



**The production performance of two pig genotypes on varying levels of dietary protein.**

By

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## Abstract

The production performance of two pig genotypes on varying levels of dietary protein

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The production performance of two pig genotypes were compared by feeding them four different protein levels, NRC +20%, NRC + 10%, NRC default and NRC –10 %. The energy (DE) levels were the same 14MJ/kg. The diets were fed *ad libitum*. Pigs of the highest protein diet were serially slaughtered at 5 days and 14 days of age, 30 kg, 50 kg, 70 kg, 90 kg and 110 kg live weight for chemical analysis of the empty body. The other pigs were slaughtered commercially at 70 kg, 90 kg and 110 kg live weight. Average daily gain (ADG), feed intake (FI) and the feed conversion ratio (FCR) were measured weekly. The pigs were weighed weekly. The dietary protein content had no significant effect ( $P<0.05$ ) on ADG, FI, FCR, live weight at slaughter, cold carcass weight or slaughter percentage. The two genotype did not differ much for ADG, FCR, FI, live weight, cold weight or slaughter percentage. The dry matter (DM) and protein:fat ratio differed significantly ( $P<0.05$ ) only in Stage one between the two genotypes. There were no other significant differences between the genotypes in any Stage for dry matter, fat, ash and protein. The data suggests that under the conditions of this study, there are no significant differences between the two genotypes.

Die produksie prestasie van twee vark genotipes op verskillende dieet proteïen vlakke.

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Die produksie prestasie van twee vark genotipes is vergelyk deur die varke vier verskillende proteïen vlakke te voer, NRC +20%, NRC +10%, NRC standaard en NRC -10%. Die energie (VE) vlakke is konstant gehou op 14MJ/kg. Vark wat die hoogste proteïen dieet ontvang het, is gebruik vir die vergelykende slag tegniek. Vark is vir chemiese analises geslag op 5 dae, 14 dae, 30kg, 50kg, 70kg, 90kg en 110kg lewendige massa. Die ander varke is kommersieel geslag op 70kg, 90kg en 110kg lewendige massa. Die gemiddelde daaglikse toename (GDT), voerinnome (VI), voeromset verhouding (VOM) en die massas van die varke is weekliks bepaal. Die verskillende proteïen diëte het nie 'n betekenisvolle ( $P < 0.05$ ) invloed op GDT, VI, VOM, lewendige massas, koue massas of uitslagpersentasies gehad nie. Die twee genotipes het nie betekenisvol verskil ten opsigte van GDT, VOM, VI, lewendige massa, koue massa en uitslag persentasies. Die droë materiaal en proteïen: vet verhouding het betekenisvol verskil tussen die twee genotipes in die eerste Stadium. Daar was geen verdere betekenisvolle verskille in die ander Stadiums tussen die genotipes nie. Die genotipes het nie betekenisvol van mekaar verskil vir vet, as of proteïen nie. Uit die data bleik dit dat die twee genotipes nie betekenisvol van mekaar verskil nie.



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## Chapter 1

### Introduction

The profitability of a pig production unit depends on a lot of different aspects. Good management, adequate facilities, the correct feeding strategy for the genotype used, a genotype that would be the best for the market and a marketing strategy. This study will concentrate on two genotypes and their differences in production performance and chemical composition. This is necessary to distinguish between the genotypes which performs the best. A genotype with higher average daily gains, less feed intake, lower feed conversion ratio and less back fat would be the most cost effective.

Genotypes differ in their mature composition and how they develop towards it. The mature composition can be quantified by measuring the body protein weight and the degree of fatness of the animal. Genetic potential can best be described as a genetically defined limit in the response of an animal to a non-limiting environment. According to Emmans (1988) the genetic potential is the animal's potential rate of growth at a given time. Growth can be measured in terms of protein deposition rate as opposed to an increase in body mass.

Lean growth or the accretion of muscle is directly related to body protein deposition. The genetic potential of the pig determines its maximum protein deposition potential. Modern genotypes have protein deposition rates of 210 and 240 g/day (Close, 1994). Close (1994) defined three classes of genotypes: the superior, genetically improved animals with the highest protein deposition rates, normal animals and the less improved animals with the lowest protein deposition rates. Protein deposition rates follow a rainbow-like curve. Genotype and mature size determine the point of maximum protein deposition rate. The genetically improved animals have a higher peak of maximum protein deposition rates and a slower decline than less improved animals.

Body tissue contains a certain level of fat, which is best described in relation to body protein in the form of a lipid:protein ratio at maturity. An allometric coefficient relating lipid content to protein can be used as well. Different genotypes have different requirements for deposition of essential body lipids associated with protein deposition even when energy intake limits protein deposition (Whittemore, 1983; Black and de Lange, 1995). The environment and

nutrition influence the genetic characteristic of lipid growth. The diet must not limit protein growth nor allow excess fat deposition to occur.

The relative proportions of moisture and ash are less likely to vary between sexes and breeds than the lipid proportion (Moughan and Verstegen, 1988).

Genetic differences can be found between breeds, between genetic lines within a breed and between individuals within a line (Ellis, Miller and Cisneros, 1997). These authors state that the genetic relationship between appetite and lean growth can be positive as well as negative and leaner genotypes can grow faster than fatter genotypes.

Feed is used for body maintenance functions, lean tissue growth and body fat deposition. Only nutrients supplied in excess of maintenance requirements can be used for growth in the form of lean tissue and body fat.

Protein is metabolically a very active tissue, therefore most maintenance requirements is associated with protein tissue. The maintenance requirements of modern genotypes represent almost 40% of the total potential intake compared to only 25% of unimproved pigs. Faster growth can reduce the maintenance costs, by reducing the maintenance proportion of each day's growth and saving time spent in the finishing house.

Fatty tissue requires more than three times the amount of energy that is required for a unit of lean growth (Whittemore, 1994). The feed efficiency decreases when the lipid:protein ratio increases.

Residual feed intake (RFID) was defined by De Haer *et al* (1993) as the daily feed intake (FID) adjusted for predicted feed intake (pFID) based on metabolic body weight (MBW) and performance level (body weight gain and lean percentage in the carcass). RFID is a measure of feed use per kg gain at a certain level of MBW and lean percentage. A low RFID is reflective of a more efficient pig, which requires less feed than the average pig for a certain level of MBW and performance. A high level of feed intake activity does not give a high growth performance, but it does give a high RFID, which means a high overall FID and, therefore, high feed costs.



The Nil Hypothesis ( $H_0$ ) to be tested is that there are no differences between the genotypes in protein deposition, lipid:protein ratio, feed intake, feed efficiency and carcass analysis.

The aim of this study is to determine if there are any significant differences between the MPD and PIC genotypes for growth performance and carcass analysis.

## Chapter 2

### Literature review

#### 2.1. Gompertz growth function:

The method of evaluating genotypes proposed by Emmans (1981) uses the Gompertz growth function to describe the potential growth rate, which an animal desires to achieve. There are good reasons for selecting this growth function. The values for only three parameters need be known, all of which have biological meaning. The function fits the data as well as other more complex growth functions that do not have the above properties; and that allometric relationships between the chemical and physical components of the body can be defined in terms of the growth rate parameter in this function. Two assumptions are made in defining the chemical composition of the genotype. The first being that body protein, water, lipid and ash each have potential growth rates which are Gompertz functions of time, and the second being that each of these body components has the same growth rate parameter for a given genotype. Instead of predicting growth from the empty body as a whole, body protein is predicted and used as the base component to predict the remaining three components. This is done by making use of the allometric relationships that exist between protein and moisture, ash and lipid. Body protein is used to describe the current state of the pig and the subsequent growth rates of the remaining body components; body moisture, ash and lipid (Emmans and Fisher, 1986). In a study by Ferguson and Gous (1993a), they used the Gompertz function as follows:

$$P_t = P_m \times e^{-e[\log_e(-\log_e U_0) - (B \times t)]}$$

Where  $P_m$  = mature body protein weight (kg);  $U_0$  = (body protein weight at birth)/ $P_m$ ;  $B$  = rate of maturing (per day);  $t$  = age (days).

Therefore the information required to describe a pig consists of three parameters in the Gompertz function, namely, the initial and the mature body protein weight and rate of maturing. In addition to these inherent growth parameters, the allometric relationships between body protein and the other chemical components of the body need to be defined if these components are to be estimated from the protein weight. Moisture and ash are relatively constant relationships for most genotypes. The relationship between protein and lipid is a heritable characteristic specific to the genotype of pig. To quantify this inherent fatness an additional parameter is required; lipid:protein ratio. This ratio defines the desired amount of lipid at maturity relative to protein, being specific to a given genotype. The lipid:protein ratio

at maturity of fat strains of pigs is in excess of six, whereas very lean strains can have a ratio as little as two (Whittemore *et al.*, 1988).

A non-limiting environment is a prerequisite for measuring the relative proportions of these components. To ensure that the animal is not fatter than it would like to be and, thereby to overestimate the mature lipid size, it is necessary to feed the animal a high protein diet throughout the growth period. This will afford those pigs that are fatter than they intrinsically desire to be, the opportunity to lose excess fat (Ferguson and Gous, 1993a).

The ability to define an animal in terms of a few meaningful biological characteristics is desirable for a number of reasons. A more definitive strategy for genetic selection of improved strains of pigs would result, as would a more accurate means of predicting growth performances and nutrient requirements.

Ferguson and Gous (1993b) tested the procedure mentioned above. They concluded that it allows an accurate means of predicting protein growth, which is a prerequisite for accurate genetic selection and for correct formulation of nutrient requirements for different genotypes and sexes of growing pigs. To be able to obtain the protein retention of an animal at different stages of growth affords producers the opportunity of manipulating diets so as to optimise productivity, especially during the declining phase of protein retention where fat deposition is increasing.

## 2.2 Protein

Protein deposition is supposed to increase linearly with energy intake up to its maximum value (PD<sub>max</sub>) and to plateau afterwards in the model of Whittemore and Fawcett (1976). From a biological point of view, this broken line model is controversial and a curvilinear model would fit better the biological processes involved in protein deposition. In the study of Quiniou *et al.* (1999), it was unlikely that protein deposition would continue to increase at high levels of energy intake; then models with a maximum or an asymptote are most often adopted. The amount of ME<sub>g</sub> not deposited as protein is supposed to be exclusively used for lipid deposition. The broken line model between ME<sub>g</sub> and protein deposition would then imply a higher slope of lipid deposition when PD<sub>max</sub> is reached. With regard to the high energy content of lipids and the low range of ME<sub>g</sub> intake above the value corresponding to the achievement of PD<sub>max</sub>, such a difference is not observed. The relationship between lipid deposition and energy intake is considered as linear whichever the level of energy supply.

Cambell and Taverner (1988) conducted an experiment to provide information on the extent to which the growing pig's capacity for protein deposition might be affected by genotype and castration. The results showed that both factors affected, although somewhat differently, the relationship between energy intake and protein deposition and, as a consequence, growth performance and body composition. The results suggest that the intense selection of these animals under *ad libitum* feeding had raised their genetic ceiling for protein deposition beyond the upper limit of appetite. The difference in maximal protein deposition and in the relationship between energy intake and protein deposition was directly responsible for the concomitant differences between strains in growth performance and body fat content at the higher levels of feeding. As well as for the increase in the magnitude of these differences with each increase in energy intake above 7.9 Mcal DE/day.

Kem *et al.* (1991) fed three protein levels to genetically lean and obese Landrace pigs. The dietary treatments were lysine concentrations of 1.22 (T1), 1.02 (T2) and 0.83% (T3), with corresponding concentrations of crude protein (CP) of 19.7, 16.8 and 13.7%. The growth period was from 30 – 90 kg live weight. Dietary CP content had no significant effect on mean voluntary DE intakes and daily gains. Obese pigs consumed highly significantly more DE than lean pigs, and also needed highly significantly more DE/kg gain, but they had similar daily gains. The protein deposition rate curves peaked at  $\pm$  56 kg live weight (51 kg for obese gilts and 64 kg for lean boars). A reduction of 15% in dietary protein content (T2) had no apparent effect on protein deposition. Pigs from T3, fed 30% less protein than pigs from T1, deposited only 2 g (1.9%) less protein/day at 32 kg live weight, 2 g (1.6%) less at maximum deposition and 2 g (1.9%) less at 90 kg live weight. The data on live weight gains and protein deposition confirm the findings of Siebrits *et al.* (1986) that DE (feed) intake in *ad libitum* fed pigs peaks at a live weight approximately 20 kg higher than live weight gain and protein growth. Apart from type and sex effects, deposition rates not only tend to peak at a later stage in boars and lean type pigs, but also decline at a slower rate thereafter. The rate of protein deposition is only slightly reduced by the level of dietary protein, but only at the lowest of the three levels fed (T3). No advantage could or should therefore be gained by feeding a protein level higher than the 16.8% CP (T2) to pigs equal in growth potential compared to those used in their study, providing the animals consume an adequate daily amount of DE.

Quiniou *et al.* (1995) studied the effect of two dietary crude protein levels (17.7: L and 24.3: H) on nitrogen and energy retention between 20 and 50 kg live weight. The L pigs retained more energy as fat (7.96 vs. 4.94 MJ/day) and less energy as protein (3.19 vs. 4.02 MJ/day)

than H pigs. In agreement with Henry (1985), their results indicate a specific adjustment of voluntary feed intake on dietary CP content. The average daily gain of H pigs tended to be higher during the experiment (926 vs. 856 g/day). The difference in growth rate between the two groups of pigs could be explained by the variation in the daily amount of amino acids intake. In spite of their higher feed intake, the L pigs had a higher feed:gain ratio than the H pigs (1.80 vs. 1.47 kg DM/kg,  $P < 0.01$ ). This has to be related with the higher energy content of the empty body weight gain.

Rao and McCracken (1991) found that in improved genotypes: (1) the response in protein/lean tissue deposition to increasing ME intake is linear up to levels above the normal *ad libitum* consumption of energy. (2) The protein deposition potential is in excess of 210 g/day. (3) The rate of protein deposition between 35 kg and 85 kg live weight is almost constant. (4) The decrease in protein deposition due to restriction in energy intake is greater than in unimproved pigs. (5) The maintenance energy requirement is greater than in unimproved pigs.

The rate of protein deposition is only dependent on the rate of protein supply, at low levels of protein intake. At high levels of protein intake protein deposition depends only on the energy supply. This is according to Kyriazakis and Emmans (1992b) and it is consistent with a previous experiment of Kyriazakis and Emmans (1992a).

The daily rate of protein retention ( $Pr$ ) achieved by growing pigs is a function of a wide variety of environmental influences, but primarily of dietary amino acids and energy. For an individual, or for a population of like individuals, the upper limit to  $Pr$ , or the maximum potential daily rate of protein retention ( $Pf$ ) is an inherently unknowable characteristic, but if there is an upper limit, it is presumably related to the genetic constitution of the individual animal. For a population of animals of similar genetic constitution, a variance would be associated with the estimate of  $Pf$ . When limits to nutrient supply control  $Pr$ , then  $Pf$  has no relevance to performance. But for the estimation of nutrient requirements, for the calculation of optimum feeding strategies, and for genetic selection of improved strains of pigs for enhanced protein retention rate, there is interest in possible values for  $Pf$ . Not merely measured values of  $Pr$ , which may do no more than demonstrate an environmental constraint. In particular, knowledge of the age or weight at which  $Pf$  can first be achieved, and the nature of any relationship with subsequent age or weight is essential to form a view of potential growth rate. Only  $Pr$ , not  $Pf$  can be measured experimentally and only in the absence of nutritional and other environmental constraints will  $Pr$  approach  $Pf$ . This would be shown by

a plateau in the response of  $P_r$  to increasing nutrient inputs, but there is no sure method of knowing that any maximum potential has indeed been approached. It is simplistic to assume, merely because  $P_r$  does not respond further to increasing nutrient supply, that necessarily  $P_r = P_f$ . However, given that the likely negative effects of the environment are minimised and that nutrient supply is as generous as possible, then it may be hoped that  $P_r$  at least more nearly approaches  $P_f$  than would otherwise be the case. Within the bounds of experimental possibility such strategies represent the only way open to derive some guidance as to the value of  $P_f$ . A further difficulty in the interpretation of measured response is that the experimental period may cover early growth when  $P_r$  is limited by appetite ( $P_r < P_f$ ) and later when  $P_r$  is indeed a true reflection of  $P_f$ . Analysis of such data would not demonstrate a linear plateau effect, but rather a curvilinear form of response (Whittemore *et al.*, 1988).

### 2.3 Energy

The main objective of pig production is to increase lean gain while limiting fat deposition, which is a tissue with a high-energy cost and low commercial return. Physical body composition at slaughter results from the relative development of body tissues and chemical components, which are mainly determined by the appetite of animals and/or nutrient supplies. In fact, voluntary feed intake is influenced by many factors such as growth potential (genotype, sex and stage of growth) on one hand and housing conditions (temperature, air speed, relative humidity, floor type, space allowance), health status and feeding conditions on the other hand. As feeding represents one of the most expensive components of pig production, it is of major interest to optimise efficiency of utilisation of nutrient supplies. In addition, it would contribute to reduce associated nutritional losses through matching nutrient supplies to nutrient requirements. For this purpose, characterisation of the relationship between nutrient supplies and deposition rates of body components is required. In lean or fat pigs, 1g of protein deposition is associated with deposition of 3.5g to 4g of water and minerals and results in a body weight gain of 4.5g to 5g, whereas 1g of lipid deposition induced 1g of body weight gain (Quiniou and Noblet, 1995). Under optimal breeding conditions, pigs are considered to deposit protein at a level close or equal to their potential maximum level, so called  $P_{dmax}$ . The variation of  $P_{dmax}$  with body weight has been widely studied but is still controversial. Some authors considered protein deposition as constant over 20 – 110 kg body weight range in lean pigs (Whittemore, 1993; Quiniou *et al.*, 1996a). It can be concluded that the increasing body fatness with body weight results rather from an increase of lipid deposition than from a decrease of  $P_{dmax}$ .



Castration has been shown to decrease P<sub>dmax</sub> and to increase lipid deposition (Quiniou *et al.*, 1996a). The combination of results on weight and chemical composition of body tissues shows that, on average, more than 55% of protein and 80% of lipids are deposited in lean and fat tissues, respectively (Quiniou and Noblet, 1995). The relationships between protein deposition and lean gain on one hand and lipid deposition and fat gain on the other hand are affected by the growth potential of the animal. On average, over the 20 – 110 kg body weight range, 1g of protein deposition is associated with 3.2g and 2.7g of lean gain in lean and fat types of pigs respectively. By contrast, 1g of lipid deposition is associated with 1g of fat gain in any breed. Such a relationship between chemical components and tissue deposition explains why the fat gain associated to lean gain or lipid deposition associated to protein deposition is strongly affected by genotype and sex. Lean gain in gilts is comparable to barrows whereas their fat gain is comparable to that of boars, resulting in an intermediate physical composition of body weight gain in gilts between boars and barrows. Castration is associated with an increased level of fat gain and a reduced level of lean gain, this being more important in genotypes with high lean gain potential. Differences in daily gain of body components among genotypes and sex result in differences in growth rate.

Growth performance depends on the amount of energy available for growth (ME<sub>g</sub>), i.e. the remaining amount of energy when the maintenance requirements are met. This means that ME<sub>g</sub> is affected by factors that influence ME<sub>m</sub> value and (or) appetite (Quiniou *et al.*, 1999).

In a study by Bikker *et al.* (1994), they found that maximum protein and lysine deposition rates were higher at the high energy level than at the low energy level, which means that at adequate levels of protein intake, energy intake limited the protein and lysine deposition. The linear plateau relationship between protein intake and protein deposition respectively is in agreement with Cambell *et al.* (1985) for pigs 20 to 45 kg. In the linear phase, protein and lysine deposition were limited by protein intake and not by energy intake. These results support the concept of separate protein and energy dependent phases in protein and lysine deposition. The point of transition between the linear and the plateau phase increased with increasing energy intake, which implies that if energy intake is increased, protein intake must also be increased to reach maximum protein deposition. Feeding animals well below their protein requirements will not improve utilisation whereas carcass fatness will be increased. On the other hand, feeding the animals above their protein requirements will not result in any further increase in protein deposition and as a consequence nitrogen excretion will increase rapidly. Results of the experiment showed that weights of different visceral organs increased with increasing protein intake or energy intake or both. Those increased weights presumably

reflect an increased activity of these organs. Consequently an increased protein or energy intake can be expected to cause an increased maintenance heat loss.

De Greef *et al.* (1994) performed an experiment to check whether an affect of body weight and of amount of energy intake on the partitioning of energy is indeed absent when protein deposition is limited by energy intake. Two constant amounts of energy were given above maintenance requirement (L: 12.6 and H: 16.3 MJ DE per day) for production. The fatter bodies and higher lipid to protein deposition ratios in H pigs as compared with L pigs demonstrate the influence of energy intake on the partitioning of production energy into protein and lipid deposition. This is contrary to the assumption made in most growth models that, below maximal protein deposition rate and with adequate amounts of essential amino acids, there is no effect of degree of energy restriction on the ratio of lipid to protein deposition (Moughan *et al.*, 1987).

Present results indicate that the concept describing the response to energy intake in an energy-limited situation should be adjusted. The ratio between lipid deposition rate and protein deposition rate increases with an increase in energy intake. A possible approach to describe this phenomenon is to change the constant minimal ratio into a constant marginal ratio. This means that not the ratio between total lipid deposition and total protein deposition is constant, but the ratio between extra lipid deposition and extra protein deposition is constant. This modification only holds in an energy limiting situation, and is clearly supported by the data of Cambell and Traverner (1988). It can be stated that a change in partitioning between protein and lipid was a major factor causing the reduced live weight gain with increasing body weight. Other factors like water deposition and maintenance requirement can be expected to play a minor and no role, respectively. There was no significant interaction between weight range and amount of production energy on any of the parameters tested. This suggests that weight range and amount of production energy exert their effects independently and additionally. Although these results did not show a significant interaction between amount of production energy and weight range; results indicate that the difference in ratio between lipid and protein deposition rate between the two energy intake levels increases with body weight. The present work shows that, below maximal protein deposition, an increased intake of energy results in an increased ratio of lipid to protein. Furthermore, the ratio between lipid and protein deposition increases also with live weight at constant levels of energy available for production (De Greef *et al.*, 1994).

## 2.4 Energy partitioning

One of the most important factors determining body protein accretion rate is energy intake. It is generally accepted that there is a linear relationship between protein accretion and energy intake when energy intake is greater than the amount needed for maintenance and less than the amount required to maximise protein accretion (Cambell *et al.*, 1983, Cambell and Travermer, 1988). The change in protein accretion per unit increase in energy intake is called the slope. When energy is supplied in excess of what is required for the maximum rate of body protein accretion, this “excessive” energy intake will all be used to support lipid deposition. As energy intake increases above what is needed for maximum lean growth, increases occur in the ratio of fat:lean deposition, backfat thickness and lean feed conversion. As protein accretion potentials in grower-finisher pigs continue to increase, through genetic selection or better health management, energy intake is more likely to become the limiting factor for body protein deposition. Recent studies indicate that in pigs with high genetic capabilities for lean growth, the pig’s upper limit to protein retention, PD<sub>max</sub>, cannot be reached below about 80 to 90 kg live weight (Cambell and Travermer, 1988; Rao and MacCracken, 1991). The slope of protein deposition quantifies the additional amount of protein that is deposited from each additional unit of energy intake. A constant amount of additional energy is available for lipid deposition from each additional unit of energy intake. The changes in energy partitioning and rapid decline in lean growth after 90 kg live weight make it very difficult to produce uniform lean carcasses from early maturing low lean growth genotypes (Schinckel and De Lange, 1996). According to (Quiniou *et al.*, 1995) there is no clear relationship between the pig’s protein accretion potential and the slope of protein accretion on energy intake, or the protein to lipid deposition ratio, when energy intake limits protein accretion across various pig genotypes.

## 2.5 Residual feed intake

Residual feed intake (RFI) was defined by de Haer *et al.* (1993), as daily feed intake (FID) adjusted for predicted feed intake (pFID) based on metabolic body weight (MBW) and performance level (body weight gain and lean percentage). RFI is a measure of feed use per kg gain at a certain level of metabolic body weight and lean percentage. The difference between observed feed intake (FID) and this predicted feed intake (pFID) is defined as residual feed intake (RFI). The mean RFI was zero as expected by definition. In this

study there were no clear correlations between growth performance traits and eating frequency or duration. However, meal size and rate of feed intake were clearly associated with performance. Pigs with a low rate of feed intake were clearly associated with performance. Pigs with a low rate of feed intake and small meals had a high lean percentage, but a low daily weight gain and a low pFID. Meal size may also influence digestibility of nutrients and their utilisation after they have been absorbed. RFID was strongly related with frequency of eating and daily eating time.

Variation in feed intake among growing pigs may be explained by variations in feed intake for maintenance and feed intake for growth performance. Variation in feed intake for maintenance is usually predicted as a function of variation in metabolic weight and variation in feed intake for growth performance as a function of body weight gain and its components (NRC, 1988). These relationships indicate a higher RFID when feed intake activity increases. A low RFID is reflective of a more efficient pig that requires less feed than the average pig for a certain level of metabolic body weight and performance. Pigs with a low RFID had less visits and meals and spent less time eating per day than pigs with a high RFID. The variation in RFID that is was not explained by variation in feed intake activity is due to variation in basal metabolic rate, sustaining body temperature, protein turnover, health status, measurement errors etc. A high level of feed intake activity does not give a high performance but it does give a high RFID, which means a high overall FID and, therefore, high feed costs. De Haer *et al.* (1993) concluded from their study that pigs with a combination of a short daily eating time, a low eating frequency (i.e. a low RFID) and a large feed intake per visit to the feed hopper (i.e. a high growth performance and pFID) would be desired.

## 2.6 Feed intake

Genetic differences in feed intake, as well as other growth performance and carcass characteristics, can be found between breeds, between distinct genetic lines within a breed and between individuals within a line. The genetic relationship between appetite and lean growth can be positive as well as negative and leaner genotypes can grow faster than fatter genotypes. The relationship between intake and lean growth depends largely on the testing and selection program under which the genetic lines has been developed and particularly on the combination of selection criteria and feeding regime employed. The genetic relationship between growth rate, feed efficiency and carcass lean on the one hand and feed intake on the other are not fixed and vary with testing environment. The genetic correlation between

growth rate and carcass lean content is negative under *ad libitum* feeding but positive under restricted feeding regimes. In the vast majority of swine industries, feed efficiency is of greatest economic importance, followed generally by carcass content and then growth rate. Reduced feed intake is of much greater importance at lighter weights when intake may limit growth performance. Selection for reduced feed intakes as a mechanism for reducing carcass fat levels by improving feed efficiency resulted in the development of lines of pigs that can be fed to slaughter weight using *ad libitum* feeding without becoming excessively fat. Selection for lean growth rates achieved improvement in efficiency through increased growth rate with a correlated increase in feed intake and change in back fat thickness. It is obvious from this result that there is no fixed relationship between feed intake and lean growth and that these will be changing in time and in a way that is largely dependant on the selection program used (Ellis *et al.*, 1997).

Schinckel (1994) observed 30% differences in feed intake between genotypes of pigs that were fed similar diets and managed under similar conditions.

## 2.7 Growth

Growth has sometimes been defined as the rate of change in body weight, but the development of the animal, e.g., change in body composition, or in the relative importance of the different parts of the body, also needs to be included in the definition (Bastianelli and Sauvant, 1997). The actual growth rate of an animal in a given situation should be distinguished from the potential growth rate, which is an intrinsic characteristic of a given animal and represents the achievement without limitation of its genetic project through homeorhetic regulation. Actual growth is the result of factors related to the animal (genetics, sex, health, etc.), its diet (quantity, quality, and feeding plan) and its environment (climate, housing, etc.). It is assumed that there is a maximum value for daily protein deposition ( $P_{max}$ ), and therefore for growth rate. It is generally considered as a function of age or live weight. This function is assumed to be completely determined for a given animal (sex and strain). Whittemore *et al.* (1988) proposed the use of a Gompertz function. Actual protein growth is generally determined as the minimum value of (a) the accretion allowed by available dietary protein (total amount and amino acid profile); (b) the accretion allowed by available dietary energy; (c) the potential protein accretion ( $P_{dmax}$ ). Kyriazakis (1994) suggest that animals attempt to grow at a genetically determined rate, and that the level of feed intake is the quantity necessary to satisfy the needs for the first limiting factor in the

feed, except if the intake capacity is reached. If energy is not limiting then the gain is greater than the genetic potential.

## 2.8 Protein and energy

Whittemore (1983), taking into account the close relationship between protein deposition and lean gain, proposed that lean gain also vary with energy intake according a linear- plateau model. Schinkel (1994) and Quiniou *et al.* (1996b) validated this model. The fact that protein deposition and lean gain vary similarly with energy intake is to be related to the high proportion of marginal protein deposition in lean gain (Quiniou and Noblet, 1997). It resulted from the fact that the effect of energy intake on partition of protein deposition among different body tissues is limited. Taking into account the low body fat content in restrictively fed pigs, the fat gain change with energy intake is proportionally more important than lean gain change, which results in an increased body fatness. According to Quiniou *et al.* (1996b), body fatness increases with energy intake. Indeed, lean gain increases with energy intake according to a linear relationship, whose intercept is not different from zero, whereas fat gain increases also linearly with energy intake but with a negative intercept. Quiniou *et al.* (1999) found that in agreement with the observed effect on protein gain, the increase in lean gain was lower over the finishing period than over the growing period (9.6 and 12.5 g/MJ DE, respectively). This effect of stage of growth on body weight composition leads to the rejection of the hypothesis proposed by Whittemore (1993). According to which body weight composition would not be dependent on the stage of growth when pigs are restrictively fed. Then, when energy intake increases, it results in a higher increase of body fatness in heavier pigs.

## 2.9 Lipid

The effects of varying the levels of protein and energy intake on the rates of lipid deposition of the growing pig can be seen as the indirect result of the concomitant effect of protein and energy intake on the rates of protein deposition. And, thus, the amount of energy available for lipid synthesis.

The interaction between allowance of feed (protein level) and the feeding level (amount of energy) suggests that on treatment (P3H) female pigs had reached their potential for protein

deposition. Therefore did the females have more energy available for lipid synthesis than male pigs (Kyriazakis and Emmans, 1992b).

## 2.10 Types

Quiniou *et al.* (1996a) reported a higher slope in the relationship between protein deposition and MEg in boars than in barrows. The results of Quiniou *et al.* (1999) indicate, in agreement with Cambell and Taverner (1988), that the slope is higher in lean types of pigs than in fatter types of pigs. Such a result would correspond to a better efficiency of energy utilisation for protein deposition in lean types of pigs. But, according to Cambell and Taverner (1988), protein deposition increased linearly up to PDmax in conventional types of pigs, whereas PDmax seemed to remain unreached in lean types of pigs. On the other hand, the results of Quiniou *et al.* (1996a) suggest that PDmax can be reached at a lower level of energy intake in lean type of pigs than in fatter type of pigs. This suggest that a high value of the slope can be associated with a high or low PDmax or, on the contrary, a high PDmax can be associated with a high or low slope.

Genotype or sex does generally not influence the increase of lipid deposition rate with energy intake. In fact, from published results, the increase of lipid deposition averages 14.5 (-/+ 0.5) g/MJ DE over the 45 to 100 kg body weight range (Quiniou *et al.*, 1999). A similar value was calculated over the 2 to 55 kg body weight range. The increased body fatness in ad libitum fed pigs when body weight increased was not due to the decrease of PDmax but resulted from the increased lipid deposition. Although the slope between protein deposition and MEg can be the same, the intercept changes with body weight and it was found to decrease linearly with increased body weight. Concomitantly, the amount of MEg required to express PDmax increased with body weight.

It is now generally accepted that the genetic and phenotypic diversity in the performance of pigs is such that the nutrient requirements of 'the pig' cannot be specified satisfactorily without some further characterisation of the particular population of animals of interest. This characterisation should be in terms of their nutritional responses, which are determined by interactions between genetic and environmental factors (Fuller *et al.*, 1995). Work done with different genotypes and with porcine somatotropin-treated pigs suggests that animals with a greater maximum rate of protein accretion are also more efficient at sub optimal intakes. In the experiments reported by Cambell and Taverner (1987, 1988), for example, three populations of pigs, of one strain and boars and castrated males of another, were given the same 'protein -adequate' diet at daily rates from 22 MJ/day to *ad libitum*. Their responses

can be characterised by two parameters: (1) the maximum daily rates of protein accretion ( $P_{max}$ ). These rates were (approximately) > 190, 130, 80g, respectively. (2) The marginal efficiency of body protein accretion, expressed as the change in daily protein accretion (g) per MJ change, in daily metabolisable energy (MJ) at restricted intakes. These efficiencies were 5.3, 4.4 and 2.9 respectively. Thus, pigs with a high value of  $P_{max}$  also had a high efficiency of body protein accretion at sub optimal intakes. However, with only three populations in the experiment it is not clear if these two parameters are simply related or if nutritional responses can be described by just two parameters.

The intact male pigs responded to increases in dietary protein with increased rate of gain. The female and castrated pigs showed a very small response or none. For these, the low protein diet evidently sufficed to satisfy their needs for maximum growth. The changes in body composition were also as expected. Increased dietary protein resulted in greater carcass leanness and increased intake resulted in less protein and more lipids. The males had least lipid and the castrates the most, but amongst the three groups of males, differences in body composition were small and not significant.

According to results of Quiniou *et al.* (1996a), the major effect of type of pig on the voluntary feed intake was due to castration. Fuller *et al.* (1995) has also reported such conclusions. In *ad libitum* feeding conditions, their results indicate that the rate of deposition of chemical components was strongly affected by the type of pig. No significant effect of stage of growth on  $P_{Dmax}$  was found within each type of pig between 45 and 100 kg. This result is in agreement with those of Whittemore *et al.* (1988), who found that variation of protein deposition over 45 to 100 kg body weight range was significant and varied between 0 and 20 g/day. As a consequence, the increased fatness of body weight gain when body weight increased is not due to the decrease of daily protein deposition but to the increase of daily lipid deposition.

## 2.11 Genotypes

Genotypes may differ in a number of respects that affect their potential growth curves. Among these are: mature size, mature composition, including fat content, and the rates of maturing of the body chemical components. These variables all influence the daily feed intake and amounts of energy and amino acids needed for the potential to be attained. The chemical and physical composition of the body changes systematically during growth and a sufficient description of potential must deal with such changes (Gous *et al.*, 1999).



In a study conducted by Cambell and Travermer (1988), the results showed that both genotype and castration affected, although somewhat differently, the relationship between energy intake and protein deposition and, as a consequence, growth performance and body composition. The difference in maximal protein deposition and in the relationship between energy intake and protein deposition was directly responsible for the concomitant differences between strains in growth performance and body fat content at higher levels of feeding. The results of the study suggest, however, that the between strain differences in body fat content were an indirect effect of the concomitant differences in protein deposition and thus in the amount of energy available for lipogenesis. The differences in maintenance energy costs would have accentuated, particularly at the lower levels of feeding, the differences between strains in body composition, but it should reduce those differences associated with the differences in protein deposition between the two strains.

## 2.12 Maintenance

At maintenance, the animal would use body lipids to achieve protein deposition. From a biological point of view, protein deposition would then be a process having priority in growing pigs. But when body weight increases, with regard to the concomitant decrease of the intercept, the estimated protein deposition in animals fed at maintenance would decrease which could be considered as an indicator of maturity. In other words, such a variation would explain the reduced protein deposition observed at a given level of energy supplied above maintenance. However, those results need to be validated because values of the intercept were estimated from measurements performed at levels of energy intake far above maintenance (Quiniou *et al.*, 1999).

The results of Campbell and Taverner (1988), also indicate an effect of genotype or of genetic improvement in protein deposition on the growing pig's energy requirement for maintenance. This difference in maintenance energy costs would have accentuated, particularly at the lower levels of feeding; the difference between strains in body composition. It should reduce that differences reduce that differences associated with live weight gain that otherwise would be associated with the concomitant differences in protein deposition between the two strains. The higher maintenance energy requirement indicated by the present results for strain A boars, probably was associated with a considerably higher lean body mass and a higher

protein turnover rate than either strain B boars or the castrates (Campbell and Traverner, 1988).

Genetic improvement in the rate and efficiency of live weight gain appears to be associated with increase in both the ceiling for protein deposition and, to a lesser extent, the slope of the ascending linear component of the relationship between energy intake and protein deposition (Campbell and Traverner, 1988).

Maintenance energy requirements are better expressed in relation to the distribution of the major tissue groups (viscera, muscle, and fat) in the pig's body and considering the contribution of these tissue groups of the various processes that determine maintenance energy requirements. Other factors to consider in interpreting maintenance energy requirements include animal activity, maintenance of a constant body temperature and the exposure to disease causing organisms (Schinckel and De Lange, 1996).

### 2.13 Chemical composition

The rate of food intake and the genetic potential for lean gain are the two major factors influencing growth rate, food efficiency and body composition of growing animals. Whittemore (1983) suggested that the maximum daily protein deposition was 120 to 175 g for boars, 105 to 155 g for gilts and 90 to 140 g for castrated males. In work done by Campbell and Traverner (1988), it is clear that the upper end of the range is close to 200 g/day. Rao and McCracken (1990) used different protein levels to determine what the effect on energy and nitrogen balance and chemical composition of gain in growing boars would be. The daily accretion rate of ash was similar for all the dietary treatments, but the proportion of ash content in EBW was low in the boars given the high protein diets presumably because of the lower number of days to reach slaughter weight. This may be relevant to the practical problems of leg weakness in fast growing pigs, due to less calcification of bones and reduced bone strength. The daily retention of protein continued to increase and the fat retention to decrease up to the highest level of protein used (282 g CP/kg DM), indicating that the dietary levels of protein and lysine to optimise carcass quality are higher than for optimum growth. The protein concentration required for maximum protein deposition was not identified in their experiment.

## 2.14 The Nil Hypothesis ( $H_0$ )

The  $H_0$  to be tested is that there are no differences between the two genotypes in protein deposition, feed intake, feed efficiency, carcass analysis and protein:fat ratio. The following were done to accomplish this:

- a) Four diets with different protein levels were given to the two genotypes.
- b) The serial slaughter technique was used for carcass analysis.

## 2.15 Genotypes used

The MPD barrows are a cross between MPD Landrace boars and MPD Great White sows. The PIC barrows are a cross between PIC Landrace line 2 boars and PIC Great White line 3 sows (Gee, 2003).

## Chapter 3

### Materials and methods

#### 3.1 Trial Design

The experimental design was a 2 x 4 factorial design. The two factors are the two pig genotypes, PIC and MPD breeding lines of Kanhym Estates. Four treatments of different protein levels were used. Each treatment was replicated 7 times. The experimental house was divided into 7 blocks. Each block had 8 pens. The blocks were randomly allocated in the house. Each block represented a replicate. Replicate 1 consisted of the 12 pigs of each genotype, 3 per pen; thus 4 pens per genotype, which were the different treatments. The 8 pens per block were randomly allocated. At the beginning of the trial 56 pens were used with 3 pigs per pen. Block 1 housed the 12 heaviest pigs of each genotype and block 7 the lightest. The average weight of the pigs in each pen within a block was equal, within a genotype.

#### 3.2 Animals

One hundred and ninety two 21-day old barrows were received from Kanhym estates of which 96 were of the MPD and 96 of the PIC breeding lines. All pigs were weighed upon arrival. 12 pigs were slaughtered on the day of arrival (see paragraph 3.7: Slaughter procedure) and the remaining 180 were tagged and randomly allocated to 18 pens for the weaner period. The animals were grouped by genotype into 9 pens per genotype, 10 animals per pen. The pens were 1.2mx3.05m, thus 0.4575m<sup>2</sup> per pig.

The average live weight of the pigs upon arrival was 7 kg for both genotypes. One piglet died within a week of arrival, probably stress related. Another three died without signs of illness before the trial started. One pig died during the trial and was removed from the experiment.

The dietary treatments of the pre trial period are given in Tables 3.1 and 3.2. The pigs had *ad libitum* access to the diets. The ASA Premier Weaner pellets and ASA Creep pellets plus blood plasma were given at 3kg per pig and Kanhym Creep was given at 6 kg per pig, in order to limit wastage, until the feed was finished. The Kanhym Weaner diet was given to each pen after the initial diet was finished.

#### 3.3 Health management

The pigs did not receive prophylactic treatment against sickness. Ten pigs developed scours before the trial started and another 20 during the trial. The pigs were treated with an antibiotic, Advocin® (Pfizer, Danofloxacin 25 mg/ml), and Norotrim® (Pharmacia and

Upjohn, Sulphadiazine) as prescribed by the consulting veterinarian. All of the pigs responded quickly to the treatment and the cases were randomly spread through out the experimental house. The 4 pigs with joint infections were treated with Fenylbutazone® 20% (Phenix). Three pigs were treated for abscesses before the trial started with Advocin®.

### 3.4 Housing

The pigs were initially kept in a broiler house that was adapted to accommodate them. Infra red lamps were used for extra heat. The pigs were moved to the experimental house when they were 20kg live weight. The experimental house had been empty for 30 days before the pigs were moved there. The experimental house was thoroughly cleaned and disinfected. The house contained 58 pens of which 56 were used. The pens have two-thirds concrete floors and one-third slats. The pens were 1.2mx2.9m, thus 1.16m<sup>2</sup> per pig at the beginning of the trial. The house was warmed with space heaters.

### 3.5 Treatments

The diets were formulated using a matrix-type programme from the company Spesfeed to provide protein levels according to the NRC recommended inclusion levels of lysine. The diets were formulated on an “as is” basis to simulate how it is done in practise.

The dietary treatments differed in protein content. The protein levels of the treatments were Treatment 1: NRC default plus 20%; Treatment 2: NRC default plus 10%; Treatment 3: NRC default and Treatment 4: NRC default minus 10%. Dietary treatments were over 4 phases which corresponded to the following weight ranges: Phase 1: 30 kg to 50 kg; Phase 2: 50 kg to 70 kg; Phase 3: 70 kg to 90 kg; Phase 4: 90 kg to 110 kg.

The diets were a mixture of maize and soya bean oil cake. A mineral and vitamin premix was added to each mixture. (Table 3.3 to 3.6). The feed was mixed on the Hatfield experimental farm, except in the last phase, when Dalein Estates mixed one ton of each treatment.

### 3.6 Parameters

The feed intake, body weight gains and feed efficiency were measured on a weekly basis. The feed was given *ad libitum* in self-feeders and was replenished regularly. The feed intake was determined by weighing the feed remaining in the feeder and subtracting it from the

amount that was weighed into the feeder. The Feed Intake (FI) was measured for each pen. Phase 1 and 2 had three pigs per pen, Phase 3 had two pigs per pen and Phase 4 had one pig per pen. The Feed Conversion Ratio (FCR) was calculated by adding the ADG of the pigs per pen and using the FI per pen. The pigs were weighed on the same day, without prior removing feed and water. From these data the daily feed intake, average daily gain and feed conversion ratios were calculated.

### 3.7 Slaughter procedure

The serial slaughter technique was used to determine body composition. Only pigs of Treatment 1 were used for the serial slaughter technique to minimise the effect of excess energy. Three pigs per genotype were slaughtered for carcass analyses at the following stages: 5 days of age, 14 days of age, 30 kg live weight, 50 kg, 70 kg, 90 kg and 110 kg.

Of the 12 pigs slaughtered on the day of arrival, six were five days old, three of each genotype, and six were 14 days old, three of each genotype. These pigs were randomly selected. The animals that were slaughtered for carcass analysis were weighed before slaughter. The remaining pigs were slaughtered at a Commercial abattoir. One pig per pen was slaughtered at 70kg, 90kg and 110kg live weight. Commercial slaughtering was done at Enterprise Pork Packers.

The animals were stunned across the temples with an electric stunner (Jarvis Sheep Pig stunner, 170-180V, 1.3A) and the carotid artery severed. Most of the blood was collected, but there was some blood loss. The carcasses were dressed down and the intestines were cleaned. The whole dressed down carcass, blood and intestines were weighed again to obtain the empty body weight. The carcasses of the pigs from 50 kg live weight and heavier were split along the spinal cord. Only the right half was used for further processing. The other half was sold. The blood, carcass and intestines were frozen (-20°C) for preserving and further processing. The small carcasses were stored in sealed plastic bags and the bigger carcasses (from 50 kg) were wrapped in plastic, to prevent loss of moisture during frozen storage for at least a week before further processing. The frozen carcass, blood and intestines were cut into pieces with a band saw to feed into the mincer. The whole carcass was then minced and mixed thoroughly to get a representative sample of 3kg to analyse for protein, fat, moisture and ash content.

### 3.8 Chemical analysis

The samples of the minced-up carcasses were freeze dried and then ground up further in a blender, bottled and stored. These samples were then analysed for moisture, protein, fat and ash content by using the AOAC (1995) procedures.

The feed samples were grounded and bottled. The AOAC (1995) procedures were used to analyse the feed samples for moisture, protein fat, ash, phosphorus, calcium and sodium content.

A soxhlet fat extraction apparatus was used to determine the fat content of the feed and empty body samples.

The moisture content of the feed and empty body samples was determined by drying the samples for 24 hours in a 100°C oven. The loss in weight is recorded as moisture loss.

The ash content of the samples was determined by putting the samples in a 600°C oven for 4 hours.

The Makro Kjeldahl method was used to determine the N content of the samples. A Kjeltec System 1026 Distilling unit was used. The protein percentage was determined by assuming that protein has a N content of 16%.

The feed samples were prepared for the calcium, phosphorus and sodium analysis by a wet digestion process (AOAC, 1995). The phosphorus analysis was done with an Auto Analyzer™ II Technicon™. The sodium analysis was done with a Varian – Sperctr AA 50 (Atomic absorption spectrometer). The calcium analysis was done by using a Perkin Elmer 2380 (Atomic absorption spectrophotometer).

### 3.10 Statistical analysis

Analysis of variance with the GLM model (Statistical Analysis System, 1994) was used to determine the significance between different genotypes, treatments, slaughter stage and genotype x treatment x slaughter stage interactions for the slaughter data.

Least square means and standard deviations (SD) were calculated.

Significance of difference (5%) between least square means was determined by using the Bonferroni test (Samuels, 1989).

Analysis of covariance with the GLM model (Statistical Analysis System, 1994) was used for Average Daily Gain to determine the significance between different genotypes, treatments and the interactions between genotypes and treatments, with starting mass as covariant.

Analysis of variance with the GLM model (Statistical Analysis System, 1994) were used for Feed Intake, Feed Conversion Ratios and the chemical analysis of the empty body samples to determine the significance between the genotype, phases, treatments and interactions.

**Table 3.1** Composition of ASA diets given to pigs after arrival. Weaner: ASA Premier Weaner Pellets, Creep: ASA Creep Pellets Plus Blood Plasma. Analyses of diets received from ASA.

		Weaner	Creep
Crude Protein	g/Kg	197.24	186.6 6
Fat	g/Kg	83.19	70.7
Lysine	g/Kg	13.61	12.56
Vit A	MIU	15000	
Vit D3	MIU	3000	
Vit E	g/Kg	80	
Biotin	mg	150	
T.S.A.A.	g/Kg	8.39	7.57
Tryptophan	g/Kg	2.32	2.12
Threonine	g/Kg	8.49	8.18
Fibre	g/Kg	20.98	35.21
Calcium	g/Kg	7.51	8.2
Total Phosphorus	g/Kg	6.54	6.76
Avl. Phosphorus	g/Kg	5.53	5.22
Sodium	g/Kg	2.26	2.32
Potassium	g/Kg	8.78	
Chloride	g/Kg	4.96	
NE Swine	MJ/K g	11.52	9.51
DE Swine	MJ/K g	16.87	14.56
AS Lysine	g/Kg		11.26
AS Methionine	g/Kg		4.46
AS TSAA	g/Kg		6.73
AS Isoleucine	g/Kg		6.97
AS Tryptophan	g/Kg		1.75
AS Threonine	g/Kg		6.91
AS Valine	g/Kg		7.68
AS Histidine	g/Kg		4.06

Analyses of diets received from ASA.





**Table 3.2** Composition of Kanhym diets given to pigs after arrival.

		Creep	Weaner
Crude Protein	g/Kg	195.68	196.53
Crude Fat	g/Kg	52.82	52.98
Crude Fibre	g/Kg	31.65	27.4
Dry Matter	g/Kg	878.3	870.92
DE Swine	MJ/Kg	14.9	14.5
Calcium	g/Kg	7	8
Total Phosphorus	g/Kg	6.7	7.1
Arginine	g/Kg	12.08	13.68
Lysine	g/Kg	13.5	12.2
Methionine	g/Kg	4.53	4.2
Methionine,Cystine	g/Kg	8	7.69
Tryptophane	g/Kg	2.41	2.49
Glycine	g/Kg	6.77	7.87
Histidine	g/Kg	4.29	4.9
Leucine	g/Kg	15.14	17.28
Isoleucine	g/Kg	8.08	9.18
Phenylalanine	g/Kg	9.3	10.65
Phen + Tryp	g/Kg	16.34	18.87
Threonine	g/Kg	8	7.8
Valine	g/Kg	9.12	10.08
Salt	g/Kg	4	5
Avl.Phosphorus	g/Kg	5.15	6
Sodium	g/Kg	2.74	3.06

Analyses of diets received from Kanhym.



**Table 3.3** Dietary treatments for Phase 1: 30 to 50 kg live weight. Protein levels of Treatments: Treatment 1: NRC default +20%, Treatment 2: NRC default +10%, Treatment 3: NRC default and Treatment 4: NRC default -10%.

Dietary components		Treatments			
		1	2	3	4
Maize 8.0%	Kg	629	662.0	695.0	728
Soya O/C 47%	Kg	330	297.3	264.7	232
Monocalcium Phos	Kg	14.5	14.8	15.2	15.5
Salt	Kg	5.5	5.5	5.5	5.5
Lysine HCL	Kg	3.5	3.2	2.8	2.5
DL Methionine	Kg	1.4	1.1	0.8	0.5
Limestone 36	Kg	12	12.0	12.0	12
L Threonine	Kg	0.8	0.6	0.5	0.3
Tryptosine 15/70	Kg				
Pig Weaner Px	Kg	3.0	3.0	3.0	3.0
Pig Grower Px	Kg				
Total mix	kg/batch	1000.7	1000.6	1000.4	1000.3
Treatment		1	2	3	4
NE Swine	MJ/kg	9.5	9.6	9.6	9.7
DE Swine	MJ/kg	14.0	14.0	14.0	14.0
Crude Protein	g/kg	210	196.7	183.4	170
Lysine	g/kg	13.9	12.7	11.6	10.5
Methionine	g/kg	4.7	4.2	3.7	3.3
T.S.A.A.	g/kg	8.0	7.4	6.7	6.1
Isoleucine	g/kg	9.2	8.5	7.9	7.3
Tryptophan	g/kg	2.5	2.3	2.1	1.9
Threonine	g/kg	8.8	8.1	7.5	6.8
DLys (True Ileal)	g/kg	12.7	11.7	10.6	9.5
Dlys (App Ileal)	g/kg	12.1	11.1	10.1	9.0
AS Methionine	g/kg	4.2	3.8	3.4	2.9
AS TSAA	g/kg	6.8	6.3	5.7	5.1
AS Isoluecine	g/kg	7.4	6.9	6.4	5.9
AS Tryptophan	g/kg	1.9	1.8	1.6	1.5
AS Threonine	g/kg	6.9	6.3	5.8	5.2
AS Valine	g/kg	8.1	7.6	7.1	6.6
Fat	g/kg	32	32.4	33.1	34
NDF	g/kg	94	94.1	94.3	95
Fibre	g/kg	30	29.1	28.6	28
Calcium	g/kg	8.5	8.5	8.5	8.4
Avl Phosphorus	g/kg	4.5	4.5	4.5	4.5
Sodium	g/kg	2.3	2.3	2.3	2.3

**Table 3.4** Dietary treatments for Phase 2: 50 to 70kg live weight. Protein levels of Treatments: Treatment 1: NRC default +20%, Treatment 2: NRC default +10%, Treatment 3: NRC default and Treatment 4: NRC default -10%.

Dietary components		Treatments			
		1	2	3	4
Maize 8.0%	Kg	702	728.0	754.0	780
Soya O/C 47%	Kg	259	233.7	208.3	183
Monocalcium Phos	Kg	14	14.2	14.3	14.5
Salt	Kg	5.5	5.5	5.5	5.5
Lysine HCL	Kg	2.8	2.5	2.2	1.9
DL Methionine	Kg	1.1	0.8	0.4	0.1
Limestone 36	Kg	11	11.0	11.0	11
L Threonine	Kg	0.7	0.5	0.2	
Tryptosine 15/70	Kg	0.6	0.4	0.2	
Pig Weaner Px	Kg	3.0	3.0	3.0	3.0
Pig Grower Px	Kg				
Total mix	kg/batch	1000.7	1000.5	1000.2	1000
Treatment		1	2	3	4
NE Swine	MJ/kg	9.7	9.7	9.8	9.8
DE Swine	MJ/kg	14.1	14.0	14.0	14.0
Crude Protein	g/kg	182	171.6	160.9	150
Lysine	g/kg	11.8	10.7	9.7	8.7
Methionine	g/kg	4.0	3.6	3.1	2.7
T.S.A.A.	g/kg	7.0	6.4	5.8	5.2
Isoleucine	g/kg	7.8	7.3	6.8	6.4
Tryptophan	g/kg	2.2	2.0	1.8	1.7
Threonine	g/kg	7.6	7.0	6.4	5.7
DLys (True Ileal)	g/kg	10.8	9.8	8.8	7.8
Dlys (App Ileal)	g/kg	10.2	9.3	8.4	7.4
AS Methionine	g/kg	3.6	3.2	2.8	2.3
AS TSAA	g/kg	6.0	5.4	4.9	4.3
AS Isoleucine	g/kg	6.3	5.9	5.5	5.1
AS Tryptophan	g/kg	1.7	1.5	1.4	1.3
AS Threonine	g/kg	5.9	5.4	4.8	4.3
AS Valine	g/kg	7.0	6.6	6.2	5.8
Fat	g/kg	33	33.8	34.3	35
NDF	g/kg	94	94.7	95.0	95
Fibre	g/kg	29	28.3	27.9	28
Calcium	g/kg	7.8	7.8	7.8	7.7
Avl Phosphorus	g/kg	4.2	4.2	4.2	4.2
Sodium	g/kg	2.3	2.3	2.3	2.3

**Table 3.5** Dietary treatments for Phase 3: 70 to 90 kg live weight. Protein levels of Treatments: Treatment 1: NRC default +20%, Treatment 2: NRC default +10%, Treatment 3: NRC default and Treatment 4: NRC default -10%.

Dietary components		Treatments			
		1	2	3	4
Maize 8.0%	Kg	748	773.7	799.3	825
Soya O/C 47%	Kg	217	191.7	166.3	141
Monocalcium Phos	Kg	13	13.3	13.7	14
Salt	Kg	5.5	5.5	5.5	5.5
Lysine HCL	Kg	2.1	1.9	1.7	1.5
DL Methionine	Kg	0.7	0.5	0.2	
Limestone 36	Kg	10	10.2	10.3	10.5
L Threonine	Kg	0.6	0.4	0.2	
Tryptosine 15/70	Kg	0.7	0.5	0.2	
Pig Weaner Px	Kg				
Pig Grower Px	Kg	3.0	3.0	3.0	3.0
Total mix	kg/batch	1000.6	1000.6	1000.5	1000.5
Treatment		1	2	3	4
NE Swine	MJ/kg	9.8	9.8	9.9	10.0
DE Swine	MJ/kg	14.1	14.1	14.1	14.0
Crude Protein	g/kg	165	154.7	144.2	134
Lysine	g/kg	10.1	9.2	8.2	7.2
Methionine	g/kg	3.4	3.1	2.7	2.3
T.S.A.A.	g/kg	6.2	5.7	5.2	4.7
Isoleucine	g/kg	7.0	6.5	6.0	5.6
Tryptophan	g/kg	2.0	1.8	1.6	1.4
Threonine	g/kg	6.9	6.3	5.7	5.1
DLys (True Ileal)	g/kg	9.2	8.3	7.4	6.4
Dlys (App Ileal)	g/kg	8.8	7.9	7.0	6.1
AS Methionine	g/kg	3.1	2.7	2.4	2.0
AS TSAA	g/kg	5.2	4.8	4.3	3.9
AS Isoleucine	g/kg	5.7	5.3	4.9	4.5
AS Tryptophan	g/kg	1.5	1.4	1.2	1.1
AS Threonine	g/kg	5.3	4.8	4.3	3.8
AS Valine	g/kg	6.4	6.0	5.6	5.2
Fat	g/kg	34	34.8	35.3	36
NDF	g/kg	95	95.3	95.5	96
Fibre	g/kg	28	27.8	27.4	27
Calcium	g/kg	7.1	7.2	7.3	7.3
Avl Phosphorus	g/kg	4.0	4.0	4.0	4.0
Sodium	g/kg	2.3	2.3	2.3	2.3

**Table 3.6** Dietary treatments for Phase 4: 90 to 110 kg live weight. Protein levels of Treatments: Treatment 1: NRC default +20%, Treatment 2: NRC default +10%, Treatment 3: NRC default and Treatment 4: NRC default -10%.

Dietary components		Treatments			
		1	2	3	4
Maize 8.0%	Kg	772.0	800.3	828.7	857.0
Soya O/C 47%	Kg	195.0	167.0	139.0	111.0
Monocalcium Phos	Kg	12.0	12.2	12.3	12.5
Salt	Kg	5.5	5.5	5.5	5.5
Lysine HCL	Kg	1.8	1.7	1.5	1.4
DL Methionine	Kg	0.3	0.2	0.1	
Limestone 36	Kg	10.5	10.5	10.5	10.5
L Threonine	Kg	0.2	0.1	0.1	
Tryptosine 15/70	Kg	0.2	0.1	0.1	
Pig Weaner Px	Kg				
Pig Grower Px	Kg	3.0	3.0	3.0	3.0
Total mix	kg/batch	1000.5	1000.6	1000.8	1000.9
Treatment		1	2	3	4
NE Swine	MJ/kg	9.9	9.9	10.0	10.0
DE Swine	MJ/kg	14.1	14.1	14.1	14.1
Crude Protein	g/kg	155.6	144.4	133.2	122.0
Lysine	g/kg	9.0	8.1	7.2	6.3
Methionine	g/kg	2.9	2.7	2.4	2.2
T.S.A.A.	g/kg	5.6	5.2	4.8	4.4
Isoleucine	g/kg	6.6	6.1	5.5	5.0
Tryptophan	g/kg	1.8	1.6	1.4	1.2
Threonine	g/kg	6.1	5.6	5.1	4.6
DLys (True Ileal)	g/kg	8.1	7.3	6.5	5.6
Dlys (App Ileal)	g/kg	7.7	6.9	6.1	5.3
AS Methionine	g/kg	2.6	2.4	2.1	1.9
AS TSAA	g/kg	4.7	4.3	4.0	3.6
AS Isoleucine	g/kg	5.3	4.9	4.4	4.0
AS Tryptophan	g/kg	1.3	1.2	1.1	0.9
AS Threonine	g/kg	4.7	4.2	3.8	3.4
AS Valine	g/kg	6.0	5.6	5.1	4.7
Fat	g/kg	34.8	35.3	35.9	36.5
NDF	g/kg	95.5	95.7	95.9	96.0
Fibre	g/kg	27.9	27.5	27.1	26.7
Calcium	g/kg	7.1	7.0	7.0	6.9
Avl Phosphorus	g/kg	3.7	3.7	3.7	3.7
Sodium	g/kg	2.3	2.3	2.3	2.3

**Table 3.7** Chemical analyses of dietary treatments used during the trial. Phase 1: 30 to 50kg, Phase 2: 50 to 70kg, Phase 3: 70 to 90kg and Phase 4: 90 to 110 kg live weight. Protein levels of Treatments: Treatment 1: NRC default +20%, Treatment 2: NRC default +10%, Treatment 3: NRC default and Treatment 4: NRC default –10%.

	DM%	Crude Prot%	Fat%	Ash%	Na%	Ca%	P%
Phase 1							
Treatment 1	89.65	22.48	1.92	4.95	0.15	0.76	0.64
Treatment 2	89.57	22.42	1.95	4.96	0.12	0.61	0.67
Treatment 3	89.02	19.09	2.02	4.78	0.12	0.56	0.62
Treatment 4	89.27	18.56	1.92	4.60	0.10	0.61	0.65
Phase 2							
Treatment 1	89.55	18.99	2.07	4.92	0.14	0.59	0.61
Treatment 2	89.23	18.31	2.27	3.67	0.09	0.49	0.56
Treatment 3	88.61	17.09	2.13	3.50	0.07	0.51	0.57
Treatment 4	89.22	14.93	2.21	3.58	0.11	0.45	0.49
Phase 3							
Treatment 1	89.27	16.94	2.63	4.32	0.14	0.72	0.57
Treatment 2	89.50	15.41	2.65	4.42	0.15	0.67	0.59
Treatment 3	89.02	14.65	2.40	3.64	0.09	0.52	0.52
Treatment 4	89.28	13.85	3.12	3.69	0.15	0.44	0.47
Phase 4							
Treatment 1	89.15	15.16	2.70	4.10	0.13	0.60	0.52
Treatment 2	89.14	14.78	2.51	3.79	0.11	0.49	0.49
Treatment 3	89.69	12.77	2.23	4.25	0.14	0.67	0.52
Treatment 4	88.94	12.14	2.65	4.18	0.19	0.56	0.57
Phase 4 (Feed from Dalein Estates)							
Treatment 1	88.99	16.27	2.41	3.80	0.11	0.49	0.55
Treatment 2	89.02	14.77	2.60	4.46	0.13	0.62	0.53
Treatment 3	89.06	13.74	2.75	4.06	0.11	0.58	0.45
Treatment 4	89.28	13.36	2.73	4.66	0.17	0.79	0.52

## Chapter 4

### Results and Discussion:

#### 4.1 Average daily gain (ADG):

The ADGs of the two genotypes for each Phase are presented separately. The ADG of the different Phases was not compared, only the ADG for the different genotype x treatment interactions in a Phase was compared. The results shown in Table 4.1.1 are the pooled results of all treatments of Phase 1: 30 kg to 50 kg live weight, Phase 2: 50 kg to 70 kg, Phase 3: 70 kg to 90 kg and Phase 4: 90 kg to 110 kg. The results in Table 4.1.2 are pooled results of the MPD and PIC genotypes of all the Phases for the Treatments. Treatment 1 was the high protein and treatment 4 was the low protein. The results in Table 4.1.3 are the results for each Phase for genotype treatment interaction. (Tables 4.1.1 – 4.1.3).

**Table 4.1.1** Pooled results of the ADG of all treatments for the MPD and the PIC genotypes for each growth phase (Phase 1: 30 kg to 50 kg, Phase 2: 50 kg to 70 kg, Phase 3: 70 kg to 90 kg and Phase 4: 90 kg to 110 kg).

Phase	MPD	PIC
	LSMean $\pm$ SD	LSMean $\pm$ SD
1	0.88 <sup>a</sup> $\pm$ 0.1538	0.87 <sup>a</sup> $\pm$ 0.2411
2	1.05 <sup>a</sup> $\pm$ 0.1384	1.10 <sup>a</sup> $\pm$ 0.2348
3	1.06 <sup>a</sup> $\pm$ 0.1427	1.13 <sup>a</sup> $\pm$ 0.2057
4	1.00 <sup>a</sup> $\pm$ 0.1564	1.07 <sup>a</sup> $\pm$ 0.2425

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant ( $P < 0.05$ ) differences between the genotypes in anyone Phase.

**Table 4.1.2** Pooled results of the ADG of the MPD and PIC genotypes for all the treatments for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

Phase	Treatments			
	1	2	3	4
	LSMean $\pm$ SD	LSMean $\pm$ SD	LSMean $\pm$ SD	LSMean $\pm$ SD
1	0.86 <sup>a</sup> $\pm$ 0.1556	0.92 <sup>a</sup> $\pm$ 0.1708	0.88 <sup>a</sup> $\pm$ 0.2136	0.84 <sup>a</sup> $\pm$ 0.2540
2	1.10 <sup>b</sup> $\pm$ 0.1659	1.10 <sup>b</sup> $\pm$ 0.1397	1.10 <sup>b</sup> $\pm$ 0.1760	1.00 <sup>a</sup> $\pm$ 0.2587
3	1.07 <sup>a</sup> $\pm$ 0.2363	1.12 <sup>a</sup> $\pm$ 0.1830	1.07 <sup>a</sup> $\pm$ 0.1540	1.12 <sup>a</sup> $\pm$ 0.1443
4	1.00 <sup>a</sup> $\pm$ 0.2170	1.09 <sup>a</sup> $\pm$ 0.1643	1.00 <sup>a</sup> $\pm$ 0.1937	1.03 <sup>a</sup> $\pm$ 0.2450

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant differences ( $P < 0.05$ ) in ADG between the Treatments in Phases 1, 3 and 4. The LSM of the Treatment 4 in Phase 2, was significantly ( $P < 0.05$ ) lower than that of the other three Treatments.

**Table 4.1.3** Pooled results of the ADG of all treatments for the MPD and PIC genotypes for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

	MPD Treatments			
	1	2	3	4
Phase	LSMean $\pm$ SD	LSMean $\pm$ SD	LSMean $\pm$ SD	LSMean $\pm$ SD
1	0.86 <sup>ab</sup> $\pm$ 0.0930	0.91 <sup>b</sup> $\pm$ 0.1261	0.84 <sup>ab</sup> $\pm$ 0.2210	0.92 <sup>b</sup> $\pm$ 0.1434
2	1.08 <sup>abc</sup> $\pm$ 0.1413	1.08 <sup>abc</sup> $\pm$ 0.1286	1.03 <sup>ab</sup> $\pm$ 0.1458	1.03 <sup>ab</sup> $\pm$ 0.1396
3	1.12 <sup>abc</sup> $\pm$ 0.1226	1.05 <sup>abc</sup> $\pm$ 0.2003	1.02 <sup>ab</sup> $\pm$ 0.1206	1.07 <sup>abc</sup> $\pm$ 0.1003
4	0.93 <sup>a</sup> $\pm$ 0.1334	1.00 <sup>ab</sup> $\pm$ 0.1126	0.94 <sup>a</sup> $\pm$ 0.2121	1.12 <sup>ab</sup> $\pm$ 0.0961

	PIC Treatments			
	1	2	3	4
Phase	LSMean $\pm$ SD	LSMean $\pm$ SD	LSMean $\pm$ SD	LSMean $\pm$ SD
1	0.87 <sup>ab</sup> $\pm$ 0.2022	0.92 <sup>b</sup> $\pm$ 0.2111	0.92 <sup>b</sup> $\pm$ 0.2041	0.77 <sup>a</sup> $\pm$ 0.3134
2	1.12 <sup>bc</sup> $\pm$ 0.1906	1.12 <sup>bc</sup> $\pm$ 0.1502	1.18 <sup>c</sup> $\pm$ 0.1736	0.98 <sup>a</sup> $\pm$ 0.3379
3	1.01 <sup>a</sup> $\pm$ 0.3244	1.19 <sup>bc</sup> $\pm$ 0.1378	1.11 <sup>abc</sup> $\pm$ 0.1738	1.16 <sup>c</sup> $\pm$ 0.1668
4	1.08 <sup>ab</sup> $\pm$ 0.2690	1.18 <sup>b</sup> $\pm$ 0.1608	1.05 <sup>ab</sup> $\pm$ 0.1713	0.95 <sup>a</sup> $\pm$ 0.3230

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

In Phase 1 the two genotypes differed significantly ( $P < 0.05$ ) in ADG with Treatment 4. In Phase 2 the two genotypes differed significantly ( $P < 0.05$ ) in ADG with Treatment 3. It was the only significant differences ( $P < 0.05$ ) in a Phase between the genotypes for the same Treatments. All the other significant differences were between different Genotype x Treatment interactions in the same Phase.

The ADG of PIC Treatment 4 in Phase 1, was significantly lower ( $P < 0.05$ ) than PIC Treatments 2 and 3 as well as MPD Treatments 2 and 4. It was the lowest of all the Genotype x Treatment interactions. In Phase 2, PIC Treatment 4 was still the lowest and it was significantly lower ( $P < 0.05$ ) than PIC Treatments 1, 2 and 3. PIC Treatment 3 was significantly higher ( $P < 0.05$ ) than MPD Treatments 3 and 4. PIC Treatment 4 was significantly higher ( $P < 0.05$ ) in Phase 3 than PIC Treatment 1 and MPD Treatment 3. In Phase 4 PIC Treatment 2 was the highest and was significantly higher ( $P < 0.05$ ) than PIC Treatment 4 and MPD Treatments 1 and 3. The two genotypes did not differ significantly ( $P < 0.05$ ) between the same Treatments, in Phases 3 and 4. The differences between the Genotype x Treatment interactions were mostly not significant ( $P < 0.05$ ). None of the dietary



treatments increased or depressed ADG dramatically, in fact, the pigs responded more or less the same to all the diets.

Kemm *et al.* (1991) fed lean and obese Landrace pigs with three different protein levels. The lean and obese pigs did not differ significantly (736 vs. 733 g/day) in ADG. The different protein levels did not have any significant effect on growth rate measured between 30kg and 90kg live weight. Fuller *et al.* (1995) found that the ADG of castrated males did not respond significantly to increased dietary protein. This can be seen in the present study as well. Campbell and King (1982) found that dietary protein level had no significant effect on growth performance from 20 to 45 kg. However, each increase in dietary protein in the live weight period 45 to 70 kg depressed the performance of castrated pigs. In Phase 4 the ADG of the high protein treatment (Treatment 1), was lower than some of the other Treatments, although not significantly ( $P < 0.05$ ). Siebrits *et al.* (1986) found an increase in the average daily gain up to a certain live weight and then a decrease in average daily gain from 30 to 100kg live weight. Most of the genotype treatment interactions followed the same trend in the present study. The results above did not indicate a clear difference in average daily gain between the genotypes, therefore it can not be concluded that there is a significant difference between the genotypes.

#### 4.2 Feed Intake

The Feed Intakes of the two genotypes for each Phase are presented separately. The results shown in Table 1.2.1 are the pooled results of all treatments of Phase 1: 30 kg to 50 kg live weight, Phase 2: 50 kg to 70 kg, Phase 3: 70 kg to 90 kg and Phase 4: 90 kg to 110 kg. The results in Table 1.2.2 are pooled results of the MPD and PIC genotypes of all the Phases for the Treatments. Treatment 1 was the high protein and treatment 4 was the low protein. The results in Table 1.2.3 are the results for each Phase for genotype treatment interaction. (Tables 4.2.1 – 4.2.3)

The feed intake was measured per pen.

**Table 4.2.1** Pooled results of the Feed Intake of all the treatments for the MPD and PIC genotypes for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

	MPD	PIC
Phase	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	115.90 <sup>a</sup> $\pm$ 22.3907	115.00 <sup>a</sup> $\pm$ 22.5968
2	167.78 <sup>a</sup> $\pm$ 31.2785	178.02 <sup>a</sup> $\pm$ 36.4516
3	177.91 <sup>a</sup> $\pm$ 28.7548	177.00 <sup>a</sup> $\pm$ 34.5169
4	58.60 <sup>a</sup> $\pm$ 6.1612	63.76 <sup>b</sup> $\pm$ 6.3899

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant differences in Phases 1, 2 and 3. The feed intake for the PIC genotype was significantly higher ( $P < 0.05$ ) than the MPD genotype in Phase 4.

**Table 4.2.2** Pooled results of the Feed Intake of the MPD and PIC genotypes for all the treatments for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

Phase	Treatments			
	1	2	3	4
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	118.48 <sup>a</sup> $\pm$ 20.4836	120.11 <sup>a</sup> $\pm$ 22.8810	115.14 <sup>a</sup> $\pm$ 22.5248	108.07 <sup>a</sup> $\pm$ 23.6663
2	165.64 <sup>a</sup> $\pm$ 34.8111	182.39 <sup>a</sup> $\pm$ 35.1775	174.80 <sup>a</sup> $\pm$ 38.4090	168.78 <sup>a</sup> $\pm$ 28.3561
3	164.39 <sup>a</sup> $\pm$ 34.2381	181.27 <sup>a</sup> $\pm$ 29.5830	185.11 <sup>a</sup> $\pm$ 32.6597	179.05 <sup>a</sup> $\pm$ 28.5185
4	63.93 <sup>b</sup> $\pm$ 6.4136	61.97 <sup>ab</sup> $\pm$ 7.5936	57.83 <sup>a</sup> $\pm$ 6.7112	61.00 <sup>ab</sup> $\pm$ 5.2388

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant differences in Phase 1, 2 and 3 between the treatments. In Phase 4 Treatment 3 was significantly lower ( $P < 0.05$ ) than Treatment 1.

**Table 4.2.3** Pooled results of the Feed Intake of all treatments for the MPD and PIC genotypes for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

Phase	MPD Treatments			
	1	2	3	4
	LSMean $\pm$ Std dev	LSMean $\pm$ Std dev	LSMean $\pm$ Std dev	LSMean $\pm$ Std dev
1	117.87 <sup>a</sup> $\pm$ 24.9089	123.10 <sup>a</sup> $\pm$ 25.3500	106.79 <sup>a</sup> $\pm$ 17.6313	115.83 <sup>a</sup> $\pm$ 22.8097
2	165.78 <sup>a</sup> $\pm$ 33.5212	176.75 <sup>a</sup> $\pm$ 35.3996	161.40 <sup>a</sup> $\pm$ 28.5706	167.20 <sup>a</sup> $\pm$ 32.6052
3	173.99 <sup>ab</sup> $\pm$ 29.5237	178.59 <sup>ab</sup> $\pm$ 31.9687	171.57 <sup>ab</sup> $\pm$ 25.0204	187.49 <sup>ab</sup> $\pm$ 32.0725
4	61.71 <sup>bc</sup> $\pm$ 5.2448	58.85 <sup>ab</sup> $\pm$ 6.3409	52.91 <sup>a</sup> $\pm$ 3.9418	60.94 <sup>bc</sup> $\pm$ 5.6541

Phase	PIC Treatments			
	1	2	3	4
	LSMean $\pm$ Std dev	LSMean $\pm$ Std dev	LSMean $\pm$ Std dev	LSMean $\pm$ Std dev
1	119.08 <sup>a</sup> $\pm$ 16.9638	117.12 <sup>a</sup> $\pm$ 21.7002	123.50 <sup>a</sup> $\pm$ 25.0118	100.30 <sup>a</sup> $\pm$ 23.5049
2	165.49 <sup>a</sup> $\pm$ 38.7510	188.03 <sup>a</sup> $\pm$ 36.7939	188.20 <sup>a</sup> $\pm$ 44.2846	170.37 <sup>a</sup> $\pm$ 25.9455
3	154.79 <sup>a</sup> $\pm$ 38.1205	183.95 <sup>ab</sup> $\pm$ 29.2819	198.65 <sup>b</sup> $\pm$ 35.4589	170.61 <sup>ab</sup> $\pm$ 23.8176
4	66.15 <sup>c</sup> $\pm$ 7.0810	65.09 <sup>bc</sup> $\pm$ 7.8770	62.74 <sup>bc</sup> $\pm$ 5.0614	61.06 <sup>bc</sup> $\pm$ 5.2428

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There was only in Phase 4 a significant difference ( $P < 0.05$ ) between the genotypes for the same Treatment in the same Phase. They differed significantly in Treatment 3. There were significant differences ( $P < 0.05$ ) in the Phases between different Genotype x Treatment interactions.

There were no significant differences ( $P < 0.05$ ) in Phases 1 and 2. PIC Treatment 1 was significantly lower ( $P < 0.05$ ) than PIC Treatment 3 in Phase 3. PIC Treatment 1 was the lowest and PIC Treatment 3 the highest of the genotype treatment interactions for feed intake in Phase 3. MPD Treatment 3 was the lowest in Phase 4 and was significantly lower ( $P < 0.05$ ) than all the other genotype treatment interactions, except MPD Treatment 2. PIC Treatment 1 was significantly higher ( $P < 0.05$ ) than MPD Treatments 2 and 3. The Feed Intake (FI) was measured for each pen. Phase 1 and 2 had three pigs per pen, Phase 3 had two pigs per pen and Phase 4 had one pig per pen. This could be a possible reason for the non significant differences in FI between the genotypes, especially in Phases 1 and 2.

Castrated males had slightly higher ( $P < 0.05$ ) intakes on a low protein diet than on the high protein diet (Fuller *et al.*, 1995). This was not the case in the present study. In a study by Kemm *et al.* (1991), it was not possible to alter the mean daily intake and live mass gain, although dietary protein content was decreased from 19.7 to 13.7%. There were some

significant ( $P < 0.05$ ) differences in Feed Intake between the Genotype x Treatment interactions, but it was not constant over the trail period. The different protein levels did not have a major effect on the Feed Intake of the two genotypes. The Hypothesis that the two genotypes do not differ significantly in feed intake, can therefore not be rejected.

#### 4.3 Feed conversion ratios

The Feed Conversion Ratios (FCR) of the two genotypes for each Phase are presented separately. The results shown in Table 1.3.1 are the pooled results of all treatments of Phase 1: 30 kg to 50 kg live weight, Phase 2: 50 kg to 70 kg, Phase 3: 70 kg to 90 kg and Phase 4: 90 kg to 110 kg. The results in Table 1.3.2 are pooled results of the MPD and PIC genotypes of all the Phases for the Treatments. Treatment 1 was the high protein and treatment 4 was the low protein. The results in Table 4.3.3 are the results for each Phase for genotype treatment interaction. (Tables 4.3.1 – 4.3.3)

**Table 4.3.1.** Pooled results of the FCR of all treatments for the MPD and PIC genotypes for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

	MPD	PIC
Phase	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	2.31 <sup>a</sup> $\pm$ 0.2943	2.35 <sup>a</sup> $\pm$ 0.3933
2	2.77 <sup>a</sup> $\pm$ 0.2359	4.50 <sup>b</sup> $\pm$ 0.6923
3	4.39 <sup>a</sup> $\pm$ 1.1499	4.27 <sup>a</sup> $\pm$ 1.2248
4	3.53 <sup>a</sup> $\pm$ 0.6327	3.89 <sup>a</sup> $\pm$ 1.9965

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

The FCR of the PIC genotype was significantly higher ( $P < 0.05$ ) than the FCR of the MPD genotype in Phase 2. The differences between the genotypes in the other Phases were not significant ( $P < 0.05$ ).

**Table 4.3.2** Pooled results of the FCR of the MPD and PIC genotypes for all the treatments for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

Phase	Treatments			
	1	2	3	4
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	2.43 <sup>a</sup> $\pm$ 0.4107	2.24 <sup>a</sup> $\pm$ 0.2264	2.33 <sup>a</sup> $\pm$ 0.3220	2.33 <sup>a</sup> $\pm$ 0.3983
2	3.61 <sup>a</sup> $\pm$ 1.0082	3.71 <sup>a</sup> $\pm$ 1.1557	3.59 <sup>a</sup> $\pm$ 0.9882	3.63 <sup>a</sup> $\pm$ 1.0041
3	5.03 <sup>b</sup> $\pm$ 1.7009	3.89 <sup>a</sup> $\pm$ 0.6508	4.13 <sup>a</sup> $\pm$ 0.5221	4.27 <sup>ab</sup> $\pm$ 1.2285
4	3.92 <sup>a</sup> $\pm$ 0.9822	3.37 <sup>a</sup> $\pm$ 0.3638	3.51 <sup>a</sup> $\pm$ 0.6303	4.04 <sup>a</sup> $\pm$ 2.7237

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant differences ( $P < 0.05$ ) in Phases 1, 2 and 4. Treatment 1 was significantly higher ( $P < 0.05$ ) than Treatments 2 and 3 in Phase 3.

**Table 4.3.3** Pooled results of the FCR of all treatments for the MPD and PIC genotypes for each growth phase (Phase 1: 30kg to 50kg, Phase 2: 50kg to 70kg, Phase 3: 70kg to 90kg and Phase 4: 90kg to 110kg).

Phase	MPD Treatments			
	1	2	3	4
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	2.43 <sup>a</sup> $\pm$ 0.4228	2.24 <sup>a</sup> $\pm$ 0.2431	2.27 <sup>a</sup> $\pm$ 0.3492	2.32 <sup>a</sup> $\pm$ 0.0759
2	2.71 <sup>a</sup> $\pm$ 0.2168	2.72 <sup>a</sup> $\pm$ 0.2835	2.77 <sup>a</sup> $\pm$ 0.2705	2.877 <sup>a</sup> $\pm$ 0.1695
3	4.70 <sup>ab</sup> $\pm$ 1.4843	4.11 <sup>a</sup> $\pm$ 0.8083	4.02 <sup>a</sup> $\pm$ 0.5896	4.72 <sup>ab</sup> $\pm$ 1.5032
4	3.98 <sup>ab</sup> $\pm$ 0.7920	3.47 <sup>ab</sup> $\pm$ 0.2561	3.45 <sup>ab</sup> $\pm$ 0.8035	3.22 <sup>a</sup> $\pm$ 0.3253

Phase	PIC Treatments			
	1	2	3	4
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	2.44 <sup>a</sup> $\pm$ 0.4321	2.24 <sup>a</sup> $\pm$ 0.2279	2.40 <sup>a</sup> $\pm$ 0.3044	2.34 <sup>a</sup> $\pm$ 0.5812
2	4.50 <sup>b</sup> $\pm$ 0.5176	4.69 <sup>b</sup> $\pm$ 0.7349	4.42 <sup>b</sup> $\pm$ 0.6768	4.39 <sup>b</sup> $\pm$ 0.9084
3	5.36 <sup>b</sup> $\pm$ 1.9513	3.67 <sup>a</sup> $\pm$ 0.3912	4.24 <sup>ab</sup> $\pm$ 0.4637	3.81 <sup>a</sup> $\pm$ 0.7271
4	3.86 <sup>ab</sup> $\pm$ 1.2063	3.26 <sup>a</sup> $\pm$ 0.4423	3.57 <sup>ab</sup> $\pm$ 0.4551	4.86 <sup>b</sup> $\pm$ 3.7941

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

In Phase 2 the PIC feed conversion ratios were significantly higher ( $P < 0.05$ ) than the MPD feed conversion ratios. In Phase 4 PIC Treatment 4 was significantly higher ( $P < 0.05$ ) than MPD Treatment 4. All of the other significant differences ( $P < 0.05$ ) were between different Genotype x Treatment interactions.

There were no significant differences ( $P < 0.05$ ) between the genotype treatment interactions in Phase 1. PIC Treatment 1 was significantly higher ( $P < 0.05$ ) than PIC Treatments 2 and 4 and

MPD Treatments 2 and 3 in Phase 3. PIC Treatment 2 was significantly lower ( $P < 0.05$ ) than PIC Treatment 4 in Phase 4.

A Study of Fuller *et al.* (1995) showed that increased dietary protein did not elicit a significant reduction in the FCR of castrated males. The treatments in the present study did not have a real influence on the FCR. The FCRs of the PIC genotype was worse than the MPD genotype in Phase 2. This difference did not occur in the other periods. The ADG of the PIC genotype was higher in Phase 2 only for Treatments 1, 2 and 3. The Feed Intake of the PIC genotype in Phase 2 was higher for Treatments 2, 3 and 4 than the MPD genotype. Therefore the Hypothesis that the two genotypes do not differ significantly in feed efficiency, can not be rejected.

#### 4.4 Slaughter periods:

##### Commercial slaughter

The animals were slaughtered at 70 kg (S1), 90 kg (S2) and 110 kg (S3) live weight. (Tables 4.4.1-4.4.6)

**Table 4.4.1** Pooled results of the commercial slaughter of all treatments for the MPD and PIC genotypes for live weight, cold weight and slaughter percentage over all the growth phases.

Parameters	MPD	PIC
	LSMean $\pm$ Sd	LSMean $\pm$ Sd
LW	92.79 <sup>a</sup> $\pm$ 18.8259	96.64 <sup>d</sup> $\pm$ 22.6231
CW	71.75 <sup>a</sup> $\pm$ 15.2133	73.12 <sup>b</sup> $\pm$ 17.9506
SP	77.18 <sup>a</sup> $\pm$ 1.8048	75.42 <sup>b</sup> $\pm$ 2.4065

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

LW: Live weight

CW: Cold carcass weight

SP: Slaughter percentage

The two genotypes did not differ significantly ( $P < 0.05$ ) for live weight, although the PIC genotype had a higher live weight. There was a significant difference ( $P < 0.05$ ) between the cold weight of the genotypes with again the PIC genotype the higher one. The MPD genotype had a significantly ( $P < 0.05$ ) higher slaughter percentage than the PIC genotype.

**Table 4.4.2** Pooled results of the commercial slaughter of the MPD and PIC genotypes for all the treatments for live weight, cold weight and slaughter percentage over all the growth phases.

Parameters	Treatments			
	1	2	3	4
	LSMean ± Sd	LSMean ± Sd	LSMean ± Sd	LSMean ± Sd
LW	94.59 <sup>a</sup> ± 19.4561	96.28 <sup>a</sup> ± 21.6996	95.71 <sup>a</sup> ± 19.7138	92.27 <sup>a</sup> ± 22.6785
CW	71.82 <sup>a</sup> ± 15.8129	73.61 <sup>a</sup> ± 16.5219	73.89 <sup>a</sup> ± 15.6857	70.41 <sup>a</sup> ± 18.5569
SP	75.72 <sup>a</sup> ± 2.1614	76.47 <sup>ab</sup> ± 1.6759	77.09 <sup>b</sup> ± 2.1548	75.91 <sup>a</sup> ± 2.8875

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

The differences between the Treatments was not significant ( $P < 0.05$ ) for live weight and cold weight. Treatment 3 had the highest slaughter percentage and it was significantly ( $P < 0.05$ ) higher than Treatments 1 and 4.

**Table 4.4.3** Pooled results of the commercial slaughter of the MPD and PIC genotypes for all the treatments for live weight, cold weight and slaughter percentage over all the growth phases. (Period 1: at the end of growth Phase 2: 50kg to 70 kg; Period 2: at the end of growth Phase 3, 70 kg to 90 kg and Period 3: at the end of growth Phase 4, 90 kg to 110 kg)

Parameters	Slaughter periods		
	1	2	3
	LSMean ± Sd	LSMean ± Sd	LSMean ± Sd
LW	70.85 <sup>a</sup> ± 10.8632	97.35 <sup>b</sup> ± 10.2310	115.94 <sup>c</sup> ± 9.5127
CW	53.85 <sup>a</sup> ± 9.3130	74.00 <sup>b</sup> ± 8.3113	89.45 <sup>c</sup> ± 7.5635
SP	75.78 <sup>a</sup> ± 2.9999	75.97 <sup>a</sup> ± 1.8640	77.14 <sup>b</sup> ± 1.7643

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

The live weights and the cold weights differed significantly ( $P < 0.05$ ) between periods with Period 1 the lowest and Period 3 the highest. The slaughter percentage of Period 2 was higher than Period 1 but it did not differ significantly ( $P < 0.05$ ). Period 3 had the highest slaughter percentage and it differ significantly ( $P < 0.05$ ) with Periods 1 and 2.

**Table 4.4.4** Pooled results of the commercial slaughter for live weight of all treatments for the MPD and PIC genotypes for each slaughter period. (Period 1: at the end of growth Phase 2: 50kg to 70 kg; Period 2: at the end of growth Phase 3, 70 kg to 90 kg and Period 3: at the end of growth Phase 4, 90 kg to 110 kg)

	MPD Treatments			
	1	2	3	4
Periods	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
S1	71.20 <sup>ab</sup> $\pm$ 7.5961	70.67 <sup>ab</sup> $\pm$ 7.5542	73.67 <sup>b</sup> $\pm$ 13.9523	69.80 <sup>ab</sup> $\pm$ 11.8195
S2	95.86 <sup>ab</sup> $\pm$ 8.3552	94.29 <sup>ab</sup> $\pm$ 10.6570	91.33 <sup>a</sup> $\pm$ 5.2789	96.00 <sup>ab</sup> $\pm$ 7.2388
S3	111.57 <sup>ab</sup> $\pm$ 8.7723	115.43 <sup>ab</sup> $\pm$ 9.1078	111.00 <sup>a</sup> $\pm$ 5.8310	114.57 <sup>ab</sup> $\pm$ 5.3807

	PIC Treatments			
	1	2	3	4
Periods	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
S1	75.60 <sup>b</sup> $\pm$ 9.1269	71.67 <sup>ab</sup> $\pm$ 9.4956	75.60 <sup>b</sup> $\pm$ 5.6833	60.83 <sup>a</sup> $\pm$ 15.6897
S2	93.00 <sup>ab</sup> $\pm$ 8.8600	104.14 <sup>b</sup> $\pm$ 11.2461	102.57 <sup>b</sup> $\pm$ 10.5965	99.57 <sup>ab</sup> $\pm$ 13.7702
S3	120.50 <sup>ab</sup> $\pm$ 11.1131	121.57 <sup>b</sup> $\pm$ 9.3248	120.43 <sup>ab</sup> $\pm$ 13.4766	112.86 <sup>ab</sup> $\pm$ 8.6300

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

PIC Treatment 4 was significantly lower ( $P < 0.05$ ), in the first Period, than PIC Treatments 1 and 3 and MPD Treatment 3. MPD Treatment 3 had the lowest live weight in Period 2 and it was significantly ( $P < 0.05$ ) lower than PIC Treatments 2 and 3. PIC Treatment 2 was the highest in Period 3 and it differed significantly ( $P < 0.05$ ) from MPD Treatment 3 that was the lowest in live weight. There were more heavy pigs in the last Period from the PIC genotype than the MPD genotype, but it was not always significantly ( $P < 0.05$ ).



**Table 4.4.5** Pooled results of the commercial slaughter for carcass weight of all treatments for the MPD and PIC genotypes for each slaughter period. (Period 1: at the end of growth Phase 2, 50kg to 70 kg; Period 2: at the end of growth Phase 3, 70 kg to 90 kg and Period 3: at the end of growth Phase 4, 90 kg to 110 kg)

	MPD Treatments			
	1	2	3	4
Periods	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
S1	53.96 <sup>ab</sup> $\pm$ 6.9475	54.70 <sup>b</sup> $\pm$ 5.9501	56.867 <sup>b</sup> $\pm$ 11.1654	53.76 <sup>ab</sup> $\pm$ 9.7728
S2	73.03 <sup>ab</sup> $\pm$ 6.6837	72.29 <sup>ab</sup> $\pm$ 8.5205	71.07 <sup>ab</sup> $\pm$ 4.0766	73.50 <sup>ab</sup> $\pm$ 6.3128
S3	87.23 <sup>a</sup> $\pm$ 7.8370	89.457 <sup>a</sup> $\pm$ 7.0016	86.89 <sup>a</sup> $\pm$ 6.8204	89.60 <sup>a</sup> $\pm$ 5.8492

	PIC Treatments			
	1	2	3	4
Periods	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
S1	55.80 <sup>b</sup> $\pm$ 7.3253	54.33 <sup>b</sup> $\pm$ 8.1208	58.08 <sup>b</sup> $\pm$ 7.3384	44.63 <sup>a</sup> $\pm$ 13.5330
S2	69.08 <sup>a</sup> $\pm$ 7.8519	78.94 <sup>b</sup> $\pm$ 9.2417	78.17 <sup>ab</sup> $\pm$ 8.3278	74.31 <sup>ab</sup> $\pm$ 12.0210
S3	91.67 <sup>a</sup> $\pm$ 8.2187	91.80 <sup>a</sup> $\pm$ 6.7221	92.66 <sup>a</sup> $\pm$ 11.1909	86.60 <sup>a</sup> $\pm$ 7.1190

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

PIC Treatment 4 had the lowest cold weight in Period 1 and it was significantly ( $P < 0.05$ ) lower than PIC Treatments 1, 2 and 3 and MPD Treatments 2 and 3. The cold weight of PIC Treatment 1 was the lowest in Period 2 and it differed significantly ( $P < 0.05$ ) from PIC Treatment 2. There were no significant differences ( $P < 0.05$ ) in Period 3. The cold weights of the genotypes did differ, but the one genotype did not have higher or lower cold weights than the other for all the Periods. The Treatments did not affect the cold weights in such a way as to give lower or higher cold weights. The heavier live weights of the PIC genotype in the last Period led to the heavier cold weights for Treatments 1, 2 and 3, although not significantly ( $P < 0.05$ ).

**Table 4.4.6** LSMean and Standard deviations of between slaughtering periods for genotypes and treatments Pooled results of the commercial slaughter for slaughter percentage of all treatments for the MPD and PIC genotypes for each slaughter period. (Period 1: at the end of growth Phase 2, 50kg to 70 kg; Period 2: at the end of growth Phase 3, 70 kg to 90 kg and Period 3: at the end of growth Phase 4, 90 kg to 110 kg)

	MPD Treatments			
	1	2	3	4
Periods	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
S1	75.70 <sup>bc</sup> $\pm$ 2.9820	77.41 <sup>c</sup> $\pm$ 1.8067	77.12 <sup>c</sup> $\pm$ 1.9236	76.91 <sup>c</sup> $\pm$ 1.2334
S2	76.17 <sup>ab</sup> $\pm$ 1.1498	76.64 <sup>b</sup> $\pm$ 1.6221	77.82 <sup>b</sup> $\pm$ 0.9874	76.53 <sup>ab</sup> $\pm$ 2.0416
S3	78.13 <sup>a</sup> $\pm$ 1.2040	77.51 <sup>a</sup> $\pm$ 0.7606	78.20 <sup>a</sup> $\pm$ 2.5972	78.15 <sup>a</sup> $\pm$ 1.7859

	PIC Treatments			
	1	2	3	4
Periods	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
S1	73.74 <sup>ab</sup> $\pm$ 1.2652	76.22 <sup>c</sup> $\pm$ 2.2106	76.59 <sup>c</sup> $\pm$ 4.2609	72.64 <sup>a</sup> $\pm$ 4.2308
S2	74.18 <sup>a</sup> $\pm$ 1.9666	75.75 <sup>ab</sup> $\pm$ 1.2257	76.20 <sup>ab</sup> $\pm$ 1.1416	74.42 <sup>a</sup> $\pm$ 2.4505
S3	76.09 <sup>a</sup> $\pm$ 1.4773	75.54 <sup>a</sup> $\pm$ 1.7057	76.87 <sup>a</sup> $\pm$ 1.2052	76.71 <sup>a</sup> $\pm$ 1.3072

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

PIC Treatment 4 had the lowest slaughter percentage and it differed significantly ( $P < 0.05$ ) from all the other Genotype x Treatment interactions in Period 1, except PIC Treatment 1. PIC Treatment 1 was significantly ( $P < 0.05$ ) lower than PIC Treatments 2 and 3 and MPD Treatments 2, 3 and 4. MPD Treatments 2 and 3 was significantly higher than PIC Treatments 1 and 4 in Period 2. There were no significant ( $P < 0.05$ ) differences between the Genotype x Treatment interactions in Period 3. The MPD genotype had higher slaughter percentages than the PIC genotype for each Period, but it was not always significant ( $P < 0.05$ ). Treatment 4 had a lower slaughter percentage than Treatment 3 for all the Periods in both genotypes, although just once significantly ( $P < 0.05$ ).

#### 4.5 Chemical analyses of the empty body samples:

Samples for chemical analyses were taken only from pigs of Treatment 1 at the following stages: Stage 1: 5 days of age, Stage 2: 14 days of age, Stage 3: 30 kg live weight, Stage 4: 50 kg live weight, Stage 5: 70 kg live weight, Stage 6: 90 kg live weight and Stage 7: 110 kg live weight. (Tables 4.5.1-4.5.8).

**Table 4.5.1** Pooled results of the chemical analyses of treatment 1 for the MPD and the PIC genotypes for all the Stages for dry matter, fat, ash, protein and protein:fat ratio.

	MPD	PIC
	LSMean ± Sd	LSMean ± Sd
DM	35.32 <sup>a</sup> ± 8.7974	36.29 <sup>a</sup> ± 7.7075
Fat	43.44 <sup>a</sup> ± 13.5888	43.99 <sup>a</sup> ± 12.3658
Ash	9.06 <sup>a</sup> ± 3.4281	8.84 <sup>a</sup> ± 2.6935
Protein	47.51 <sup>a</sup> ± 10.4175	47.16 <sup>a</sup> ± 9.9458
Prot:Fat	1.34 <sup>a</sup> ± 0.8935	1.25 <sup>a</sup> ± 0.7058

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

DM: Dry matter

There were no significant ( $P < 0.05$ ) differences between the genotypes for the different analyses.

**Table 4.5.2** Pooled results of the chemical analyses of treatment 1 for the MPD and the PIC genotypes for all the Stages for dry matter, fat, ash, protein and protein:fat ratio. (Slaughter time 1: 4 days, 2: 15 days, 3: 30 kg, 4: 50 kg, 6: 70 kg, 7: 90 kg and 7: 110 kg)

	Stage			
	1	2	3	4
	LSMean ± Sd	LSMean ± Sd	LSMean ± Sd	LSMean ± Sd
DM	22.29 <sup>a</sup> ± 1.9577	31.87 <sup>b</sup> ± 1.3779	32.17 <sup>b</sup> ± 0.9394	34.83 <sup>c</sup> ± 1.3891
Fat	21.84 <sup>a</sup> ± 3.2929	42.67 <sup>cd</sup> ± 3.3581	37.81 <sup>b</sup> ± 1.8021	40.95 <sup>bc</sup> ± 4.1286
Ash	14.95 <sup>f</sup> ± 1.7485	8.65 <sup>ce</sup> ± 0.4502	9.93 <sup>cd</sup> ± 0.8900	8.51 <sup>cd</sup> ± 0.7398
Protein	63.22 <sup>c</sup> ± 2.4774	48.69 <sup>cd</sup> ± 3.0665	52.26 <sup>d</sup> ± 1.7327	50.55 <sup>d</sup> ± 3.5310
Prot:Fat	2.96 <sup>d</sup> ± 0.5249	1.15 <sup>bc</sup> ± 0.17945	1.39 <sup>c</sup> ± 0.1096	1.25 <sup>bc</sup> ± 0.2135

	Stage		
	5	6	7
	LSMean ± Sd	LSMean ± Sd	LSMean ± Sd
DM	39.18 <sup>d</sup> ± 1.4296	43.26 <sup>e</sup> ± 1.9766	48.37 <sup>f</sup> ± 2.5390
Fat	45.96 <sup>d</sup> ± 5.9520	56.39 <sup>e</sup> ± 2.9354	62.56 <sup>f</sup> ± 3.1178
Ash	7.99 <sup>bc</sup> ± 1.9657	6.54 <sup>ab</sup> ± 0.6182	5.66 <sup>a</sup> ± 0.7011
Protein	46.05 <sup>c</sup> ± 4.4935	37.07 <sup>b</sup> ± 2.7675	31.78 <sup>a</sup> ± 2.6348
Prot:Fat	1.03 <sup>b</sup> ± 0.2849	0.66 <sup>a</sup> ± 0.0832	0.51 <sup>a</sup> ± 0.0630

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

The DM content of the samples tended to increase from the first sample taken to the last. The DM of Stage 2 was lower than the DM of Stage 3, although not significantly ( $P < 0.05$ ). The fat content of the samples followed more or less the same trend than the DM, except that Stage 2 and 4 did not differ significantly ( $P < 0.05$ ). Stage 3 and 4 did not differ significantly and Stage 2 and 5 did not differ significantly ( $P < 0.05$ ).

The ash content tended to decrease from Stage 1 to 7.

Protein content decreased as well and it seems the older pigs had less ash and protein, proportionally.

The protein:fat ratio decreased from Stage 1 to 7. The fat portion increased and the protein portion decreased.

Tables 4.5.3 – 4.5.7 LSMeans and Standard deviations between genotypes for the periods and parameters.

**Table 4.5.3** Pooled results of the chemical analyses for the MPD and the PIC genotypes for all the Stages for dry matter.

DM	MPD	PIC
Stage	LSMean ± Sd	LSMean ± Sd
1	20.87 <sup>a</sup> ± 1.1108	23.70 <sup>b</sup> ± 1.5255
2	31.34 <sup>a</sup> ± 1.8001	32.40 <sup>a</sup> ± 0.8143
3	31.79 <sup>a</sup> ± 0.4674	32.54 <sup>a</sup> ± 1.2484
4	35.43 <sup>a</sup> ± 1.3437	34.22 <sup>a</sup> ± 1.3858
5	38.86 <sup>a</sup> ± 1.7494	39.49 <sup>a</sup> ± 1.3222
6	41.56 <sup>a</sup> ± 1.1384	44.40 <sup>a</sup> ± 1.5198
7	49.49 <sup>a</sup> ± 3.2591	47.24 <sup>a</sup> ± 1.3116

LSMeans with different superscripts in the same row indicate a significant difference (P<0.05).

The MPD genotype had significantly (P<0.05) less dry matter in Period 1 than the PIC genotype. The two genotype did not differ significantly (P<0.05) in the other Periods.

**Table 4.5.4** Pooled results of the chemical analyses for the MPD and the PIC genotypes for all the Stages for fat.

Fat	MPD	PIC
Stage	LSMean ± Sd	LSMean ± Sd
1	19.86 <sup>a</sup> ± 1.9990	23.81 <sup>a</sup> ± 3.3779
2	41.70 <sup>a</sup> ± 4.9780	43.64 <sup>a</sup> ± 0.7679
3	37.00 <sup>a</sup> ± 2.321	38.62 <sup>a</sup> ± 0.8700
4	42.57 <sup>a</sup> ± 3.9683	39.32 <sup>a</sup> ± 4.3562
5	48.25 <sup>a</sup> ± 0.4080	43.67 <sup>a</sup> ± 8.5231
6	54.52 <sup>a</sup> ± 2.2274	57.64 <sup>a</sup> ± 2.9821
7	63.86 <sup>a</sup> ± 4.1100	61.26 <sup>a</sup> ± 1.5211

LSMeans with different superscripts in the same row indicate a significant difference (P<0.05).

There were no significant differences (P<0.05) between the two genotypes in any Period. MPD did not have a higher Fat content than PIC throughout the Periods and vice versa.

**Table 4.5.5** Ash Pooled results of the chemical analyses of treatment 1 for the MPD and the PIC genotypes for all the Stages for ash.

Ash	MPD	PIC
Stage	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	15.95 <sup>a</sup> $\pm$ 2.1462	13.95 <sup>a</sup> $\pm$ 0.1914
2	8.70 <sup>a</sup> $\pm$ 0.4484	8.59 <sup>a</sup> $\pm$ 0.5450
3	9.97 <sup>a</sup> $\pm$ 0.8804	9.88 <sup>a</sup> $\pm$ 1.0946
4	8.51 <sup>a</sup> $\pm$ 1.0404	8.51 <sup>a</sup> $\pm$ 0.5346
5	7.66 <sup>a</sup> $\pm$ 0.9958	8.32 <sup>a</sup> $\pm$ 2.8877
6	6.27 <sup>a</sup> $\pm$ 0.0636	6.72 <sup>a</sup> $\pm$ 0.7965
7	5.41 <sup>a</sup> $\pm$ 0.4590	5.91 <sup>a</sup> $\pm$ 0.9101

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant differences ( $P < 0.05$ ) between the genotypes in any Period.

**Table 4.5.6** Protein Pooled results of the chemical analyses for the MPD and the PIC genotypes for all the Stages for protein.

Protein	MPD	PIC
Stage	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	64.19 <sup>a</sup> $\pm$ 0.9920	62.24 $\pm$ 3.3924
2	49.60 <sup>a</sup> $\pm$ 4.5242	47.77 <sup>a</sup> $\pm$ 0.7269
3	53.02 <sup>a</sup> $\pm$ 1.4755	51.50 <sup>a</sup> $\pm$ 1.8921
4	48.92 <sup>a</sup> $\pm$ 2.9345	52.17 <sup>a</sup> $\pm$ 3.8278
5	44.09 <sup>a</sup> $\pm$ 1.2092	48.00 <sup>a</sup> $\pm$ 6.1246
6	39.23 <sup>a</sup> $\pm$ 2.2981	35.63 <sup>a</sup> $\pm$ 2.2188
7	30.73 <sup>a</sup> $\pm$ 3.6631	32.83 <sup>a</sup> $\pm$ 0.7804

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

There were no significant differences ( $P < 0.05$ ) between the genotypes in any Period.

**Table 4.5.7** Protein:Fat ratio Pooled results of the chemical analyses for the MPD and the PIC genotypes for all the Stages for protein:fat ratio.

Protein:Fat	MPD	PIC
Stage	LSMean $\pm$ Sd	LSMean $\pm$ Sd
1	3.25 <sup>a</sup> $\pm$ 0.3409	2.67 <sup>b</sup> $\pm$ 0.5600
2	1.21 <sup>a</sup> $\pm$ 0.2635	1.10 <sup>a</sup> $\pm$ 0.0332
3	1.44 <sup>a</sup> $\pm$ 0.1260	1.33 <sup>a</sup> $\pm$ 0.0778
4	1.16 <sup>a</sup> $\pm$ 0.1871	1.34 <sup>a</sup> $\pm$ 0.2321
5	0.91 <sup>a</sup> $\pm$ 0.0310	1.15 <sup>a</sup> $\pm$ 0.3992
6	0.72 <sup>a</sup> $\pm$ 0.0716	0.62 <sup>a</sup> $\pm$ 0.0725
7	0.48 <sup>a</sup> $\pm$ 0.0854	0.54 <sup>a</sup> $\pm$ 0.0250

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

The MPD genotype had a significant ( $P < 0.05$ ) higher Protein:Fat ratio than the PIC genotype in Period 1. The protein:fat ratios did not differ significantly between the genotypes in the other Stages.

**Table 4.5.8** Pooled results of the chemical analyses of treatment 1 for the MPD and the PIC genotypes for all the Stages for dry matter, fat, ash, protein and protein:fat ratio.

	MPD Stage			
	1	2	3	4
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
DM	20.87 <sup>a</sup> $\pm$ 1.1108	31.34 <sup>c</sup> $\pm$ 1.8001	31.79 <sup>cd</sup> $\pm$ 0.4674	35.43 <sup>e</sup> $\pm$ 1.3437
Fat	19.86 <sup>a</sup> $\pm$ 1.9990	41.70 <sup>bc</sup> $\pm$ 4.9780	37.00 <sup>b</sup> $\pm$ 2.3208	42.57 <sup>bcd</sup> $\pm$ 3.9683
Ash	15.95 <sup>f</sup> $\pm$ 2.1462	8.70 <sup>de</sup> $\pm$ 0.4484	9.97 <sup>e</sup> $\pm$ 0.8804	8.51 <sup>cde</sup> $\pm$ 1.0404
Protein	64.19 <sup>g</sup> $\pm$ 0.9920	49.60 <sup>ef</sup> $\pm$ 4.5242	53.02 <sup>f</sup> $\pm$ 1.4755	48.92 <sup>def</sup> $\pm$ 2.9345
Prot:Fat	3.25 <sup>f</sup> $\pm$ 0.3409	1.21 <sup>cd</sup> $\pm$ 0.2635	1.44 <sup>d</sup> $\pm$ 0.1260	1.16 <sup>bcd</sup> $\pm$ 0.1871

	MPD Stage		
	5	6	7
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
DM	38.86 <sup>f</sup> $\pm$ 1.7494	41.56 <sup>fg</sup> $\pm$ 1.1384	49.49 <sup>h</sup> $\pm$ 3.2591
Fat	48.25 <sup>de</sup> $\pm$ 0.4080	54.52 <sup>ef</sup> $\pm$ 2.2274	63.86 <sup>g</sup> $\pm$ 4.1100
Ash	7.66 <sup>bcd</sup> $\pm$ 0.9958	6.27 <sup>abc</sup> $\pm$ 0.0636	5.41 <sup>a</sup> $\pm$ 0.4590
Protein	44.09 <sup>dc</sup> $\pm$ 1.2092	39.23 <sup>bc</sup> $\pm$ 2.2981	30.73 <sup>a</sup> $\pm$ 3.6631
Prot:Fat	0.91 <sup>bc</sup> $\pm$ 0.0310	0.72 <sup>abc</sup> $\pm$ 0.0716	0.48 <sup>a</sup> $\pm$ 0.0854

	PIC Stage			
	1	2	3	4
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
DM	23.70 <sup>b</sup> $\pm$ 1.5255	32.40 <sup>cd</sup> $\pm$ 0.8143	32.54 <sup>cd</sup> $\pm$ 1.2484	34.22 <sup>de</sup> $\pm$ 1.3858
Fat	23.81 <sup>a</sup> $\pm$ 3.3779	43.64 <sup>cd</sup> $\pm$ 0.7679	38.62 <sup>bc</sup> $\pm$ 0.8701	39.32 <sup>bc</sup> $\pm$ 4.3562
Ash	13.95 <sup>f</sup> $\pm$ 0.1914	8.59 <sup>de</sup> $\pm$ 0.5450	9.88 <sup>e</sup> $\pm$ 1.0946	8.51 <sup>cde</sup> $\pm$ 0.5346
Protein	65.05 <sup>g</sup> $\pm$ 2.9976	47.77 <sup>de</sup> $\pm$ 0.7269	51.50 <sup>ef</sup> $\pm$ 1.8921	52.17 <sup>ef</sup> $\pm$ 3.8278
Prot:Fat	2.67 <sup>e</sup> $\pm$ 0.5600	1.10 <sup>cd</sup> $\pm$ 0.0321	1.33 <sup>d</sup> $\pm$ 0.0778	1.34 <sup>d</sup> $\pm$ 0.2322

	PIC Stage		
	5	6	7
	LSMean $\pm$ Sd	LSMean $\pm$ Sd	LSMean $\pm$ Sd
DM	39.49 <sup>f</sup> $\pm$ 1.3222	44.40 <sup>g</sup> $\pm$ 1.5198	47.24 <sup>h</sup> $\pm$ 1.3116
Fat	43.67 <sup>cd</sup> $\pm$ 8.5231	57.64 <sup>f</sup> $\pm$ 2.9821	61.26 <sup>fg</sup> $\pm$ 1.5211
Ash	8.32 <sup>cde</sup> $\pm$ 2.8877	6.73 <sup>abcd</sup> $\pm$ 0.7965	5.91 <sup>ab</sup> $\pm$ 0.9101
Protein	48.00 <sup>def</sup> $\pm$ 6.1246	35.63 <sup>ab</sup> $\pm$ 2.2188	32.83 <sup>a</sup> $\pm$ 0.7804
Prot:Fat	1.15 <sup>cd</sup> $\pm$ 0.3992	0.62 <sup>ab</sup> $\pm$ 0.0725	0.54 <sup>a</sup> $\pm$ 0.0250

LSMeans with different superscripts in the same row indicate a significant difference ( $P < 0.05$ ).

The DM and Fat content of both the genotypes increased from Stage 1 to Stage 7. The differences between the Stages were expected to be significant ( $P < 0.05$ ), with each Stage

higher than the previous one. This was not the case in Table 1.6.8. The differences between the genotypes are given in Tables 1.6.3 to 1.6.7 and only differences within a genotype will be discussed here.

The DM content of MPD Stage 2 was not significantly ( $P < 0.05$ ) lower than MPD Stage 3. MPD Stage 6 was not significantly ( $P < 0.05$ ) higher than MPD Stage 5. PIC Stages 2, 3 and 4 did not differ significantly ( $P < 0.05$ ), although the DM content increased from PIC Stage 2 to PIC Stage 4.

MPD Stages 2, 3 and 4 did not differ significantly ( $P < 0.05$ ) in fat contents. MPD Stage 3 was the lowest of the three Stages, and MPD Stage 4 the highest. MPD Stage 4 was not significantly ( $P < 0.05$ ) lower than MPD Stage 5 and MPD Stage 6 was not significantly ( $P < 0.05$ ) higher than MPD Stage 5. The fat content of PIC Stages 2, 3, 4 and 5 did not differ significantly ( $P < 0.05$ ), with PIC Period 3 the lowest of the four, then PIC Period 4. PIC Period 2 was higher than PIC period 4 and PIC period 5 was the highest of the four. PIC Period 7 was not significantly ( $P < 0.05$ ) higher than PIC Period 6.

The ash and protein content of the two genotypes decreased in relation to the fat and DM content from Stage 1 to Stage 7. The ratio between the decreasing protein content and increasing fat content, decreased from Stage 1 to Stage 7. The different stages of each parameter did not always differ significantly ( $P < 0.05$ ) from the previous stage of that parameter and not all of the stages were lower than the previous stages. The differences between the genotypes are indicated in Tables 1.6.3 to 1.6.7. The differences within the genotypes will be discussed below.

MPD Stages 2, 3 and 4 did not differ significantly ( $P < 0.05$ ) in ash content, with Stage 4 the lowest and Stage 3 the highest of the three. MPD Stages 4, 5 and 6 did not differ significantly ( $P < 0.05$ ), although the ash content decreased from Stages 4 to 6. MPD Stage 7 was not significantly ( $P < 0.05$ ) lower than MPD Stage 6. PIC Stages 2, 3, 4 and 5 did not differ significantly ( $P < 0.05$ ) in ash content. Stage 3 was the highest, followed by Stage 2, Stage 4 was higher than Stage 5. PIC Stage 6 was not significantly ( $P < 0.05$ ) lower than PIC Stages 4 or 5. PIC Stage 6 was not significantly ( $P < 0.05$ ) higher than PIC Stage 7.

The protein content of MPD Stages 2, 3 and 4 did not differ significantly ( $P < 0.05$ ), with Stage 3 the highest, followed by Stage 2. MPD Stage 5 was not significantly ( $P < 0.05$ ) lower than

MPD Stage 4, and MPD Stage 5 was not significantly ( $P < 0.05$ ) higher than MPD Stage 6. PIC Stages 2, 3, 4 and 5 did not differ significantly ( $P < 0.05$ ) in Protein content. Stage 4 was the highest followed by Stage 3 and then Stage 5. PIC Stage 7 was not significantly ( $P < 0.05$ ) lower than PIC Stage 6.

The protein:fat ratio of MPD Stages 2, 3 and 4 did not differ significantly ( $P < 0.05$ ), Stage 3 was the highest followed by Stage 2. MPD Stages 2, 4, 5 and 6 did not differ significantly ( $P < 0.05$ ), although the protein:fat ratio decreases from Stage 2 to Stage 6. MPD Stage 7 was not significantly ( $P < 0.05$ ) lower than MPD Stage 6. The protein:fat ratio of PIC Stages 2, 3, 4 and 5 did not differ significantly ( $P < 0.05$ ). Stage 4 was the highest followed by Stage 3 and then Stage 5. PIC Treatment 6 was not significantly ( $P < 0.05$ ) higher than PIC Stage 7.

The increase in fat and DM content were also found in SA Landrace sows (Coetzee, 1991). Campbell and Taverner (1988) found a decrease in the protein and ash contents and an increase in the fat and DM content of the castrated males they tested from 45 to 90kg live weight. Whittemore *et al.* (1988) studied the protein growth in pigs. The DM and fat content of the castrated males increased from 22kg live weight to 110kg live weight. The protein and ash content decreased. The results above give the impression that there is almost no significant difference in the chemical composition of the two genotypes. The Hypothesis that there is no significant difference between the genotypes for protein:fat ratio and protein deposition can therefore not be rejected.





## Chapter 5

### Conclusion:

Feeding the different protein levels in the diets did not have a significant effect on the two genotypes. The animals achieved the same results with all the diets. The growth performance and chemical analysis indicated that the two genotypes do not differ significantly for feed intake, feed conversion ratio, protein deposition, carcass analysis and protein:fat ratio. The Nil Hypothesis can therefore not be rejected.

## Chapter 6

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