

Gonadal development and the relationship to body development of pig genotypes in South Africa

by

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DEDICATION

Dedicated to my mother, Babedi Gofhamodimo, who has worked so hard to ensure that I get a proper education.

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DECLARATION

I declare that this dissertation which I submit to the University of Pretoria for the degree MSc (Agric) has not been submitted by me for a degree at any other University.

.....

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...../...../ 2005

Date

ABSTRACT

Gonadal development and the relationship to body development of pig genotypes in South Africa

The effect of genotype and slaughter age on gonadal development, body development and the correlations between these measurements were studied in five pig genotypes (Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5) consisting of 112 gilts and 112 boars with initial live weights varying between 25 – 30 kg. The pigs were group-housed in commercial type grower houses and fed a diet consisting of 14 MJ/kg energy, 18 % CP and 1.1 % lysine during the growth period up to a live weight of 65 kg, followed by a diet consisting 13.5 MJ/kg, 16 % CP and 0.9 % lysine from 65 kg to 90 kg and then a diet consisting of 13.2 MJ/kg, 15 % CP and 0.7 % lysine from 90 kg onwards. Pigs were slaughtered at 116, 130, 144, 158, 172, 186, 200 and 214 days of age. Gonadal growth and development were measured in gilts (ovary length, ovary width, ovary thickness, ovary weight, ovary volume, follicle number, and size of the largest follicle), boars (testis length, testis width, testis weight and testis volume) and body development parameters (slaughter weight, warm carcass weight, carcass length, chest depth, dressing percentage and P2 backfat thickness) were compared. Differences between means were tested using breed, sex and slaughter age as fixed effects, while the relationships between gonadal and body development parameters were evaluated by means of correlation analysis. Genotype 5 had a significantly shorter ovary length than Genotype 4 and Genotype 2. Genotype 2 gilts also had heavier ovaries and larger ovary volumes than Genotype 5 gilts. In boars, Genotype 2 had significantly heavier testes weights than Genotype 5 boars. Genotype 5 boars also tended to have smaller testis volumes than Genotype 2 boars. In body development, Genotype 2 gilts and boars were superior to the Genotype 5 in terms of slaughter and warm carcass weights, while Genotype 3 seconded Genotype 2. The average P2 backfat thicknesses were 11.88 mm and 13.68 mm for boars and gilts respectively. Correlations between gonadal and body development parameters were low to moderate in the gilts ($r = -0.305$ to 0.555) and moderate to high in boars ($r = 0.560$ to 0.871). However, dressing percentage, follicle number and size of the largest follicle correlated poorly with all other measurements. It is concluded from the study that although Genotype 5 do not grow to the same size and at the same rate compared to the other genotypes, they appear to be the most

suitable for the production of top quality pork in terms of its low backfat thickness. Genotype 5 pigs were also characterized with a slower gonadal growth and body development compared to Genotype 2 pigs. Results from this study suggest that selecting against backfat may delay gonadal development and sexual maturation in pigs.

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LIST OF ABBREVIATIONS

- ACTH:** adrenocorticotrophic hormone
- AI:** Artificial Insemination
- AMP:** chlormadinone acetate
- ANOVA:** Analysis of Variance
- ANPI:** Animal Nutrition and Products Institute
- ARC:** Agricultural Research Council
- °C:** degrees Celsius
- cm:** centimeter
- cm³:** centimeter cubed
- CP:** Crude protein
- eCG:** equine chorionic gonadotrophin
- FAO:** Food Agricultural Organization
- Fig.:** Figure
- FSH:** Follicle stimulating hormone
- g:** gram
- g/day:** grams per day
- GH:** Growth hormone
- GLM:** General Linear Model
- GnRH:** Gonadotrophin Releasing Hormone
- hCG:** human chorionic gonadotrophin
- HGP:** Hennessy Grading Probe
- IGF-I:** Insulin-like growth factor I
- kg:** kilogram
- kg/ton:** kilogram per tonne
- LSmeans:** Least square means
- LH:** Luteinizing hormone
- MAP:** methylacetoxyprogesterone
- MEg:** Metabolisable energy for growth
- MEM:** Metabolisable energy for maintenance
- MJ/kg:** Mega joules per kilogram

ml: millilitre

mm: millimeter

NDA: National Department of Agriculture

ng/ml: nano grams per millilitre

PGE2 α : Prostaglandin E2 alpha

PGF2 α : Prostaglandin F2 alpha

ppm: parts per million

RMRDT: Red Meat Research and Development Trust

RTV: Rietvlei

SA Landrace: South African Landrace

SAS: Statistical Analytical System

SE: Standard error

STD: Standard

TNF: tumour necrosis factor

Thrip: Technology and Human Resources for Industry Program

vs.: versus

CHAPTER 1

1.1 Introduction

World over, efficient production of lean meat for the consumer is the primary objective of commercial pig production. However, as pig units have become larger and more specialized, the needs of population have placed increased demands on reproductive performance. At the same time, large-scale confinement production itself created new problems which need to be solved in order to achieve and maintain optimum reproductive performance (Gordon, 1997). The economics of pig production depend very much on reproductive efficiency. Maintaining high levels of reproductive efficiency is essential in pig production, as improving reproductive efficiency will increase the overall efficiency of pig production. Genetics play an integral role in the control of these reproductive traits (Rothschild, 1996). Male reproductive traits or performance may be measured by testicular size (length, width, circumference, volume and weight) sperm volume, concentration of sperm, sperm quality, libido and breeding aggressiveness. Female reproductive traits on the other hand include litter size, number of piglets born alive, litter birth weight, number of piglets weaned, age at puberty, weaning to estrus interval and farrowing interval (Rothschild, 1996). Ovulation rate is also of prime importance since it determines the litter size, the most important reproductive trait from an economic point of view. Also, the number of viable piglets per litter is considered important since it generally defines prolificacy in pigs and is an important limiting factor affecting sow productivity (McCoard *et al.*, 2003). In South Africa, pig production has been structured to use F₁ crossbred sow to measure reproductive performance and then in theory, a sire line is used on such females to maximize the efficiency of lean production. The major sire lines currently used in South Africa are Large White, Landrace, and to a much lesser extent the Duroc and Hampshire (Rossouw, 1998).

Testicular and ovarian growth may be an indicator of reproductive performance in boars and gilts respectively. Research has shown that male pigs selected for larger testes at a constant age have greater sperm numbers and superior mating efficiency (Schinkel *et al.*, 1983). Growth and development of the ovaries and testes are of prime importance as these are the main reproductive organs in the female and boars respectively. Growth has traditionally been quantified by measuring the changes in body mass (live weight) various linear body dimensions

such as height, hip width and girth, or more recently the chemical components of body over time (Ferguson and Kyriaziz, 2003). The purpose of growth is to reach maturity. Two things are responsible for the maturation of an animal or an organ; an increase in body mass (growth) and change in body conformation (development) (Belt, 1988). Growth occurs through accretion of bone, fatty and lean tissue in the body while development relates to changes in shape, form and function as growth progresses.

1.2 Motivation

Due to increased consumer demand for lean meat, commercial pig producers tend to rely to a large extent on the use of imported pig breeds for the production of lean carcasses. On the other hand, the increased growth in the human population and urban expansion have resulted in an increased demand for animal products, which further necessitates the need to use these improved breeds. The majority of these improved breeds have been selected for high lean growth potential and reduced backfat thickness. The success of any pig production system depends on a high reproductive efficiency and performance; so, improving reproductive efficiency will increase the overall efficiency of pig production. There has however, been an increasing awareness of the possible conflict between selection for increasingly leaner pigs and reproductive performance of pigs. Research suggests that selecting for high lean growth in pigs tend to delay their sexual maturity, because the pigs tend to reach maturity at a later chronological age (O'Dowd *et al.*, 1997; Schinckel *et al.*, 1983; Whittemore, 1998). In breeding sows, the consequences of such selection strategies have been an increased mature body size, live weight and maintenance at any given age. A reduction in voluntary feed intake has also been reported in these lean lines and this makes it more difficult to meet the nutrient requirements from dietary input at times of high metabolic demands such as during lactation (O'Dowd *et al.*, 1997). Other negative effects of selecting for lean tissue includes lower litter size, reduced milk yield and reduced longevity (O'Dowd *et al.*, 1997). A decreased level of male hormones (5α -androstenone and testosterone) has also been reported in the systematic plasma of boars selected for low backfat and a high rate of lean gain, compared to those selected for fatness and a low rate of gain (Schinckel *et al.*, 1983).

Despite the fact that many studies were done worldwide, the effects of selecting for lean tissue growth rate on the reproductive development of pigs has not been adequately researched in

South Africa. Available literature covers production traits in pigs (Browne, 1994) and breed evaluations for phenotypic and production traits (Rossouw, 1998) which were obtained from performance testing results. Most studies focus on growth and meat and carcass characteristics due to their economic importance. Reproductive traits such as litter size at birth and piglet survival at 21 days of age are also reported in most studies. There is therefore a need to study the effects of these improved breeds on the reproductive development of pigs in South Africa, and also to see how these reproductive traits relate to body development traits. The aim of this study was therefore to study the gonadal development of commercial pig genotypes used in South Africa, as well as to study the correlations between gonadal development and body development.

1.3 Objectives

The objectives of the present dissertation were:

- ◆ to study the gonadal development of male and female pigs of different genotypes slaughtered at different target weights or ages

- ◆ to study the correlations between gonadal and body development of different pig genotypes.

Note:

The present research formed part of a larger pig research project conducted by the Agricultural Research Council (ARC) at Irene, South Africa. Since the ARC agreed not to disclose the names of the various pig genotypes used in the project, the breeds are also referred to as Genotype 1, 2, 3, 4 and 5 in the present thesis.

CHAPTER 2

2. LITERATURE REVIEW

2.1 The pig industry in South Africa

South Africa's climate is ideally suited for stock farming, and it is the most viable agricultural activity in a large part of the country. Almost 70% of the 122.3 million hectares of land surface in South Africa are suitable for raising livestock, particularly cattle, sheep, pigs and goats (SAMIC, 1998b). Stock farming in South Africa is therefore one of the oldest industries and it has played an important role in the history and development of the country (SAMIC, 1998a).

Pigs used by farmers today are descendants of wild pig (*Sus scrofa*). Pigs originated from Asia, Europe and Africa. They are descendants of the common wild boar, which existed in Europe throughout the middle ages (SAMIC, 1998a; Food Agricultural Organization (FAO) 2000). In the 1800s, the Chinese pig was introduced to Britain and while the Italian pig type breed was introduced later in the century. The Chinese pig tended to be small and white while the Italian pig was large and black. Nowadays, most of the modern pig breeds have a mixture of these characteristics. As with cattle and sheep, pigs were introduced to America and many other parts of the world by explorers and settlers (SAMIC, 1998a).

As in many developed countries, South Africa is currently based primarily on intensive systems of pig production. These intensive systems are designed to make the maximum use of space within controlled environment buildings with minimum labour requirements (Gordon, 1997). Today more than 25% of all pig production units in South Africa consist of 200 to 1000 sows, something that was non-existent twenty to thirty years ago (Agricultural Research Council (ARC) 1993).

Like in the rest of the world, the size of pig production units is increasing while the number of pig producers is decreasing (ARC, 1993). In 1984, there were 2000 producers who produced approximately 1 673 000 pigs (836 pigs per producer) while in 1990, there were 900 producers who produced 2 142 000 pigs (i.e. 2 380 pigs per producer) (ARC, 1993). The reason for this reduction in producer population is because of increased costs which comes along with

intensification, and the increased demand for improved quality of meat by consumers (Grimbeek, 2002). Intensive pig farming is more capital intensive and requires technical input and skill (Grimbeek, 2002) and it is for this main reason that many pig producers decided to quit. Despite the decreasing producer population, pig numbers continue to increase. It is estimated that pig numbers increased from 1,531 million in August 1999 to 1,556 million in August 2000 (National Department of Agriculture (NDA) 2000) while sow numbers in the intensive pig production sector are now estimated at 100 000 (SAMIC, 1998b). Throughout South Africa, pigs are found predominantly in the Eastern Cape, Western Cape and Mpumalanga regions. The majority of these pigs (95%) are kept mainly for pork while the remainder is kept for breeding purposes (NDA, 2000).

2.1.1 Marketing of pigs

Pig producers in South Africa belong to South African Pork Producers' Organisation (SAPPO), which serves as the mouthpiece of commercial pork producers in South Africa (SAMIC, 2005). SAPPO has a total of 46 registered pig abattoirs and is financed by a voluntary levy, which is paid per sow per month by more than 70 % of commercial pork producers in the country.

According to the latest classification system, pigs with a live mass up to 20kg are marketed as suckling pigs. Between the live weights of 21 and 90 kg, pigs are classified according to their backfat thickness, which is used to predict percentage of lean meat in the carcass (see Table 2.1). Pigs with live weights of 60 to 70 kg and that are 14 to 16 weeks old are marketed as porkers while those with live weights of 70 to 90 kg are marketed as baconers. Pigs with a live weight of 91 kg and higher are marketed as sausage pigs (ARC, 1993). Classification regulations specify that boars be castrated to qualify as baconers, which are marketed at a mass of 86 to 90 kg (ARC, 1993).

The average slaughter weight of pigs in South Africa is currently between 70 and 90 kg live weight. However, over the last decade, the genetic quality of pigs in terms of growth rate and feed conversion ratio has improved dramatically. This implies marketing heavier pigs in a cost-effective manner because slaughter pigs will reach slaughter weight age at a relatively young age. This will also bring South Africa on par with European counterparts where average slaughter weight is 119kg (Pieterse *et al.*, 2000). Selling at heavier weights may even be more beneficial in terms of processing since the ideal carcass mass for use by processors would

rather be 80kg. Carcasses of this weight provide a good proportion of meat to fat as compared to the average national carcass mass of about 65 to 69 kg (Unknown (a) 1994).

Table 2.1: Different classes of pigs showing their age, mass, feed intake and protein content requirement

Stage	Suckling pigs	Weaners	Large pigs	Finishing pigs	Dry sows and boars	Lactating sows
Age (days)	0 – 35	35 – 56	56 – 110	110 – 160	285days/yr	80days/yr
Mass (kg)	1.5 – 9	9 – 20	20 – 50	50 – 90	140 – 200	140 – 200
Intake (g/day)	50 – 520	About 800	About 1900	About 3000	About 4kg/sow/day from weaning to breeding. Generally 2kg/sow/day for the term of pregnancy	2kg plus 0.4kg/piglet to 8kg/day
Protein (%)	21	18	16	15	13 – 14	16

Source: National Department of Agriculture, 1997

2.1.2 Pig feeds used in South Africa

Major determinants of economic efficiency of a pig production unit are feed costs (Klindt *et al.*, 2001). Traditionally, pig feeds in South Africa were composed of maize meal or fishmeal with a little bran, some minerals and vitamins. However, fishmeal has since become expensive and scarce therefore making it necessary to look for alternative protein sources such as oilcake meals (ARC, 1993). Oilcake meals are vegetable protein sources made from the residues after extraction from oilseeds such as sunflower seeds, soya beans, groundnuts and cottonseed. Although protein quality of these vegetable sources is reported to be low, this can be corrected by addition of synthetic amino acids to improve protein quality in these feeds. Furthermore, vegetable sources often contain anti-nutritional factors which have to be rendered harmless by processing, a method which was also found to be expensive (ARC, 1993). According to Viljoen and Ras (1991) the fat component of these alternative protein sources also contain high proportions of unsaturated fatty acids. These unsaturated fatty acids have the potential of producing soft fatty tissue in the baconer, even though it is realized that the sex of the pig can also have an effect on the backfat fatty acid composition of the pig. Apart from maize, other grains such as sorghum, wheat, barley and triticale are used in pig feeds. Other protein sources apart from the ones mentioned above include bonemeal, milk products, lupins, beans and peas (NDA, 1997).

2.1.3 Pork consumption patterns in South Africa

Pigs belong to the least versatile group of farm animals since they only have one major product which is pork (Rossouw, 1998). However, the pig's positive attributes of early sexual maturity, high prolificacy and short generation interval compared to other farm animals have contributed to its competitiveness as a meat producer. Although pork has the potential to be an excellent source of protein, its average *per capita* consumption in South Africa is relatively low when compared to European countries. South Africa's average *per capita* consumption in 1990 was approximated at 3.62kg whereas *per capita* consumption in Europe was between 30 to 45kg/year (ARC, 1993). The United Kingdom and Denmark *per capita* pork consumptions have been reported to be 24.1 and 64.5kg/year respectively, as compared to South Africa's current *per capita* consumption of 3.5kg/year (De Kock *et al.*, 2001).

Table 2.2: *Per capita* consumption of various meat types in South Africa during 1990 and 2000

Meat type	Consumption (kg/year)	
	1989/90	1999/00 ¹
Beef	18.00	12.32
Mutton ²	5.0	3.8
Pork	3.4	3.1
White meat	16.72	22.91

¹ = Preliminary results

² = value includes mutton, lamb and goat meat

Source: Statistics South Africa, 2001

The low average *per capita* consumption has been attributed to two reasons, these being (a) competition with other meat types in the market and (b) pork acceptance or popularity to consumers (Grimbeek, 2002). These reasons are briefly discussed below.

a) Competition of pork with other meat types

The other meat types that pork has to compete with are beef, lamb, mutton and goat meat and chicken (see Table 2.2). Currently, the average *per capita* consumption in South Africa of beef and veal is 12.4 kg/year, consumption of lamb, mutton and goat meat being 3.6 kg/year while pork consumption is 3.2 kg/year. The total average *per capita* consumption of beef and veal, lamb, mutton and goat meat and pork is therefore 19.2 kg/year (SAMIC, 1998a). Another recent study on market research on South African consumers has shown that chicken is by far the most purchased and consumed meat type (98 % eaten and 92 % purchased) followed by beef (88 % eaten and 74 % purchased) and then fish (84 % eaten and 58 % purchased). Lamb occupies the fourth place (79 % eaten and 49 % purchased) then followed by pork (57 % eaten and 34 % purchased) (Unknown (b) 2000). Availability of beef, sheep meat and pork in the formal sector amounts to an average of 475 000 tonnes, 100 000 tonnes and 130 000 tonnes per annum respectively. This is based on an estimated annual slaughter of 1.95 million cattle, 4.5 million sheep and 2.00 million pigs at registered abattoirs (SAMIC, 1998b). It is further estimated that slaughterings in the informal sector could amount to a further 20 to 25 % in the case of cattle, 25 to 30 % in the case of sheep and 10 % in the case of pigs (SAMIC, 1998b). Besides these slaughterings, there is still more red

meat, which is imported into South Africa. Pork imports are reported to have increased by 39.4 % from 6,979 tons in 1998 to 9,727 tons in 1999. Of these 9046 tons of imported pork in the year 1999, 93 % came from European Union and Hungary (NDA, 2000).

b) Acceptance of pork by consumers

The population of South Africa has a heterogeneous composition in which various black ethnic groups (75 %) whites (14 %) and coloureds (9 %) make up the majority of the population. These groups represent the South African pork eating population and have different meat eating habits and attitudes towards pork in general (De Kock *et al.*, 2001). Average *per capita* consumption of pork by the different South African population groups varies with whites representing the highest *per capita* consumption at 16.2 kg, coloureds being average with 3.4 kg and blacks being the lowest at 0.6 kg (De Kock *et al.*, 2001). From these results, it is evident that pork does not make a significant part of the black consumers' diet despite the fact that they constitute a large proportion of the population in the country. Traditionally among black consumers, pork was reserved for special occasions. Currently, it has been suggested that the black consumers can be divided up into 3 groups; the first group being those who eat pork because it is fashionable, the second being those who eat pork because it is cheaper than other meats and the third group being those who do not eat pork because of religious beliefs or for other reasons (Unknown (a) 1994). Taking the low average *per capita* consumption among black consumers into consideration, it makes sense to assume that that there is a significant number of black consumers in the third group.

2.1.4 Pig breeding in South Africa

The South African Pig Industry breeding objectives can be divided into three classes. These are:

1. To improve the production traits such as economy of gain, high lifetime growth rate from birth to slaughter.
2. To improve the suitability of carcass quality for the available markets in terms of marketing weight, skin colour, carcass length, carcass lean percentage, backfat measurement, stress free pigs and the eating quality of pork.
3. To aim for high sow productivity, which is assessed by number of piglets born per litter (Rossouw, 1998).

Like in most countries worldwide, artificial insemination (AI) is widely practiced in piggeries in South Africa, where it is adopted as an on-farm practice. This means that the producer uses his own boar semen on his own sows. If semen is sold to another farmer, then the AI has to be registered by the National Department of Agriculture (Robinson, 2002).

Traditionally, pigs are only selected for breeding purposes if they have sufficient number of teats, with the threshold often set to 14 (Rydhmer, 2000). However, in South Africa, the threshold for number of teats is set at twelve, meaning that each sow must have at least six well-placed functional teats on each side (NDA, 1997). The number and quality of teats represent the main phenotypic characteristics for reproduction and maternal ability in the sow. Generally, pig production in South Africa has been structured to use F₁ crossbred sows or gilts, as these are the most productive females in modern intensive piggeries. F₁ crossbreds express more hybrid vigour (heterosis) than any other further crosses. The most popular F₁ in South Africa in most cases is the first cross between two major pig breeds in South Africa; the Large White and the South African Landrace (Robinson, 2002). The major sire lines to date have been the Large White and Landrace, and to a much lesser extent Duroc and Hampshire boars (Rossouw, 1998).

2.1.4.1 Crossbreeding and heterosis

In animal breeding, the method of crossbreeding is used on one hand to develop new breeds or populations, and on the other hand, the production of commercial hybrid animals as final products. Pig producers have long known that crossbreeding is an effective means of improving reproductive performance in pigs. Some examples of crossbreeding are crossing of two different breeds, crisscrossing and triple crossing. Crossing two different breeds involves the mating of purebred boars to pure bred or high-grade sows of another breed. In crisscrossing (two-breed rotational crossing) boars of two different breeds are used in alternate generations. Crossbred sows are retained in each generation and bred to boars of the same breed as the grandsire on the dam side. Crossbred vigour usually results in an increase in litter size, livability and growth rate (Eusebio, 1980). In triple crossing (3-breed rotation) the first cross gilts are mated to a boar of the third breed. Using a sire selected from each of the 3-breeds in rotation continues the process. Some pig producers in South Africa use a three-breed system with one specific breed being nominated as the ‘special’ sire line. The Duroc has been mainly used this way to improve the economic returns in the slaughter generation (Rossouw, 1998).

2.1.4.2 Advantages of crossbreeding

a) Use of hybrid vigour or heterosis

Crossbreeding is mainly practiced so as to take advantage of heterosis in crossbred animals. Majority of pigs marketed nowadays worldwide (80 to 90 %) are crossbreds (Eusebio, 1980). In South Africa, an increasing proportion of pigs slaughtered in the late 80s and 90s were crossbreds between Large White and South African Landrace (Browne, 1994). This large percentage indicates that pig producers have found crossbreeding to be an essential and profitable management practice (Eusebio, 1980). Maximum benefits of crossing are obtained when the sire, dam and progeny are all crosses of unrelated strains (Dunkin and Taverner, 1996).

b) Improving reproductive efficiency

Crossbreeding can be one of the most efficient ways to increase reproductive efficiency in pigs. Lowly heritable traits like reproductive characteristics (e.g. litter size at birth, litter size at weaning, individual piglet mass at weaning and piglet survival rate) respond best to crossbreeding (ARC, 1993; Browne, 1994). While heterosis improves fertility in traits that are lowly heritable, combining breed strengths through

crossbreeding can improve traits that are more highly heritable (ARC, 1993). However, each performance trait responds differently depending on the amount of heterosis exhibited.

c) Breed traits complementarity

In addition to heterosis, there are extra benefits to be gained from crossing if the lines producing the sires and the lines producing the dams have somewhat different attributes that can compliment each other in the cross. In practice, this is achieved by the development of specialized sire lines (developed from meaty breeds and selected for lean growth alone) and specialized dam lines (developed at least partly from prolific breeds and selected for reproductive ability and lean growth) (Dunkin and Taverner, 1996).

d) Reduction of inbreeding

The other advantage of crossbreeding is to avoid the deleterious effects of inbreeding which otherwise occur especially in pure breeding of small populations. Inbreeding increases the frequency of deleterious recessive genes, which are otherwise hidden in the population. According to Rothschild (1996) the effects of inbreeding in pigs are negative on average as they reduce hybrid vigour and lessen reproductive efficiency. This reduction, referred to as inbreeding depression, is generally larger for lowly heritable traits (e.g. reproductive traits) and increases with additional amounts of inbreeding. According to ARC (1993) economic traits that are most adversely affected by inbreeding are the ones that show the greatest response when crossbreeding is practiced. The greatest effects of inbreeding are seen in reduced survivability of piglets. In the male, the effects of inbreeding can cause reduction in sperm numbers and sexual aggressiveness. Inbreeding has also been found to delay onset of puberty by several weeks (Pond and Maner, 1974, Gee, 1977). Inbreeding can therefore only be considered if it is practiced in a wide genetic base and accompanied by strong selection to remove unwanted reproductive effects (Rothschild, 1996).

2.1.5 Pig breeds in South Africa

The demand for lean meat by consumers has meant that improved pig genotypes with high lean growth potential been imported into the country. On the other hand, human population growth and urban expansion have resulted in an increased demand for animal products, further necessitating the need for these high-producing pigs. Registered South African pig breeds include Chester White, Duroc, Large Black, Large White, Hampshire, Hamline, Pietrain, South African Landrace, Welsh, Kolbroek, Domesticated pig, Feral pig and Robuster (ARC and NDA, 1997). However, only the most popular breeds used in South Africa will be briefly discussed below.

2.1.5.1 Large White

The Large White is the most popular breed in South Africa (ARC, 1993). It originates in the United States. Characteristically it has white hair and pink skin. Sows are prolific and give birth to large litters. The Large White breed is very popular for its excellent mothering and rearing ability of the females, which is combined with docility. In a study by Browne (1994) Large White sows produced more litters than the Landrace, Hampshire, Duroc and Large White and Landrace crosses although the results were only significant for the Hampshire breed. The Large White also had a high percentage of piglet survival at 21 days (Browne, 1994). Moreover, an F₁ breeding sow of Landrace X Large White has excellent mothering and rearing ability. Compared to other breeds in South Africa, the Large White has high performance figures and it is less susceptible to stress than the Landrace (ARC, 1993). However, according to Eusebio (1980) this breed has been found to sometimes experience problems of pale, soft muscles.

2.1.5.2 Landrace

The Landrace is the second most popular breed in South Africa after the Large White (ARC, 1993). It is said to originate in Denmark. Like the Large White, it has white hair and pink skin which is sometimes freckled (Eusebio, 1980). Well known for its superiority in maternal performance (Iowa Purebred Swine Council, 2002) the Landrace has excellent litter size, mothering ability and confinement adaptability. In a comparison study done by Browne (1994) the South African Landrace was superior to Hampshire and the Duroc in terms of number of piglets born alive and had the highest 21-day piglet weight compared to Large White, Duroc and Hampshire. Among all South African pig breeds, it is said to exhibit the best mothering traits, and has therefore been used largely for crossing with the Large White to produce F₁ gilts

(ARC, 1993). Studies in Iowa, USA, have shown that the Landrace excelled all breeds in carcass length (Iowa Purebred Swine Council, 2002). Its docile nature makes it easy to handle. However, the Landrace is reported to be more stress susceptible than other breeds in South Africa (ARC, 1993).

2.1.5.3 Duroc

The Duroc breed originates from the United States of America (Eusebio, 1980). However, the first imported Duroc pigs into South Africa were from Canada in 1980/81 (ARC, 1993). According to Browne (1994) the lack of sire breed in South Africa for three-breed crossbreeding prompted the importation of this breed, together with the Hampshire. The Duroc breed has a distinct characteristic rusty red colour, although other shades can vary from light to dark. Duroc pigs excel all other pig breeds in muscle quality (ARC, 1993; Eusebio, 1980) and are well known for their ability to grow faster on less feed (Iowa Purebred Swine Council, 2002). The Duroc's ability to display rapid growth rate, coinciding with efficient conversion of pounds of feed to pounds of red meat, has been found to be unequalled by any other breed (Iowa Purebred Swine Council, 2002). Duroc piglets are heavier at 21 days of age and have better survival than SA Landrace and Hampshire (Browne, 1994). Duroc pigs are well renowned for their strong bone development hence their extensive use as sire line. The Duroc pig's skeletal structure, which stand up in all kinds of environment, combined with natural leanness, produce a fast growing, efficient product that is acceptable to the retailer and the consumer (Iowa Purebred Swine Council, 2002). Duroc boars are reported to have good libido and rarely give trouble when mating (ARC, 1993). In South Africa, the Duroc has mainly been used for crossbreeding purposes. According to ARC (1993) the Duroc breed has almost negligible stress susceptibility when compared to other pig breeds and therefore its terminal offspring can be transported and marketed with lesser problems.

2.1.5.4 Hampshire

The Hampshire pig breed was also imported into South Africa around the same time as the Duroc in the 1980/81 (ARC, 1993). It originates from England (Eusebio, 1980). The Hampshire pig breed is characteristically black in colour, with a white belt around the body and the shoulders. The breed is mainly used for crossbreeding purposes as a terminal sire in commercial pig production (ARC, 1993). Hampshire sows give birth to large litters but compared to the white breeds (i.e. Large White and Landrace) it has average mothering and

rearing ability (ARC, 1993). A study by Browne (1994) showed that the Hampshire was inferior to all the breeds in the study (Large White, Duroc, South African Landrace (SA Landrace) and reciprocal crosses of Large White and Landrace) in terms of piglet survival at 21 days of age and number of piglets born. Sows and boars of Hampshire breed exhibit placid temperament. Hampshire boars generally do not have good libido and it is common for boars of this breed to lack sexual aggressiveness (ARC, 1993; Iowa Purebred Swine Council, 2002). However, F₁ boars of Hampshire and Large White possess good libido, convey heterosis to the offspring and produce carcass possessing a good percentage of lean meat. (ARC, 1993). According to Iowa Purebred Swine Council (2002) Hampshire pigs are productive, lean, meaty and profitable to raise.

2.1.5.5 Large Black

The Large Black breed used to be the most popular pig breed in South Africa in the past until the arrival of the white breeds. Originally from England (Whittemore, 1980) the Large Black was imported into South Africa in 1910 possibly from Europe. The breed is characterized by a black hair colour. It has tall and excessively fat body and droopy ears. The Large Black sow is well known for its excellent mothering and rearing ability and it rears large litters. It is almost stress-free and shows symptoms of MMA-syndrome (ARC, 1993). Today the breed represents a small fraction of the total pig population in South Africa (ARC, 1993). However, according to FAO (2000) this breed is considered extinct in South Africa.

2.1.5.6 Robuster

The Robuster is a South African synthetic pig breed developed from crosses between Large White, Duroc and Belgian Landrace (Rossouw, 1998). White in colour, the Robuster was developed to have strong, well-boned legs, which may be used in sub-optimal and less extensive production units. It is also said to exhibit extra stress resistance (Rossouw, 1998). According to Rossouw (1998) the Robuster compares favourably with existing breeds in productive and reproductive performance in crosses with F₁ females, producing fast growing pigs of a high quality carcass type. In phenotypic evaluation studies for production traits by Rossouw (1998) the Robuster sired progeny showed superior growth rate (615.19 g/day) compared to the SA Landrace sired progeny (515.80 g/day). The Robuster sired progeny also out-performed the progeny bred from Large White, SA Landrace and Duroc in the production of high quality carcass (12.57 mm backfat and 69.37 % lean). The Duroc and the Robuster

three-breed crosses out-performed the other two breeds as far as reproduction figures were concerned.

2.1.5.7 Chester White

White in colour with a pinkish skin, the Chester White was imported into South Africa in 1983 from United States of America. However, it is claimed that the Chester White originated from the Yorkshire and the Lincolnshire in England (Eusebio, 1980). The Chester White is highly prolific and has good mothering and rearing ability, and as such it is mainly used as a dam line (ARC, 1993). The carcass quality is intermediate as it is shorter and contains more fat on average than other breeds (Eusebio, 1980). However, carcasses have been found to produce a good size of loin muscle and a high percentage of ham. Compared to the other white breeds, the Chester White has slower growth rate (Eusebio, 1980). Boars have good libido and are generally well built with well-developed bone structure. The Chester White breed is said to be virtually free of the stress gene (Iowa Purebred Swine Council, 2002).

2.2 Normal reproductive cycle and endocrinology in pigs

2.2.1 Reproduction in pigs

The economics of pig production is based on the ability of the sow to reproduce and to raise her piglets efficiently at the estimated cost and in good time (Eusebio, 1980). The pig is widely recognized as a non-seasonal polyestrous species, meaning that the pigs can reproduce at any time of the year and they are litter-producing animals (Dunkin and Taverner, 1996).

2.2.1.1 The female reproductive system

The reproductive system of a mature sow consists of paired ovaries weighing between 3 to 10g, oviducts between 14 to 30 cm long connecting the ovaries to a bicornuate uterus, which consists of two horns (40 to 110 cm long) and a 5 cm body (see Fig. 2.1). The body of the uterus is joined to the vagina by a muscular cervix measuring around 10 to 23 cm long and 2 to 3 cm in diameter (Dunkin and Taverner, 1996). The oviducts, uterus, cervix, vagina and the vulva are all part of the duct system of the female reproductive tract (Bearden and Fuquay, 2000). The ovaries are lobular owing to follicles in varying stages of development, and vary in weight from 3 to 10 g and in diameter from 2 to 4 cm. There may be 10 to 25 individual mature

follicles each 8 to 12 mm in diameter (Pond and Maner, 1974). The ovary is composed of an inner medulla and its outer shell, the cortex. The medulla is composed primarily of blood vessels, nerves and connective tissue. The cortex contains the cell and tissue layers associated with ovum and hormone production (Bearden and Fuquay, 2000).

The ovaries (female gonads) are considered the primary reproductive organs of the female because they produce gametes (ovum) and the female sex hormones (estrogens and progestins). The cow, mare and the ewe are considered monotocous because they normally give birth to one offspring per each gestation period, therefore one ovum is produced in each estrous cycle. The sow on the other hand is polytocous, producing 10 to 25 ova in each estrous cycle and giving birth to several young each gestation period (Bearden and Fuquay, 2000). The ovaries in the sow are slightly larger than those in the ewe, and appear like a ‘cluster of grapes’ because of extensive follicle growth and associated *corpora lutea*.

2.2.1.2 The estrous cycle

Immediately after reaching puberty, gilts exhibit spontaneous ovarian and estrous cycles about every 21 days throughout their lives, interrupted only by pregnancy or lactation (Dunkin and Taverner; 1996, Hughes and Varley, 1980). The estrous cycle is characterized by periods of reproductive quiescence, lasting 18 to 20 days followed by short periods of sexual receptivity, having a mean duration of 53 hours (range: 12 – 72 hours) (Hughes and Varley, 1980). The porcine estrous cycle can be divided into 3 distinct phases, namely:

- 1) Di-estrus, which is the luteal phase
- 2) Pro-estrus, which is the follicular phase
- 3) and the estrus period, which is the period of sexual receptivity (Dial and Britt, 1986).

During the estrus, which is also known as the heat period, the female’s vulva becomes swollen and reddish, and this is accompanied by a discharge of mucus from the vulva. The estrus female will go in search of a boar and will stand close to the boar in readiness for mating (standing heat reflex). The mature ova are normally released from the ovary during the second half of the estrus period, normally within the range of 38 to 42 hours after the onset of estrus. Ovulation in the pig occurs 38 to 42 hours after the onset of estrus. It occurs within a period of 3.8 hours and with the range in ovulation rate of 10 to 25 ova (Hughes and Varley, 1980).

Immediately following ovulation, the walls of the ruptured follicle collapse, usually around the central blood clot and the diameter reduces to 4 to 6 mm. The granulosa cells of the ruptured follicle then undergo changes known as luteinization. Hypertrophy of these luteal cells results in rapid increase in the size of the collapsed follicle as it develops into a functional *corpus luteum* of 8 to 9 mm in diameter by day 7 of the cycle. Little morphological changes then occur until day 14 or 15 of the cycle. Assuming no fertilization occurred, there is a rapid decline in *corpus luteum* diameter to 6mm in 2 to 3 day period. This is associated with the complete breakdown of the lutein cells and the collapse of the accompanying capillaries (Hughes and Varley, 1980). The regressing corpora changes colour from pink to white scar tissue. Subsequently, these inactive *corpora lutea* further generate to small masses of scar tissue known as *corpora albicans* (Hughes and Varley, 1980). However, a sow cannot conceive if mating does not take place during the appropriate period of the estrus cycle. Morphological changes in the ovary are responsible for hormonal changes in the peripheral circulation during the estrous cycle (Dunkin and Taverner, 1996).

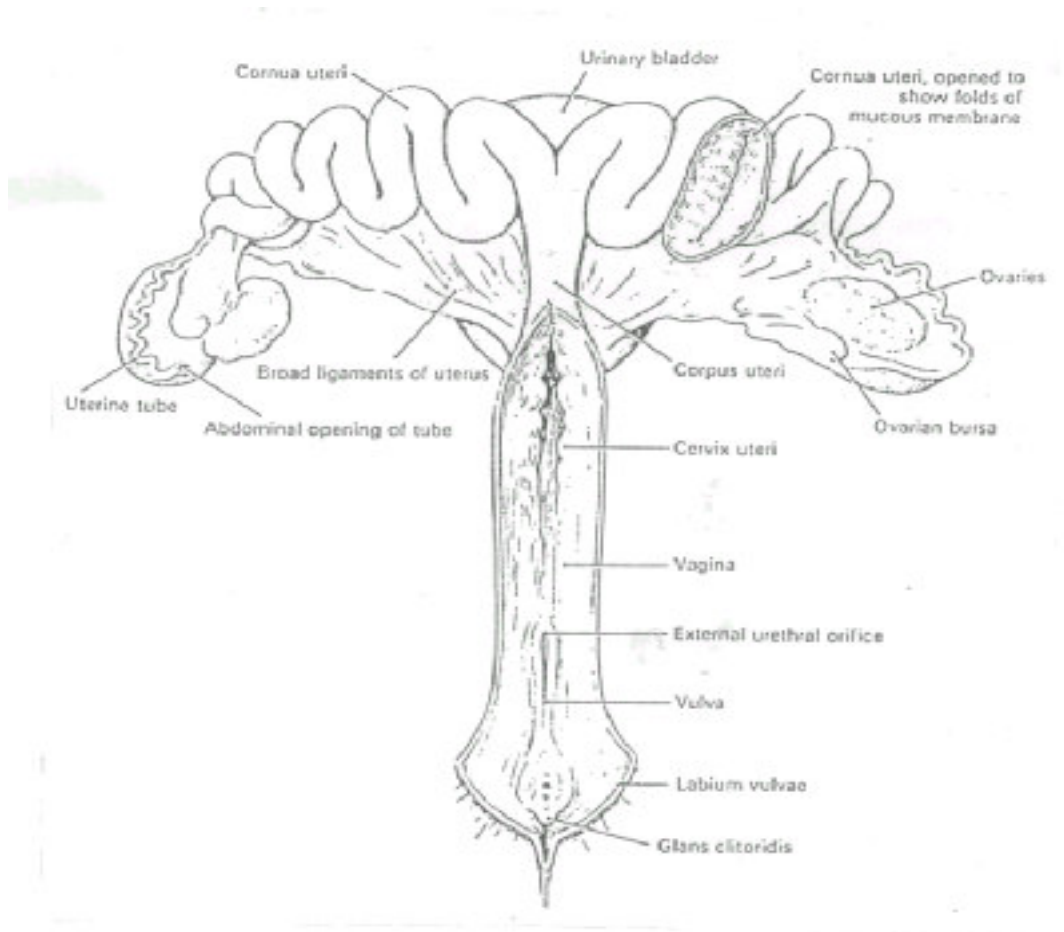
2.2.1.3 The male reproductive system

The male reproductive system consists of the scrotum, spermatic cords, testes, accessory glands, penis, prepuce, and the male duct system (see Fig. 2.2). The duct system includes *vasa efferentia* located within the testis along with the *epididymis*, *vas deferens* and urethra external of the testis. The testes are the primary organs of reproduction in the male (male gonads) just as the ovaries are in the female. They are the primary organs of reproduction because they produce male gametes (spermatozoa) and male sex hormones (androgens) (Bearden and Fuquay, 2000). The testes differ from the ovaries in that all potential gametes are not present at birth. Germ cells, located in the seminiferous tubules undergo continual cell divisions, forming new spermatozoa throughout normal reproductive life of the male. The testes also differ from the ovaries in that they do not remain in the body cavity. They descend from their site of origin (near the kidneys) down through the inguinal canals into the scrotum (Bearden and Fuquay, 2000).

The testis occupies a conspicuous position in the scrotum (Cole and Foxcroft, 1982) and it is attached to the vaginal process along the line of its epididymal attachment (Hafez and Hafez, 2000). The position in the scrotum and the orientation of the long axis of the testis differ with the species. For a mature boar, the testis is a large organ, about the same size as that of a bull

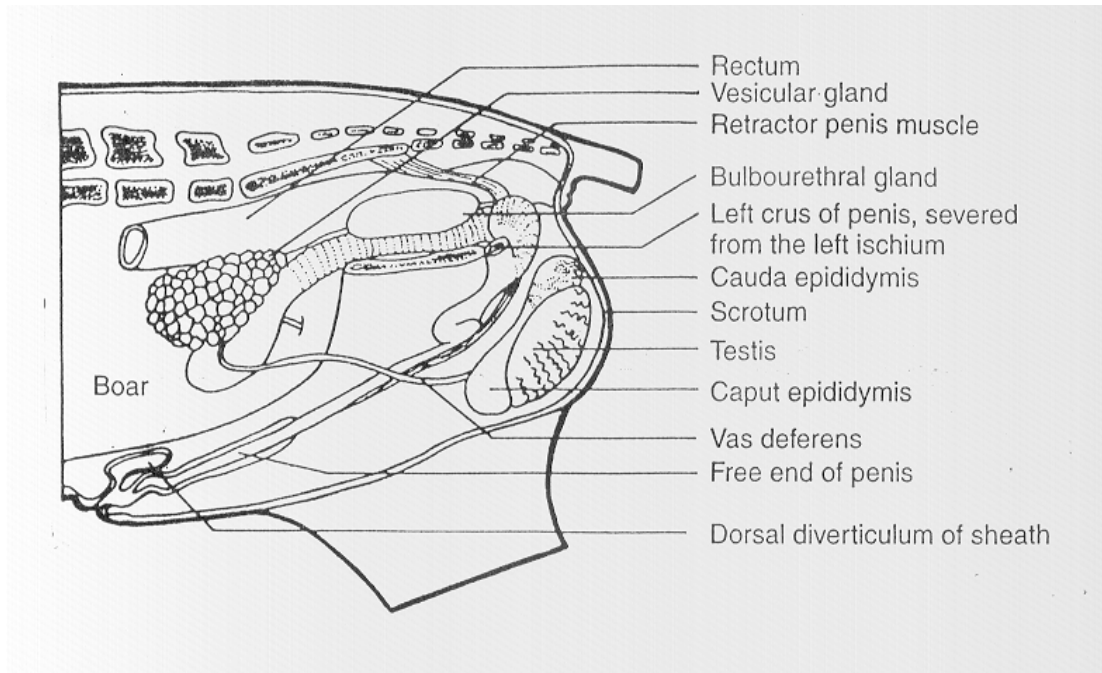
and weighs as much as 300 to 500 g. It is about 10 to 13 cm long and 5 to 6.5 cm wide (Cole and Foxcroft, 1982; Bearden and Fuquay, 2000).

Figure 2.1: The female reproductive system of a sow



Source: Pond and Maner, 1974

Figure 2.2: The male reproductive system of a boar



Source: Bearden and Fuquay, 2000

2.2.2 Endocrinology of the female pig

2.2.2.1 Endocrinology of the fetal and pre-pubertal gilt

In the pre-pubertal gilt, there is a gradual maturation of ovarian follicles during the later stages of gestation and the first few postnatal months. Tertiary (antral) follicles capable of endocrine function do not develop until 60 to 90 days of age, the age at which ovaries of pre-pubertal gilts can respond to exogenous gonadotropins. Normal circulation concentrations of estradiol remain relatively low from the time of birth until 150 to 210 days of age (Dial and Britt, 1986; Evans and O'Doherty, 2001). On the other hand, plasma progesterone concentrations of 2ng/ml was reported for female pigs of 90 days of age while the levels rose to 2.4ng/ml of plasma during the last month prior to puberty. This therefore indicates a gradual rise in progesterone concentration in the plasma from birth to puberty (Gee, 1977).

The maturation of the hypothalamus-pituitary axis is thought to regulate the rate of ovarian development through the release of gonadotropins. Circulating levels of Luteinizing hormone (LH) in the gilt are highest around time of birth and then decrease gradually until mid-puberty (Dial and Britt, 1986; Evans and O'Doherty, 2001). Basal plasma LH level of 0.8 to 1.6 ng/ml of plasma has been reported for pre-pubertal gilts, with its fluctuations becoming high during the luteal phase in mature female pigs (Gee, 1977).

In contrast, concentrations of follicle stimulating hormone (FSH) increase from the time of birth and remain elevated throughout pre-pubertal period. It has been reported that at 17 days of age, the pituitary contains a higher level of FSH and LH than during normal estrous cycle (Gee, 1977). As puberty approaches, there is an increased pulsatile release of gonadotropins (FSH and LH) that stimulate the progressive development of ovarian follicles (Dial and Britt, 1986; Hughes and Varley, 1980). According to Dial and Britt (1986) it is possible that both gonadotropins likely act in concert to promote follicular development and consequently steroidogenesis.

In addition to the changes that occur in the ovary, there are also synthesis and secretion of ovarian steroid hormones namely estrogens and progesterone through the process of steroidogenesis. These steroid hormones are produced by developing follicles and *corpora lutea* respectively (Hughes and Varley, 1980). Thus the output of estrogen reaches a maximum just prior to ovulation, when follicular growth is at its greatest. Progesterone, on the other hand,

attains a maximum rate of release once *corpus luteum* formation is completed (Hughes and Varley, 1980). Gonadotropin-stimulated increases in circulatory levels of 17β -Estradiol (an estrogen) triggers the pre-ovulatory release of LH that initiates ovulation at puberty (Dial and Britt, 1986). Estradiol has primarily negative feedback effects, but positive feedback develops as sexual maturation advances. Positive feedback of Estradiol stimulates the surge release of LH that is necessary for ovulation (Evans and O'Doherty, 2001). The pre-ovulatory surge of LH initiates many morphological and biochemical processes, resulting in transformation of follicle into a *corpus luteum*. In summary, fluctuations of LH concentrations are closely related to maturation of the reproductive tract and first ovulation in the gilts (Evans and O'Doherty, 2001).

2.2.2.2 Endocrinology of the pubertal and mature gilt

Circulating hormones originating from the pituitary, the ovary and the uterus mainly control reproduction in the female pig. These hormones that control the morphological changes occurring during the estrous cycle includes:

a) Ovarian hormones

The ovary secretes two steroid hormones: estrogen and progesterone which are produced by the growing follicles and the *corpora lutea* respectively. The estrogen levels are low for most of the estrous cycle but begin to rise on day 17 onwards to a maximum level on day 19 or 20. This rise occurs at the time when follicular growth and maturation are at their maximum and culminates in the pre-estrus estrogen peak. The circulating levels of estrogen are primarily responsible for the behavioral patterns and the vulval changes seen just prior to, and during the estrous period (Hughes and Varley, 1980). Following ovulation, the major sources of estrogen, the antral follicles are converted to the *corpora lutea* of the luteal phase of the estrous cycle. As these develop, they begin to secrete progesterone in increasing quantities (Hughes and Varley, 1980).

b) Uterine hormones

The uterus produces prostaglandin hormones ($\text{PGF}_2\alpha$ and PGE_2) which are involved in the estrous cycle. PGE_2 has luteotrophic properties while $\text{PGF}_2\alpha$ has luteolytic properties. Prostaglandin hormones are actually released from the uterus throughout the cycle but $\text{PGF}_2\alpha$ shows one distinct peak just prior to the end of the luteal phase. The

PGF2 α is then carried in the uterine vein and transported to the ovarian artery via a counter-current exchange system. This localized transfer of this hormone ensures that the hormone reaches the ovary in an active state as it would rapidly breakdown in the lungs if it is to travel to the ovaries via the general circulation (Hughes and Varley, 1980).

c) Pituitary hormones

The pituitary secretes three hormones that are of prime importance in the control of estrous cycle. These are LH, FSH and prolactin (Hughes and Varley, 1980). The LH secretion is at its minimum for the majority of the cycle, with only one significant peak of release which follows the pre-estrus estrogen peak, and it occurs just prior to ovulation. The FSH displays a slightly more erratic secretion pattern. The circulating levels of FSH are fairly low for most of the estrous cycle, but they show two distinct peaks around the time of ovulation. The first and smaller peak occurs at the same time as the pre-ovulatory LH peak. The second FSH peak is considerably larger and occurs on day 2 to 3 of the cycle (Hughes and Varley, 1980). It has been suggested that FSH may be involved in the selection of preantral follicles once ovulation has occurred or it works synergistically with prolactin to initiate the selection of preantral follicles from the germ pool (Hughes and Varley, 1980). During follicular phase, gonadotrophic stimulation causes an increased secretion of estrogen (Hughes and Varley, 1980). The LH and FSH are primarily responsible for the stimulation of follicular growth. However, during the luteal phase, the high circulating levels of progesterone prevent the secretion of large quantities of the gonadotropins (negative effect) up to day 16 of the cycle, thereby minimizing the follicular growth up to around day 16 (Hughes and Varley, 1980).

The secretion pattern of prolactin is less well known; however, it has been reported that a peak prolactin release occurs after the pre-ovulatory LH peak, followed by a second peak on day 2 of the cycle. Several other peaks of prolactin also occur during the estrous cycle (Hughes and Varley, 1980). Several *in vitro* studies have revealed that prolactin has profound effects on the luteal function during the first days of the porcine estrous cycle. Also, results from Ciereszko *et al.* (2002) indicate that prolactin may be involved in the regulation of the hypothalamic-pituitary-ovarian axis at the beginning of

the luteal phase of the porcine estrous cycle. Prolactin is regarded as the most important constituent of a luteotrophic complex in rodents and carnivores (Ciereszko *et al.*, 2002). In sows however, the luteotrophic role of prolactin is suggested to be limited to the early luteal phase of the estrous cycle and pregnancy. Moreover, prolactin is said to maintain progesterone secretion for a prolonged period (10 days) in hypophysectomised-hysterectomised gilts (Ciereszko *et al.*, 2002). The effect of prolactin on the developing follicles has been reported to be closely related to follicle size. In small follicles (1 – 2 mm) progesterone synthesis is inhibited while in larger follicles (>6 mm) progesterone production is stimulated (Gordon, 1997).

Results from *in vitro* studies and the existence of luteal prolactin receptors in the pigs indicate that prolactin may directly affect steroidogenesis. Several mechanisms may be involved in mediating the luteotrophic effect of prolactin in sows, including its action on the hypothalamus, pituitary and the ovary (Ciereszko *et al.*, 2002). The different effects of prolactin (i.e. stimulatory and inhibitory) are probably dependent on species, sex, reproductive status and prolactin concentrations in plasma or medium (Ciereszko *et al.*, 2002). Whittemore (1998) has also suggested that prolactin may be involved with prostaglandins in the breakdown of *corpora lutea* (luteolytic) a complete reversal of its role in *corpora lutea* support during pregnancy and lactation (luteotrophic). Upon luteolysis, prolactin levels fall, and thereafter with PGF₂α in attendance, rise during estrus.

Studies performed on several species including pigs revealed that prolactin receptors are widely distributed in the hypothalamus and/or the pituitary. Therefore, the direct or indirect action of prolactin on Gonadotropin releasing hormone (GnRH) neurons and/or gonadotrophes cannot be excluded. The indirect action could involve the opioid system since high prolactin concentrations in rats were suggested to induce a decrease in hypothalamus beta-endorphin content and opioids, in certain situations, inhibited GnRH or LH secretion in pigs (Ciereszko *et al.*, 2002). In pigs, prolactin has been found to affect plasma LH in three different ways depending on the physiology and reproductive status of the animal. For instance, in pre-pubertal pigs, plasma LH was enhanced, in boars inhibited while plasma LH was unaltered in late luteal and follicular phase of the cyclic sows. Therefore, it is difficult to explain the effects of prolactin on pituitary cells

without relation to the physiological and reproductive status of the animal (Ciereszko *et al.*, 2002).

2.2.2.3 Endocrinology of the estrous cycle

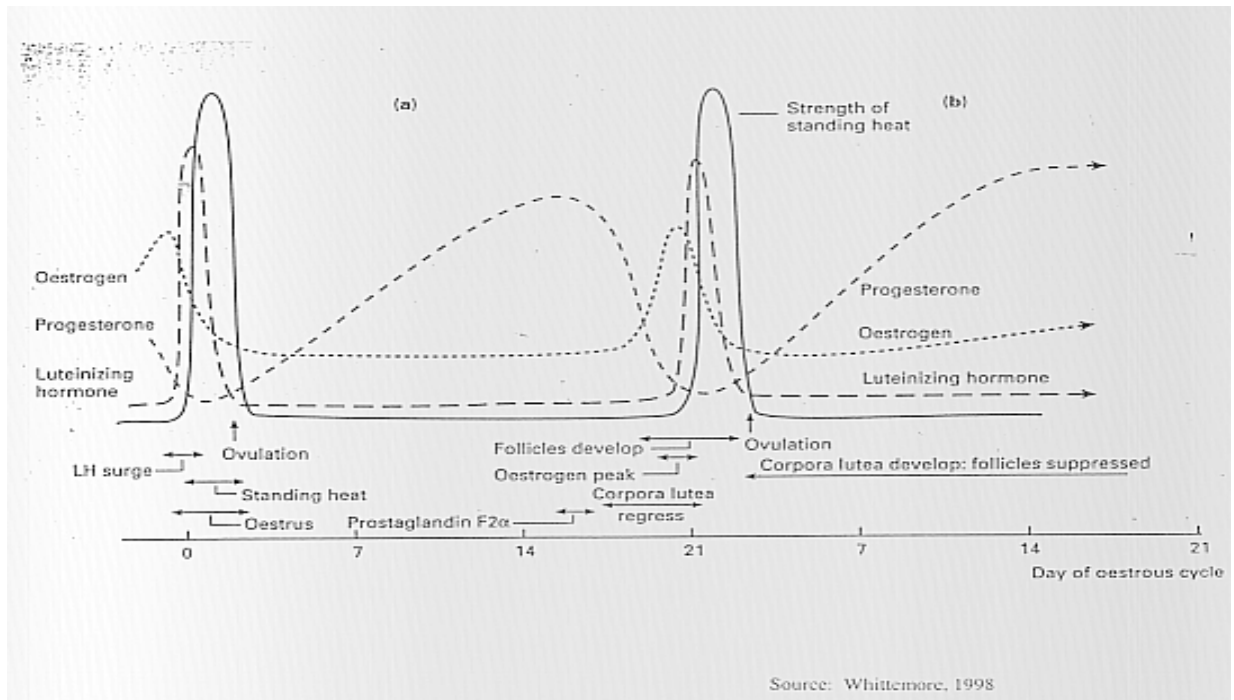
Estrus behavior is triggered by high levels of estrogens released by developing pre-ovulatory follicles in the ovary. During the luteal or follicular phase of the sow's estrous cycle, there are about 50 small follicles (2 to 5 mm in diameter) on the ovarian surface. Within day 16 to day 18 of the cycle, about 10 to 20 follicles begin to grow rapidly to greater than 8 mm in diameter (pre-ovulatory follicles). It is these follicles which secrete the rising levels of estrogen seen in plasma during the pre-estrus and estrus phases of the estrous cycle. Pre-ovulatory follicles are selected from the pool of the small follicles by a gonadotropin-dependent mechanism that is still poorly understood. It is believed that the stimulus for the development of pre-ovulatory follicles probably involves adequate FSH levels and increased secretion of LH (Dunkin and Taverner 1996). The rising level of estrogen secreted from the follicles build up to reach a threshold and act on the hypothalamus to increase the LH secretion through a positive feedback. Unlike in cows and ewes in which estradiol stimulates the LH surge by acting both on the central nervous system and the anterior pituitary, estradiol apparently stimulates the pre-ovulatory surge in pigs by acting primarily on the central nervous system (Gordon, 1997). The LH levels then increase to reach a pre-ovulatory peak about 8 hours after peak levels of the estrogen. Peak levels of LH cause changes in the follicle wall, which eventually result in the release of the potential ovum from the follicle in the process of ovulation (Dunkin and Taverner 1996; Gordon, 1997; Whittemore, 1998). Whittemore (1998) has suggested that the massive dose of LH stimulates the local intrafollicular prostaglandin secretion which initiates the rupture of the follicles (ovulation) thereby releasing the ova from the follicles.

Ovulation in the pig occurs 38 to 42 hours after the onset of estrus, occurring within a period of 3.8 hours. The granulosa cells of the ruptured follicle then undergo changes known as luteinisation. Hypertrophy of these luteal cells results in rapid increase in the size of the collapsed follicle as it develops into a functional *corpus luteum* of 8 to 9 mm in diameter by day 7 of the cycle. The luteal cells secrete increasing amount of progesterone resulting in a rise in peripheral levels of this hormone within day 3 to 4 (Dial and BeVier, 1986). Concentrations of progesterone increase linearly until a maximum is reached during mid to late di-estrus (Dial and BeVier, 1986; Gordon, 1997). The plasma progesterone levels have been recorded as

reaching a peak in the region of 25 – 35 ng/ml at about 8 to 10 days of the cycle (Gordon, 1997). After peaking, concentrations of progesterone may fluctuate dramatically in individual animals from day to day until they begin to decline coincident with luteolysis on days 13 to 15 of the cycle in case of no pregnancy. The lysis of the *corpora lutea* is accompanied by increased circulating levels of PGF2 α (Dial and Britt, 1986).

In the absence of a viable embryo in the uterus by day 11 or 12, PGF2 α from the uterus travels to the ovary by a countercurrent mechanism involving the utero-ovarian vein and the ovarian artery and causes the regression of the *corpus luteum* (Dunkin and Taverner 1996). Plasma progesterone concentrations decrease sharply after day 15, falling from around 30 ng/ml to 1 ng/ml or less within the next 2 days. Studies in Germany (Gordon, 1997) also suggested that in the process of luteolysis in pigs, *corpora lutea* are invaded by macrophages which produce tumour necrosis factor (TNF). This TNF then inhibits estradiol production and due to this inhibition, an indirect estradiol mediated luteotrophic effect of PGF2 α is prevented, and its direct luteolytic (i.e. its progesterone inhibitory) effect becomes dominant. It was also concluded that both TNF and PGF2 α act synergistically during the process of luteolysis in the pig. The *corpora lutea* regresses into avascular non-functional white structures known as corpora albicantia, which eventually disappear as scar tissue on the ovarian surface. The decline in progesterone levels which accompanies luteolysis is associated with the start of rapid growth of developing follicles and rising levels of estrogens which lead to behavioral estrus and ovulation again via the mechanism described above (Dunkin and Taverner 1996). Figure 2.3 shows the relative hormone levels of the estrous cycle (a) in the absence of pregnancy, and (b) during pregnancy.

Figure 2.3: Relative changes in blood concentrations of hormones in the estrous cycle, (a) in the absence of pregnancy, and (b) during pregnancy.



Source: Whittemore, 1998

2.2.3 Endocrinology of the boar

2.2.3.1 Endocrinology of the fetal and pre-pubertal boar

The testis is composed of two major tissue types, these being the Leydig cells and the seminiferous tubules. The primary function of these two tissues are, respectively, the synthesis and secretion of the male gonadal hormone testosterone (steroid hormone) and the development of mature spermatozoa from primitive germ cells (Hughes and Varley, 1980). In addition, the seminiferous tubules are made up of two basic cell types, these being germ cells (ranging in degree of maturity) and the supporting sertoli cells (Hughes and Varley, 1980).

During the late fetal and neonatal periods, there is a rapid differentiation of the leydig, sertoli and germ cell components of the testes. The differentiation is accompanied by an increase in steroid production. At birth, circulating levels of testosterone, LH and FSH are elevated and reach a maximum at about the second week of farrowing (Dial and Britt, 1986). Following this peak, concentrations of steroids and gonadotropins decrease until a second rise in testosterone levels is observed between 40 to 220 days of age. An increase in peripheral levels of estradiol lags behind but parallels the rise in testosterone concentrations. Estradiol is produced by either the sertoli cells of the testes from steroid precursors synthesized in the leydig cells or by the leydig cells itself (Dial and Britt, 1986). Testosterone is produced within the leydig cells. Estradiol is thought to act in concert with testosterone in the maturing boar to stimulate the development of the accessory sex glands and the onset of sexual behavior (Dial and Britt, 1986). After their postnatal decline, circulating concentrations of LH and FSH remain relatively constant throughout the pre-pubertal period. As the boar matures, there is a curvilinear increase in testicular size and weight, and presumably an increase in the steroidogenic tissue (Dial and Britt, 1986). In addition, there also appears to be an increase in the sensitivity of the leydig cells to gonadotropins during sexual maturation (Dial and Britt, 1986). Thus, circulating levels of testicular hormones increase during puberty even though mean serum LH concentrations does not change substantially as the boar matures (Dial and Britt, 1986).

2.2.3.2 Endocrinology of the mature boar

Androgens produced primarily in the leydig cells play a vital role in sustaining spermatogenesis, in supporting development of the penis and accessory sex glands, and in stimulating the development of secondary sex characteristics. Even though functionally very important, testosterone becomes quantitatively less important as the boar matures. The principal steroids found in the peripheral circulation of older boars are the 16-Androstenes (16-unsaturated C19 steroids). This class of steroids is responsible for the 'boar taint' or the musk smell that characterize the mature male. The compounds are the pheromones of the pig and can be used to facilitate the induction of estrus behavior when presented in the aerosol form to the females (Dial and Britt, 1986). The 16-Androstenes accumulate in the sub-maxillary salivary glands of the boar and are released into the saliva. Like those of the stallion, the boar testes secrete substantial amounts of estrogen. These estrogens are thought to originate from the aromatization of the androgens. Estrogens have been shown to enhance the behavioral response of castrated boars to testosterone (Dial and Britt, 1986).

2.2.4 Attainment of sexual maturity (puberty) in pigs

2.2.4.1 Puberty in the gilt

Puberty may be defined as that phase which links immaturity and maturity in animals. According to Dunkin and Taverner (1996) puberty in gilts represent the start of the animal's reproductive lifetime as it refers to the occurrence of the first behavioral estrus or period of sexual receptivity (Hughes and Varley, 1980) coinciding with first ovulation. Puberty occurs as the combined effect of genetic and environmental factors that contribute to its attainment. Once puberty has been reached, gilts will normally experience regular estrous cycles of around 21 days duration. Age at puberty is regulated by internal (e.g. breed, live weight, backfat depth) and management factors (e.g. nutrition, boar exposure, environmental factors) both of which are mediated via the endocrine reproductive axis (Evans and O'Doherty, 2001). However, with the recent trend towards rearing pigs in intensive confinement, the problem of delayed puberty in gilts has become apparent, and it is now recognized as an important component of economic loss in pig production (Esbenshade *et al.*, 1982).

Although puberty is an abrupt process in the gilt, the pubertal commencement of ovarian and estrus activity is preceded by gradual maturational changes in each of the components of the hypothalamus-pituitary-ovarian axis. These maturational changes commence prior to birth,

continue at differing rates and culminate in a series of endocrine events resulting in the onset of puberty (Dial *et al.*, 1986).

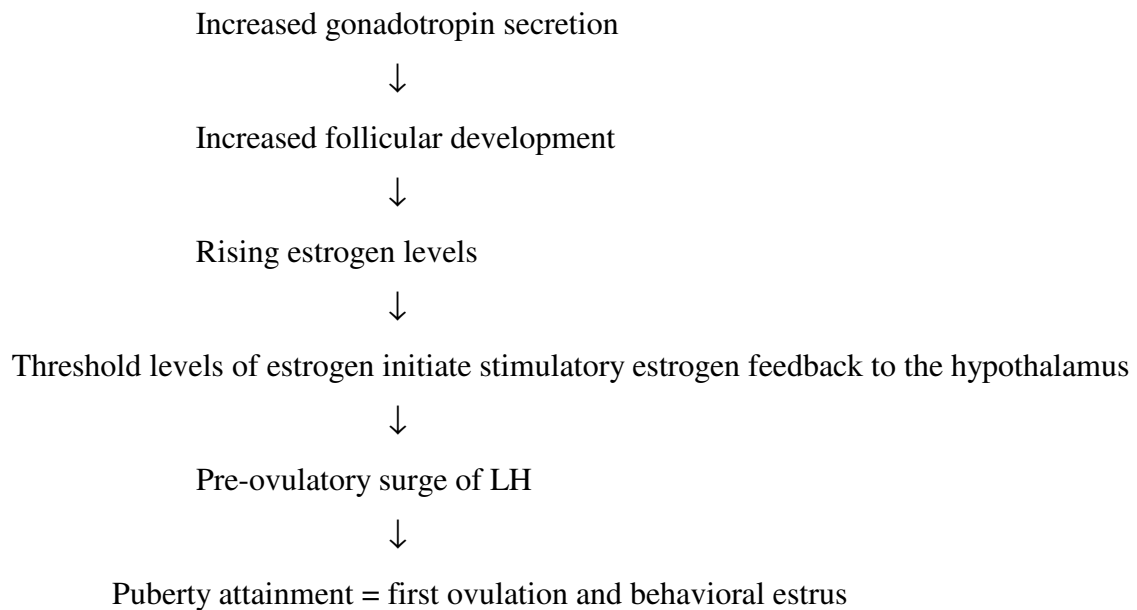
Gilts are anatomically ready for reproduction by 4 to 5 months of age, but puberty does not normally occur before 6 to 7 months of age (O’Leary *et al.*, 2002). This is accompanied by a series of physiological and behavioral changes culminating in receptivity to mating (O’Leary *et al.*, 2002). Domestic gilts can reach puberty from as young as 106 days to as old as 350 days, with the average age at puberty being around 200 to 210 days (Dunkin and Taverner; 1996; Hughes and Varley, 1980). Such variations in age at puberty are due to stimulatory and inhibitory influences originating in both the external and internal environments (Hughes and Varley, 1980). The number of ova released per estrus increases gradually over the first several estrus cycles. Studies have observed that there appears to be a block to physiological maturity, and hence puberty, possibly until a pre-determined age or weight is achieved. With this in mind, nutrition may have a part to play since the level of nutrition is a major factor controlling weight at any given age. Many factors such as genotype, season and social environment have been shown to profoundly affect the onset of puberty in the gilt and have been subject of many recent investigations (Dunkin and Taverner 1996).

Dramatic changes in the growth of the reproductive organs occur around the time of puberty in the gilt. Between the ages of 169 and 189 days, the mean length of the uterine horns has been reported to increase by 58 %, the mean weight of the uterus by 72 % and the mean weight of the ovaries by 32 % (Dunkin and Taverner 1996). These morphological changes result from changes in the secretion, metabolism and biological activity of the reproductive hormones (see Fig. 2.4). According to Dunkin and Taverner (1996) the following are possible triggers, which result in the initial rise in the gonadotropin levels:

- i) a reduction in the sensitivity of the hypothalamic-pituitary unit to the inhibitory effects of estradiol – the Gonadostat theory
- ii) a reduction in the metabolic clearance rate of estrogens,
- iii) increased gonadotropin releasing hormone (GnRH) from the hypothalamus
- iv) increased pituitary sensitivity to GnRH.

These changes, along with possible modifications in ovarian gonadotropin receptors, and/or steroidogenesis and LH pulse frequency, and/or amplitude changes may be taken as the central events in the initiation of puberty attainment in pigs (Dunkin and Taverner, 1996).

Figure 2.4: Endocrine changes occurring in the gilt around the time of puberty



(Source: Dunkin and Taverner, 1996)

2.2.4.2 Puberty in the boar

The attainment of puberty in the male is considered to have occurred once free spermatozoa have appeared in the seminiferous tubules and are present in the *cauda epididymis* (Hughes and Varley, 1980). However, Dial *et al.* (1986) defines the actual age of puberty in boars as the time at which a maturing boar is able to produce sufficient sperm to impregnate a female. Boars usually have spermatozoa in their testes at about 4 to 5 months and others at 8 to 9 months of age (Dial *et al.*, 1986).

While the onset of puberty is abrupt in the gilt, it is a more gradual process in the boar. Like that of the gilt, sexual maturation is a culmination of gradual maturational changes in each of the components of the hypothalamus-pituitary-gonadal axis (Dial *et al.*, 1986). The descent of the testis from the body cavity into the scrotal sac begins in the fetal boar around 60 days after conception, and is completed soon after birth (Dunkin and Taverner, 1996). Sexual maturity in the boar is a gradual process in which sperm production and sexual desire begin concurrently in increasing intensity beginning at about 4 months of age (Pond and Maner, 1974).

Testis weight increases markedly from the onset of puberty around 125 days of age to reach mature size at about 250 days of age. Testosterone concentrations in the peripheral plasma correspond to changes in testicular size, increasing sharply from around 125 days to 200 days of age. Estradiol levels also show a similar steady increase through pubertal development to maturity, whereas LH levels remain more constant over this period (Dunkin and Taverner, 1996). The development of spermatozoa (spermatogenesis) begins somewhere between the ages of 60 and 90 days of age and the spermatogenetic cycle requires 34.5 days for completion. The sperm then requires 10.2 days for transit through the *epididymis* and acquisition of fertilizing capacity (Dunkin and Taverner, 1996).

During the normal development of a boar, the immature spermatozoa first appear in the testis at three months of age. The first mature sperm are present at 5 to 6 months of age and the first ejaculation may occur at this time (ARC, 1993). According to Nalbandou (1976) as quoted by Young *et al.* (1986) the average age at which spermatozoa first appear in the seminiferous tubules of the domestic boars is 147 days. Others reported that mature sperm were ready to be shed into the lumen at a mean age of 154 days (Young *et al.*, 1986). However, Dunkin and Taverner (1996) reported that by the age of 140 days, there were sufficient sperm present in the

epididymis for fertile mating (2.5×10^9 mobile sperm). The epididymal sperm concentration increases further with age to 21×10^9 mobile sperm at 154 days and to 96×10^9 mobile sperm at 255 days at maturity. The age at which boars become capable of fertile mating varies between individuals (125 to 175 days or more) as does the development of sexual behavior. Mounting and erection often occur as early as 120 days, whereas coordinated mating behavior, including ejaculation, is not usually seen until after 150 to 160 days of age.

It has been reported that after puberty, the fertility of a boar still increases until the age of 12 months, which is the maximum reproductive potential (ARC, 1993). Mature size in boars is therefore not reached until about 1 year of age. Increases in total semen and sperm production accompany both the development of sexual maturity and the increase in body size until about 1 year of age (Pond and Maner, 1974). Unlike in females, age appears to be the prime factor governing the time at puberty in male pigs, and nutrition has little influence.

2.3 Genetic basis of reproductive cycle in pigs

2.3.1 Ovulation rate

The term ovulation rate is used to describe the number of ova shed at any particular estrous period. It thus represents the potential litter size of the pig from that particular estrous, although losses will subsequently occur due to failure of fertilization and embryo mortality. It is estimated that the ovulation rate is 35 – 45 % higher than actual litter size (O’Leary *et al.*, 2002). Ovulation rate is influenced by two main groups of factors, those which are intrinsic to the individual animal (such as age and genotype) and those which may be modified in the short-term (such as nutrition, climatic environment and exogenous hormones) (Hughes and Varley, 1980). The relative influences of both groups of factors are briefly reviewed below.

2.3.1.1 The influence of age on ovulation rate

The influence of age on ovulation rate may be considered at 3 categories. These are chronological age, sexual age (i.e. heat number) and parity (Hughes and Varley, 1980). Most reports suggest that ovulation rate increases as chronological age of the female increases. However, most of the influence of chronological age is due to sexual age and therefore the influence of chronological age is relatively small (Hughes and Varley, 1980). In contrast, the

influence of sexual age on ovulation rate is well documented. Ovulation rate tends to be low at the pubertal estrus, increasing rapidly up to at least the 4th estrous period. However, the actual number of ova shed at any given estrus will also be influenced by such factors as breed and nutrition. For gilts, the mean number of eggs increases from 8 to 10 at the first post pubertal estrus with an average of 15 by the third estrus (Hughes and Varley, 1980). Moreover, ovulation rate increases with parity and multiparous sows will ovulate more than 15 oocytes (O'Leary *et al.*, 2002). Research has shown that there is a marked increase in ovulation rate over the first four parities, reaching peak at fifth parity (Whittemore, 1998) and reaching a plateau at about the sixth parity (Hughes and Varley, 1980). Once a plateau is reached, there is no apparent drop in the sow's ovulation rate, although litter size is likely to be reduced as a result of increase in embryo mortality (Hughes and Varley, 1980).

2.3.1.2 The influence of weight and body condition on ovulation rate

Since age and parity are usually associated with increases in body weight, it might be expected that ovulation rate would also be correlated with the weight of the animal. However, body weight is a manifestation of many interactions such as age, breed, nutrition and health status, and thus its association with ovulation rate is not an easy task. In addition, any observed influence of weight on ovulation rate may be due to absolute body weight or the dynamic effect of weight change (Hughes and Varley, 1980). An increase in ovulation rate of 0.73 ova for every 10kg of the sow's service weight has been reported (Hughes and Varley, 1980). However, the influence of body weight on ovulation rate may mainly be due to dynamic weight change. Research has indicated that ovulation rate increase when sows were fed high plane diet prior to mating (Hughes and Varley, 1980). However, since this effect was only seen in relatively thin sows, it seems likely that it was attributable to increasing body weight and condition.

2.3.1.3 Nutrition influence on ovulation rate

Effects of nutrition on ovulation rate are of considerable importance from a practical point of view. The present evidence indicates that the level of energy received by the female pig is the dietary factor primarily responsible for alterations in ovulation rate (Hughes and Varley, 1980).

However, under certain conditions, protein level may also exert some influence. Increasing protein content of the diet has been found to slightly increase the ovulation rate in pigs (White

and Wheeler, 1995). Under most nutritional circumstances though, the influence of protein level on ovulation rate may be considered minimal (Hughes and Varley, 1980).

The effect of energy on ovulation rate may be considered in 2 categories. These are the long term influences which relates to prepubertal, estrous cycle and weaning to remating nutrition and the short term influences resulting from manipulation of dietary energy content at or around the time of estrus (Hughes and Varley, 1980).

Long term effects – Gilts and sows that are on a high plane diet have been reported to have high ovulation rates. Hughes and Varley (1980) reported that gilts that were on ad-libitum feeding had higher ovulation rate at their first and second heats than similar gilts which were fed 70% of the ad-lib feed from age 70 days onwards. However, optimum duration of high energy feeding prior to estrus was 11 to 14 days (Hughes and Varley, 1980).

Short term feeding (flushing) – Two factors influence the flushing effect; the timing of increased feed intake and the basal level of feeding prior to flushing. Considering the fact that ovulation rate is normally in excess of requirements in sows, it therefore seems more likely that short term flushing will have little effect on the sow's ovulation rate. However, flushing may have beneficial effects on the ovulation rate of gilts as their ovulation rates usually low (Hughes and Varley, 1980). Gilts on low caloric intake will ovulate on average two oocytes less than their counterparts with high caloric intake in the seven days preceding ovulation (O'Leary *et al.*, 2002). In the gilt, effective ovulation is dependent upon an adequate level of body fatness, and a harmonized physiological system not stressed by under-satisfied demands for continuing body tissue growth. Reduced feeding levels will both diminish body lipid level and deny optimum muscle growth (Whittemore, 1998). In such gilts, increased feeding levels will improve upon the situation and help to lift what would otherwise been a depressed ovulation rate.

2.3.1.4 The influence of genotype on ovulation rate

Ovulation rate is greatly influenced by breed type of the pig. Like most of the reproductive traits, ovulation rate is of low heritability (about 10 %) (Hughes and Varley, 1980) but can be improved by genetic selection (Hughes and Varley, 1980; Whittemore, 1998; O'Leary *et al.*, 2002). In general, white breeds tend to be associated with higher ovulation rates than black

breeds (Hughes and Varley, 1980). Also, certain breeds of pigs such as the Chinese Meishan ovulate up to 40 % more oocytes than European breeds, indicating a genetic influence on ovulation rate (O’Leary *et al.*, 2002). Evidence from other studies indicate that ovulation rate of Meishan and the Western breeds was similar at puberty and early estrous cycles but diverged in favour of the Meishan as they underwent more estrous cycles and later parities (White and Wheeler, 1995). White and Wheeler (1995) have also suggested that uterine and fetal interactions may have a role in the prolificacy of the Meishan females. The differences in ovulation between breeds are thought to be due to inherent variations in hormone levels and/or ovarian sensitivity to circulating gonadotropins (Hughes and Varley, 1980).

Inbreeding and crossbreeding may also influence ovulation rate. Inbreeding has an adverse effect on ovulation rate, a reduction of 0.6 to 1.7 ova per 10 % inbreeding increase in pigs has been reported by Hughes and Varley (1980). On the other hand, crossbreeding may result in an increase in ovulation rate (White and Wheeler, 1995; Whittemore, 1998). In a study by White and Wheeler (1995) crossbred gilts had higher ovulation rate (17.3 ovulations) as compared to purebred Meishan gilts (14.2 ovulations) and purebred Yorkshire gilts (12.5 ovulations). The high ovulation rate in crossbred gilts compared to purebred Meishan and Yorkshire gilts could have been due to hybrid vigour. Rothschild (1996) also reported an increase of 2 – 4 % in conception rates and nearly the same for ovulation rate, 0.23 more piglets per litter at birth and 0.70 more pigs at weaning for crossbreds than for purebreds.

2.3.1.5 The influence of climatic environment on ovulation rate

The influence of climatic environment on ovulation rate can be considered at 3 categories; ambient temperature, daylength and season. Of these three, the influence of temperature seems to be the most significant one. It has been reported by Hughes and Varley (1980) that ovulation rate was significantly reduced as ambient temperature was increased. Ovulation rates of 14.2, 13.7 and 13.1 ova were observed as temperature increased from 26.7, 30.0, and 33.3 °C respectively. Since a rise in temperature of 6.6 °C resulted in 45 % reduction in food intake (Hughes and Varley, 1980) it is possible that the reduced ovulation rate observed in high temperature pigs could be due to reduced energy intake rather than ambient temperature *per se*. On the other hand, the effects of daylength on ovulation rate have been reported to be very minimal (Hughes and Varley, 1980). It has also been confirmed that increased daylength rather affects litter size positively, not through increased ovulation rate but rather through increased

embryo survival (Hughes and Varley, 1980). The effects of season on ovulation are still not clear, and studies on this are not certain since some have reported seasonal effects while others have reported none (Hughes and Varley, 1980).

2.3.1.6 The influence of exogenous hormones on ovulation rate

Research has shown that ovulation rate can be increased by the use of exogenous hormones. The most predominant hormones used for increased ovulation are a combination of equine chorionic gonadotropin (eCG) and human chorionic gonadotropin (hCG). These are normally given during the follicular phase of the estrous cycle in order to promote development of more follicles to ovulatory size at time of ovulation (Hughes and Varley, 1980). Administration of these hormones during the luteal phase has been found to be less effective and results in adverse side effects. The number of ovulations follows a typical dose response relationship to the amount of hormone injected. Although administration of these hormones can increase ovulation rate, it has been reported that litter size following such treatments is not significantly increased (Hughes and Varley, 1980). The reasons for this could be the inability of the uterus to maintain large numbers of embryos and induction of the ovarian secretion by the administered hormones.

2.3.2 Litter size

Litter size is a major factor limiting profitability in the pig industry. The potential litter size as indicated by ovulation rate is rarely realized and is estimated to be 35 – 45% higher than actual litter size (O’Leary *et al.*, 2002). Losses in potential litter size occur due to failure of fertilization, embryo and fetal loss during pregnancy and losses at parturition. Litter size is therefore considered to be the reproductive trait with the highest economic value in pig production hence the reason for it being recorded in most studies. Litter size and piglet weight at birth are not only important measures of the sow and the boar’s genetic capacity for reproduction, but also determine conditions for each piglet born (Rossouw, 1998). Although a large litter is the breeding goal for litter size, number of piglets born alive is the selection trait of choice in South Africa, as well as in many other countries. The motivation for choosing litter size at birth, instead of at weaning stems from the attitude that the number of piglet born is the upper limit set by the sow, thereafter it is the responsibility of the producer to keep the piglets alive (Rossouw, 1998). Like with other reproductive measures, genetic variability exists for average litter size among breeds. It varies from 4 to 16 piglets per litter. Moreover, genetic

variability within breed also exist, which suggest the possibility of genetic improvement in reproduction through selection (Rothschild, 1996).

Although litter size potential is mainly determined by ovulation rate, any limitation due to litter size (of which probably due to ovulation rate) is more likely to occur in the gilt than in the sow. This is because in most cases, the sow ovulates more eggs than she is capable of maintaining as viable embryos through to parturition. In contrast, ovulation rate may be a primary factor limiting litter size in the gilt. If ovulation rate is below the target ovulation of 12 – 14 ova, then litter size will be lower than 11 – 12 piglets (Hughes and Varley, 1980).

2.3.3 Litter birth weight and piglet survival

Despite the improvement in management over the years, piglet mortality is still a substantial problem in the pig industry (Leenhouwers *et al.*, 2002, Rydhmer, 2000). Birth weight is one of the most important factors influencing piglet survival. In general, prenatal and postnatal survival increase with increasing birth weight (Leenhouwers *et al.*, 2002). The low birth weight in piglets could be a result of inadequate nutrition in *utero* or due to low fibre number (Whittemore, 1998). Studies (Leenhouwers *et al.*, 2002) have shown that genetic differences in piglet survival are related to the degree of development or maturity of piglet at birth, as opposed to the progress of parturition or early neonatal piglet behaviour leading earlier post-partum ingestion of colostrum. Genetic improvement of sow productivity has mainly been focusing on litter size. However, an increase in litter appears to be associated with decrease in piglet birth weight and survival. Therefore in order to increase the number of piglets weaned per sow through genetic improvement, further reproductive traits of the sow including birth weight, average piglet weight at birth and 21-day litter should be included in breeding programs (Hermesch *et al.*, 2000). A study conducted by Browne (1994) showed some breed differences in 21-day piglet survival, the Large White out-performed the Landrace, Duroc and Hampshire while the Hampshire had the lowest 21-day piglet survival. However, other studies have reported that differences exist between breeds in fetal survival. A study by White and Wheeler (1995) showed that embryonic survival up to 20 days of gestation was highest in the Meishan and lowest for Yorkshire sows, while their previous studies showed that Large White sows were inferior to Meishan in 20-day fetal survival. This was an indication of existence of breed differences in fetal survival.

There is a positive genetic correlation between litter birth weight and average piglet weight at birth, which indicate that selection for leanness will also improve litter weight traits (Hermesch *et al.*, 2000). However, selection for increased litter size decreases birth weight with possible concomitant delayed maturation of the gut and the central nervous system (Leenhouwers *et al.*, 2002). Selection for lean tissue growth rate leads to heavier but less mature piglets at birth while selection for piglet survival leads to somewhat lighter but more mature piglets at birth (Leenhouwers *et al.*, 2002). Selection for piglet survival may ultimately lead to piglets that closely resemble piglets from genetically obese lines such as the Meishan (Leenhouwers *et al.*, 2002).

2.3.4 Litter weaning weight

Most factors including birth weight affects weaning weight, which in turn affect post weaning growth and growth to slaughter (see Table 2.3). According to Hoffman (2001) weaning piglets at a heavier weight will result in better growth. Research has also demonstrated that heavier weaning piglets attain market weight faster than lighter weaning piglets (Noble *et al.*, 2005). Although piglet growth rate can be regarded as a measure of the sow's reproductive capacity, the genotype of the piglets obviously influences their growth as well. For piglet growth, the sire could be expected to have larger genetic influence owing to its genetic capacity for growth, and may also be the case to suckling behavior (Rydhmer, 2000). Piglet growth is unfavourably related to litter size, but the genetic correlation is low, $r = -0.03$ (Rydhmer, 2000). Palatability and digestibility of feed are also of paramount importance for the young pig, both during the suckling period and crucial periods after weaning as these can affect the daily gain to slaughter by up to 30 % (Hoffman, 2001).

Apart from birth weight, the other two factors that have the greatest bearing on the weaning weight are sow milk production and creep feed. These two are the source of nutrients to the piglet as nutrition is essential to sustain growth. It has been reported too that differences in the composition of growth and its efficiency arise from changes in nutritional status (Lister *et al.*, 1976). The amount and composition of milk that a sow produces has been found to have a significant impact on the growth, health and development of her offspring. In swine, the volume of solids from the sow's milk accounts for 44 % of growth of suckling pigs (Bleck *et al.*, 2005). Another important factor in piglet growth is stress reduction. Unlike in ruminant growth, a young piglet cannot have compensatory growth. Therefore, if exposed to a period of

poor growth due to either nutrition, disease or growth check at weaning, then it will always be on the lower growth curve and never make up for the loss (Hoffman, 2001). According to Kemm *et al.* (1991) body type (lean and obese) sex and live weight of the pigs also influence the rate, composition and pattern of growth. Hence the absolute amount and pattern of feed intake is primarily dictated by the animal's inherent rate, pattern and composition (protein: fat) of growth.

Table 2.3: Effect of weaning weight on realized slaughter weight

Weaning weight (kg)	5	5.5	6	6.5	7	7.5	8	8.5	9
Daily weight gain (g/day)	720	725	730	735	738	743	747	752	757
Slaughter weight (kg)	87.90	88.91	89.91	90.92	91.92	92.92	93.94	94.94	95.94

Source: Hoffman, Porcus Dec. 2001

2.3.5 Estrus synchronization and superovulation

Hormones that have been used in attempts to promote follicular growth and induce ovulation in the pig include eCG, hCG, LH, FSH, GnRH and its synthetic analogs, PGF2 α and prostaglandin analogs and estrogens (Dial and BeVier, 1986).

2.3.5.1 Estrus synchronization

Because of intensive rearing conditions that typify the modern swine production, it is usually essential that females are bred and farrowed in relative synchrony. Therefore, the main principal reason for controlling the estrous cycle of the pig is to synchronize the onset of estrus in groups of females. This allows breeding females to be treated as groups rather than as individuals, thereby allowing same time mating and farrowing (Hughes and Varley, 1980). The two methods of synchronizing estrous are by delaying the onset of estrus (heat) or by prematurely inducing it. Estrus inhibition or delay is usually accomplished by extending the luteal phase of the cycle via the administration of progesterone or synthetic progestones. On the other hand, a variety of methods have been used to induce estrus during the cycle. These include treatments with eCG, hCG, FSH, GnRH and prostaglandins (Hughes and Varley, 1980; Gordon, 1997).

a) Delaying the onset of estrus (Estrus inhibition)

The easiest way which estrus and ovulation may be inhibited is by the suppression of gonadotropin release. In theory, this may be achieved by either maintaining the *corpora lutea* beyond the normal point of regression, by providing the equivalent *corpora lutea* via the administration of progesterone or synthetic progestins, or by administering compounds which simply inhibit gonadotropin release (Hughes and Varley, 1980).

i) Administration of estrogens

The ability to induce prolonged luteal phase (*pseudopregnancy*) followed by a timed luteolysis would be a valuable tool for timing the entry of gilts into the breeding herd. During the luteal phase of pigs, estrogens are very luteotrophic (Gordon, 1997). A prolonged luteal phase can therefore be established by the exogenous administration of estrogens, the signal for maternal recognition of pregnancy (Soede *et al.*, 2001). In normal pregnancy, porcine blastocysts are capable of estrogen synthesis as early as day 10 after conception and are the main source of the compounds in *utero* during early

pregnancy (Gregoraszczyk and Michas, 1999). An injection or oral administration of estrogen has been reported to prolong the lifespan of *corpora lutea* for several weeks (Gordon, 1997). Pseudopregnancy in pigs can be induced by the administration of a single dose of hCG at day 12 of the estrous cycle. Administration of hCG at day 12 of the estrous cycle resulted on elevated follicular fluid concentration of estrogen at day 13 and day 16 of the cycle. The duration of inter-estrous interval is affected by the dose administered, the timing of administration and duration of exposure to estrogen (Soede *et al.*, 2001). In a different study, administration of estrogen on day 6 was found to have no effect while administration on day 16 gave variable results. Estrogen administration on day 11 of the estrous cycle resulted on long delay of estrus, which occurred probably due to the prolonged life of the *corpora lutea* rather than just the suppression of gonadotropin release (Hughes and Varley, 1980). The average inter-estrous interval though could not be increased beyond 36 days in the study by Soede *et al.* (2001) while average follicle size at day 18 after 2 treatments of hCG was 8.4 mm compared to average size of 5.9 mm at day 17 after one treatment.

Several possible mechanisms for estrogen effect on *corpora lutea* have been suggested. Soede *et al.* (2001) have suggested that the mechanism involves the induction of follicular development and the endogenous production of estradiol. Hughes and Varley (1980) suggested that estrogen prolong the life of *corpora lutea* by stimulating the release of endogenous luteotrophic hormones such as prolactin. Alternatively, it is possible that exogenous estrogens emulate the role of embryonic estrogen by redirecting prostaglandin flow from the uterine vein to the lumen of the uterus (Hughes and Varley, 1980). However, Gregoraszczyk and Michas (1999) reported that estradiol may inhibit prostaglandin synthesis and modify the PGE₂: PGF₂ α ratio during establishment of pregnancy in pigs. Estrogen is thought not only to protect the *corpus luteum* by decreasing uterine PGF₂ α secretion during early pregnancy but also preferably increase secretion of PGE₂ over PGF₂ α (Gregoraszczyk and Michas, 1999).

The use of exogenous estrogens as a means of controlling the estrous cycle in pigs is however limited by two factors, the first factor is that it is only effective during a short period of the cycle and secondly it may result in the development of cystic follicles.

ii) Suppression of gonadotropin release

Attempts to suppress gonadotropin release have involved the use of progesterone or synthetic progestagens. The administration of these compounds provides the female with an artificial luteal phase, preventing any major release of gonadotropin. This is due to the negative feedback influence of progesterone on GnRH release from the hypothalamus (Hughes and Varley, 1980). Work by many researchers has shown that progesterone may suppress the onset of estrus. Most progesterone treated females have been reported to return to estrus 4 to 8 days following the withdrawal of the treatment. The fertility of these progesterone-induced estruses have been reported as variable, others have reported normal ovulation whereas others indicate sharp decline in subsequent fertility (Hughes and Varley, 1980). The incidence of cystic follicles has been reported to increase markedly in progesterone treated females (Hughes and Varley, 1980).

Another approach to the suppression of gonadotropin release has been the use of synthetic progestagens. The compounds used include methylacetoxyprogesterone (MAP) chlormadinone acetate (AMP) and allyloestronol (A-35957). These compounds are administered at varying levels. The optimum treatment to sows and gilts appears to be a dose of 50 – 100 mg/day given over a 15 day period (Hughes and Varley, 1980).

Progesterones and synthetic progestagens suppress follicular development in cyclic females (Dial and BeVier, 1986). The parental administration of progesterone at doses of at least 100 mg/day for 14 consecutive days will delay onset of estrus and allow estrous synchronization (Dial and BeVier, 1986). However, most of the synthetic progestins have been found to have significant adverse effects such as the production of cystic follicles, the failure to synchronize onset of estrus tightly, the reduction of litter size and reduction in conception and farrowing rates (Dial and BeVier, 1986).

Although the use of progesterone and synthetic progesterones do facilitate the control of estrous cycle in the pig, it appears that their adverse effects on subsequent fertility will limit their likely use. These adverse effects are the high rate of cystic follicles and the reduced rate of fertilization (Hughes and Varley, 1980). The occurrence of cystic follicles following progesterone or progestagens treatment appears to be due to failure to

completely inhibit the release of gonadotropins during treatment. Therefore, it seems likely that progesterone or progestagens only inhibits the release of large quantities (surge-type release) of gonadotropins, while allowing small episodic releases to continue as normal during the luteal phase of a normal estrous cycle (Hughes and Varley, 1980). Thus, follicular growth does continue but rather slowly throughout the period of estrus inhibition. The result of this is that follicular growth continues beyond the normal ovulatory size and the follicle eventually becomes cystic.

b) Induction of luteolysis

During the estrous cycle in the pig, pulsatile secretion of PGF2 α (luteolytic) and PGE2 (luteotrophic) increase markedly from day 13 to 16 of the estrous cycle. However, due to their antagonistic functions, the concentration of PGE2 remains at least 3-times lower than that of PGF2 α (Gregoraszczyk and Michas, 1999). It has therefore been observed that the porcine *corpora lutea* remains unresponsive to the acute administration of PGF2 α or its analogs until 12 to 14 days after ovulation. Whittemore (1998) has reported that the sow's *corpora lutea* are insensitive to luteolytic factors such as PGF2 α until they are at least 10 days old. Prior to this, treatment with prostaglandins causes no decline or only a transient decline in circulating levels of progesterone (Dial and BeVier, 1986). As porcine *corpora lutea* typically commences its natural regression because of endogenously secreted prostaglandins 14 to 17 days after estrus, there is only a transient period during which exogenous prostaglandins are effective in initiating luteolysis in the pig (Dial and BeVier, 1986).

2.3.5.2 Superovulation

The other potential advantage of controlling the estrous cycle of the pig is that it facilitates the use of artificial insemination and superovulation, both of which can be important management aids. The gilt is first able to respond to exogenous gonadotropins at approximately 100 days of age. There is a gradual improvement in ovulatory response as puberty is approached, with optimal response occurring at 160 days of age or older ages (Dial and BeVier, 1986). To date, eCG has been the most commonly used and the most effective therapy in the induction of follicular growth, ovulation and estrus in pigs. Studies have shown that superovulation of prepubertal gilts can be induced with 250, 500, 1000 or 2000 IU of eCG followed by 500 IU hCG. The average number of ovulations in each of the above eCG treatments was 7.2, 12.5,

19.6, and 45.8 respectively (Shimatsu *et al.*, 2000). An increased dosage of eCG in prepubertal domestic gilts and sows resulted in an increased number of ovulations although there were individual variations. In a study by Shimatsu *et al.* (2000) the average number of ovulations in miniature pigs that received 1,2 and 3 vials of product PG600 (made of 400 IU eCG, and 200 IU hCG) followed by 500IU hCG was 20.8, 36.7, and 55.8 respectively. The eCG has also been reported to promote follicular development by inhibition of granulosa cell apoptosis. Follicular atresia in the pig, as in other animals (sheep and cattle) is characterized by apoptosis of large numbers of granulosa cells (Liu *et al.*, 2003).

The split-dose and combination treatments appear to induce comparable rates of ovulation and estrus (Dial and BeVier, 1986). It has been observed that when eCG is given without hCG, ovulation rates and the percent of gilts showing estrus are reduced relative to treatments utilizing hCG in conjunction with eCG (Dial and BeVier, 1986). The time of ovulation following treatments with eCG/hCG combination is approximately 110 – 120 hours. When either hCG or GnRH is given following eCG, ovulation occurs at approximately 40 – 44 hours following second injection. Although conception rates of 40 – 90 % are common following gonadotropin induced ovulation, less than 60 % are typically able to maintain successful pregnancies. It is thought that in some gilts, induced *corpora lutea* are not able to retain their viability 20 to 30 days after ovulation (Dial and BeVier, 1986) hence the return to estrus.

The value of exogenous estrogens and progesterones in terms of increasing litter size is however minimal, although research has shown that there may be beneficial effects when treating sows with a history of low litter size. The effect of these treatments is also influenced by the stage of estrous cycle where they are administered. Administration of eCG to gilts during the follicular phase of the cycle was found to result in an estrus of normal fertility 3 to 4 days later. On the other hand, the same treatment during the luteal phase may not result in spontaneous ovulation, and estrus exhibition is often suppressed (Hughes and Varley, 1980). In addition, if AI is carried out at this induced estrus, fertility is generally low, with frequent abnormalities in fertilization and transport occurring. These adverse effects are attributable to the high circulating levels of progesterone apparent in gilts treated with gonadotropins during the luteal phase of the estrous cycle (Hughes and Varley, 1980).

2.4 Relationship between growth and gonadal development in pigs

2.4.1 Growth in pigs

Growth may be defined as an increase in size. However, this is a crude definition of growth as the lung may increase in size after filling with air yet it cannot be said to be growing. Young (1950) as quoted by Belt (1988) defined growth as “*the addition of material to that which is already organized into a living pattern*”. Inadequacies to this definition have been pointed out e.g., in many instances; material may be removed as rapidly as it is produced with no resultant size increase. There may also be no change in total size but only in the arrangement of the units making up the total e.g., cell constituent proportions (Belt, 1988). A better biological and more plausible definition of growth was proposed by Von Bertalanffy (1960) as quoted by Belt (1988). He defined growth as “*the quantitative increase of a living system which results from the prevalence of anabolism over catabolism*”. This definition is not far from that of Brody (1945) as quoted by Pond and Maner (1974) where he defined growth as “*the constructive or assimilatory synthesis of one substance at the expense of another (nutrients) which undergoes dissimulation*”.

The term ‘growth’, as applied to animal production is considered to be synonymous with increase in body weight. This increase in body weight is brought about by cell multiplication (as in prenatal cleavage) and cell enlargement (as in postnatal muscle growth) (Whittemore, 1998). As a part of cell enlargement, there may also be simple incorporation of material directly to cells (e.g. the inclusion of lipid to fatty tissue). Pond and Maner (1974) reported that most cell division (hyperplasia) in muscle is complete at birth or shortly thereafter and that most postnatal body growth is associated with increase in cell size. The reason for this could be that muscle fibre number is largely determined at the point of birth, so any observed animal growth in lean tissue postnatally would primarily be due to muscle fibre size (Whittemore, 1998). Early pregnancy nutrition has been implicated in influencing muscle fibre number while late pregnancy nutrition influences mostly muscle size (birth weight). Studies on longissimus muscles of pigs have indicated that most of the increase in cross-sectional area of the loin eye as the pig grows is related primarily to an increase in diameter of individual muscle fibres, rather than to an increase in number of fibres. From birth to 25 days of age, fibre diameter has been shown to increase by 100 %, but from 100 to 125 days of age, the increase is only 10 percent (Pond and Maner, 1974). Growth of cells is dependent upon the difference between

anabolic processes and catabolic processes. If a body increases in size, its surface increases with the second power to its length, but its volume and mass with the third power (Belt, 1988). Growth rate of animals is therefore not a function of time or age, but rather is a function of body size. Furthermore, growth rate is different in spatial dimensions. When growth of an organism is considered, time and body size are the only variables used (Belt, 1988).

The purpose of growth is to reach maturity and the impulsion for growth is from current mass, age and most importantly, nutrient supply (Whittemore, 1998). Two things are responsible for the maturation of animals: an increase in body mass, referred to as growth and change in body conformation referred to as development (Belt, 1988). Therefore growth is of quantitative nature and development is of a qualitative nature. The two are not independent of each other but are, in fact interdependent upon each other (Belt, 1988). Growth occurs through the accretion of bone, fatty and lean tissues in the body. It is a result of positive difference between continuous anabolic and catabolic processes associated with tissue turnover. On the other hand, development relates to changes in the shape, form and function of animals as growth progresses. For example, the blocky Pietrain pig types develop in a way that results in meatier hams and larger loin muscles, while fatty strains will develop in such a way as to be a different shape from lean strains

Growth, as indicated by increase in body weight can be expressed as absolute gain in given period or as relative gain. Absolute gain gives the average growth rate over time while relative growth rate declines steadily as the animal approaches maturity (Pond and Maner, 1974). Morphogenetic changes in a growing animal take place primarily by relative growth, where certain components increase at a higher or lower rate than others (Belt, 1988). In addition, relative growth is concerned with the ratio of growth rates in several components of the living system. Relative growth is represented by the ratio of gain of weight during a given unit of time to average weight of an organism during the time period (Belt, 1988).

2.4.1.1 Growth curves

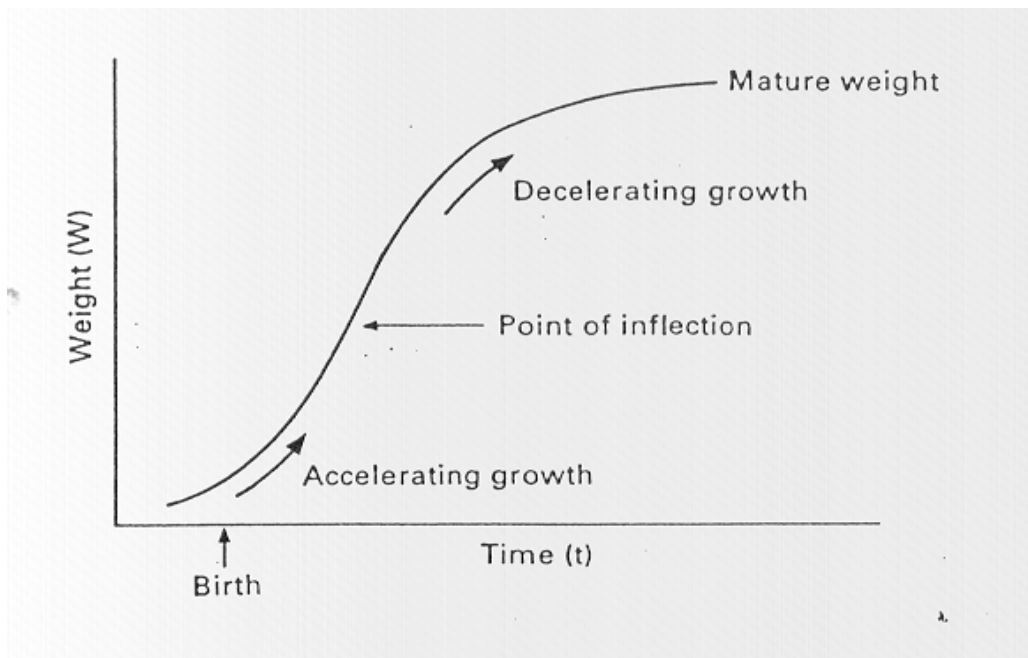
Given adequate environmental conditions, animals will increase their weight with increasing time as they grow to attain mature size. Typical growth curves are sigmoid in shape (see Fig. 2.5) and have the following characteristics:

- a) A self accelerating phase from time of birth

- b) A point of inflection where growth rate is maximum
- c) A phase where growth rate is self inhibiting (decelerating)
- d) A limiting value (asymptote) a mature weight towards which the growth curves tends (Brody (1945); Wilson (1977) as quoted by Belt, 1988).

Genetics, as well as environment, nutrition and pregnancy will affect the shape of the curve. Modern strains of improved pigs have been shown to have higher growth rates and greater mature size than unimproved strains. For individual pigs however, the shape of the potential curve is an intrinsic character, but genetic selection can be imposed on a population to artificially move intrinsic characteristics of the pig population (Whittemore, 1998). The slope of the curve describes the rate of increase in weight while the asymptote describes the mature size (Whittemore, 1998). Maximum slope or rate of growth (point of inflection) occurs at approximately one third of adult mass in most animals, except in humans where it occurs at two thirds of adult mass (Belt, 1988). In the Gompertz curve, the point of inflection occurs at 0.37 of mature size (Whittemore, 1998). The graph of the slope against time i.e. the first derivative of mass/time or dx/dt is approximately bell-shaped, but is not symmetrical. The asymmetry infers that there is little or no relationship between the cause of high growth rate at the onset and that of subsequent retardation in growth rate (Belt, 1988).

Figure 2.5: Typical growth curve showing response of weight over time



Source: Whittemore, 1998

In general, growth rate decreases during animal growth and eventually the animal reaches the constant adult state. All growth curves are similar in shape, any differences being accounted for by changes in the values of the equation parameters (Belt, 1988). Mathematical descriptions of growth by functions of this nature may be used to interpolate, and, if care is taken, extrapolate, but they have no theoretical meaning. Constants of these expressions have no physiological meaning (Belt, 1988). A collection of growth curve mathematical functions used in the descriptions are presented in Table 2.4 below:

Table 2.4: Mathematical growth curve functions and their owners

Growth function	Owner
$W = \exp [-\exp (-k (t - t'))]$	Gompertz, 1825
$W = A / [1 + \exp (-k (t - t'))]$	(Logistic) Robertson, 1923
$W = w \exp (\alpha t)$ $0 < t < a$	Brody, 1945
$W = A [1 - \exp (-k (t - t'))]$ $a < t$	Brody, 1945
$W = N/n - \exp [(-1-m) n (t - t')]^{1/(1-m)}$	Von Bertalanffy, 1957
$W = w_0 \exp [(A/k) (1 - \exp (1 - at))]$	Laird 1966
$W - w = (A - w) [1 - \exp (- (BC) (t - t') (1 - D/C) (1 - \exp -t/t'))]$	Parks, 1970

(Source: Belt, 1988)

Where W = live weight, t = time, k = constant. w_0 = weight at starting time, n = constant of anabolism, x = constant of catabolism, m = some power of weight W .

2.4.2 Organ growth

The body is the sum of its parts. Accordingly, its size may be determined by the innate limitations in the growth of one or more of its component organs. Yet, each part of the body is a predictable proportion of the whole, and their sizes may therefore be a function of the overall body mass (Lister *et al.*, 1976). There can be little doubt that the growth of organs is at least in part a function of the physiological demands impinging on them. However, it is equally obvious that such influences cannot account for the entire development of an organ. The relative sizes of organs have evolved as genetic adaptations to physiological needs. The growth rates of the different parts of the pig are not identical, the head and the shoulders have been found to reach maturity before posterior parts of the body. Similarly, individual organs and tissues grow at different rates, and the same applies to individual cell types within a single organ (Pond and Maner, 1974). Thus, in the evolutionary and developmental sense, it is true, as Brody has pointed out that ‘the organism changes geometrically, so as to remain the same physiologically’ (Lister *et al.*, 1976).

Growth of tissues, organs and of the whole organism occur in two phases, first there is the increase in number of cells (hyperplasia) and secondly an increase in size of cells (hypertrophy) (Pond and Maner, 1974). Immediately after conception, most growth is by hyperplasia. During late prenatal life and early postnatal life, the two phases occur concurrently. Finally, at some point in postnatal life, cell division ceases (except in some tissues) and growth occurs only by hypertrophy (Pond and Maner, 1974). The potential for an organ growth can be divided into four increments, which are:

1. **Basic size** – this represents that size which forms in pre-functional stages of development and persists after disuse and atrophy.
2. **Normal dimensions** – this equals the sum of its basic size plus such additional growth that may take place in response to ordinary physiological activities
3. **Compensatory hypertrophy** – this is the reversible adaptive growth caused by functional overload e.g. in the case on one kidney.
4. **Pathological growth** – this refers to excessive overgrowth so excessive as to be irreversible (Lister *et al.*, 1976).

Disuse of an organ almost always leads to atrophy, but almost never brings about the total disappearance of an organ or tissue (Lister *et al.*, 1976). Reproductive organs, like other parts

of the body grow in relation to the animal body size. Each component of the reproductive tracts of all farm animals grows in size relative to overall body size and undergoes histologic differentiation. Functional competence is not achieved simultaneously in all components of the reproductive system (Hafez and Hafez, 2000) indicating the different relative growth rates of the components. Thus in the bull, the capacity for erection of the penis precedes the appearance of sperm in the ejaculate by several months.

2.4.3 Follicular growth

It is well known that the mammalian ovary contains a huge number of non-growing oocytes, of which only a small number grow to their final size mature and are ovulated. During the mammalian reproductive life, 70 to 99 % of ovarian follicles, depending on the species undergo a degenerative process called atresia, and only a few ovulate (Liu *et al.*, 2003). There are two patterns of follicular growth in mammals. In one pattern, found in humans, rats and pigs, the development of ovulatory-sized follicles is suppressed except during the follicular phase of the cycle. In the other pattern, e.g. cattle, sheep and horses, development of follicles to ovulatory or near ovulation size is not confined to follicular phase, but occurs throughout the cycle (Gordon, 1997). The porcine and bovine oocytes grow from 30 μ m (not including the zona pellucida) to 120 μ m at maturity (Miyano, 2003). Oocyte growth accompanied by follicular development is a lengthy processes; the period from initiation of primordial follicle growth to final pre-ovulatory stage last 3 months in pigs and 6 months in cattle. It has been recently identified that oocytes play a key role in initiating their growth in regulating follicular development and oocyte growth by secreting certain factors (Miyano, 2003). The expression of these factors by oocytes begins in primordial or primary follicles in the mouse, the ovine and bovine ovaries.

In vitro studies in different mammalian species have revealed that oocytes growth and development of somatic cells of the follicles are highly coordinated, and the communication between oocytes and somatic cells is bi-directional (Miyano, 2003). The oocyte depends on surrounding granulosa and thecal cells, which support its growth and regulate the expression of meiosis. The oocyte also promotes granulosa cell proliferation and differentiation throughout follicular development (Miyano, 2003). Oocyte growth and follicular development are co-regulated through gap junctions between oocyte and granulosa cells and also co-regulated in paracrine fashion. It has been suggested that these paracrine factors secreted by the follicular

somatic cells regulate many important aspects of follicular development in mammals (Miyano, 2003). Theca cells in the pig provide substrate for production of steroids in granulosa cells as well as synthesizing estrogens and androgens. They in turn affect follicular maturation (Slomczynska and Tabarowski, 2001).

Growth of ovarian follicles, ovulation and formation of *corpora lutea* all involve dramatic changes in their cellular components. In the follicular phase of the pig's cycle, those vesicular follicles that are destined to ovulate grow from about 4 – 5mm diameter on day 15 to an ovulatory diameter of 9 – 11 mm (Gordon, 1997). The pre-ovulatory surge of LH initiates many morphological and biochemical processes, resulting in transformation of follicle into a *corpus luteum*. In most species, there is a decrease in both androgen and estrogen production when follicular cells are luteinized (Slomczynska and Tabarowski, 2001).

2.4.3.1 Influence of androgen on folliculogenesis

Androgen is one of the well-recognized regulators of folliculogenesis. It appears to act in three distinct ways; as an enhancer of follicular differentiation, as an inhibitor of folliculogenesis and as a substrate for estrogen synthesis (Slomczynska and Tabarowski, 2001). Therefore, cellular mechanisms through which androgen acts in the ovary are likely to be important in female reproductive physiology. Ovarian androgens may act locally to modulate follicular and luteal function in various species. The presence of androgen receptors in the granulosa cells in the ovary has been demonstrated in several species including humans, pigs, sheep, rats and chickens (Slomczynska and Tabarowski, 2001). Androgen may act through androgen receptors to modulate ovarian function, as androgens enhance FSH-induced progesterone and estradiol synthesis in granulosa cells (Slomczynska and Tabarowski, 2001).

Androgens synergistically enhance FSH-responsive genes, including cytochrome. They serve as a substrate once P450arom is induced by androgens and FSH. Aromatase activity consequently increases as follicles mature. On the other hand, androgens may also inhibit follicular differentiation and induce follicular atresia (Slomczynska and Tabarowski, 2001).

2.4.3.2 Influence of nutrition on folliculogenesis

Nutrition has been reported to influence follicular growth and maturation e.g. the percentage of small (1 to 3 mm) healthy follicles and the ovulation rate are decreased under feed restriction (Prunier and Quesnel, 2000a,b). It has been observed that feed restriction alters the repartitioning of healthy follicles between size classes. According to Prunier and Quesnel (2000a) the proportion of 0.4 to 1.0 mm healthy follicles to the total number of antral follicles is increased whereas the proportion of 1.0 to 2.9 mm healthy follicles is decreased in feed restricted lactating sows. This could explain the delayed return to estrus and the lower ovulation rate after weaning in lactating sows (Prunier and Quesnel, 2000a). Diameter, antral volume, and estradiol synthesis of the largest follicles were reduced in feed restricted pre-pubertal gilts. These effects occurred despite similar plasma concentrations of LH and FSH, suggesting that nutrition influences folliculogenesis, atleast in part, through gonadotropin-independent mechanisms (Prunier and Quesnel, 2000b). Nutrition may also influence the ovarian activity via other pathways. For instance, feed intake may affect the hepatic clearance rate of steroid hormones and hence the circulating concentrations of these hormones which, may act on folliculogenesis (Prunier and Quesnel, 2000b).

2.4.4 Growth curves in muscle nucleic acid and protein

2.4.4.1 Chemical components of growth

Chemical composition of the whole body of the growing pig varies with the lean:fat:bone ratio, but averages about 64 % water, 16 % protein, 16 % lipid and 3 % ash, with a small amount of (liver) carbohydrate (Whittemore, 1998). In pig production, the main objective is to increase lean growth while limiting fat deposition, which is a tissue with high-energy cost and low commercial return. Protein content is found to be a much more stable a proportion of the total body than fat content, the former usually range from 14 and 18 % while the latter ranges from 5 to 40% (Whittemore, 1998). Most of the variation in fat growth is due to nutrient supply, the more feed, the fatter the pig. Growth in animals can only occur if there is sufficient provision of nutrients. Therefore, this makes feed intake a prime determinant of the rate of weight gain, body composition and carcass quality in meat producing animals. Body weight and tissue gains result from the chemical components deposition rates and their localization. The effect of genotype and sex on daily gain of body components result in differences in growth rate (Quiniou *et al.*, 1999). Physical body components at slaughter results from the relative

development of body tissues and chemical components, which themselves are mainly determined by the appetite of the animals and/or nutrient supplies.

a) Protein deposition

Among chemical components, protein deposition appears to be a major component of growth (Quiniou *et al.*, 1999). Protein deposition is considered important because it controls both production efficiency and product quality. Lean tissue comprises of 70 – 75 % water, 5 – 15 % fat and 20 – 25 % protein. Mature protein mass of modern pig genotypes selected for fast lean tissue growth rate appears to be in the region of 35 – 55 kg (Whittemore, 1998). Therefore, this makes lean tissue mass a better measure of mature weight rather than the total mass which shows high variability due to the nutrition-dependant fatty tissue.

Under optimum breeding conditions, pigs are considered to deposit proteins at a level close or equal to their potential maximum level (PDmax). According to many studies done, PDmax would remain almost constant between 30 and 130 kg body weight and would then decrease (Quiniou *et al.*, 1999). It has been suggested that PDmax varied quadratically with body weight and that its maximum value depended on growth potential. However, in most studies, variation of PDmax did not exceed 20 g/day over the range of weights studied (Quiniou *et al.*, 1999). In literature, both PDmax and lipid depositions vary over a wide range of values and are influenced by the sex and genotype of the pig (Quiniou *et al.*, 1999). Castration has been shown to decrease PDmax and increase the lipid deposition through metabolic differences induced especially by androgen hormone secretion and circulation (Quiniou *et al.*, 1999).

Protein deposition is supposed to increase linearly with energy intake up to its maximum value (PDmax) and to plateau afterwards. In young pigs, the response of protein retention to feed intake is linear up to maximum appetite, while in slightly older pigs, protein retention reaches a plateau at higher levels of feed intake (Whittemore, 1998). The plateau occurs at the maximum growth potential for the animal and at high feed intakes, it may be attained relatively early in life. However, from a biological point of view, it has been argued that this broken line model is controversial and a curvilinear model would fit better to the biological processes involved in protein deposition

(Quiniou *et al.*, 1999). The slope for protein deposition curve is higher in lean type pigs than for fatter pigs which is due to a better efficiency of energy utilization for protein deposition in lean types of pigs (Quiniou *et al.*, 1999).

PDmax is reached at lower level of energy intake in leaner type of pigs than in fatter types of pigs (Quiniou *et al.*, 1999). In fact, differences in association or protein deposition slope and PDmax can be attributed to the amount of metabolisable energy for growth (MEg) required to express PDmax which depend on growth potential of the pig (Quiniou *et al.*, 1999). Although both lean and fat deposition rates increase with level of energy intake, the increase in lean gain was rather variable and ranged from 10 to 20 g per extra MJ DE intake. On the contrary, the fat gain change with energy was less variable and ranged from 8 to 12 g/ MJ DE (Quiniou *et al.*, 1999). The sex of the pig can also influence the rate of lean gain tissue. In pigs, the entire male has a much higher potential for lean tissue growth than either the female or the castrate pig. In Whittemore (1998) the lean tissue growth rates of entire males, females and castrate males were reported as 700, 600 and 500 g/day respectively.

b) Lipid deposition

Most pigs lay down two-thirds of their fat as external subcutaneous fat, and about one-third internally. Most of the internal laid fat is deposited as intermuscular fat and fat around the kidneys and intestines. Although subcutaneous fat is deposited differently between different parts of the body, the balance of fat in various fat depots is similar between young and old pigs (Whittemore, 1998). The most rapid phase of fat growth in the pig most likely occur in the first 4 weeks of life due to high fat gain potential in little pigs and high milk fat content (8 %) in the sow's milk. Fatty tissue contains 10 – 25 % water, 2 % protein and 70 – 80 % lipid (Whittemore, 1998).

In normal course of growth, pigs become fatter as they grow larger and the animal body shape is strongly influenced by the extent and the position of the fat cover (Whittemore, 1998). However, the point at which fattening begins is highly dependent on sex, genotype and feed level (Whittemore, 1998). Improved male pigs may contain no more than 12 % of lipid over the totality of a 10 – 110 kg growth phase, while unimproved castrates may readily achieve 25 % over the same growth increment (Whittemore,

1998). Unlike protein deposition, lipid deposition has been found to increase linearly with energy intake, but genotype and sex generally do not influence the increase in lipid deposition rate as energy intake increases (Quiniou *et al.*, 1999). Therefore at any given weight and sex, higher levels of feeding will increase the percentage of fat (Whittemore, 1998). The following nutritional conditions are those that will cause fattening to occur:

- 1) when the diet is imbalanced (at any pig weight) providing excess energy in relation to protein;
- 2) when feed intake exceeds the needs of maintenance and lean tissue growth (at any pig weight);
- 3) when, for sound physiological reasons, the body places fat accretion above lean accretion in its order of priority e.g. during pregnancy when preparing for lactation
- 4) when mature lean mass is achieved and ingested feed has no function to satisfy other than fat growth (Whittemore, 1998).

2.4.5 Growth as it relates to reproductive performance in pigs

2.4.5.1 Ovarian development in fetal and pre-pubertal gilts

At mid-gestation, all of the ova of the fetal gilt are concentrated in discreet areas of the ovary called egg nests. There is a progressive decrease in the percentage of egg nests as fetal age increases, until the egg nests are seldom observed after 20 days of postnatal age. Egg nests are replaced during mid to late gestation by undifferentiated primordial follicles, which will differentiate into primary follicles. During the period from birth to puberty, considerable growth and differentiation of the ovary occurs. Furthermore, it appears that ovarian growth is not uniform at this time: more follicles grow during the early pre-pubertal period and these follicles develop faster than in the older immature female (Hughes and Varley, 1980). Secondary follicles become differentiated at about time of birth and increase in number postnatally. Tertiary follicles do not form until 40 to 60 days of age (Dial *et al.*, 1986). Coincident with the development of antral follicles, is an abrupt increase in ovarian size. From about 70 to 110 days of age, the ovaries of pre-pubertal gilts undergo a period of rapid follicular development and become able to respond to exogenous gonadotropins with increased steroidogenesis and with ovulation. The number of small antral follicles (1 to 3 mm) increases linearly between 70 to 110 days of age, after which time, their numbers decrease. This decrease is accompanied by the rapid development of large antral follicles (>3 mm). These reach their

highest number at approximately 140 days of age and then remain relatively constant until puberty (Dial *et al.*, 1986).

From birth to 200 days of age, gilt ovaries increase markedly in size. The average ovarian measurements at one day of age are reported to be 6.00mm in length, 4.00 mm in width and 2.1mm thickness versus 24.9, 18.9 and 12.7 mm respectively at 210 days of age (Gee, 1977). The actual pattern of ovarian development varies considerably between species. For example, graafian follicles have been observed at birth in a heifer, whereas vesicular follicles do not appear until atleast 70 days after birth in the gilt (Hughes and Varley, 1980). However, irrespective of species, a full set of graafian follicles is produced by the pre-pubertal female in readiness for ovulation at puberty (Hughes and Varley, 1980).

2.4.5.2 Testicular development in fetal and pre-pubertal boar

Androgen-stimulated descent of testes from the abdomen commences at about mid-gestation and is completed shortly after birth. During the last weeks of gestation and the initial few weeks following birth, there is a rapid phase of testicular development that is attributable largely to leydig cell proliferation. At about one month of age, the leydig cells begin regressing and cause a decrease in the testicular weight. The negative feedback of the gonadotropin secretion by testicular steroids appears to be established at this age (Dial *et al.*, 1986). During the perinatal period, there is also an increased lengthening and coiling of the semeniferous tubules. The sertoli cells develop their morphologically distinct forms at about 40 days of age and form the blood-testes barrier at 110 to 120 days of age. At about 100 days of age, a period of rapid testicular growth commences. This testicular growth is due to tubular development and a substantial increase in the number of germ cells. After approximately 110 days of age, spermatogenesis is completed in many semeniferous tubules. By 120 to 150 days of age, there are often sufficient spermatozoa in the *epididymis* to impregnate gilts. Testicular size increases throughout the pre-pubertal period and continues to increase following puberty (Dial *et al.*, 1986).

2.4.6 Genetic effects on growth curves of pigs

In pigs, growth potential is influenced by numerous factors such as genotype and sex and stage of growth (Bastianelli and Sauvant, 1997). Certain breeds of pigs grow faster, because of this, mature live weight of different breed and strains of pigs may vary from 150 to 400 kg

(Whittemore, 1998). On the other hand, intact males grow faster than females and castrates. At any given weight and level of feeding, the intact male is considerably leaner than castrated male, with the female being intermediate. Their carcass lean percentages have been reported as 60 %, 56 % and 58 % respectively (Whittemore, 1998). In addition, growth rate and body weight composition are influenced by energy intake which effect differently between types of pigs and body weight range (Quiniou *et al.*, 1999). Growth rate depends on the amount of energy available for growth (MEg). Since maintenance requirements for energy (MEM) are always satisfied first, this means that MEg is affected by factors that influence MEM value or appetite. The MEM has been shown to depend on genotype, sex and stage of growth as well as unproductive energy requirements for thermoregulation, physical activity or stress (Quiniou *et al.*, 1999)

2.4.6.1 Protein and lipid deposition relationship

The combination of results on weight and chemical composition of body tissues shows that on average, more than 55 % of proteins and 80 % of lipids are deposited in lean and fat tissues respectively. However, the relationship 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 (Quiniou *et al.*, 1999). Genetically lean and obese pigs differ in their growth potential, hence in their protein and lipid deposition. On average, over 20 to 110 kg body weight range, 1 g of protein deposition is associated with 3.2 g and 2.7 g of lean gain in lean and fat types of pigs respectively. By contrast, 1 g of lipid deposition is associated with 1 g of fat gain in any breed (Quiniou *et al.*, 1999). 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. Therefore, as a consequence, it can be concluded that the increasing body fatness with body weight results rather from an increase in lipid deposition than from a decrease in PDmax (Quiniou *et al.*, 1999).

2.5 Factors affecting reproductive development in pigs and the physiological mechanisms mediating their effects on reproduction

Many factors contribute to the sexual development of pigs. These include internal (e.g. breed, live weight, backfat depth, etc.) and management factors (e.g. nutrition, boar exposure, and environmental factors) both of which are mediated via the endocrine reproductive axis. The influences of some of these factors on sexual maturity are discussed below.

2.5.1 Age, weight and growth rate

The weight and growth rates of pigs are usually a reflection and are influenced largely by genotype and nutrition. Because age, weight and growth rate are intimately related, it is difficult to distinguish their relative contributions to the onset of puberty (Dial *et al.*, 1986, Hughes and Varley, 1980). Nonetheless, there is a consensus among researchers that chronological age is a more accurate indicator of sexual maturity than weight or growth rate (Dial *et al.*, 1986). Robertsons *et al.* (1951) as quoted by Hughes and Varley (1980) have shown that although chronological age does not provide a definitive method of determining stage of maturation, it does however appear to be more closely associated with physiological age than is the body weight of the animal. Although age is considered an important factor in puberty attainment, it is however widely accepted that unstimulated gilts may reach puberty from as early as 170 days to as late as 260 days of age (Evans and O’Doherty, 2001). Part of the variability of age at puberty appears to be related to the genetic background of the gilts and to other aspects of the environment in which they are kept. Consequently, age is not considered to be an accurate predictor of puberty in gilts (Evans and O’Doherty, 2001).

According to Evans and O’Doherty (2001) gilts must reach a minimum weight of 75 kg before puberty can be attained. However, like age, great variability exists. Recently it was suggested that live weight on its own does not account for the induction of puberty, but contributes as one of the factors leading to puberty. Body weight is a measure of the combined components of the different elements of body composition, and consideration needs to be given to the fatness and leanness of the gilts if it is to be considered in relation to age at puberty (Evans and O’Doherty, 2001).

When body weight and gilt age are combined, then growth rate of the animal can be calculated. However, growth rate is considerably altered by nutritional status (Hughes and Varley, 1980). Brody (1945) as quoted by Hughes and Varley (1980) concluded that the attainment of sexual maturity is closely connected with the point of inflection of the growth curve. He further concluded that the level of early nutrition would alter the age, but not the weight at which the point of inflection occurs. However, Hughes and Varley (1980) reported that other researchers found this not to be the case in gilts.

2.5.2 Nutrition

Live weight and rate of growth are interrelated with the nutrient supply to the animal and, thus, it might be expected that both plane of nutrition and composition of the diet would influence puberty attainment in pigs (Hughes and Varley, 1980). In general, poor nutrition in livestock caused by inadequate, excess or imbalanced nutrient intake may adversely affect the various stages of reproductive development. The impact of poor nutrition on reproduction may range from delayed puberty, reduced ovulation and lower conception rates, through high embryonic to fetal losses and to excessively long post-partum anoestrous, poor lactation, high prenatal mortality and poor neonatal performance (Smith, and Akinbamijo, 2000).

Adequate nutrition during growth is required for normal development of reproductively competent females (Klindt *et al.*, 2001). According to Prunier and Quesnel (2000a,b) inadequate nutritional intake may influence reproductive performance of female pigs in various ways: it may delay puberty attainment and return to estrus after weaning or decrease ovulation rate and embryo survival. Nutrition may influence reproduction at the three levels of the hypothalamus-pituitary-ovarian axis via neuroendocrine pathways and/or variation in metabolic clearance of reproductive hormones. For instance, under-nutrition impairs the GnRH pulse generator while re-feeding restores the LH secretion (Prunier and Quesnel, 2000a). Theoretically, a nutrient deficiency or toxicity may affect a variety of organs and still results in non-specific decreased reproductive performance (Geoloff and Morrow, 1986). Deficiencies or toxicity may primarily affect the anterior pituitary or hypothalamus, thus interfering with normal LH and FSH production. Alternatively, other endocrine glands could be affected as in the case with iodine and the thyroid gland. Some other deficiencies and toxicities, and nutrient imbalances may have a direct effect on the gonads and genitalia. (Geoloff and Morrow, 1986).

This therefore can result in altered sperm development and survival in the male or decreased ovum or embryo survival in the female (Geoloff and Morrow, 1986).

Since weight and growth rate appear to have little influence on the onset of puberty, it might be expected that the nutrition of the gilt would similarly have an insignificant effect on the pubertal process. However, this is not always the case. Depending upon the study, level of feeding and composition of the diet may or may not influence the age at puberty. Studies on restricted feeding of gilts have been controversial. Some studies have reported that gilts on restricted feeding have delayed onset of puberty relative to their full fed counterparts, while other studies have noted no influence by plane of feeding on onset of puberty. Similarly, restricted energy intake was found to delay onset of puberty in some studies but in others, no influence was observed (Dial *et al.*, 1986, Evans and O'Doherty, 2001). Nonetheless, most studies agree that deficit in protein intake or amino acid imbalances can increase the age at puberty.

Nutrition of gilts during the rearing period has effects on subsequent reproduction, both in the short-term and in the long-term basis (Evans and O'Doherty, 2001). The plane of nutrition used in rearing gilts influences the age at which first estrus is shown (Evans and O'Doherty, 2001). Modern pig genotypes are extremely sensitive to modest reductions in feed intake, which can delay puberty by more than 3 weeks. It has been suggested that the decrease in fat in modern genotypes has partly resulted in gilts that are less able to deal with simultaneous demands of growth, pregnancy and subsequent lactation (Evans and O'Doherty, 2001).

The effects of under nutrition on reproductive efficiency of the female pig maybe related to physiological mechanisms acting at various points along the hypothalamic–pituitary–ovarian–uterine axis. They may be mediated nutrients, hormones or neuropeptides primarily involved in the control of nutritional function (Prunier and Quesnel, 2000a). Prunier and Quesnel (2000a) reported that in pre-pubertal intact gilts, as well as ovariectomised mature gilts, LH and FSH release after GnRH were higher in animals having the lower feed intake. Therefore these observations suggest that feed restriction inhibit LH release more than LH synthesis. This might result in an increase in the pituitary stores of gonadotropins in undernourished females. Therefore, inhibition of GnRH pulse generator system could be one of the mechanisms

explaining the effects of nutrition on reproduction in the female pig (Prunier and Quesnel, 2000a).

Since ovarian function is under the control of gonadotropin hormones, LH and FSH, any effect of nutrition on the hypothalamic-pituitary axis may have consequences on the folliculogenesis and ovulation (Prunier and Quesnel, 2000b). In addition, nutrition may influence the sensitivity of the ovaries to metabolic hormones through altering receptor numbers (Prunier and Quesnel, 2000b). Ovaries are relatively small organs whose contribution to the overall metabolic demand is low. However, they represent a very active tissue with permanent re-organization implying a high rate of cell proliferation, growth and differentiation. Therefore hormones which control nutrient uptake and utilization (e.g. insulin, GH, cortisol, thyroid hormone etc.) as well as cell mitogenesis and growth (e.g. insulin, IGF-I etc) are likely to influence ovarian activity (Prunier and Quesnel, 2000b).

In growing males, severe restricted feed intake has been found to delay puberty as well. Severe feed intake restriction has been reported to result in a reduction in the diameter of the seminiferous tubules of the testis as well as a reduction in its total growth. The effect is reversible by returning the animal to a normal level of feed intake (Pond and Maner, 1974).

2.5.3 Genotype (breed)

Breed and mating systems have long been recognized to influence puberty attainment in female pigs. Breeds such as the Landrace have been shown to reach puberty at a younger age than other breeds. On the other hand, certain Chinese breeds (e.g. Meishan) are well known to exhibit exceptional prolificacy and reach sexual maturity at an earlier age compared to western breeds. Several studies have indicated that increased prolificacy of the Meishan sows is primarily due to low levels of embryonic and fetal mortality (McCoard *et al.*, 2003) while White and Wheeler (1995) have suggested that uterine and fetal interactions may have a key role to play in Meishan prolificacy. Notably, Meishan pigs also farrow on average three or four more pigs per litter, possess more teats, and reach sexual maturity 60 – 90 days sooner than their European counterparts (McCoard *et al.*, 2003). Sun *et al.* (2002) reported that the Chinese Meishan pigs reach puberty at less than 100 days of age, have smaller placental size and larger litter size as compared to the British continental breeds. In a study by White and Wheeler (1995) Meishan gilts reached puberty at the youngest age of 103 days as compared to 223 days

of Yorkshire gilts, and reciprocal crosses of Yorkshire and Meishan were intermediate in age at puberty with 148 to 150 days. However, there is a considerable variation in the relative rates at which the other breeds achieve puberty. The variability between genetic lines and within a breed makes absolute ranking of the breeds difficult. Large differences between breeds in the age at puberty have been reported, with the average being greatest in Duroc gilts and lowest in Meishan gilts. Besides these differences between breeds, considerable variations also exist within breed. These variations maybe explained by variations in genotypes within breeds and in management regimes.

Heterosis on the other hand has got its influence on age at puberty. It has been observed that, because of heterosis, crossbred gilts generally reach puberty at an earlier age and have fewer non-reproductive days compared with their purebred counterparts (Evans and O'Doherty, 2001). Rothschild (1996) reported an average reduction in age at puberty of 14.3 days in crossbred gilts, an improvement which was due to hybrid vigour. Some insight into the possible mechanism which produce crossbreeding influences and between-breed differences in pubertal age were suggested by Dailey *et al.* (1970) as quoted by Hughes and Varley (1980). They reported that pituitary gland weight was significantly higher, at the same age and weight, for crossbreds than it was for purebreds. Thus, increased pituitary activity may well be responsible for earlier maturity in the crossbred gilt. A similar mechanism has been suggested to be operative to produce between-breed differences in age at puberty.

In summary, it can be said that breed influences age at puberty, but among commercial breeds, management factors can have a greater effect than breed on the onset of reproductive capability (Evans and O'Doherty, 2001).

2.5.4 Social environment and confinement

The environment in which gilts are reared can influence the age at which they commence estrus cyclicity. According to De Jonge *et al.* (1996) poor rearing conditions facilitate the development of social stress in subdominate pigs reared in group-houses. These subdominate pigs then develop physiological deviations associated with chronic stress exposure such as delayed onset of puberty, elevated saliva cortisol and a smaller live weight gain.

The current rearing systems of large confinement pig units also have not been without their disadvantages in pig reproductive performance. Rearing replacement gilts in environmentally regulated buildings has decreased the reproductive efficiency of swine operations because the incidence of delayed puberty has increased. It has been reported by Caton *et al.* (1986) that gilts raised in confinement were older at puberty than non-confined gilts, therefore, rearing gilts in confinement can cause a retardation of the onset of puberty. The age at which confinements starts to affect the gilts is unknown though (Caton *et al.*, 1986).

As pig production practices moves towards energy efficient housing that reduces or eliminates exposure to sunlight, gilts may not receive adequate exposure to light during pre-pubertal development. It has been reported that gilts exposed to supplemental lighting during decreasing day length but not increasing day lengths attained puberty at an earlier age than gilts that did not receive supplemental lighting (Diekman *et al.*, 1997). Numerous environmental factors such as light and space are often suggested as causes of poor reproductive performance in confined pigs (Caton *et al.*, 1986).

The number of gilts per pen and stocking density may also influence onset of puberty since it has been found that overcrowding may decrease the percentage of gilts showing estrus. Gilts maintained with a space allowance of 1m² per pig had significantly elevated free corticosteroid concentrations and lower proportions displayed estrus and were successfully mated compared with those pigs allowed 2 or 3m² of space. Restricted space also resulted in delayed growth in gilts (Evans and O'Doherty, 2001). While crowding may be a factor in controlling age at puberty, group size also appears to be an additional variable. Gilts housed in small groups (three or less) reached puberty later than gilts housed in larger groups (nine or more) at the same density (Christenson (1986) as quoted by Evans and O'Doherty (2001)). Gilts reared individually show delays in commencement of puberty (Dial *et al.*, 1986). On the other hand, restriction of movement in gilts by tethering during the late growing period has been reported to result in a delay of several days in onset of first estrus. The estrus period has also been reported as less recognizable in tethered than in non-tethered gilts (Pond and Maner, 1974).

As far as boars are concerned, it appears that the influence of rearing conditions on the attainment of puberty by the boar has not been well documented. However, it has been reported by Dial *et al.* (1986) that seemingly, boars reared in groups reach puberty at an earlier age and

demonstrate greater sexual aggressiveness as compared with boars raised individually (Dial *et al.*, 1986). According to Dial *et al.* (1986) pre-pubertal rearing experiences have no influence on the development either the testis or the reproductive tract of the boars. Contrary to what is commonly believed, rearing boars in confinement apparently has no effect on sexual development relative to boars maintained in pasture lots.

2.5.5 Exposure of gilts to the boar

The exposure of pre-pubertal gilt to the boar can accelerate the maturational changes leading to the onset of puberty. According to Patterson *et al.* (2002) the boar effect on gilts is largely due to the synergistic actions of the boar stimuli components, which are visual, tactile, olfactory and auditory. The presence of a boar has been shown to exert physiological effect on the female at breeding. Circulating oxytocin levels in the sow increase when sows are subjected to olfactory and tactile stimulation, which stimulate uterine and oviductal contractions (Patterson *et al.*, 2002). When gilts are exposed to boars at about 160 days of age, the majority of pigs commence estrus within 2 to 3 weeks. However, the exposure of gilts to boars at less than 140 days of age results in an increased interval from first boar contact to puberty and no advancement of age of puberty (Dial *et al.*, 1986). The introduction of gilts to a boar at >170 days of age causes gilts to reach puberty at an older age relative to gilts of 140 to 170 days of age. Gee (1977) also reported similar kind of results where the presence of a boar tended to decrease the age at first estrus (puberty). Gilts that were older when exposed to boars had decreased age at first estrus compared to those gilts that were younger when exposed to boars. It appears that boar introduction when gilts are approximately 160 days of age minimize both the interval from first boar contact to puberty and age at which gilts reach puberty (Dial *et al.*, 1986). However, it has been reported by Pedersen *et al.* (1997) that behavioural response of the gilt is of great importance for the stimulatory effect of the boar to be effective. Gilts showing fearful behaviour during boar stimulation were observed to have lower probability of reaching puberty within 10 days after the initial boar contact (Pedersen *et al.*, 1997).

In addition to induction of puberty in gilts, introduction of boars to gilts has been shown to result in decreased estrous cycles and increased ovulation rate (Turner *et al.*, 1998). While the increased ovulation rate could have been due to stimulation provided by direct physical contact with the boar, it is also possible that it could have been due to a direct effect on the hypothalamus-pituitary-adrenal axis on reproduction, since acute stress can stimulate

reproduction. Although it is not clear on the mechanism for the stimulatory effect of the hypothalamus-pituitary-adrenal axis on reproduction, it has been suggested that it may be due to a direct stimulatory effect of adrenocorticotrophic hormone (ACTH) on neurons that secrete GnRH (Turner *et al.*, 1998). The introduction of boar to gilts is stressful and therefore this can lead to stimulation of adrenal cortex to secrete its hormones.

The type of male exposed to gilts also influences the age of onset of puberty in gilts. Females that are raised with intact boars reach puberty earlier than when raised with barrows. Boars that are 11 months of age and above are more effective in inducing puberty in gilts than younger males. While tactile, auditory and visual cues from the boar may be involved in the pubertal response, pheromonal stimulation has an important role in stimulating puberty in gilts (Evans and O'Doherty, 2001). This is due to the fact that pheromones produced by the mature boar are potent sensory stimuli for the gilt (Dial *et al.*, 1986).

The timing of puberty in gilts is also markedly influenced by the age at which they come in contact with the boar. Therefore, for maximum boar stimulation, it is essential that gilts be reared out of sight and sound of boars until they are about 165 days of age. At this age, they are transported to a new location and are regularly exposed to a sexually active boar. This has the effect of bringing them to heat within 7 to 20 days (Evans and O'Doherty, 2001). The different methods of boar exposure (e.g. direct boar contact or fence-line boar contact) also affect puberty induction in gilts. In a study by Patterson *et al.* (2002) they demonstrated that direct boar contact is more effective in inducing puberty in gilts than fence-line contact. On the other hand, introducing the boar and removing it for some time is reported to be more effective than maintaining the boar in constant fence-line exposure. According to Dial *et al.* (1986) daily boar exposure for 30 minutes also appears to be just as effective and in some cases even more effective than continuous exposure for the induction of puberty. For optimal boar effect, gilts need to be exposed to a mature boar with high libido for one or more 30 minutes periods per day after 165 days of age (Evans and O'Doherty, 2001). Boar exposure to gilts increases their LH concentrations but the role of cortisol and the mechanism by which this occurs is unclear (Evans and O'Doherty, 2001).

A study by Turner *et al.* (1998) showed that introduction of gilts to boars resulted in significant transient increase in plasma concentrations of cortisol while there was no significant effect of

using back pressure test on plasma cortisol. It has also been found that females that were not sexually receptive had higher levels of plasma cortisol 15 minutes after boar introduction compared to those that were sexually receptive. From this it can therefore be concluded that while boar introduction to females may be sexually stimulating, it may also be stressful, particularly to those females that are not sexually receptive to the boar (Turner *et al.*, 1998).

In the pig, poor follicle development, failure to ovulate and ovarian cysts formation have been reported in association with elevations of glucocorticoids concentrations. Both hypothalamic-pituitary axis and the ovary are suggested as possible sites of glucocorticoid action (Viveiros and Liptrap, 1995). However, it has been suggested that the ability of the glucocorticoids to influence hypothalamic-pituitary axis may however be dependent on the stage of the estrous cycle. Viveiros and Liptrap (1995) reported that administration of glucocorticoids to sows during the luteal phase of the estrous cycle does not alter the pulsatile secretion of LH and FSH. However, plasma and follicular fluid estrogen levels are reduced and an increased percentage of follicles fail to reach ovulatory size, indicating a possible action directly on the ovary. The presence of cortisol binding protein in porcine follicular fluid and glucocorticoid receptors in ovarian cells also suggests the ovary as a possible site of glucocorticoid action (Viveiros and Liptrap, 1995).

Experiments have indicated that the effects of ACTH on the reproductive system are mediated by increased concentrations of glucocorticoids which results in altered follicular fluid hormone levels. The affected follicular fluid hormone balance may then have negative implications on subsequent follicular development (Viveiros and Liptrap, 1995). Glucocorticoids have been shown to directly influence granulosa cell differentiation as reflected by suppression of FSH-stimulated LH/hCG receptor expression and estrogen production but enhanced progesterone production. Treatment of sows/gilts with ACTH lead to elevated endogenous glucocorticoid concentrations prior to follicle recruitment, therefore leading to higher proportion of follicles with reduced number of viable granulosa cells. This therefore may account for the altered follicular fluid steroid hormone profile observed in stressed pigs (Viveiros and Liptrap, 1995). The dynamic changes in the follicle population during the luteal phase occur in the absence of detectable differences in pulsatility of LH and FSH, therefore suggesting that ovarian paracrine and autocrine factors play an important role prior to follicle recruitment. Any disruption in these factors, which include local steroid hormones, may interfere with subsequent follicle

development. It has therefore been suggested that the recruitment period maybe vulnerable to the exposure of high glucocorticoids hormone levels (Viveiros and Liptrap, 1995).

2.5.6 Exposure of gilts to an estrus female

Although not as effective as boar exposure, it has also been reported that gilts exposed to an estrual female resulted in induced puberty (Sterle and Lamberson, 1996). According to Sterle and Lamberson (1996) this effect has also been reported in mice and heifers where puberty was induced with introduction of mature female and cow respectively. It has been proposed that the female-induced stimulation may be caused by a combination of stress and pheromones; but the exact mechanism is not yet known (Sterle and Lamberson, 1996). In addition, the exposure of pre-pubertal boars to mature sows and gilts during the sexual maturation has been reported to improve post pubertal sexual behavior. Both sexually receptive and non-receptive females are able to promote the behavioral aspects of sexual development. However, the exposure to females appears to have a greater effect on mating behavior than exposure to other males (Dial *et al.*, 1986).

2.5.7 Climatic environment

Transition from one season to another encompasses changes in photoperiod, temperature and other environmental components. Each of these factors influences sexual maturity of the gilt. Although the domestic pig is known to reproduce throughout the year, there is seasonal decrease in its breeding performance. The domestic pig has been found in recent years to have pronounced seasonal breeding patterns. One manifestation of seasonal breeding patterns in pigs may be the timing of the onset of puberty. Gilts that farrowed in the fall have been found in some studies to reach puberty earlier than females born in the spring (Dial *et al.*, 1986; Andersson *et al.*, 1998). The adult females show a decrease in fertility and a diminished ability to return to estrus following weaning during summer. In domestic boars, steroid synthesis, sperm count and libido were reported to be lower in summer compared with the winter optima (Andersson *et al.*, 1998). Plasma testosterone levels of five months old boars were also found to increase as natural photoperiod decreases. One explanation of the delayed onset of puberty in spring-born gilts is that they reach pubertal age during the ‘non-breeding season’ and are thus less likely to commence ovarian cyclicity during the summer anestrus period. However, not all studies have observed seasonal changes in the onset of puberty (Dial *et al.*, 1986).

2.5.7.1 Effect of photoperiod on sexual maturity

Photoperiod is the amount of time the world is exposed to light. Several studies have shown that lighting regimes with 12 or more hours of daylight induce an earlier onset of puberty in gilts than when it occurs with daylight of 6 hours or less. Thus, long hours of daylight would appear to be conducive of the early maturation of the gilts (Dial *et al.*, 1986). Although optimal light: dark schedule for the advancement of puberty has not been elucidated, it appears that exposure to natural changes in photoperiod or to artificial photoperiods of at least 12 hours of light will allow for normal onset of puberty (Dial *et al.*, 1986). For the European Wild Boar (*Sus scrofa*) which is a short day breeder, photoperiod together with food availability are considered the most important environmental factors affecting the timing of reproductive events (Andersson *et al.*, 1998).

In farm animals that are photo-responsive i.e. ewe and mare, reproductive responses to photoperiod cues are mediated through the secretion of melatonin from the pineal gland. Since melatonin is thought to inhibit the synthesis and/or release of gonadotropins from the pituitary, this appears to provide a possible mechanism of action of light on reproductive function (Hughes and Varley, 1980; Whittemore, 1998). An increase in daily diurnal period of melatonin secretion is associated with a decrease in GnRH release and subsequent regression of the gonads in long-day breeders (e.g. horse) but results in an increase in GnRH and an activation of the gonads in short-day breeders (e.g. sheep) (Gerlarch and Aurich, 2000). Extended daylength would therefore result in a reduction of the inhibitory influence of melatonin on the production of gonadotropins. According to Diekman *et al.* (1997) the secretory patterns of melatonin during light: dark cycle have remained controversial in the domestic pig with some researchers reporting the absence while others reported the presence of nocturnal rise in serum melatonin. Gilts however have been found to respond to exogenous melatonin consumption which resulted in reduced age of puberty or overcame the seasonal inhibition of attainment of puberty (Diekman *et al.*, 1997).

2.5.7.2 Effect of temperature on sexual maturity

Elevated environmental temperatures are another aspect of the seasonal environment that can also delay sexual maturation of gilts. A study by Flowers *et al.* (1989) on heat stressed gilts reported increased rectal temperature, respiration rates and water consumption due to thermal stress on the heat stressed gilts. The elevated rectal temperature was a response to high

environmental temperature while the increased water consumption and respiratory rate were attempts by the heat stressed animals to compensate for elevated body temperatures. Plasma concentrations of progesterone were slightly (although not significant) higher in heat stressed gilts. The source of this additional production of progesterone in heat stressed gilts has not been documented fully but adrenal gland has been implicated (Flowers *et al.*, 1989). It is known that both estrogens and progesterone are produced by the adrenal cortex and that stimulation of the adrenal with ACTH causes an increase in production of these hormones. Therefore, severe stress may increase production of both estrogen and progesterone by the adrenal cortex (Gee, 1977; Bearden and Fuquay, 2000). In the study by Flowers *et al.* (1989) 18 control gilts reached puberty at 204.5 days whereas 16 heat stressed gilts reached puberty by 230 days of age. This therefore indicates that chronic exposure to elevated ambient temperatures may delay sexual maturation in female pig. In the adult female, elevations in environmental temperature cause a decrease in estrus activity as well as decreased farrowing rates and embryonic survival (Dial *et al.*, 1986). Although evidence is limited, the high temperatures that characterize the summer months may also adversely affect the initiation of puberty.

According to Flowers *et al.* (1989) ovaries in heat stressed females are capable of responding to gonadotropic stimulation, which could imply that heat stress may be delaying pubertal development in part, through suppression of FSH and LH. Results of this study showed that the delay could be four weeks or longer. The effect of elevated environmental temperature on puberty may be due to either reduced concentrations of gonadotropins, to decreased responsiveness of the ovary to these hormones or to a combination of both factors (Flowers *et al.*, 1989). Hence, the attainment of sexual maturation in gilts is subject to seasonal variation, as puberty is delayed in summer than in winter.

Thus, it seems clear that both environmental temperature and daylength (photoperiod) affect puberty attainment, and these two combines to produce seasonal effects. Increasing daylength results in the earlier attainment of puberty whereas increasing temperatures appears to delay the onset of sexual maturity. When considering the combined influences of photoperiod and temperature, it becomes apparent that spring-born gilts may be sexually activated by increasing the photoperiod and inhibited by decreasing temperatures. The seasonal influence on age at puberty of gilts in commercial herds may therefore be pronounced or absent, depending upon the environment within the building in which the gilts are housed (Dial *et al.*, 1986).

2.5.8 Management stimuli

When gilts of about pubertal age are removed from familiar environments, moved to the breeding herd and exposed to boars, estrus often occurs within a few days. Typically, 20 to 60 % of the pre-pubertal gilts show synchronous estrus within 2 weeks of movement, the effect of mixing and transport in this case inducing synchronous estrus (Gee, 1977; Evans and O'Doherty, 2001). In addition, transportation of gilt increases the response to boar stimulation, but transportation alone (not followed by boar stimulation) is insufficient to stimulate early estrous behavior (Hughes *et al.*, 1997 as quoted by Evans and O'Doherty, 2001); Gee, 1977). It is not clear what part the transportation, the change in environment, or the changes in social environment each play in the induction of puberty.

However, the procedures of transportation, relocation and regular boar exposure are stressful to gilts, and therefore stress is likely to be an integral component of the boar effect (Evans and O'Doherty; 2001, Hughes and Varley, 1980). Stress causes stimulation of the adrenal glands and it has both short term and long term effects on the animal (Gee, 1977). In the short-term effect, the result is an emergency or flight-and-fight response accompanied by increased sympathetic nervous activity, an increased output of epinephrine (adrenaline) and norepinephrine (noradrenaline) from the adrenal medulla. Consequently, there will be changes in heart beat, blood distribution, skin temperature etc. The long-term reaction to the stressors involves the release of ACTH from the pituitary, which in turn stimulates the adrenal cortex to release glucocorticoids. These changes are followed by a variety of secondary effects amongst which are increased corticosteroid levels, enlargement of adrenals, the development of gastric ulcers, catabolic changes leading to restricted growth or weight gain, increased susceptibility to diseases and interference in the reproductive processes (Gee, 1977).

2.5.9 Poor air quality

According to Evans and O'Doherty (2001) poor air quality delays the onset of puberty in gilts. When gilts were exposed to aerial ammonia concentrations of either <5 or 20 ppm from 10 to 40 weeks of age, greater proportion of gilts reared in the cleaner environment attained puberty by 26 weeks of age. In another study, gilts reared in an environment of 20 ppm ammonia attained puberty later than gilts reared in an environment of <10 ppm ammonia (Evans and O'Doherty, 2001). The mechanism whereby poor air quality delays the onset of puberty in gilts

is unknown, but it appears that odorous gases, such as ammonia may diminish the ability of the gilts to perceive olfactory cues from boars (Evans and O'Doherty, 2001).

2.5.10 Modern genotypes and body composition

According to Evans and O'Doherty (2001) animals that are selected for lower backfat grow bigger as compared to animals of small mature size, are leaner at the same body weight because they have a lower proportion of their mature body size, and are physiologically younger. As well as reduced backfat, voluntary food intake of selected animals has been reduced (O'Dowd *et al.*, 1997; Evans and O'Doherty, 2001). Based on measures of live weight, backfat thickness and average daily gain, it has been proposed that puberty occurs only after the attainment of a minimum level of leanness, fatness, or the ratio of fat: lean (Evans and O'Doherty, 2001). Genetic changes in body composition and appetites are associated with reduced reproductive performance in pigs selected for different components of efficient lean growth. Animals selected for low daily food intake and/or a high lean to food conversion ratio show a delay in age at puberty (Evans and O'Doherty, 2001) lower litter size, reduced milk yield and reduced longevity (O'Dowd *et al.*, 1997).

It has been suggested that puberty is delayed in extremely fast growing gilts because the increase in growth rate is primarily due to a more rapid accumulation of lean rather than fat tissue (Evans and O'Doherty, 2001). It has also been determined that a minimum threshold of 6mm in backfat thickness needs to be achieved for the attainment of puberty (Evans and O'Doherty, 2001). Hence, body fat and muscle content are associated with age at puberty, and there appears to be genotype specific minimum levels of body fat and muscle required for the onset of puberty to occur. More recently, the rate of fat deposition and/or protein deposition has been linked to physiological maturity and reproductive performance of gilts (Edwards, 1998 as quoted by Evans and O'Doherty, 2001) and this rate is suggested to be more a determinant for puberty than age, weight or boar exposure. (Evans and O'Doherty, 2001).

2.5.11 Induction of puberty by exogenous hormones

Attempts to induce puberty in gilts using exogenous hormones have involved the use of gonadotrophic stimulation. These include the eCG followed by hCG. The system is used because eCG is known to contain high concentrations of FSH, whereas hCG contains predominantly LH (Hughes and Varley, 1980; Whittemore, 1998). However, effectiveness of

these depends on the age of the gilt at the time of application. Puberty and subsequent cyclicity can only be induced when all the components parts of the hypothalamic-pituitary-ovarian system are approaching maturity, hence are able to respond to the stimulus (Hughes and Varley, 1980). An alternative approach to the induction of early puberty in the gilt is the use of exogenous estrogens. Such a system is dependent for its success on the maturation of the positive loop, by the administered estrogen (Hughes and Varley, 1980). Research has shown that early-synchronized estrus may be triggered in 60 % of the gilts when treatment is given at 140 days. Furthermore, the number of gilts responding to exogenous estrogens should increase as the gilts increase in their degree of maturity (Hughes and Varley, 1980). In conclusion, their application of these in stimulating puberty is of limited use since they depend on the female reproductive system (hypothalamus, pituitary, and ovaries) being ready for reproduction. They however can be used as a last resort when all methods fail to trigger puberty, and after the age of 9 months when puberty is long overdue.

CHAPTER 3

3. MATERIALS AND METHODS

3.1 Experimental animals

A total of 224 pigs (112 gilts and 112) of five different genotypes (see Table 3.1) from an ongoing growth study at the Agricultural Research Council – Animal Nutrition and Products Institute (ARC-ANPI) at Irene were used in this study. The five pig genotypes were obtained from five different commercial pig farmers in South Africa. The pigs entered the trial at an age of 9 – 10 weeks, and weighed approximately 25 – 30 kg.

Table 3.1: Experimental design showing number of pigs by genotype and sex in the two treatments

Genotype	Group housing (n = 224)	
	Gilts n = 112	Boars n = 112
Genotype 1	22	23
Genotype 2	22	23
Genotype 3	23	23
Genotype 4	23	21
Genotype 5	22	22

3.2 Housing

The pigs were housed in commercial type grower houses with temperature controlled self-opening curtains, and the temperature was maintained at about 20 °C. Eight pigs of the same sex were kept in a pen and each pen was equipped with a self-feeder and an automatic water nipple.

3.3 Management of the animals

The pigs were weighed on a weekly basis to determine their average daily growth rates. The backfat thickness of the pigs was measured using an ultrasound P2 Fat-O-meter during the weighing period. However, for the purpose of this study, only final P2 backfat thicknesses at slaughter and slaughter weights were used.

3.4 Diet

Animals were fed a diet that was divided into four phases. At the beginning of the trial (phase 1) a commercial grower diet containing 18 % crude protein (CP) 1.1 % lysine and 14 MJ/kg digestible energy and 10 % oxytetracycline at 2 kg/ton were fed for the first 14 days of arrival. Phase 2 diet was the same as phase 1 diet, but without oxytetracycline. This diet (phase 2) was fed until the pigs reached an average live weight of 65 kg. Thereafter (Phase 3) a diet containing 16 % CP, 0.9 % lysine and 13.5 MJ/kg digestible energy was fed until the pigs reached average live weight of 90 kg. From 90 kg live weight onwards (phase 4) the nutritive value of the diet was reduced to 15 % CP, 0.7 % lysine and 13.2 MJ/kg digestible energy, and this diet (phase 4) was fed until the last pigs were slaughtered. The four diets are shown in Table 3.2.

Table 3.2: Ingredient and nutrient composition of phase 1 to phase 4 diets

	Unit of measure	Diet 1 (Phase 1)	Diet 2 (Phase 2)	Diet 3 (Phase 3)	Diet 4 (Phase 4)
Ingredient composition					
Yellow maize	%	68.97	68.82	67.44	63.84
Soya bean oilcake meal (47 % CP ¹)	%	11.61	12.73	11.86	7.53
Sunflower oilcake meal (38 % CP ¹)	%	3.38	3.20	11.30	12.00
Fishmeal (65 % CP ¹)	%	7.99	7.29	-	-
Wheaten bran	%	5.00	5.00	5.00	12..35
Synthetic lysine	%	0.09	0.09	0.32	0.31
Synthetic methionine	%	-	-	0.04	-
Synthetic threonine	%	-	-	0.08	0.06
Monocalcium phosphate	%	0.94	1.01	1.68	1.61
Feed lime	%	1.18	1.20	1.49	1.52
Fine salt	%	0.24	0.26	0.39	0.39
Vitamin & Mineral premix	%	0.40	0.40	0.40	0.40
Oxytetracycline (10 %)	%	0.2	-	-	-
Nutrient composition (minimum requirement)					
DE swine	MJ/kg	14.00	14.00	13.50	13.20
Crude protein	%	18.00	18.00	16.00	15.00
Digestible lysine	%	0.90	0.90	0.81	0.71
Digestible methionine	%	0.31	0.31	0.29	0.25
Digestible TSAA ²	%	0.56	0.56	0.53	0.46
Digestible tryptophan	%	0.17	0.17	0.15	0.13
Digestible threonine	%	0.59	0.59	0.57	0.50
Calcium	%	0.90	0.90	0.90	0.90
Phosphorous	%	0.60	0.60	0.60	0.60
Available phosphorus	%	0.40	0.40	0.40	0.40
Sodium	%	0.18	0.18	0.18	0.18
Calculated Nutrient composition (as formulated)					
DE swine	MJ/kg	14.00	14.00	13.50	13.20
Crude protein	%	18.00	18.00	16.00	15.00
Total lysine	%	1.03	1.03	0.92	0.82
Total methionine	%	0.38	0.37	0.34	0.29

Total sulphur containing amino acids	%	0.67	0.67	0.63	0.57
Total tryptophan	%	0.22	0.22	0.20	0.19
Total threonine	%	0.71	0.71	0.68	0.61
Digestible lysine	%	0.90	0.90	0.81	0.71
Digestible methionine	%	0.34	0.33	0.30	0.25
Digestible TSAA ²	%	0.57	0.56	0.53	0.47
Digestible tryptophan	%	0.18	0.18	0.16	0.15
Digestible threonine	%	0.59	0.59	0.57	0.50
Fat	%	3.80	3.74	3.18	3.29
Fibre	%	3.00	3.00	4.21	4.65
Calcium	%	0.90	0.90	0.90	0.90
Total phosphorous	%	0.68	0.69	0.68	0.69
Available phosphorous	%	0.40	0.40	0.40	0.40
Sodium	%	0.18	0.18	0.18	0.18
Chloride	%	0.29	0.29	0.34	0.34
Linoleic acid	%	1.54	1.55	1.55	1.58

¹CP = Crude protein (N X 6.25)

²TSAA= Total Sulphur Amino Acids

3.5 Slaughter weights

The animals were slaughtered at different slaughter weights ranging from *ca.* 65 to *ca.* 148 kg, based on the assumption that animals will grow at an average daily gain of 850 g/day (see Table 3.3). The first slaughter date was on the 24th June 2002 at 116 days of age, then 130 days, 144 days, 158 days, 172 days, 200 days, while the last slaughter date was on the 30th September 2002 at 214 days of age. There were a total of 8 slaughter groups, which were slaughtered at Rietvlei (RTV) Abattoir in Benoni, Gauteng Province.

Table 3.3: Estimated slaughter information with an assumed average daily gain of 850 g/day

Slaughter group	Slaughter Date	Est. slaughter age (days)	Est. live weight (kg)	Number of days into trial	Est. carcass weight (kg)	Abattoir
1	24 Jun 2002	116	65	49	49	RTV
2	08 July 2002	130	77	63	58	RTV
3	22 July 2002	144	89	77	67	RTV
4	05 Aug 2002	158	101	91	76	RTV
5	19 Aug 2002	172	113	105	84	RTV
6	02 Sept 2002	186	125	119	93	RTV
7	16 Sept 2002	200	136	133	102	RTV
8	30 Sept 2002	214	148	147	111	RTV

3.6 Slaughter technique

In order to minimize the amount of stress on the pigs before slaughter, the pigs were sent to the abattoir at least 2 hours before slaughter. Minutes before slaughter, the pigs were sprinkled with cold water to clean the dirt off their coats and calm their nerves. The pigs were stunned by means of an electric stunner set at 220 volts, with a current flow of six seconds. The electrodes were positioned at the base of each ear. After stunning, the pigs were exsanguinated by severing the jugular vein on the neck with a knife and pushing it deep into the throat. After this the pigs were then shackled and hoisted, and scalding (in 60 °C hot water) commenced about 5 minutes after stunning. The pigs were scalded for 3 to 5 minutes as a preparation for dehairing and hoof removal. Dehairing was carried out in a special machine while hooves were then removed by means of a hook. After the scalding and scraping of hair and hoof removal, the carcasses were cleaned-up and subsequently opened by cutting longitudinally along the belly down to the larynx. To remove the testicles from boars, a cut was made along the central pelvic line from the penis to the anus, which leaves the testicles exposed. The skin is also cut along the inner side of the hind leg until these cuts meet the central cut. Testicles from each boar were then cut off and placed into labeled (with pig identity number) plastic bags. Abdominal organs (stomach, intestines, liver, heart, lungs, uterus and ovaries etc) were then cut free from the cavity walls down to the diaphragm, around which a cut is made through the tissue that

connects the diaphragm membrane. The gonadal organs (uterus and the ovaries) of each sow were then cut off from the abdominal organs and placed into a labeled (with pig identity number) plastic bag. The pigs' identity numbers on the vacutainer tubes and plastic bags were labeled using a permanent marker pen so as to prevent the numbers from fading before measurements on the organs can be performed. The carcasses were again washed and body development measurements like warm carcass weight, and P2 fat thickness recorded. Following this, the carcasses were hung in a cold storage room (4 °C) for at least 12 hours after which other body development measurements such as backfat thickness measurements, carcass length and chest depth were recorded.

3.7 Handling of gonadal organs

Gonadal organs from gilts and boars were collected and transferred to the laboratory at the University of Pretoria. Due to time constraints, samples were kept in the refrigerator at 4 °C overnight and measurements taken the following day.

3.8 Parameters measured

3.8.1 Testicular measurements

Before measurements were done, the membranes covering the testicles (the *epididymis*, the *tunica vaginalis* and the scrotal sac) were removed with a longitudinal cut to obtain only the testicle. Measurements recorded were:

- ◆ Testicular weight (left and right testis) – Each testis was weighed using a digital weighing scale measuring to one decimal place in grams
- ◆ Testicular lengths (left and right testis) – Testicular length of each testis was measured from the head to the tail of the testis using a vernier caliper. Measurements were made to two decimal places in centimeters.
- ◆ Testicular widths (left and right testis) – Testicular width of each testis was measured at the middle of the testis using a vernier caliper. Measurements were made to two decimal places in centimeters.
- ◆ Testicular volumes (left and right testis) – Testicular volume of each testis was measured by submerging the testis into a known volume of water in a 2000 cm³-measuring cylinder (measuring to the nearest 20 cm³). The difference in volume readings was taken as the volume of the testis.

3.8.2 Ovarian measurements

To take measurements on the ovaries, each ovary was removed from the oviduct using a knife, and then trimmed of excess oviduct tissue. Measurements recorded were:

- ◆ Ovary weight (left and right) – The weight of each ovary was measured using a digital weighing scale measuring to one decimal place in grams
- ◆ Ovary length (left and right) – The length of each ovary was measured along the longest side of the ovary using a vernier caliper. Measurements were made to two decimal places in centimeters
- ◆ Ovary width (left and right) – The width of each ovary was measured on the shortest side of the ovary using a vernier caliper. Measurements were made to two decimal places in centimeters.
- ◆ Ovary thickness (left and right) – The ovary thickness was measured using a vernier caliper measuring to two decimal places in centimeters

- ◆ Ovary volume (left and right) – The volume of each ovary was determined by submerging the ovary into a known volume of water in a 100 cm³-measuring cylinder. Measurements were made to the nearest 2 cm³.
- ◆ No of follicles in each ovary were counted individually on the ovary. Only follicles > 0.2 cm (2 mm) in size were counted.
- ◆ Size of the largest follicle in each ovary – For each ovary, the size of the largest follicle was measured using a vernier caliper to two decimal places in centimeters.

3.8.3 Body development measurements

Body development measurements were taken from the main growth study mainly on group housing animals. The body development measurements that were used were:

- ◆ Slaughter weight – taken the morning before pigs were sent to the abattoir
- ◆ Warm carcass weight (complete with head, trotters, tail and kidneys but with inside fat removed)
- ◆ P2 fat thickness measurements, measured 45 mm from dorsal midline using P2 fat-o-meter which is an ultrasound meter (P2 ultrasound) (live animal) intrascope and Hennessy grading probe (HGP) on the carcass.
- ◆ Carcass length – measured from the cranial of the pubic symphysis to the cranial edge of the first rib at the angle curvature
- ◆ Chest depth – measured as the distance between the spine and the widest part of the ribs on one half of the carcass

3.9 Data Analysis

The data for gilts and boars were analyzed separately except when body development measurements were compared across the two sexes. Equality of variances and occurrence of outliers were investigated, but outliers were left in place due to fear of losing genotype data. Results were analyzed by means of ANOVA procedures with Statistical Analysis System (SAS[®] 2001, Version 8.2). Due to the unbalanced nature of the results, General Linear Model (GLM) procedures were used to analyze for the effects of genotype and slaughter age on gonadal and body development measurements. Furthermore, from STD ERROR / PDIFF information, probabilities of differences between the genotypes and slaughter ages LSmeans were determined. Gonadal measurements were averages of the left and right ovarian or testicular measurements. For the gilts, these were ovary length, ovary width, ovary thickness, ovary weight, ovary volume, follicle number and the size of the largest follicle. Gonadal measurements of boars were testis length, testis width, testis weight and testis volume. The body development measurements included were slaughter weight, warm carcass weight, dressing percentage, carcass length, chest depth and P2 backfat thickness measurements, which were measured by ultrasonography (P2 (ultrasound)) intrascope (P2 (intrascope)) and Hennessy Grading Probe (HGP) (P2 (HGP)). Dressing percentage was calculated as a percentage of warm carcass weight divided by slaughter weight. Correlations between all the measurements as stated in section 3.8 (gonadal and body development) were performed for both gilts and boars. Moreover, correlations for body development measurements between gilts and boars were performed. Statistical significance was recorded if the probability values associated with the models were $P \leq 0.05$.

CHAPTER 4

4. RESULTS AND DISCUSSION

The effects of genotype, slaughter age, P2 (ultrasound) backfat and their interactions on all the measurements for gilts and boars are presented in Tables 4.1a,b. In all the analyses that were done, comparisons between individual genotypes and slaughter ages were determined by means of the PDIFF procedure (pair wise comparisons) of SAS (SAS, 2001) to identify any difference that could have been hidden in the overall genotype and slaughter age effect.

4.1 Influence of genotype on gonadal measurements

4.1.1 Influence of genotype on the gonadal measurements of gilts

Results on the influence of genotype on the gonadal measurements of gilts are presented in Table 4.2. This table shows the least square means and standard errors (SE) of all the gonadal measurements of gilts from the five genotypes, when slaughter age was used as a covariate. The effect of genotype had no significant influence on ovary width, ovary thickness, ovary weight, ovary volume and the size of the largest follicle. Only the ovary length was significantly influenced by genotype influence ($P = 0.0568$) (see Table 4.1a). Least square means comparison between individual genotypes by the PDIFF procedure indicated some significant differences between certain individual genotypes in some gonadal measurements. Genotype 5 gilts had significantly shorter ovary lengths than the Genotype 2 ($P = 0.010$) and the Genotype 4 ($P = 0.0123$) but was not significantly different from Genotype 1 and Genotype 3 gilts (see Figure 4.1a). The Genotype 5 gilts tended to have shorter ovary widths than the Genotype 2 gilts ($P = 0.0609$). With regard to ovary weight and ovary volume, Genotype 2 gilts had significantly heavier ovary weights ($P = 0.0467$) and larger ovary volumes ($P = 0.0181$) than Genotype 5 gilts (see Figures 4.1b,c). Furthermore, Genotype 4 gilts also tended to have heavier ovary weights ($P = 0.0698$) and larger ovary volumes ($P = 0.0726$) than Genotype 5 gilts but the results were only significant at the $P < 0.10$ significance level. Genotype 2 gilts had the highest values for ovary weight (4.48 g) and ovary volume (4.50 cm³) followed by Genotype 4 gilts (4.37 g) and (4.18 cm³) respectively while Genotype 5 gilts had the lowest ovary weights (3.45 g) and ovary volumes (3.31 cm³). With largest follicle size, Genotype 3 gilts had significantly bigger follicle sizes than the Genotype 2, Genotype 4 and Genotype 5 gilts, but were not significantly different to Genotype 1 gilts. One possible explanation for this

could have been hybrid vigor, which could have resulted in the increased follicle development in Genotype 3 gilts. The effect of genotype on ovary thickness and follicle number was not significant ($P \geq 0.05$).

Table 4.1a: Significance levels of genotype, slaughter age and their interactions on gonadal and body development measurements of gilts, (with slaughter age as a covariate)

Variable	N	Genotype	Slaughter age	Genotype X Slaughter age
P2 (intrascope) backfat	37	NS	*	NS
P2 (HGP) backfat	41	NS	*	NS
P2 (Ultrasound) backfat	31	NS	#	NS
Slaughter weight	41	*	***	#
Warm carcass weight	54	*	***	*
Dressing percentage	41	NS	*	NS
Carcass length	41	NS	***	NS
Chest depth	41	NS	***	NS
Ovary length	62	#	*	*
Ovary width	62	NS	*	*
Ovary thickness	62	NS	NS	NS
Ovary weight	62	NS	*	#
Ovary volume	62	NS	*	#
Follicle number	52	NS	NS	NS
Largest follicle size	52	NS	*	#

* = P < 0.05 ** = P < 0.001 *** = P < 0.0001 # = P < 0.10 NS = not significant

P2 (intrascope) backfat = P2 backfat measurement by an intrascope, P2 (HGP) = P2 backfat measurement by Hennessy Grading Probe

P2 (ultrasound) = P2 backfat measurement by ultrasound meter,

Table 4.1b: Significance levels of genotype and slaughter age and their interactions on gonadal and body development measurements of boars, (with slaughter age as a covariate)

Variable	N	Genotype	Slaughter age	Genotype X Slaughter age interaction
<i>P2 (intrascope) backfat</i>	23	NS	*	NS
P2 (HGP) backfat	31	NS	*	NS
P2 (Ultrasound) backfat	31	NS	*	NS
Slaughter weight	31	*	***	NS
Warm carcass weight	54	**	***	NS
Dressing percentage	31	NS	NS	NS
Carcass length	38	NS	***	NS
Chest depth	38	NS	**	NS
Testis length	54	NS	**	NS
Testis width	54	NS	***	NS
Testis weight	54	NS	***	NS
Testis volume	54	NS	***	NS

* = P < 0.05 ** = P < 0.001 *** = P < 0.0001 # = P < 0.10 NS = not significant

P2 (intrascope) backfat = P2 backfat measurement by an intrascope, P2 (HGP) = P2 backfat measurement by Hennessy Grading Probe

P2 (ultrasound) = P2 backfat measurement by ultrasound meter,

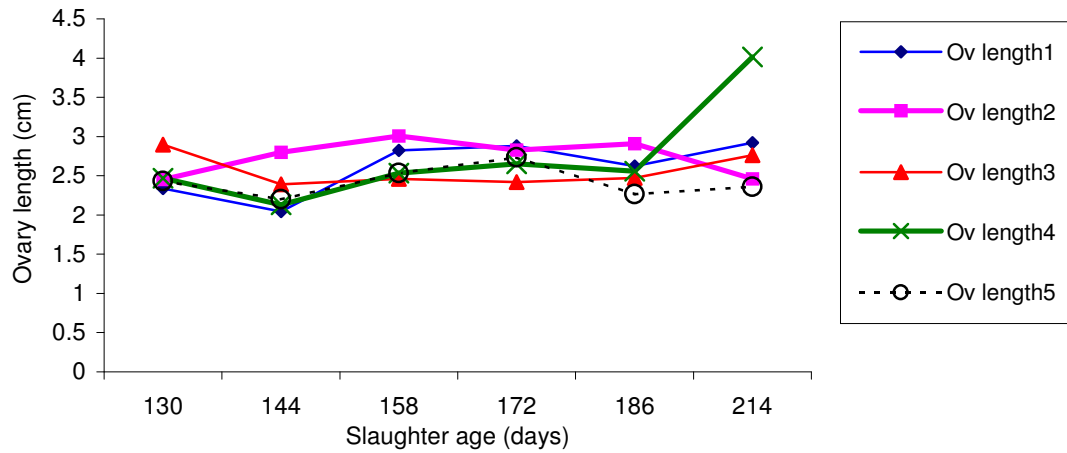
Table 4.2: Gonadal measurements (LSmeans and SE) of gilts from different genotypes (Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5) with slaughter age as a covariate

Genotype	Ovary length (cm) n = 62 (SE)	Ovary width (cm) n = 62 (SE)	Ovary thickness⁺ (cm) n = 62 (SE)	Ovary weight (g) n = 62 (SE)	Ovary volume (cm³) n = 62 (SE)	Follicle number⁺ n = 52 (SE)	Largest follicle size n = 52 (SE)
Genotype 1	2.60 ^{ab} (0.07)	1.79 (0.06)	1.01 (0.05)	4.16 ^{ab} (0.33)	4.07 ^{ab} (0.31)	13.80 (2.94)	0.59 ^{ab} (0.04)
Genotype 2	2.74 ^b (0.08)	1.94 (0.07)	0.98 (0.06)	4.48 ^b (0.37)	4.50 ^b (0.35)	16.75 (3.14)	0.55 ^a (0.04)
Genotype 3	2.56 ^{ab} (0.07)	1.84 (0.06)	0.98 (0.05)	4.02 ^{ab} (0.31)	3.98 ^{ab} (0.29)	11.71 (2.77)	0.67 ^b (0.03)
Genotype 4	2.72 ^b (0.08)	1.84 (0.07)	1.03 (0.06)	4.37 ^{ab} (0.35)	4.18 ^{ab} (0.34)	17.43 (3.21)	0.52 ^a (0.04)
Genotype 5	2.41 ^a (0.07)	1.74 (0.06)	0.95 (0.05)	3.45 ^a (0.33)	3.31 ^a (0.32)	15.43 (2.80)	0.54 ^a (0.03)

^{a-b}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

Figure 4.1a: Ovary length of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 gilts



Ov length1 = Ovary length of Genotype 1 gilts

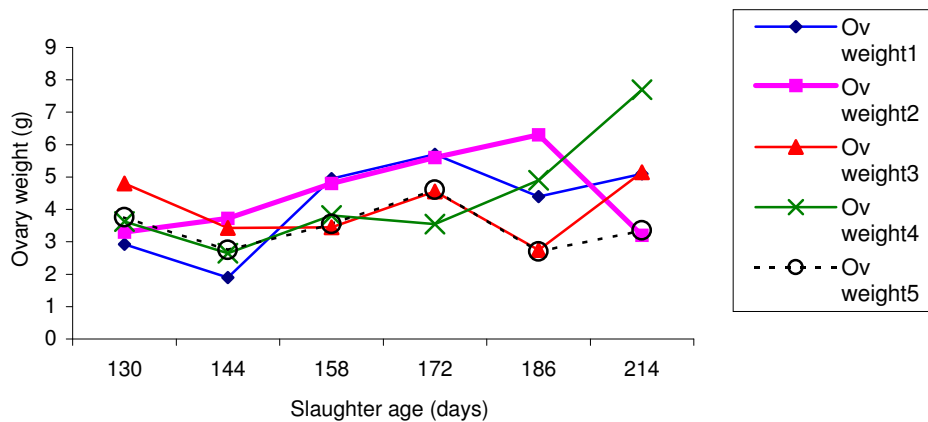
Ov length2 = Ovary length of Genotype 2 gilts

Ov length3 = Ovary length of Genotype 3 gilts

Ov length4 = Ovary length of Genotype 4 gilts

Ov length5 = Ovary length of Genotype 5 gilts

Figure 4.1b: Ovary weight of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 gilts



Ov weight1 = Ovary weight of Genotype 1 gilts

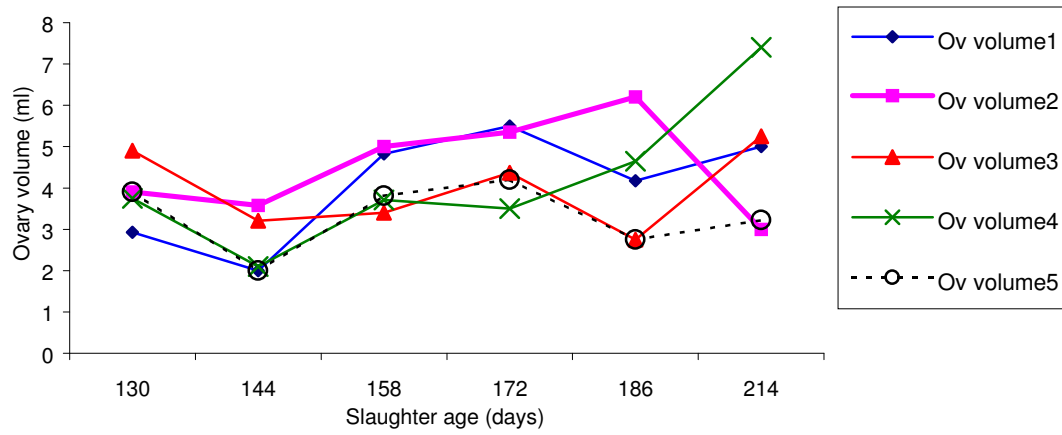
Ov weight2 = Ovary weight of Genotype 2 gilts

Ov weight3 = Ovary weight of Genotype 3 gilts

Ov weight4 = Ovary weight of Genotype 4 gilts

Ov weight5 = Ovary weight of Genotype 5 gilts

Figure 4.1c: Mean ovary volume of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 gilts



Ov volume1 = Mean ovary volume of Genotype 1 gilts
 Ov volume2 = Mean ovary volume of Genotype 2 gilts
 Ov volume3 = Mean ovary volume of Genotype 3 gilts
 Ov volume4 = Mean ovary volume of Genotype 4 gilts
 Ov volume5 = Mean ovary volume of Genotype 5 gilts

4.1.2 Influence of genotype on the gonadal measurements of boars

Results on the influence of genotype on the gonadal measurements of boars are presented in Table 4.3. These results show least square means and standard errors (SE) of gonadal measurements of boars from the five genotypes, using slaughter age as a covariate. There was no overall genotype influence on testis length, testis width, testis weight, and testis volume (Table 4.1b). However, least square means comparisons of individual genotypes by the PDIFF procedure revealed significant differences between certain individual genotypes in testis weight and testis volume (see Figures 4.2a,b). For testis weight, Genotype 2 boars had significantly heavier testis weights ($P = 0.0482$) than Genotype 5 boars. There was also a tendency of differences ($P = 0.0899$) between Genotype 1 crossbred boars and Genotype 2 boars in testis weights, with the Genotype 2 having heavier testis weights (241.20 g) than Genotype 1 crossbred boars (208.58 g). Genotype 2 boars also tended ($P = 0.0549$) to have larger testis volumes than Genotype 5 boars (243.45 cm³ vs. 189.60 cm³).

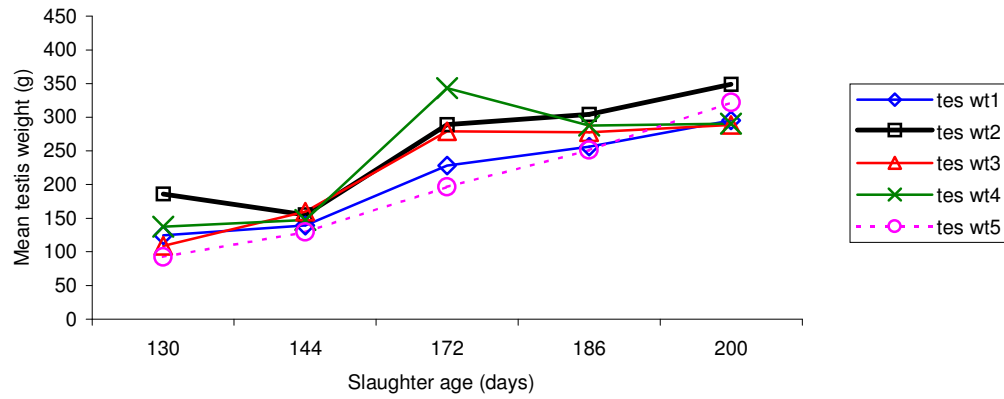
Table 4.3: Gonadal measurements (LSmeans and SE) of boars from different genotypes (Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5) with slaughter age as a covariate

Genotype	Testis length (cm) [#] n = 54 (SE)	Testis width (cm) n = 54 (SE)	Testis weight (g) n = 54 (SE)	Testis volume (cm ³) n = 54 (SE)
Genotype 1	9.57 (0.39)	6.11 (0.27)	208.58 ^{ab} (17.77)	200.35 (16.91)
Genotype 2	10.35 (0.45)	6.65 (0.32)	256.55 ^b (20.77)	243.45 (19.77)
Genotype 3	9.82 (0.39)	6.43 (0.27)	222.92 ^{ab} (18.09)	213.31 (17.22)
Genotype 4	10.14 (0.42)	6.55 (0.29)	241.20 ^{ab} (19.19)	229.75 (18.26)
Genotype 5	9.48 (0.42)	6.14 (0.29)	198.20 ^a (19.19)	189.60 (18.26)

^{a - b}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

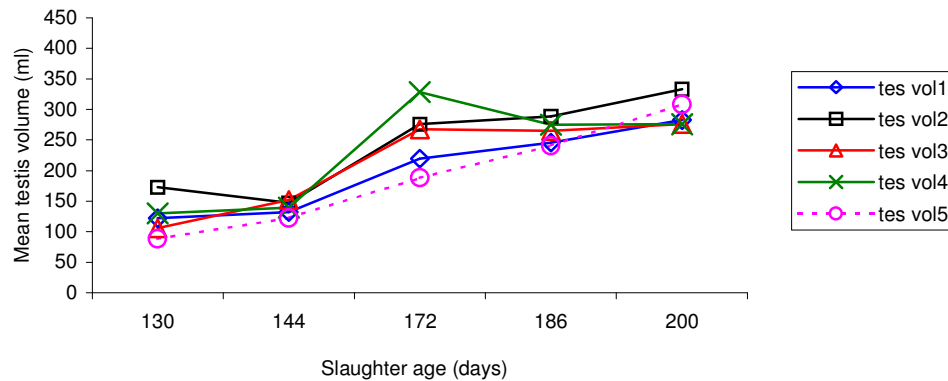
[#]: GLM model significant $P < 0.10$

Figure 4.2a: Testis weight of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 boars



Tes wt1 = Testis weight of Genotype 1 boars
 Tes wt2 = Testis weight of Genotype 2 boars
 Tes wt3 = Testis weight of Genotype 3 boars
 Tes wt4 = Testis weight of Genotype 4 boars
 Tes wt5 = Testis weight of Genotype 5 boars

Figure 4.2b: Testis volume of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 boars



Tesvol1 = Testis volume of Genotype 1 boars
 Tes vol2 = Testis volume of Genotype 2 boars
 Tes vol3 = Testis volume of Genotype 3 boars
 Tes vol4 = Testis volume of Genotype 4 boars
 Tes vol5 = Testis volume of Genotype 5 boars

4.2 Influence of genotype on body development measurements

4.2.1 Influence of genotype on body development measurements of gilts

Due to the problem of missing values, the analysis of the effect of genotype on the body development measurements of gilts could not be done with both the Genotype 2 and Genotype 5 gilts present in the same model. Therefore, two sets of analyses were done to evaluate the effect of genotype on gilts body development measurements. The first analysis compared only four genotypes; Genotype 1, Genotype 2, Genotype 3 and Genotype 4 (excluding Genotype 5) while the second analysis compared Genotype 1, Genotype 3, Genotype 4 and Genotype 5 (excluding Genotype 2). The results of these analyses are presented in Tables 4.4a and 4.4b respectively. These tables show the least square means and standard errors (SE) of the body development measurements of gilts from the five genotypes, using slaughter age as a covariate. In the first comparison that excluded Genotype 5 gilts, genotype had a significant influence on slaughter weights ($P = 0.0041$) and warm carcass weights ($P = 0.0020$). The least square means comparison between individual genotypes showed that Genotype 2 and Genotype 3 gilts were significantly heavier at slaughter than Genotype 1 gilts ($P < 0.01$) and Genotype 4 gilts ($P < 0.05$). Genotype 1 gilts had the lightest slaughter weights although they were not statistically different from Genotype 4 gilts. For warm carcass weight, Genotype 1 gilts had significantly lighter ($P < 0.05$) warm carcass weights than the Genotype 2, Genotype 3 and Genotype 4 gilts while Genotype 2 and Genotype 3 gilts had the heaviest warm carcass weights and Genotype 4 gilts were intermediate. There were no significant differences between the four genotypes (Genotype 1, Genotype 2, Genotype 3 and Genotype 4) in carcass lengths, chest depths and dressing percentages. All GLM models for P2 backfat thicknesses (intrascope, HGP and ultrasound) were not significant.

In the second analysis comparing Genotype 1, Genotype 3, Genotype 4 and Genotype 5, genotype also significantly influenced on slaughter weight and warm carcass weights. In this instance, Genotype 3 gilts had significantly ($P < 0.01$) heavier slaughter weights than Genotype 1, Genotype 4 and Genotype 5 gilts. Genotype 3 had the heaviest warm carcass weights (87.73 kg) followed by Genotype 4 gilts (80.20 kg) and the lightest were Genotype 1 gilts (77.35 kg) and Genotype 5 gilts (75.96 kg). From both analyses comparisons, it was found that Genotype 3 gilts were heavier at slaughter than both Genotype 1 and Genotype 4 gilts. Also, Genotype 3 gilts had heavier ($P < 0.05$) warm carcass weights than Genotype 1 and Genotype 5 gilts.

Average slaughter and warm carcass weights of gilts from the five genotypes are plotted graphically in Figures 4.3a,b. Dressing percentages also tended to be influenced by genotype ($P = 0.0710$). Least square means comparisons by the PDIFF procedure showed that Genotype 3 gilts had significantly lower ($P < 0.05$) dressing percentages than Genotype 1 and Genotype 4 gilts, while Genotype 5 gilts were intermediate. Genotype had no significant influence on P2 (intrascope) backfat thicknesses, P2 (ultrasound meter) backfat thicknesses, P2 (HGP) backfat thicknesses, carcass lengths, and chest depths at $P \leq 0.05$. However, least square means comparisons showed the Genotype 4 to have thicker P2 (ultrasound meter) backfat than Genotype 5 gilts (14.20 vs. 11.70 mm). For P2 (intrascope) backfat thickness, Genotype 5 gilts had thinner P2 (intrascope) backfat thickness (12.68 mm) than in other genotypes, although the difference was only numerical

Table 4.4a: Body development measurements (LSmeans and SE) of gilts from different genotypes (Genotype 1, Genotype 2, Genotype 3 and Genotype 4) with slaughter age as a covariate

Genotype	Slaughter weight (kg) n = 41 (SE)	Warm carcass weight (kg) n = 54 (SE)	Dressing percentage⁺ (%) n = 41 (SE)	Carcass length (cm) n = 41 (SE)	Chest depth (cm) n = 41 (SE)	P2-U/s.meter^{x+} (mm) n = 31 (SE)	P2-Intrascop^{y+} (mm) n = 37 (SE)	P2-HGP^{z+} (mm) n = 41 (SE)
Genotype 1	103.60 ^a (1.94)	81.52 ^a (1.52)	78.58 (0.45)	85.35 (0.87)	19.07 (0.32)	12.50 (0.88)	16.08 (1.34)	15.68 (1.22)
Genotype 2	114.87 ^b (2.03)	90.27 ^b (1.59)	78.28 (0.47)	85.50 (0.91)	19.64 (0.34)	-	17.58 (1.50)	16.14 (1.28)
Genotype 3	113.67 ^b (1.94)	89.22 ^b (1.46)	77.75 (0.45)	85.00 (0.87)	19.14 (0.32)	13.85 (0.88)	17.00 (1.42)	16.71 (1.22)
Genotype 4	107.17 ^a (2.12)	86.45 ^b (1.54)	79.14 (0.49)	85.07 (0.95)	19.21 (0.35)	-	16.58 (1.50)	16.65 (1.34)

^{a-b}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

^x: P2 backfat measurement by ultrasound meter

^y: P2 backfat measurement by an intrascop

^z: P2 backfat measurement by Hennessy Grading Probe

Table 4.4b: Body development measurements (LSmeans and SE) of gilts from different genotypes (Genotype 1, Genotype 3, Genotype 4 and Genotype 5) with slaughter age as a covariate

Genotype	Slaughter weight (kg) n = 32 (SE)	Warm carcass weight (kg) n = 49 (SE)	Dressing percentage (%) n = 31 (SE)	Carcass length (cm) n = 32 (SE)	Chest depth (cm) n = 32 (SE)	P2-U/s.meter^x (mm) n = 31 (SE)	P2-Intrascop^y (mm) n = 25 (SE)	P2-HGP^{z#} (mm) n = 31 (SE)
Genotype 1	98.31 ^a (2.02)	77.35 ^{ab} (1.36)	78.58 ^b (0.40)	84.10 (0.96)	19.10 (0.36)	12.50 ^{ab} (0.64)	15.87 (1.09)	15.64 (1.22)
Genotype 3	112.46 ^b (2.18)	87.73 ^c (1.39)	77.01 ^a (0.43)	84.20 (1.04)	19.10 (0.39)	13.40 ^{ab} (0.69)	17.25 (1.34)	16.08 (1.32)
Genotype 4	99.26 ^a (2.47)	80.20 ^b (1.51)	78.63 ^b (0.49)	83.40 (1.18)	18.90 (0.45)	14.20 ^b (0.78)	14.87 (1.44)	15.00 (1.49)
Genotype 5	97.66 ^a (2.13)	75.96 ^a (1.28)	77.70 ^{ab} (0.43)	84.10 (0.96)	18.80 (0.36)	11.70 ^a (0.69)	13.87 (1.34)	12.68 (1.32)

^{a-c}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

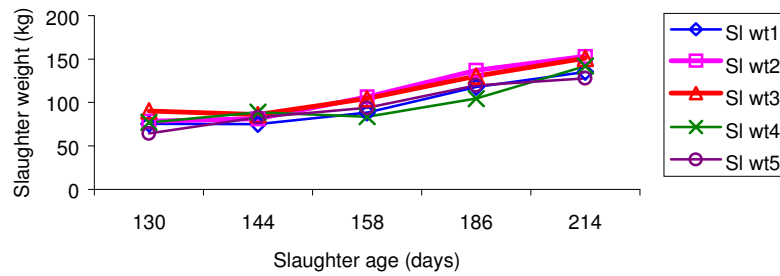
[#]: GLM model significant at $P < 0.10$

^x: P2 backfat measurement by ultrasound meter

^y: P2 backfat measurement by an intrascop

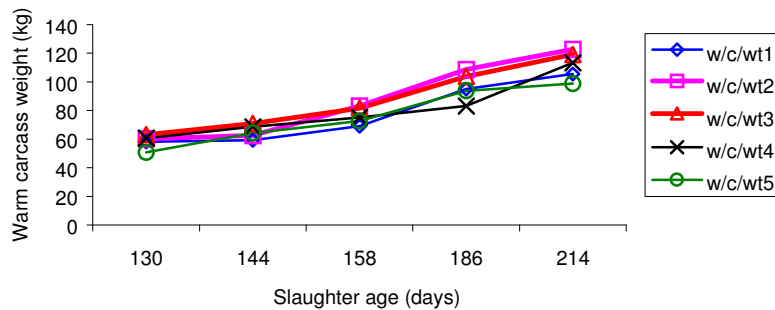
^z: P2 backfat measurement by Hennessy Grading Probe

Figure 4.3a: Average slaughter weights of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 gilts at different slaughter ages



SI wt1 = Average slaughter weight of Genotype 1 gilts
 SI wt2 = Average slaughter weight of Genotype 2 gilts
 SI wt3 = Average slaughter weight of Genotype 3 gilts
 SI wt4 = Average slaughter weight of Genotype 4 gilts
 SI wt5 = Average slaughter weight of Genotype 5 gilts

Figure 4.3b: Average warm carcass weights of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 gilts at different slaughter ages

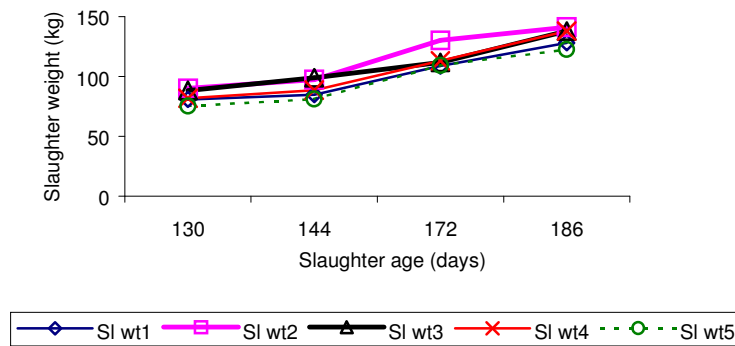


w/c/wt1 = Average warm carcass weight of Genotype 1 gilts
 w/c/wt2 = Average warm carcass weight of Genotype 2 gilts
 w/c/wt3 = Average warm carcass weight of Genotype 3 gilts
 w/c/wt4 = Average warm carcass weight of Genotype 4 gilts
 w/c/wt5 = Average warm carcass weight of Genotype 5 gilts

4.2.2 Influence of genotype on body development measurements of boars

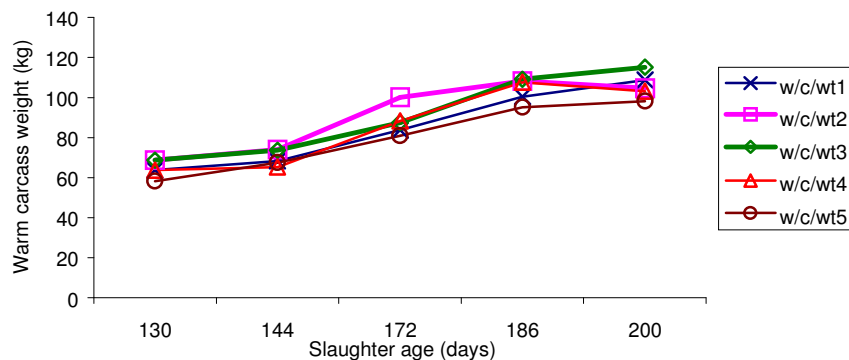
Table 4.5 shows the results of body development measurements of boars. These results show least square means and standard errors of all body development measurements of boars in the five genotypes, with slaughter age as a covariate. Genotype had a significant influence on the slaughter and warm carcass weights of boars. Genotype 2 ($P < 0.01$) and Genotype 3 boars were heavier ($P < 0.05$) at slaughter than Genotype 1 and Genotype 5 boars while Genotype 4 boars were intermediate. Although Genotype 4 boars were intermediate in terms of average slaughter weight, they were significantly different ($P < 0.05$) from to Genotype 2 boars (heaviest) and Genotype 5 (lightest) boars as far as average slaughter weights are concerned. The same applied to warm carcass weight; Genotype 2 (91.17 kg) and Genotype 3 boars (90.85 kg) had significantly ($P < 0.05$) heavier warm carcass weights than Genotype 4 (85.60 kg), Genotype 1 (84.95 kg), and Genotype 5 boars (80.00 kg) with Genotype 5 boars having the lightest warm carcass weights. Also, Genotype 1 boars tended ($P = 0.0591$) to have heavier warm carcass weights than Genotype 5 boars. Graphical representation of slaughter and warm carcass weights of boars from the five genotypes are plotted in Figures 4.4a,b. Although the overall effect of genotype was not significant for P2 (intrascope) backfat and P2 (HGP) backfat (see Table 4.1b) comparisons based on the PDIFF procedure revealed more differences between genotypes in these measurements. Genotype 5 boars had lower ($P = 0.0278$) P2 (HGP) backfat thicknesses than Genotype 1 boars (12.25 vs. 16.05 mm) while other genotypes were intermediate. Genotype 5 boars also tended to have lower P2 (intrascope) backfat thicknesses than all the other genotypes but all results were significant at the $P < 0.10$ probability level. The GLM models for dressing percentages and P2 (ultrasound) backfat thicknesses of boars were not significant.

Figure 4.4a: Average slaughter weight of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 boars at different slaughter ages



SI wt1 = Average slaughter weight of Genotype 1 boars
 SI wt2 = Average slaughter weight of Genotype 2 boars
 SI wt3 = Average slaughter weight of Genotype 3 boars
 SI wt4 = Average slaughter weight of Genotype 4 boars
 SI wt5 = Average slaughter weight of Genotype 5 boars

Figure 4.4b: Average warm carcass weight of Genotype 1, Genotype 2, Genotype 3, Genotype 4 and Genotype 5 boars at different slaughter ages



w/c/wt1 = Average warm carcass weight of Genotype 1 boars
 w/c/wt2 = Average warm carcass weight of Genotype 2 boars
 w/c/wt3 = Average warm carcass weight of Genotype 3 boars
 w/c/wt4 = Average warm carcass weight of Genotype 4 boars
 w/c/wt5 = Average warm carcass weight of Genotype 5 boar

Table 4.5: Body development measurements (LSmeans and SE) of boars from different genotypes (Genotype 1, Genotype 2, Genotype 3 and Genotype 4) with slaughter age as a covariate

Genotype	Slaughter weight (kg) n = 31 (SE)	Warm carcass weight (kg) n = 54 (SE)	Dressing ⁺ percentage (%) n = 31 (SE)	Carcass length (cm) n = 38 (SE)	Chest depth (cm) n = 38 (SE)	P2-U/s.meter ^{x+} (mm) n = 31 (SE)	P2-Intrascopy ^y (mm) n = 23 (SE)	P2-HGP ^{z#} (mm) n = 31 (SE)
Genotype 1	100.60 ^{ab} (2.25)	84.95 ^{ab} (1.71)	78.68 (1.61)	83.90 (1.16)	19.15 (0.40)	13.25 ^{ab} (0.86)	15.50 (0.81)	16.05 ^b (1.06)
Genotype 2	114.73 ^c (2.66)	91.17 ^c (2.00)	76.43 (1.90)	86.80 (1.42)	19.60 (0.50)	10.87 ^{ab} (1.02)	15.66 (1.15)	14.30 ^{ab} (1.25)
Genotype 3	109.18 ^{bc} (2.47)	90.85 ^c (1.74)	78.32 (1.76)	86.50 (1.34)	19.80 (0.47)	13.50 ^b (0.95)	15.33 (0.94)	15.60 ^{ab} (1.16)
Genotype 4	105.30 ^b (2.47)	85.60 ^b (1.84)	76.96 (1.76)	86.20 (1.26)	19.70 (0.44)	12.75 ^{ab} (0.95)	15.50 (1.05)	13.80 ^{ab} (1.16)
Genotype 5	96.97 ^a (2.25)	80.00 ^a (1.84)	78.13 (1.61)	86.50 (1.26)	19.20 (0.44)	10.62 ^a (0.86)	12.66 (0.94)	12.25 ^a (1.06)

^{a-c}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

[#]: GLM model significant at $P < 0.10$

^x: P2 backfat measurement by ultrasound meter

^y: P2 backfat measurement by an intrascopy

^z: P2 backfat measurement by Hennessy Grading Probe

4.3 Influence of age on gonadal measurements

Results of the effect of slaughter age on gonadal and body development measurements are shown in Tables 4.6 – 4.9.

4.3.1 Influence of slaughter age on gonadal measurements of gilts

Presented in Table 4.6 are the results of the effect of slaughter age on gonadal measurements of gilts. These results show least square means and standard errors (SE) of gilts reproductive measurements at different slaughter ages, using genotype as a covariate. Slaughter age influenced ($P < 0.05$) ovary length, ovary width, ovary weight, ovary volume and largest follicle size (see Table 4.1a). There was a general trend for these measurements to increase with age. (See Figure 4.1a,b,c for plotted graphs of ovary length, ovary weight and ovary volume of the five genotypes respectively). Despite this general trend, certain slaughter ages were not significantly different from each other in terms of certain gonadal measurements. For example, slaughter age of 144 days had the shortest ovary length (2.31 cm) while ovary length was longest at the oldest age of 214 days (2.90 cm). The slaughter ages of 130, 158, 172 and 186 days were intermediate between those of 144 and 214 days in ovary lengths. One possible reason for low mean ovary length values in gilts at a slaughter age of 144 days could have been a result of change in the diet since diets were changed after the pigs reached a certain average weight. Energy and crude protein content of the feed was reduced from 14 MJ/kg to 13 MJ/kg and from 18 % CP to 16 % CP for energy and crude protein respectively after the pigs reached an average live weight of 65 kg. These were further reduced to 13.2 MJ/kg and 15 % CP after pigs reached an average live weight of 90 kg. In an animal, reproduction needs are only attended to after maintenance and growth needs have been satisfied, therefore, the reduction in energy and crude protein content from 14MJ/kg to 13MJ/kg and from 18% CP to 16% CP in the live weight band of 65 – 90 kg could have affected the ovarian development in gilts at the age of 144 days since that age (144 days) was reached in the 65 to 90 kg weight band. The other factor to consider is the fact that the majority of those gilts slaughtered at this age (144 days) were of Genotype 3, so it is not known whether this could have affected the results somehow. There were ten (10) Genotype 3 gilts as compared to one or two gilts from other genotypes. The ovary widths and ovary volumes were also significantly lower ($P < 0.05$) at the slaughter age of 144 days than at all the other ages while the other slaughter ages did not significantly differ between each other, although they increased with slaughter age; despite the fact that slaughter age of 130 days was the youngest in gilts and not 144 days. However, ovary volumes at 214 days tended (P

= 0.0924) to be larger than at 130 days. Ovary weights also had the lowest measurements at 144 days and highest at 214 days followed by mean ovary weights at 172 days of age. Similarly, it is most probable that these slaughter ages coincided with change in diet. Nevertheless, ovary weights were significantly different between the ages of 130, 144 and 214 days although it was not different between the slaughter ages of 130 and 144 days. The sizes of the largest follicle were significantly larger ($P < 0.05$) at the age of 172 days than at the slaughter ages of 144, 158, and 186 days. Compared to slaughter age of 214 days, the largest follicle sizes had a strong tendency to be bigger at 172 days ($P = 0.0642$) than at 214 days. This increase in largest follicle sizes at the age of 172 days could have coincided with the start of puberty in most gilts or it be an evidence to show that most gilts had already started cycling at the age of 172 days. The GLM models for ovary thickness and follicle number were not significant.

Table 4.6: Gonadal measurements (LSmeans and SE) of gilts at different slaughter ages, with genotype as a covariate

Slaughter age (days)	Ovary length (cm) n = 62 (SE)	Ovary width (cm) n = 62 (SE)	Ovary thickness ⁺ (cm) n = 62 (SE)	Ovary weight (g) n = 62 (SE)	Ovary volume (cm ³) n = 62 (SE)	Follicle number ⁺ n = 52 (SE)	Largest follicle size (cm) n = 52 (SE)
130	2.51 ^{ab} (0.09)	1.80 ^b (0.08)	0.96 ^{ab} (0.06)	3.68 ^{ab} (0.39)	3.87 ^b (0.37)	-	-
144	2.31 ^a (0.08)	1.55 ^a (0.07)	0.87 ^a (0.06)	2.89 ^a (0.37)	2.57 ^a (0.35)	17.81 ^{ab} (2.99)	0.55 ^a (0.04)
158	2.67 ^{bc} (0.07)	1.86 ^b (0.06)	0.95 ^{ab} (0.05)	4.11 ^{bc} (0.33)	4.14 ^b (0.31)	13.31 ^{ab} (2.64)	0.50 ^a (0.03)
172	2.70 ^{bc} (0.09)	1.92 ^b (0.08)	1.02 ^{ab} (0.06)	4.80 ^{bc} (0.39)	4.58 ^b (0.37)	9.60 ^a (3.33)	0.70 ^b (0.04)
186	2.56 ^{ab} (0.08)	1.90 ^b (0.07)	1.02 ^{ab} (0.06)	4.21 ^{bc} (0.37)	4.10 ^b (0.35)	18.95 ^b (2.94)	0.50 ^a (0.04)
214	2.90 ^c (0.08)	1.95 ^b (0.07)	1.11 ^b (0.06)	4.90 ^c (0.37)	4.77 ^b (0.35)	15.45 ^{ab} (2.94)	0.61 ^{ab} (0.04)

^{a-c}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

4.3.2 Influence of slaughter age on the gonadal measurements of the boar

Results of slaughter age influence on the gonadal measurements of the boar are presented in Table 4.7. This table shows LSmeans and standard errors (SE) of boar reproductive measurements at different slaughter ages, using genotype as a covariate. Only five slaughter ages (130, 144, 172, 186 and 200 days) were used in the analysis for slaughter age effect because of some missing values for certain slaughter ages. Slaughter age had a highly significant ($P < 0.001$) influence on all gonadal measurements (testis length, testis width, testis weight and testis volume) of boars. Although there was a growth trend in gonadal measurements as slaughter age increases, only testis weights and testis volumes had been plotted graphically in Figures 4.2ab because these two measurements also had significant different performances between certain genotypes. Least square means comparisons between slaughter ages showed that boar reproductive measurements at 130 and 144 days were significantly lower ($P < 0.01$) in size compared to those measurements of older ages (i.e. 172, 186, and 200 days) while there was no significant difference in boar reproductive measurements at these older ages.

Table 4.7: Gonadal measurements (LSmeans and SE) of boars at different slaughter ages, with genotype as a covariate

Slaughter age (days)	Testis length[#] (cm) n = 54 (SE)	Testis width (cm) n = 54 (SE)	Testis weight (g) n = 54 (SE)	Testis volume (cm³) n = 54 (SE)
130	8.42 ^a (0.42)	5.19 ^a (0.29)	129.95 ^a (19.19)	123.75 ^a (18.26)
144	8.88 ^a (0.33)	5.48 ^a (0.23)	146.18 ^a (15.25)	138.51 ^a (14.52)
172	10.46 ^b (0.42)	6.81 ^b (0.29)	267.03 ^b (19.19)	255.85 ^b (18.26)
186	10.62 ^b (0.45)	6.98 ^b (0.31)	275.30 ^b (20.52)	263.00 ^b (19.52)
200	10.98 ^b (0.45)	7.42 ^b (0.31)	308.99 ^b (20.52)	295.35 ^b (19.52)

^{a-b}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

[#]: GLM model significant $P < 0.10$

4.4 Influence of slaughter age on body development measurements

4.4.1 Influence of slaughter age on body development measurements of gilts

The results on the effects of slaughter age on the body development measurements of the gilts are presented in Table 4.8a and 4.8b. These tables show least square means and standard errors (SE) of body development measurements of gilts at different slaughter ages, with genotype as a covariate. For purposes of this discussion, only results from Table 4.8a will be discussed because the analysis of these results included all the slaughter ages. Slaughter age had a highly significant influence ($P < 0.0001$) on slaughter weights, warm carcass weights, carcass lengths, and chest depths (see Table 4.1a). The GLM model for dressing percentages was significant at $P = 0.0638$ and slaughter age had a significant influence on dressing percentages ($P = 0.0061$). Generally, these body development measurements increased with age, with younger animals having lower measurements than the older animals. The slaughter and warm carcass weights of gilts are plotted graphically in Figures 4.3ab.

Comparisons of least square means based on the PDIFF procedure revealed some significant differences between certain slaughter ages in some body development measurements. In carcass length, gilts older than 158 days of age had significantly longer carcass lengths ($P < 0.05$) than those gilts of 158 days and younger. A similar trend was also observed with chest depth. Gilts older than 158 days had significantly deeper chest depths than gilts of 158 days and younger age. Dressing percentages were only significantly different between ages of 130 days, 144 & 158 days, 186 & 200 days. There were no significant differences between dressing percentages at 214 days and 144 days and this is due to the fact that dressing percentages were highest at 186 and 200 days and not at 214 days. Slaughter weight and warm carcass weight were significantly different between the different slaughter ages and this also applied to intermediate ages. Unlike the results obtained from carcass lengths and chest depths, there were significant differences in slaughter and warm carcass weights at ≤ 144 days, 158 days to 200 days and at 214 days. The GLM models for slaughter age effect on P2 backfat by intrascope, HGP and ultrasound meter were not significant

Table 4.8a: Body development measurements (LSmeans and SE) of gilts at different slaughter ages, with genotype (Genotype 1, Genotype 2, Genotype 3 and Genotype 4) as a covariate

Slaughter age (days)	Slaughter weight (kg) n = 41 (SE)	Warm carcass weight (kg) n = 54 (SE)	Dressing [#] percentage (%) n = 41 (SE)	Carcass length (cm) n = 41 (SE)	Chest depth (cm) n = 41 (SE)	P2-U/s.meter ^{x+} (mm) n = 31 (SE)	P2-Intrascop ^{y+} (mm) n = 37 (SE)	P2-HGP ^{z+} (mm) n = 41 (SE)
130	80.26 ^a (2.63)	60.57 ^a (2.06)	75.70 ^a (0.61)	78.50 ^a (1.18)	17.00 ^a (0.44)	12.33 ^a (1.30)	12.75 ^a (1.74)	13.65 ^a (1.66)
144	82.78 ^a (2.63)	65.30 ^a (1.92)	77.86 ^b (0.61)	81.00 ^a (1.18)	18.12 ^a (0.44)	11.66 ^a (1.46)	-	12.10 ^a (1.66)
158	95.81 ^b (2.84)	77.26 ^b (1.94)	78.01 ^b (0.66)	81.87 ^a (1.28)	18.37 ^{ab} (0.47)	12.50 ^{ab} (1.46)	13.00 ^a (1.88)	13.20 ^a (1.79)
172	115.15 ^c (2.63)	90.95 ^c (2.06)	78.95 ^{bc} (0.61)	88.00 ^b (1.18)	19.62 ^b (0.44)	13.00 ^{ab} (1.46)	15.75 ^{ab} (1.74)	15.35 ^{ab} (1.66)
186	122.48 ^{cd} (2.63)	97.63 ^{cd} (2.06)	79.74 ^c (0.61)	87.00 ^b (1.18)	20.37 ^b (0.44)	12.66 ^{ab} (1.30)	16.37 ^{ab} (1.74)	16.70 ^{ab} (1.66)
200	127.00 ^d (2.63)	101.20 ^d (2.06)	79.63 ^c (0.61)	88.50 ^{bc} (1.18)	19.50 ^b (0.44)	16.83 ^{bc} (1.46)	19.87 ^{bc} (1.74)	20.40 ^{bc} (1.66)
214	145.32 ^e (2.63)	115.13 ^e (2.06)	79.19 ^{bc} (0.61)	91.75 ^c (1.18)	21.87 ^c (0.44)	17.66 ^c (1.30)	23.12 ^c (1.74)	22.70 ^c (1.66)

^{a-e}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

[#]: GLM model significant at $P < 0.10$

^x: P2 backfat measurement by ultrasound meter

^y: P2 backfat measurement by an intrascope

^z: P2 backfat measurement by Hennessy Grading Probe

Table 4.8b: Body development measurements (LSmeans and SE) of gilts at different slaughter ages, with genotype (Genotype 1, Genotype 3, Genotype 4 and Genotype 5) as a covariate

Slaughter age (days)	Slaughter weight (kg) n = 32 (SE)	Warm carcass weight (kg) n = 49 (SE)	Dressing percentage (%) n = 31 (SE)	Carcass length (cm) n = 32 (SE)	Chest depth (cm) n = 32 (SE)	P2-U/s.meter ^x (mm) n = 31 (SE)	P2-Intrascop ^y (mm) n = 25 (SE)	P2-HGP ^{z+} (mm) n = 31 (SE)
130	76.68 ^a (2.52)	58.20 ^a (1.70)	76.27 ^a (0.50)	77.75 ^a (1.20)	17.00 ^a (0.45)	11.25 ^a (0.80)	12.00 ^a (1.34)	13.05 ^a (1.53)
144	83.18 ^a (2.52)	65.73 ^b (1.59)	78.05 ^{bc} (0.50)	80.25 ^{ab} (1.20)	17.87 ^a (0.45)	10.87 ^a (0.80)	-	11.50 ^a (1.53)
158	92.59 ^b (2.45)	74.59 ^c (1.33)	77.82 ^{ab} (0.50)	82.12 ^b (1.20)	18.12 ^a (0.45)	12.75 ^a (0.80)	13.12 ^a (1.34)	13.10 ^a (1.53)
186	118.15 ^c (2.30)	93.88 ^d (1.56)	79.50 ^c (0.45)	88.00 ^c (1.10)	20.50 ^b (0.41)	12.12 ^a (0.73)	14.37 ^a (1.22)	14.40 ^a (1.39)
214	139.00 ^d (2.52)	109.13 ^c (1.56)	78.27 ^{bc} (0.50)	91.62 ^d (1.10)	21.37 ^b (0.41)	17.75 ^b (0.80)	22.37 ^b (1.34)	22.20 ^b (1.53)

^{a-c}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

^x: P2 backfat measurement by ultrasound meter

^y: P2 backfat measurement by an intrascop

^z: P2 backfat measurement by Hennessy Grading Probe

4.4.2 Influence of slaughter age on body development measurements of boars

Results of the effect of slaughter age on body development measurements of boars are presented in Table 4.9. The table shows LSmeans and standard errors (SE) of body development measurements of boars at different slaughter ages, with genotype as a covariate. Slaughter age had a significant influence on slaughter weights, warm carcass weights, carcass lengths ($P < 0.0001$) chest depths ($P < 0.001$) P2 (intrascope) backfat thicknesses, and P2 (HGP) backfat thickness ($P < 0.05$) (see Table 4.1b). Similar to the observations recorded for gilts, the body development measurements of boars increased in size as slaughter age increases (see Figures 4.4a,b). Least square means comparisons between different slaughter ages based on the PDIFF procedure showed that there were some significant differences in body development measurements of boars between certain slaughter ages. Slaughter weights and warm carcass weights were lower at 130 days compared to at 144 days while slaughter weights and warm carcass weights at these two slaughter ages were smaller than those measurements at 172 days. Warm carcass weights at 186 days were not significantly different from warm carcass weights at 200 days of age. Carcass lengths and chest depths of boars had similar growth patterns. Carcass lengths and chest depths at 130 days were smaller to those measurements at 144 days and highest at 200 while the middle slaughter ages were intermediate in carcass length and chest depth. Although results of P2 (intrascope) backfat showed a growth trend, these results should be treated with caution as only 3 slaughter ages (130, 172 and 186 days) were used in its analysis. P2 (intrascope) backfat thicknesses were higher at 186 days ($P < 0.01$) than at 130 and 172 days of age. P2 (HGP) backfat thicknesses only increased slightly with slaughter age and it was lowest at 130 days (12.76mm) and highest at 186 days (18.24mm). This could be due to the fact there was no data for P2 (HGP) backfat thickness at 200 and 214 days slaughter ages. The P2 (HGP) backfat in boars was not statistically different between the slaughter ages 130, 144 and 172 days.

Table 4.9: Body development measurements (LSmeans and SE) of boars at different slaughter ages, with genotype as a covariate

Slaughter age (days)	Slaughter weight (kg) n = 31 (SE)	Warm carcass weight (kg) n = 54 (SE)	Dressing ⁺ percentage (%) n = 31 (SE)	Carcass length (cm) n = 38 (SE)	Chest depth (cm) n = 38 (SE)	P2-U/s.meter ^{x+} (mm) n = 31 (SE)	P2-Intrascopy ^y (mm) n = 23 (SE)	P2-HGP ^{z#} (mm) n = 31 (SE)
130	83.24 ^a (2.13)	64.60 ^a (1.84)	77.60 (1.52)	76.70 ^a (1.26)	17.25 ^a (0.44)	12.00 (0.82)	12.80 ^a (0.74)	12.76 ^a (1.00)
144	90.14 ^b (2.13)	69.72 ^b (1.46)	78.32 (1.52)	82.90 ^b (1.26)	18.90 ^b (0.44)	11.40 (0.82)	-	13.28 ^a (1.00)
172	114.55 ^c (2.13)	88.10 ^c (1.84)	76.89 (1.52)	86.60 ^b (1.26)	20.20 ^{bc} (0.44)	10.60 (0.82)	13.70 ^a (0.74)	13.32 ^a (1.00)
186	133.51 ^d (2.28)	104.15 ^d (1.97)	78.01 (1.62)	91.30 ^c (1.34)	20.40 ^c (0.47)	14.80 (0.87)	18.30 ^b (0.80)	18.24 ^b (1.07)
200	-	106.00 ^d (1.97)	-	92.40 ^c (1.34)	20.70 ^c (0.47)	-	-	-

^{a-d}: LSmeans in the same column with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

[#]: GLM model significant at $P < 0.10$

^x: P2 backfat measurement by ultrasound meter

^y: P2 backfat measurement by an intrascopy

^z: P2 backfat measurement by Hennessy Grading Probe

4.5 Genotype and slaughter age interactions on gonadal measurements

The significance levels of genotype, slaughter age, P2 (ultrasound) and their interactions on gonadal and body development measurements are shown in Tables 4.1a,b. In the gilts, genotype and slaughter age interaction was only significant with ovary length ($P = 0.0055$) and ovary width ($P = 0.0100$). For ovary weight, ovary volume and size of the largest follicle, genotype and slaughter age interactions were significant at $P < 0.10$. These interactions resulted from the fact that in most cases, gonadal measurements of gilts were lower at 144 days than at the other slaughter ages while on the other hand Genotype 2 gilts had larger gonadal measurements than Genotype 5 gilts. Also, gonadal measurements of gilts were higher at the ages of 172 and 214 days of age. There were no genotype and slaughter age interactions on any of the gonadal measurements of boars.

4.6 Genotype and slaughter age interactions on body development measurements

In the gilts, there was tendency ($P = 0.0662$) of a genotype and slaughter age interaction, but non-significant. The observed interaction was located in a model where only four genotypes (Genotype 1, Genotype 2, Genotype 3 and Genotype 4) were used. In another model that (excluded Genotype 2) only had Genotype 1, Genotype 3, Genotype 4 and Genotype 5, P2 (ultrasound) backfat thickness had a significant ($P = 0.0257$) genotype and slaughter age interaction. However, these results should be treated with caution since the model did not include Genotype 2 gilts and slaughter ages of 172 and 200 days in the analysis. These results could therefore be misleading especially since the time interval between the slaughter ages was now greater. Boars had no genotype and slaughter age interaction on any of the body development measurements.

4.7 The influence of sex on body development measurements

Results of body development measurements comparisons between gilts and boars are presented in Table 4.10. This table shows least square means and standard errors (SE) of body development measurements for gilts and boars. There were no significant differences in slaughter weights, warm carcass weights, dressing percentages, carcass lengths and chest depths between both gilts and boars. Moreover, the GLM models for these variables were not significant. However, there were significant differences in P2 backfat thicknesses

(by ultrasound, intrascope and HGP) between gilts and boars. In all the three P2 backfat thicknesses (P2 (ultrasound) P2 (intrascope) and P2 (HGP)) P2 backfat measurements were highest for gilts while boars had the least P2 backfat thickness. A graphical representation of P2 (HGP) backfat thickness between gilts and boars is shown in Figure 4.5. This graph shows gilts to have thicker P2 backfat while boars had the least P2 backfat. Of the three equipments used to measure P2 backfat thickness, the intrascope recorded highest P2 backfat measurements while the ultrasound meter recorded lowest values of P2 backfat measurements and the HGP recorded intermediate measurements. The pattern was the same for both gilts and boars; P2 (intrascope) backfat was highest, followed by P2 (HGP) backfat, then lastly P2 (ultrasound) backfat. The difference in P2 backfat measurements recorded by the three devices could reflect the accuracy of these three equipments in measuring P2 backfat thickness in pigs. The device that recorded highest values, in this case being the intrascope, could reflect its high accuracy in measuring P2 backfat while the one with lowest values (the P2-ultrasound meter) could reflect its lower accuracy in measuring P2 backfat depth. These differences in P2 measurement accuracies between the three devices could be due to the fact the intrascope and the HGP measures P2 backfat thicknesses on the carcass while the ultrasound meter measures backfat thickness on a live animal.

Table 4.10: Least square means and standard errors (SE) of body development measurements of gilts and boars

Variable	n	LSmeans	
		Gilts (SE)	Boars (SE)
P2 (intrascope) backfat (mm)	171	16.51 ^{ab} (0.79)	14.62 ^a (0.76)
P2 (HGP) backfat (mm)	146	15.65 ^a (0.76)	14.07 ^a (0.72)
P2 (Ultrasound) backfat (mm)	172	13.63 ^b (0.55)	11.88 ^a (0.51)
Slaughter weight ⁺ (kg)	175	107.93 (3.91)	106.93 (3.80)
Warm carcass weight ⁺ (kg)	210	82.22 (2.58)	81.38 (2.51)
Dressing percentage ⁺ (%)	174	78.24 (0.46)	77.28 (0.44)
Carcass length ⁺ (cm)	163	85.31 (0.93)	86.82 (0.94)
Chest depth ⁺ (cm)	177	19.29 (0.35)	19.36 (0.33)

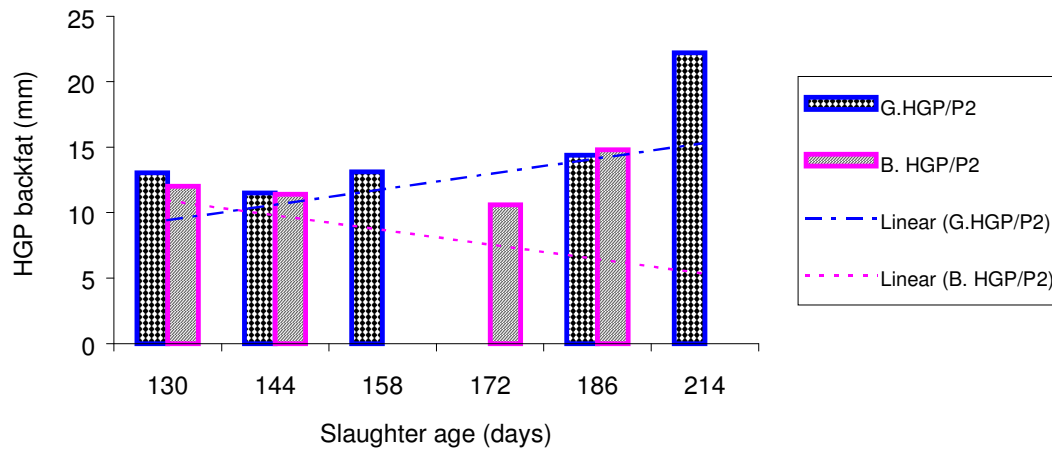
^{a-c}: LSmeans in the same row with different superscript differ ($P \leq 0.05$)

⁺: GLM model not significant (i.e. $P > 0.05$)

P2 (intrascope) backfat = P2 backfat measurement by an intrascope, P2 (HGP) = P2 backfat measurement by Hennessy Grading Probe

P2 (ultrasound) = P2 backfat measurement by ultrasound meter.

Figure 4.5: Average P2 backfat thicknesses for gilts and boars measured using Hennessy Grading Probe (HGP) at different slaughter ages



G.HGP/P2 = P2 backfat thickness by HGP for gilts

B.HGP/P2 = P2 backfat thickness by HGP for boars

Linear (G.HGP/P2) = Linear trend for P2 backfat thickness by HGP in gilts

Linear (B.HGP/P2) = Linear trend for P2 backfat thickness by HGP in boars

4.8 Correlations between measurements

4.8.1 Correlations between body development measurements

Correlation coefficients between all measurements (carcass and gonadal) for gilts and boars are shown in Tables 4.11 and 4.12 respectively. Correlations between body development measurements of gilts (excluding dressing percentage) were positive and ranged from moderate to high ($r = 0.514$ to $r = 0.995$; $P < 0.001$). The only exception was dressing percentage, which had low correlation coefficients with other body development measurements. Correlation coefficients for dressing percentage in gilts ranged from 0.189 to 0.447 and these were correlation coefficients between dressing percentages and P2 (ultrasound) backfat thicknesses and between dressing percentages and warm carcass weights respectively. However, the correlation coefficient between dressing percentages and P2 (ultrasound) backfat thickness in gilts ($r = 0.189$) was not significant, $P = 0.202$. With the exclusion of dressing percentages, body development measurements in boars were also moderate to highly correlated ($r = 0.409$ to $r = 0.986$). All correlation

coefficients of boars body development measurements were highly significant ($P < 0.0001$) except for correlation between carcass lengths and P2 (ultrasound) backfat thicknesses which had correlation of $r = 0.409$, $P < 0.03$. Dressing percentages in boars were also poorly correlated with all the other body development measurements, $r = -0.079$ to $r = 0.104$. P2 (intrascope) backfat thickness ($r = -0.10$) P2 (HGP) backfat thickness ($r = -0.079$) slaughter weight ($r = -0.043$) and carcass length ($r = -0.041$) were negatively correlated with dressing percentage in boars. In addition to this, dressing percentage correlations with other body development measurements were non-significant ($P > 0.05$). The low and non-significant correlations of dressing percentage with other body development measurements show that there is little or no relationship between dressing percentage and other body development measurements. Dressing percentage values could also have been affected by the fact that warm carcass weight was used to calculate dressing percentage. Normally, cold carcass weight is used in the calculation of dressing percentage after drip loss has occurred.

Table 4.11: Pearson Correlation coefficients between body development and gonadal measurements of gilts, with probabilities and number of observations

*		P2- intrascope ^y	P2- HGP ^z	P2- u/s.meter ^x	Slaughter weight	Warm carcass weight	Dressing percentag e	Carcass length	Chest depth	Ovary Length	Ovary. Width	Ovary thickness	Ovary weight	Ovary volume	Follicle number	Largest follicle size
P2- intrascope ^y	r	1.0000	0.965	0.829	0.733	0.736	0.313	0.576	0.585	0.446	0.308	0.345	0.362	0.323	-0.121	0.240
	P		< .0001	< .0001	<0.0001	< 0.0001	0.046	< .0001	<0.0001	0.003	0.049	0.027	0.019	0.039	0.485	0.163
	N	41	41	41	41	41	41	41	41	41	41	41	41	41	41	45
P2- HGP ^z	r	0.965	1.0000	0.853	0.739	0.743	0.338	0.642	0.598	0.396	0.321	0.320	0.346	0.333	-0.071	0.200
	P	< 0.0001		< .0001	< .0001	< 0.0001	0.017	< .0001	< .0001	0.005	0.026	0.026	0.015	0.020	0.650	0.202
	N	41	49	47	49	49	49	49	49	48	48	48	48	48	42	42
P2- u/s.meter ^x	r	0.829	0.853	1.0000	0.607	0.606	0.189	0.524	0.514	0.555	0.464	0.317	0.424	0.416	0.029	0.175
	P	< 0.0001	<0.0001		< .0001	< 0.0001	0.202	0.0002	0.0002	< .0001	0.001	0.031	0.003	0.004	0.857	0.278
	N	41	47	47	47	47	47	47	47	46	46	46	46	46	40	40
Slaughter weight	r	0.733	0.739	0.607	1.0000	0.995	0.359	0.903	0.855	0.354	0.359	0.350	0.364	0.350	-0.024	0.193
	P	< 0.0001	<0.0001	< .0001		< 0.0001	0.010	< .0001	< .0001	0.011	0.010	0.012	0.009	0.012	0.874	0.207
	N	41	49	47	51	50	50	50	50	50	50	50	50	50	44	44
Warm carcass weight	r	0.736	0.743	0.606	0.995	1.0000	0.447	0.891	0.847	0.371	0.372	0.342	0.383	0.373	0.070	0.220
	P	< 0.0001	< .0001	< .0001	< .0001		0.001	< .0001	< .0001	0.002	0.001	0.004	0.001	0.001	0.592	0.091
	N	41	49	47	50	68	50	51	51	67	67	67	67	67	60	60
Dressing percentage	r	0.313	0.338	0.189	0.359	0.447	1.0000	0.298	0.405	-0.029	0.001	-0.064	-0.060	-0.057	-0.081	-0.305
	P	0.046	0.017	0.202	< .0001	0.001		0.035	0.003	0.842	0.991	0.659	0.680	0.694	0.601	0.046
	N	41	49	47	50	50	50	50	50	49	49	49	49	49	43	43
Carcass length	r	0.576	0.642	0.528	0.903	0.891	0.298	1.0000	0.823	0.259	0.266	0.295	0.289	0.277	-0.004	0.078
	P	< 0.0001	< .0001	0.0002	< .0001	< 0.0001	0.035		< .0001	0.068	0.061	0.037	0.041	0.050	0.975	0.613
	N	41	49	47	50	51	50	51	51	50	50	50	50	50	44	44
Chest depth	r	0.585	0.598	0.514	0.855	0.847	0.405	0.823	1.0000	0.224	0.152	0.188	0.171	0.136	-0.064	-0.010
	P	< 0.0001	< .0001	0.0002	< .0001	< 0.0001	0.003	< .0001		0.116	0.292	0.189	0.232	0.345	0.675	0.948
	N	41	49	47	50	51	50	51	51	50	50	50	50	50	44	44
Ovary length	r	0.446	0.396	0.555	0.354	0.371	-0.029	0.259	0.224	1.0000	0.813	0.722	0.877	0.759	-0.104	0.117
	P	0.003	< .0001	< .0001	0.011	0.002	0.842	0.068	0.116		< .0001	< .0001	< .0001	< 0.0001	0.311	0.255
	N	41	49	46	50	67	49	50	50	105	105	105	105	105	96	96
Ovary width	r	0.308	0.321	0.464	0.359	0.372	0.001	0.266	0.152	0.813	1.0000	0.699	0.864	0.776	0.026	0.050
	P	0.049	0.026	0.001	0.010	0.001	0.991	0.061	0.292	< .0001		< .0001	< .0001	< 0.0001	0.795	0.622
	N	41	48	46	50	67	49	50	50	105	105	105	105	105	96	96
Ovary thickness	r	0.345	0.320	0.317	0.350	0.342	-0.064	0.295	0.188	0.722	0.699	1.0000	0.861	0.774	-0.076	0.118
	P	0.027	0.026	0.031	0.012	0.004	0.659	0.037	0.189	< .0001	< .0001		< .0001	< 0.0001	0.458	0.250
	N	41	48	46	50	67	49	50	50	105	105	105	105	105	96	96
Ovary weight	r	0.362	0.346	0.424	0.364	0.383	-0.060	0.289	0.171	0.877	0.864	0.861	1.0000	0.866	-0.009	0.185
	P	0.019	0.015	0.003	0.009	0.001	0.680	0.041	0.232	< .0001	< .0001	< .0001		< 0.0001	0.926	0.070
	N	41	48	46	50	67	49	50	50	105	105	105	105	105	96	96
Ovary volume	r	0.323	0.333	0.416	0.350	0.373	-0.057	0.277	0.136	0.759	0.776	0.774	0.866	1.0000	-0.054	0.211
	P	0.039	0.020	0.004	0.012	0.001	0.694	0.050	0.345	< .0001	< .0001	< .0001	< .0001		0.599	0.038
	N	41	48	46	50	67	49	50	50	105	105	105	105	105	96	96
Follicle number	r	-0.121	-0.071	0.029	-0.024	0.070	-0.081	-0.004	-0.064	-0.104	0.026	-0.076	-0.009	-0.054	1.0000	-0.007
	P	0.485	0.650	0.857	0.874	0.592	0.601	0.975	0.675	0.311	0.795	0.458	0.926	0.599		0.942
	N	35	42	40	44	60	43	44	44	96	96	96	96	96	96	96
Largest follicle size	r	0.240	0.200	0.175	0.193	0.220	-0.305	0.078	-0.010	0.117	0.050	0.118	0.185	0.211	-0.007	1.0000
	P	0.163	0.202	0.278	0.207	0.091	0.046	0.613	0.948	0.255	0.622	0.250	0.070	0.038	0.942	
	N	35	42	40	44	60	43	44	44	96	96	96	96	96	96	96

^x = P2 backfat measurement by ultrasound meter, ^y = P2 backfat measurement by an intrascope, ^z = P2 backfat measurement by Hennessy Grading Probe

Table 4.12: Pearson Correlation coefficients between body development and gonadal measurements of boars, with probabilities and number of observations

*		P2(intrascop)e ^b ackfat ^y	P2(HGP) backfat ^z	P2(u/s.meter) ^x backfat	Slaughter weight	Warm carcass weight	Dressing percentage	Carcass length	Chest depth	Testis length	Testis width	Testis weight	Testis volume
P2 intrascop ^y backfat	r	1.0000	0.939	0.854	0.817	0.819	-0.010	0.610	0.712	0.696	0.687	0.690	0.688
	P		< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.945	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	45	45	45	45	45	45	40	45	45	45	45	45
P2 (HGP) ^z backfat	r	0.939	1.0000	0.907	0.752	0.751	0.043	0.549	0.669	0.652	0.666	0.654	0.625
	P	< 0.0001		< 0.0001	< 0.0001	< 0.0001	0.757	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	45	54	54	54	54	54	48	54	54	54	54	54
P2- (u/s.meter) ^x backfat	r	0.854	0.907	1.0000	0.673	0.654	-0.079	0.409	0.532	0.582	0.587	0.561	0.560
	P	< 0.0001	< 0.0001		< 0.0001	< 0.0001	0.568	0.003	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	45	54	54	54	54	54	48	54	54	54	54	54
Slaughter weight	r	0.817	0.752	0.673	1.0000	0.986	-0.045	0.890	0.839	0.841	0.849	0.870	0.871
	P	< 0.0001	< 0.0001	< 0.0001		< 0.0001	0.745	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	45	54	54	54	54	54	48	54	54	54	54	54
Warm carcass weight	r	0.819	0.751	0.654	0.986	1.0000	0.104	0.897	0.849	0.786	0.822	0.851	0.853
	P	< 0.0001	< 0.0001	< 0.0001	< 0.0001		0.451	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	45	54	54	54	72	54	50	56	72	72	72	72
Dressing percentage	r	-0.010	0.043	-0.079	-0.043	0.104	1.0000	-0.041	0.050	0.042	0.013	0.025	0.028
	P	0.945	0.757	0.568	0.745	0.451		0.780	0.715	0.760	0.923	0.855	0.837
	N	45	54	54	54	54	54	48	54	54	54	54	54
Carcass length	r	0.610	0.549	0.409	0.890	0.897	-0.041	1.0000	0.834	0.727	0.719	0.755	0.755
	P	< 0.0001	< 0.0001	0.003	< 0.0001	< 0.0001	0.780		< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	40	48	48	48	50	48	50	50	50	50	50	50
Chest depth	r	0.712	0.669	0.532	0.839	0.849	0.050	0.834	1.0000	0.721	0.709	0.714	0.714
	P	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.715	< 0.0001		< 0.0001	< 0.0001	< 0.0001	< 0.0001
	N	45	54	54	54	56	54	50	56	56	56	56	56
Testis length	r	0.696	0.652	0.582	0.841	0.786	0.042	0.727	0.721	1.0000	0.911	0.931	0.929
	P	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.760	< 0.0001	< 0.0001		< 0.0001	< 0.0001	< 0.0001
	N	45	54	54	54	72	54	50	56	56	115	115	115
Testis width	r	0.687	0.666	0.587	0.849	0.822	0.013	0.719	0.709	0.911	1.0000	0.964	0.963
	P	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.923	< 0.0001	< 0.0001	< 0.0001		< 0.0001	< 0.0001
	N	45	54	54	54	72	54	50	56	115	115	115	115
Testis weight	r	0.690	0.654	0.561	0.870	0.851	0.025	0.755	0.714	0.931	0.964	1.0000	0.998
	P	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.855	< 0.0001	< 0.0001	< 0.0001	< 0.0001		< 0.0001
	N	45	54	54	54	72	54	50	56	115	115	115	115
Testis volume	r	0.688	0.652	0.560	0.871	0.853	0.028	0.755	0.714	0.929	0.963	0.998	1.0000
	P	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.837	< 0.0001	< 0.0001	< 0.0001	< 0.0001	< 0.0001	
	N	45	54	54	54	72	54	50	56	115	115	115	115

^x = P2 backfat measurement by ultrasound meter, ^y = P2 backfat measurement by an intrascop, ^z = P2 backfat measurement by Hennessy Grading Probe

4.8.2 Correlations between gonadal measurements

In gilts, correlations between ovary length, ovary width, ovary thickness, ovary weight and ovary volume were positive and relatively high, $r = 0.699$ to 0.877 ; ($P < 0.0001$) (see Table 4.15). However, the correlations between these gonadal measurements with follicle number and size of the largest follicle were very poor, $r = 0.164$ to $r = 0.211$. In addition, correlation coefficients of follicle number and largest follicle size were not significant $P > 0.05$, except for correlation between size of the largest follicle and ovary volume which had $r = 0.211$ and $P = 0.038$. The results also show that follicle number and size of the largest follicle are not dependent on ovary length, ovary width, ovary weight, ovary thickness and ovary volume. The follicle number, to some extent depends on the genotype (breed) and also on the stage of the estrous cycle. If the gilts were slaughtered during follicular phase, then there would be higher chances for more follicles of bigger size than when the gilt was slaughtered during luteal phase.

The testicular measurements of boars were also highly and positively correlated, $r = 0.911$ to $r = 0.998$; $P < 0.0001$ (see Table 4.16) to each other. The correlations between gonadal measurements of boars were higher than correlations between gonadal measurements of gilts. This shows that boars testicular measurements are very closely related to each other than gilts ovarian measurements. Of particular interest was the extremely high correlations between testis weight and testis volume, $r = 0.998$; $P < 0.0001$. However, it is not surprising though that these two testicular measurements are highly correlated because weight estimates volume, which can be calculated as length X width X height, while width and length are just linear measurements (Schinkel *et al.*, 1983). These high correlations with each other indicates that selecting for one will invariably mean selecting for the other, and also the prediction of the other could be possible with one measurement known.

4.8.3 Correlations between gonadal and body development measurements

In gilts, correlations between gonadal and body development measurements ranged from low to moderate. The correlation coefficient values ranged from $r = -0.305$ to $r = 0.555$ (see Tables 4.11 and 4.12). Dressing percentages of gilts were also poorly correlated with gonadal measurements and they were negatively correlated with ovary lengths ($r = -0.029$) ovary thicknesses ($r = -0.064$) ovary weights ($r = -0.060$) ovary volumes ($r = -0.057$) follicle numbers ($r = -0.081$) and size of the largest follicle ($r = -0.305$). All correlation coefficients

between dressing percentages and gonadal measurements of gilts were poor and non-significant ($P > 0.05$) except for correlations between dressing percentages and size of the largest follicle, $r = -0.305$; $P = 0.046$. Extremely low correlations were also found between follicle number and size of the largest follicle with other body development measurements, while these two measurements had low correlations with all body development measurements. Follicle number had negative correlations with P2 (intrascope) ($r = -0.121$) P2 (HGP) ($r = -0.071$) slaughter weight ($r = -0.024$) dressing percentage ($r = -0.081$) carcass length ($r = -0.004$) and chest depth ($r = -0.064$) and some of these correlations were low and close to zero. Except for correlation between dressing percentage and size of the largest follicle ($r = -0.305$; $P = 0.046$) and between size of largest follicle and ovary volume ($r = 0.211$; $P = 0.038$) correlation coefficients between follicle number and size of the largest follicle with other gilts carcass or gonadal measurements were not significant ($P > 0.05$). This therefore indicates that follicle number and size of the largest follicle are not influenced by ovary length, ovary width, ovary thickness, ovary weight and ovary volume or any of the body development measurements in the study.

Apart from the low correlations between dressing percentage with gonadal measurements in boars, correlations between gonadal measurements and body development measurements of boars were positive and ranged from moderate to high ($r = 0.560$ to $r = 0.871$; $P < 0.0001$) (see Table 4.16). Slaughter weights seemed to be highly correlated with gonadal measurements of boars ($r = 0.841$ to $r = 0.871$; $P < 0.0001$) than other carcass measurement, while P2 (ultrasound) backfat thickness had moderate correlation coefficients (range of $r = 0.560$ to $r = 0.587$) with gonadal measurements of boars. This shows that testicular measurements of boars grow linearly with increasing live weight. Therefore, selection based on testicular measurements will automatically select for higher live weight at slaughter in boars. Also, high correlations between gonadal and body development measurements can make their predictions possible.

4.9 Discussion

The purpose of this study was to compare and correlate gonadal development and body development measurements of commercial pig genotypes found in South Africa. Some of these genotypes used in the study were crossbred pigs that take advantage of more heterosis while other genotypes were synthetic pig breeds or lines developed from other purebreds.

The first genotype (Genotype 1) was a cross between two most popular breeds in South Africa. These are chosen for their superior maternal qualities and growth potential with outstanding carcass characteristics. The F1 sows of these breeds are commonly used to produce slaughter generation pigs since they exhibit maximum hybrid vigour (Rossouw, 1998). The second genotype (Genotype 2) is one of selected lines which are selected to maintain six different maternal lines and five different sire lines, which can be combined to satisfy different markets (Antunes, 2001). Unfortunately, very little information is available on what type of Genotype 2 has been imported to South Africa. Genotype 3 pigs were meat type pigs where a meat type terminal sire has been used to produce slaughter generation. In South Africa, this genotype is commonly used as meat-type terminal sire in South Africa because of its superior growth and high efficiency in conversion of feed to lean meat. The fourth genotype (Genotype 4) was a meat-type synthetic pig breed developed for the local production environment. Being a synthetic breed, it has the advantage of additive genetic effects of the qualities of the parent breeds and some heterosis effect (Rossouw, 1998). Genotype 5 pigs were a line which have been developed by sampling the world's diverse pig populations including Large White, Landrace, Duroc, Hampshire, Berkshire, Pietrain, Meishan and others (Sygen International plc, 2002).

4.9.1 The influence of genotype on gonadal development

In summary, the overall influence of genotype effect on gonadal measurements was not significant. One possible explanation for this non-significant genotype influence on gonadal measurements of both boars and gilts could be the small sample sizes of animals used to represent genotypes at different ages. However, least square means comparisons between individual genotypes by the PDIFF procedure revealed slight differences between genotypes. For gonadal development of gilts, the Genotype 5 gilts (2.41 cm) were shorter than Genotype 4 and Genotype 2 gilts in ovary lengths, which had ovary lengths of 2.72 cm and 2.74 cm respectively. The Genotype 2 gilts also out-performed the Genotype 5 gilts, with 4.48 g vs. 3.45 g and 4.50 cm³ vs. 3.31 cm³ for ovary weights and ovary volumes respectively. From an economic point of view, the high ovarian development in Genotype 2 gilts as compared to Genotype 5 gilts can be taken to imply early gonadal development in Genotype 2 gilts, therefore reaching puberty earlier than Genotype 5 gilts.

In gonadal development of boars, the Genotype 5 was found to be inferior to the Genotype 2 in testis weight and testis volume. Research has shown that testicular growth may be an indicator of the reproductive performance and that boars with larger testes at a constant age generally have greater sperm numbers and superior mating efficiency (Schinkel *et al.*, 1983). Therefore, this suggests that the Genotype 2 boar with its heavier testes and larger testes volume can be assumed to have greater sperm production potential and high mating efficiency than Genotype 5 boar.

Although other genotypes were not different from each other in gonadal development for both boars and gilts, it is evident that Genotype 5 had smaller gonads compared to Genotype 2.

4.9.2 The influence of genotype on body development

With body development measurements, the influence of genotype was only significant for slaughter and warm carcass weights. Significant ($P < 0.05$) differences were obtained between the Genotype 2 and Genotype 5 pigs. Gilts and boars from Genotype 2 were superior to Genotype 5 pigs in terms of slaughter weights and warm carcass weights. Genotype 3 came second to Genotype 2 in slaughter and warm carcass weights and was also heavier than Genotype 5 pigs. These results show that Genotype 2 pigs have higher growth potential than Genotype 5 pigs for both gilts and boars.

Carcass lengths and chest depths in boars and gilts were not different between genotypes. As far as P2 backfat thicknesses were concerned, Genotype 5 pigs were the leanest, with lower backfat thicknesses than other genotypes. Genotype 2, which proved to have a higher growth potential than the Genotype 5, also had thicker P2 backfat thicknesses than Genotype 5 pigs. However, the Genotype 2 pigs were not significantly different to the Genotype 1, Genotype 3 and Genotype 4 pigs in P2 backfat thickness. Genotype 2 boars had thicker backfat (15.66 mm vs. 12.66 mm) thickness compared to Genotype 5 boars when using an intrascope ($P = 0.0805$). Backfat thickness (intrascope) for Genotype 2 boars did not differ much from those reported in a comparative study by Antunes (2001). In this comparative study, the male progeny of Genotype 2 hybrids had average backfat thickness of 17.4mm (high growth rate line) and 16.10mm (high meat production line). In another Brazilian study also reported by Antunes (2001) the Genotype 2 progeny of high growth rate sires had average intramuscular backfat thickness of 15.40mm while other genotypes had an average backfat thickness of 19.70mm. Genotype 2 backfat thickness from the Brazilian study is very close to the backfat thickness from this current study, 15.40mm vs. 15.66mm respectively. Fat is essential for adequate reproduction, therefore thinner backfat may be related to poor reproductive capacity. It has been hypothesized that leptin (a newly discovered hormone secreted by the adipose tissue into the blood stream) may be a regulator for satiety, metabolic activity and reproductive function (Cook *et al.*, 2005). Reports have indicated that the ovary also appears to be the target organ for this hormone because ovarian leptin receptors have been identified in some species including pigs. In rodents, leptin is reported to increase total number of follicles and synthesis of estrogen but decreases progesterone in the ovary of rodents in vitro (Cook *et al.*, 2005). Maybe this hormone may also explain the differences of gonadal development between the different genotypes in this study.

4.9.3 The influence of sex on backfat thickness measurements

As already mentioned, gilts had the highest P2 backfat thickness than boars, in pooled results which did not consider individual genotype. Gilts had thicker P2 backfat thicknesses than boars. The results obtained from this study are in accordance with those obtained by Browne, (1984); Walstra, (1980); Beltranena *et al.*, (2004). In Browne (1984)'s comparative study of purebred and crossbred gilts and boars slaughtered at 90kg and 120kg, all the male progeny had highly significant thinner backfat than female progeny at both 90kg and 120kg slaughter

weights. Walstra (1980) also reported similar results in a study investigating growth and carcass composition from birth to maturity in relation to feeding level and sex in Dutch Landrace pigs. In the report of Walstra (1980) castrates and sows had more backfat thickness than boars. Also, backfat thickness was lower in boars than in sows and castrates while in a number of cases sows showed a smaller backfat depth than castrated pigs. Compared to males and females, castrated pigs have a higher propensity to gain fat. On the other hand, backfat thickness is influenced by nutritional level and stage of growth. High nutritional level would increase backfat thickness and also as the animal approaches maturity, more fat is deposited in the body as backfat and therefore this explains the thicker backfat in castrated pigs.

The current change in consumer attitude towards higher demand for leaner meat would mean that castrates would no longer be desirable because of their high fat gain. However, the rearing of entire males in swine production is hampered by the existence of boar taint, a problem that is encountered only in pigs. Castration of meat producing males has been widely used in livestock production for a very long time, for a number of reasons. These reasons include easier control of behaviour, high propensity of castrates to deposit fat, a commodity that has been in high demand until quite recently. In other livestock like cattle and sheep, the change of consumer attitude towards a higher demand for leaner meat and the lower production cost associated with entire males have led to the elimination of castration. Entire males need less feed to grow, have better feed conversion efficiency and may sometimes grow faster than castrates (Bonneau and Squires, 2000). The decreased amount of adipose tissue in intact males is another important advantage of intact males. Thinner backfat thickness in males would therefore result in higher grading carcasses. The decreased intermuscular fat and meat cuts from intact males are usually more appealing to the consumer. Research has shown that the characteristics of muscle and adipose tissue differ between intact males and castrates. The lower lipid and the higher content of unsaturated fatty acids in adipose tissue of intact males may be regarded as favourable from the dietic point of view (Bonneau and Squires, 2000).

4.9.4 The influence of sex on other body development measurements

The influence of sex on other body development measurements (slaughter weight, warm carcass weight, dressing percentage, carcass length and chest depth) besides P2 backfat was non-significant. However, it should be borne in mind that the results are pooled data for all

genotypes and only comparisons between gilts and boars could be done. Perhaps the results could have been different if comparisons were made within genotype. A study by Walstra (1980) on Dutch Landrace pigs showed that as pigs reach maturity, boars become longer than gilts. Also, depending on the feeding level, there were differences in carcass length of gilts, boars and castrates. Boars were reported to be longer than castrates at both high plane feeding and low plane feeding based on carcass weight but they were shorter at a given muscle and bone weight. Furthermore, castrates were shorter than sows at the restricted feeding level when based on carcass weight. The results from this current study did not show any difference in carcass length between gilts and boars. The reason for this could be the small sample size of animals of different sexes representing different genotypes at different slaughter ages in this study. This therefore warranted the pooling of the results together and not to consider sex effect on individual genotypes.

4.9.5 Growth in gonadal and body development measurements

Generally, all measurements (both gonadal and body development measurements) increased as slaughter age increased. This study was designed in such a way that animals will be slaughtered at a certain average weight, therefore it was to be expected that some changes would occur in measurements. Nonetheless, the pattern of growth was somewhat different between gonadal measurements and body developments. Body development measurements growths seemed to be more linear than gonadal measurements growth. This could be attributed to the significant changes of ovarian and testicular measurements at the time of puberty. Differences in growth pattern between genotypes may be due to different genetic growth potential of these genotypes, and also due to individual variation within genotype. Of particular interest were the low gonadal measurements at 144 days, particularly in gilts. This could have been due to change of diet, which may have affected ovarian developments in gilts.

4.9.6 Relationship between gonadal and body development measurements

In commercial pig production, efficient production of better quality pork is one of the most outstanding objectives. However, maintaining high levels of reproductive efficiency is also essential, as improving reproductive efficiency will increase overall efficiency of pig production. This is why it is important to have economic production traits being positively correlated with reproductive traits. A selection improvement in either production traits or

reproduction traits should not negatively affect or compromise the other. Body development measurements such as length, chest depth, loin eye muscle, colour, backfat thickness, firmness of fat etc, are indicators of body composition and meat quality of the animal. On the other hand testicular and ovarian growth may be indicators of reproductive performance in boars and gilts respectively (Schinkel *et al.*, 1983). Growth and development of the testes and ovaries are of prime importance as these are the main reproductive organs in boars and gilts respectively.

For both sexes (gilts and boars) body development measurements, excluding dressing percentage, had positive and moderate to high correlations with each other $r = 0.409$ to $r = 0.995$. Dressing percentages on the other hand had low to moderate correlations ($r = -0.079$ to $r = 0.559$) with other body development measurements. Dressing percentages correlations coefficients were non-significant ($P > 0.05$) with other carcass measurement. One possible reason for these non-significant dressing percentage coefficients in gilts and boars could be the fact that warm carcass weight was used to estimate dressing percentage instead of the normal cold carcass weight. This means that the dressing percentage from this study could not be compared with those from other studies because of the compounding effect of drip loss (which had not occurred in warm carcass weight).

With gonadal measurements, correlations were different between gilts and boars. For gilts, correlations ranged from low to high, $r = 0.164$ to $r = 0.887$. The low correlations in gonadal measurements of gilts were found between follicle number and size of the largest follicle with other ovarian measurements. These low phenotypic correlations between these two measurements (follicle number and size of the largest follicle) with other body development measurements indicate that there is little or no relationship between them and other ovarian measurements. In contrast, gonadal measurements of boars were more highly correlated ($r = 0.911$ to $r = 0.998$) than gonadal measurements of gilts. This only shows the close relationship between testicular measurements of boars. Heritability traits of testes length have been estimated at $h^2 = 0.33$ ($h^2 = 0.30 - 0.39$) while h^2 for testes weight is 0.44 ($h^2 = 0.24 - 0.73$) (Rothschild and Bidanel (1998) as quoted by Moeller, 2002). Correlations between testes weight and sperm production is reported as a positive one. Therefore, selection for testes weight can result in lower age at puberty, increased daily sperm production and increased

sperm concentration (Moeller, 2002). Improving one trait would therefore result in an improvement in the other.

Correlations between body development measurements and gonadal measurements in the gilts were moderate ($r = -0.305$ to $r = 0.555$) although follicle number was negatively correlated to some body development measurements. On the other hand, gonadal measurements of boars were more highly correlated to body development measurements ($r = 0.560$ to $r = 0.871$). The high and positive correlation between gonadal measurements and body development measurements of boars indicate the close relationship between gonadal and carcass traits in boars.

CHAPTER 5

5. CONCLUSION

It is concluded from the study that although overall genotype did not influence gonadal development in these commercial genotypes, there were differences in gonadal development between the Genotype 2 and Genotype 5 pigs. Genotype 2 boars and gilts had larger gonads than their Genotype 5 counterparts, while Genotype 4, Genotype 3 and Genotype 1 pigs were intermediate. With respect to body development measurements, the effect of genotype was only significant for slaughter weight and warm carcass weight. For both gilts and boars, there were significant differences between the Genotype 2 and Genotype 5 pigs. Genotype 2 pigs were superior to Genotype 5 pigs in slaughter and warm carcass weights and this shows the difference in growth potential of the two genotypes. Genotype 5 pigs turned out to be the leanest pig genotype as reflected by its thinner P2 backfat thickness.

Consumers nowadays want lean and high quality meat. On the other hand, body fat in pigs is costly to produce because it has to be eliminated in the process of providing lean retail meat cuts, which consumers prefer. Therefore, any effort to improve leanness of pigs is beneficial to both pig producers and consumers in providing lean meat as thinner backfat results in higher carcass classification. In this respect, Genotype 5 therefore seems most suitable for the production of top quality pork for both producers and consumers because of its low backfat content. On the other hand, it could be argued that the low backfat thickness in Genotype 5 genotype was because of its slow grow potential because Genotype 5 tended to have lighter slaughter weight than other genotypes. If the classification was based on the liveweight at slaughter, then Genotype 5 pigs would fetch lower prices than other genotypes whereas if the classification is determined by the backfat thickness, then Genotype 5 would grade higher by producing lean pork. The low backfat in Genotype 5 pigs supports findings from other studies that selecting against high backfat delays sexual maturation because Genotype 5 pigs had thinner P2 backfat thickness, and slower gonadal development.

With the exclusion of follicle number, size of the largest follicle and dressing percentage, strong positive relationships existed between gonadal and body development measurements.

The relationship was stronger in boars than in gilts, indicating that testicular traits in boars are dependent on the growth rate of the animal. Not many studies of this kind have been carried out locally; therefore it is recommended that similar studies be carried out to get more information regarding gonadal development of commercial pig genotypes in South Africa.

CHAPTER 6

6. CRITICAL EVALUATION

Firstly, I would like to express my gratefulness for the scientific experience and knowledge that this project has afforded me. I am very optimistic that the knowledge and experience I have gained doing this project will benefit my present and future career in a positive way.

Coming back to the study, it has come to my observation that there is very little literature material available on this topic locally. There haven't been many similar studies of this kind before hence the little literature available. It is therefore imperative that other similar studies are carried out in the near future so that more information can be generated regarding gonadal development of commercial pig genotypes in South Africa. These studies will also help to us to see how selection against high backfat thickness has affected gonadal development and meat and carcass traits in local commercial pigs.

The following problems were encountered during the execution of this study:

- Due to the natural design of the study where animals were slaughtered at two abattoirs, shortage of labour became a major problem. This was more pronounced when animals were slaughtered at Reitvlei (RTV abattoir) which is a commercial abattoir. Because of large numbers of pigs that have to be slaughtered within a limited time during the day, it sometimes got so problematic to the extent that the collection of gonadal organs for all pigs became extremely difficult. Slaughtering at ARC meat center would have been more ideal, but because of the limitation on capacity of the facilities, most of the animals had to be slaughtered at Reitvlei abattoir.
- On two occasions, animals were slaughtered without our presence at Reitvlei abattoir therefore this led to loss of gonadal data because I missed collecting blood samples and gonadal organs. The slaughter ages whose data were missed were on 158 days and 186 days.

- Although blood samples were collected, hormonal assay on blood plasma was not performed due to shortage of facilities and time constraint although all the effort had been made to collect blood samples and centrifuge to extract blood plasma. It must be pointed out at this juncture that blood samples collection was not an easy task at Reitvlei because the process of killing the animals was very fast and some animals passed without their blood samples being collected. There was also a possibility of blood samples contamination between different animals. This is because blood collection had to be made fast and quickly before the animal got hoisted away, therefore leaving no time to clean the hands in between the blood collections.
- There were possible sources of variation and error within the experiment that could have affected the results somehow. In some cases, there were unbalanced numbers of animals between the two housing systems. This led to unbalanced data for genotypes at different slaughter ages. Because of this problem, the data for individual housing pigs could not be analyzed due to small and unbalanced genotype sample size.

CHAPTER 7

7. REFERENCES

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