182:221-224.

Pond, W. G., D. R. Su, and H. J. Mersmann. 1997. Divergent concentrations of plasma metabolites in swine selected for seven generations for high or low plasma total cholesterol. J. Anim. Sci. 75:311-316.

Pond, W. G., J. T. Yen, and R. N. Lindvall. 1980. Early protein deficiency: Effects on later growth and carcass composition of lean or obese swine. J. Nutr. 110:2506-2513.

Pond, W. G., J. T. Yen, R. N. Lindvall, and D. Hill. 1981. Dietary alfalfa meal for genetically obese and lean growing pigs: Effect on body weight gain and on carcass and gastrointestinal tract measurements and blood metabolites. J. Anim. Sci. 51:367-373.

Preedy, V. R., and P. J. Garlick. 1986. The response of muscle protein synthesis to nutrient intake in postabsorbtive rats: The role of insulin and amino acids. Biosci. Rep. 6:177-183.

Prince, T. J., S. B. Jungst, and D. L. Kuhlers. 1983. Compensatory responses to short-term feed restriction during the growing period in swine. J. Anim. Sci. 56:846-852.

Ramsay, T. G., X. Yan, and C. Morrison. 1998. The obesity gene in swine: Sequence and expression of porcine leptin. J. Anim. Sci. 76:484-490.

Rayner, D. V., G. D. Dalgliesh, J. S. Duncan, L. J. Hardie, N. Hoggard, and P. Trayhurn. 1997. Postnatal development of the ob gene system: elevated leptin levels in suckling fa/fa rats. Am. J. Physiol. 273:446-450.

Robinson, D. W. 1964. The plane of nutrition and compensatory growth in pigs. Anim. Prod. 6:227-236.

Rompala, R. E., S. D. M. Jones, J. G. Buchanan-Smith, and H. S. Bayley. 1985. Feedlot performance and composition of gain in late-maturing steers exhibiting normal and compensatory growth. J. Anim. Sci. 61:637-646.

Rule, D. C., S. B. Smith, and H. J. Mersmann. 1989. Glycerolipid biosynthesis in porcine adipose tissue in vitro: Effect of adiposity and depot site. J. Anim. Sci. 67:364-373.

Ryan, W. J., I. H. Williams, and R. J. Moir. 1993. Compensatory growth in sheep and cattle. I. Growth pattern and feed intake. Aust. J. Agric. Res. 44:1609-1616.

Smith, J. W., 2nd, M. D. Tokach, P. R. O'Quinn, J. L. Nelssen, and R. D. Goodband. 1999. Effects of dietary energy density and lysine:calorie ratio on growth performance and carcass characteristics of growing-finishing pigs. J. Anim. Sci. 77: 3007-3015.

Stamataris, C., G. M. Hillyer, C. T. Whittemore, G. C. Emmans, A. G. Taylor, and P. Phillips. 1985. Performance and body composition of young pigs following a period of growth retardation by food restriction. Anim. Prod. 40:536. (Abstr.)

Steele, N. C., L. T. Frobish, and M. Keeney. 1974. Lipogenesis and cellularity of adipose tissue from genetically lean and obese swine. J. Anim. Sci. 39:712–718.

Valaja, J., T. Alaviuhkola, K. Suomi, and I. Immonen. 1992. Compensatory growth after feed restriction during the rearing period in pigs. Agric. Sci. Finland. 1:15-20.

Wangsness, P. J., W. A. Acker, J. H. Burdette, L. F. Krabill, and R. Vasilatos. 1981. Effect of fasting on hormones and metabolites in plasma of fast-growing, lean and slow-growing obese pigs. J. Anim. Sci. 52:69-74.

Whalstrom, R. C., and G. W. Libal. 1983. Compensatory responses of swine following protein insufficiency in grower diets. J. Anim. Sci. 56:118-124.

Wilson, P. N., and D. F. Osbourn. 1960. Compensatory growth after undernutrition in mammals and birds. Biol. Rev. 35:324.

Wood, J. D., N. G. Gregory, G. M. Hall, and D. Lister. 1977. Fat mobilization in Pietrain and Large White pigs. Brit. J. Nutr. 37:167-176.

Wright, I. A., and A. J. F. Russel. 1991. Changes in the body composition of beef cattle during compensatory growth. Anim. Prod. 52:105-110.

Wyllie, D., V. C. Speer, R. C. Ewan, and V. W. Hays. 1969. Effects of starter protein level on performance and body composition of pigs. J. Anim. Sci. 29:433-438.

Yambayamba, E. S. K., M. A. Price, and G. R. Foxcroft. 1996. Hormonal status, metabolic changes, and resting metabolic rate in beef heifers undergoing compensatory growth. J. Anim. Sci. 74:57-69.

Yu, M. E., and F. E. Robinson. 1992. The application of short-term feed restriction to broiler chicken production: A review. J. Appl. Poult. Res. 1:147-149.

Zimmerman, D. R., and S. Khajarern. 1973. Starter protein nutrition and compensatory responses in swine. J. Anim. Sci. 36:189-194.

Zubair, A. K., and S. Leeson. 1994. Effect of varying period of early nutrient restriction on growth compensation and carcass characteristics of male broilers. Poult. Sci. 73:129-136.

APPENDICES

Appendix A: Principle of the Cholesterol Analysis (Diagnostic Chemicals Ltd., Oxford)

All cholesterol esters present in serum or plasma are hydrolyzed quantitatively into free cholesterol and fatty acids by microbial cholesterol esterase.

The reaction proceeds as follows:

$$\text{Cholesterol esters} + H_20 \xrightarrow[\text{esterase}]{\text{Cholesterol}} \text{Cholesterol} + \text{fatty acids}$$

In the presence of oxygen, free cholesterol is oxidized by cholesterol oxidase to cholest -4- en -3- one.

$$\text{Cholesterol} + 0_2 \xrightarrow[\text{oxidase}]{\text{Cholesterol}} \text{Cholest -4- en -3- one} + H_20_2$$

The H_20_2 reacts, in the presence of peroxidase (POD), with phenol and 4-aminophenazone to form an O-quinoneimine dye.

$$2H_20_2 + \text{4- aminophenazone} + \text{phenol} \xrightarrow{\text{POD}}$$

$$\text{4-(}p\text{-benzoquinone-monoimino)-phenazone} + 4\ H_20$$

The intensity of color formed is proportional to the cholesterol concentration and can be measured photometrically.

Appendix B: Principle of the Triglyceride Analysis (Diagnostic Chemicals Ltd., Oxford)

Triglycerides in serum are assayed entirely by a series of enzymatic reactions. After hydrolysis by microbial lipases, triglycerides yield glycerol and free fatty acids (FFA). Glycerol is phosphorylated by adenosine-5'-triphosphate (ATP) to glycerol-1-phosphate (G-1-P) in a reaction catalyzed by glycerol kinase (GK). The G-1-P is oxidized to dihydroxyacetone phosphate (DAP) in a reaction catalyzed by the enzyme glycerol phosphate oxidase (GPO). In this reaction, hydrogen peroxide (H_2O_2) is produced in equimolar concentration to the level of triglycerides present in the sample. In the indicating Trinder type reaction, H_2O_2 reacts with 4-aminoantipyrine (4-AAP) and N-Ethyl-N-(3-Sulfopropyl)-m-anisidine (ESPAS), which is catalyzed by peroxidase (POD). The result of this oxidative coupling is a quinoneimine red colored dye. The absorbance at 550 nm of this dye in solution is proportional to the concentration of triglycerides in the sample. The series of reactions involved in the assay are indicated below.

$$\text{Triglycerides} \xrightarrow{\text{Lipase}} \text{Glycerol} + \text{FFA}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{GK}} \text{G -1- P} + \text{ADP}$$

$$\text{G-1-P} + O_2 \xrightarrow{\text{GPO}} \text{DAP} + H_2O_2$$

$$2\ H_2O_2 + 4\text{AAP} + \text{ESPAS} \xrightarrow{\text{POD}} \text{Quinonimine dye} + 4\ H_2O$$

Appendix C: Principle of the Total protein Analysis (Diagnostic Chemicals Ltd., Oxford)

At an alkaline pH, the protein reacts with copper in the biuret reagent, causing an increase in absorbance. The increase in absorbance at 540 nm because of the formation of the colored complex is directly proportional to the concentration of protein in the reaction.

$$\text{Protein} + Cu^{++} \longrightarrow \text{Blue-violet complex}$$

Appendix D: Principle of the Albumin Analysis (Diagnostic Chemicals Ltd., Oxford)

At pH 4.1, albumin displays a sufficient cationic character to be able to bind with bromocresol green (BCG), an anionic dye, to form a blue-green complex.

$$\text{Albumin} + \text{BCG} \xrightarrow{\text{pH 4.1}} \text{albumin BCG complex}$$

The color intensity of the blue-green color is directly proportional to the albumin concentration and can be determined photmetrically.

Appendix E: Principle of the Glucose Analysis (Diagnostic Chemicals Ltd., Oxford)

Glucose is phosphorylated by hexokinase (HK) in the presence of adenosine triphosphate (ATP) and magnesium to form glucose-6-phosphate (G-6-P) and adenosine diphosphate (ADP). The G-6-P is then oxidized by glucose-6-phosphate dehydrogenase (G-6-PDH) in the presence of nicotinamide adenine dinucleotide (NAD) producing 6-phosphogluconate and NADH. The formation of NADH causes an increase in absorbance at 340 nm which is directly proportional to the concentration of glucose in the sample.

$$\text{Glucose} + \text{ATP} \xrightarrow[Mg^{2+}]{\text{Hexokinase}} \text{Glucose-6-phosphate} + \text{ADP}$$

$$\text{Glucose-6-phosphate} + NAD^{+} \xrightarrow{\text{G-6-PDH}} \text{6-phosphogluconate} + \text{NADH} + H^{+}$$

Appendix F: Statistical models for the analysis of metabolite data at the end of the grower phase in Exp. 1[a]

	Response criteria				
Item	Cholesterol	Triglycerides	Total protein	Albumin	Glucose
Grower	1	1	1	1	1
Genotype	1	1	1	1	1
Sex	1	1	1	1	1
Bldg[b]	1	1	1	1	1
Grower x genotype	1	1	1	1	1
Grower x bldg	-	-	1	1	-
Sex x bldg	1	1	-	-	-
Grower x bldg x sex	-	-	2	-	-
Grower x genotype x sex	-	-	2	-	-
Initial weight	1	1	1	1	1
Error	24	24	20	24	25
Total	31	31	31	31	31

[a] Covariates are italicized.
[b] Building.

Appendix G: Statistical models for the analysis of metabolite data at the end of the finisher phase in Exp. 1[a]

	Response criteria				
Item	Cholesterol	Triglycerides	Total protein	Albumin	Glucose
Genotype	1	1	1	1	1
Sex	1	1	1	1	1
Grower	1	1	1	1	1
Finisher	1	1	1	1	1
Bldg[b]	1	1	1	1	1
Grower x finisher	1	1	1	1	1
Genotype x grower	1	1	1	1	1
Genotype x finisher	1	1	1	1	1
Genotype x sex	-	-	-	-	1
Sex x finisher	-	-	-	1	-
Bldg x sex	1	1	-	-	-
Bldg x grower	-	-	1	-	-
Bldg x finisher	-	1	-	-	-
Bldg x genotype x grower	-	-	2	-	-
Bldg x genotype x sex	-	3	-	-	-
Bldg x sex x grower	-	-	-	-	3
Genotype x grower x finisher	1	1	1	1	1
Genotype x sex x grower	-	-	-	-	2
Final weight	1	1	1	1	1
Error	20	16	18	20	15
Total	31	31	31	31	31

[a]Covariates are italicized.
[b]Building.

Appendix H: Statistical models for the analysis of metabolite data at the end of the grower and finisher phases in Exp. 2[a]

	Response criteria				
Item	Cholesterol	Triglycerides	Total protein	Albumin	Glucose
Genotype	1	1	1	1	1
Sex	1	1	1	1	1
Diet	3	3	3	1	3
Replication	1	1	1	1	1
Replication x diet	3	-	3		-
Replication x genotype	-	1	-	1	-
Replication x sex	1	-	1		-
Genotype x diet	3	3	3	1	3
Genotype x sex	1	-	-	-	-
Replication x genotype x sex	-	-	-	-	-
Replication x sex x diet	6	-	-	-	-
Initial concentration[b]	1	1	1	1	1
Error	10	20	17	24	21
Total	31	31	31	31	31

[a]Covariates are italicized.
[b]Initial concentration of metabolites at the beginning of the experiment.