CHAPTER 1

INTRODUCTION

1.0 Introduction

1.1 Background

Chicken production plays an important role in many rural house holds. Chicken production is an important source of income and employment and contributes substantially to food security among rural people in Africa (Yami, 1995). Chickens provide humans with food in the form of meat and eggs. Chicken meat and eggs provide protein of high biological value. Chicken meat and eggs are also good sources of vitamins, especially thiamin, ribloflavin and niacin (Robert, 1992).

Allowing birds an unlimited supply of food can result in consumption in excess of the bird's requirements for maintenance and production, and in excess energy being converted into fat (Fontana *et al.,* 1992). Excessive fat reduces carcass quality and feed efficiency. Reducing feed intake and fat deposition in broiler chicken production through supplementation with *Hoodia gordonii* meal could be of major nutritional importance, however, information on the effects of *Hoodia gordonii* meal on food intake and fat deposition in broiler chickens are not yet known.

The ingredient *Hoodia gordonii* a dietary supplement is a cactus-like plant with medicinal uses. It grows naturally in Namibia and in the Kalahari deserts of South Africa to about 1 meter of height and to reach this, it takes between three to four years. Originally the plant was used by the indigenous people of the areas to treat indigestion and infections. *Hoodia* is a natural substance which has been used by native San people

of Kalahari for centuries to help ward off hunger and thirst during extended travels or hunting trips in the desert (BBC, 2003). Recently, *Hoodia gordonii* has been marketed as an appetite suppressant (Engler, 2007). *Hoodia* (also known as P 57) is easily broken down by the liver, and this call for curiosity or the question of whether a *Hoodia gordonii* supplement contain enough of the active ingredient to have an impact on appetite suppression. The Council for Scientific and Industrial Research (CSIR) joined forces with the South African San Council and agreed to work together on the usage of indigenous plants including *Hoodia gordonii* for the benefit of both parties. They also committed themselves to the conservation of natural resources (Stahl, 2004). In 1977, CSIR isolated the ingredient in *Hoodia*-now known a P57 that is responsible for its appetite-suppressant effect, and patented it in 1996.

1.2 Problem statement

Broiler chickens are selected for high food consumption for the production of high muscle mass. Thus, modern commercial broiler chicken strains selected for rapid growth and high meat yield have lost the ability to regulate voluntary intake commensurate with energy requirements (Richards, 2003). However, increased growth rate has caused a greater incidence of metabolic disorders such as ascites, sudden death syndrome, skeletal abnormalities and increased fat deposition (Urdaneta and Leeson, 2000)

Excessive fat is one of the main problems faced by the broiler industry, since it does not just reduce carcass quality and feed efficiency but also causes rejection of the meat by the consumers and difficulties in processing the meat (Macajova *et al.*, 2003). The results of

many human studies have related high dietary fat intake to incidents of cardiovascular diseases and cancer (Lichtenstein, 1999). Coronary heart diseases and arteriosclerosis are strongly related to the dietary intake of cholesterol and saturated fatty acids and are among the most important causes of human mortalities (Sacks, 2002).

1.3 Motivation

An effective mechanism for regulating feed intake could greatly enhance the profitability of the poultry industry. A feed restriction program is one strategy that can be used to decrease to some extent feed consumption and hence, growth rate, in order to alleviate the occurrence of metabolic disorders, skeletal abnormalities and excessive fat deposition in broiler chickens. One feed restriction method that has been used to suppress feed intake in broiler chickens is the use of chemicals or pharmacological agents with anorectic activity. Thus, appetite control in poultry could be of importance in reducing fat deposition and hence acceptability of such meat by the consumers. *Hoodia gordonii* has been used by San people to suppress appetite during hunting expeditions in the Kalahari desert (Holt, 2005). The chemical P57 in *Hoodia gordonii* mimics adenosine triphosphate (ATP) in the hypothalamus to cause satiety in rats, thus, reducing appetite (McLean and Luo, 2004).

1.4 Aim and objectives

1.4.1 Aim

The aim of this study was to investigate the potential of improving carcass characteristics of broiler chickens through the use of *Hoodia gordonii* meal as a feed supplement at finisher stage.

1.4.2 Objectives

The objectives of this study were to:

1. Determine the effect of level of *Hoodia gordonii* meal supplementation at finisher stage on productivity and carcass characteristics of male and female Ross 308 broiler chickens.
2. Determine the effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on productivity and carcass characteristics of Ross 308 broiler chickens.

CHAPTER 2

LITERATURE REVIEW

2.1 Introduction

Modern commercial broiler chicken is the product of intensive selection over many generations for rapid growth and enhanced muscle mass. Selection for these economically important traits has been accompanied by an increase in voluntary feed intake, resulting in birds that do not adequately regulate feed intake and energy balance (Richard, 2003). Concentrated energy diets are used to raise broiler chickens to maximize growth rate and reduce number of days needed to reach market weight. However, feed energy in excess of maintenance correlates positively with daily fat deposition in most animals (Leeson, 2000). This problem most commonly occurs in broiler chickens that are fed *ad libitum* (Pasternak and Shalev, 1983).

2.2 Regulation of feed intake

The level of feed consumption is important in determining growth rate and body composition in animals. In animals, the body is subject to homeostatic control mediated by adjustments in feed intake and energy expenditure (Ferket and Gernat, 2006; McMinn *et al.,* 2000). Boswell (2005) research that focused on the neurochemical basis of the regulation of energy homeostasis by the hypothalamus, established the existence of a neuronal network of particular importance for the regulation of food intake and energy balance. The circuitry involves centers in the hypothalamus on the arcuate nucleus (infundibular nucleus in avian) of the medio-basal hypothalamus that contains two separate populations of neuronal cell types. One of which expresses neuropeptide Y

(NPY) and agouti-related protein (AGRP) messenger ribonucleic acid's (mRNA's), while others produce α-melanocyte-stimulating hormone (α-MSH) and β-endorphin from a common precursor, pro-opiomelanocortin (POMC), cocaine and amphetamine-regulated transcript (CART). Neuropeptide Y/agouti-related protein neurons and POMC/ CART neurons exert opposing influences on food intake, metabolic rate and body mass, with the former being orexigenic (stimulating food intake) and the latter anorexigenic (inhibiting food intake).

Regulation of feed intake has two key components: one that involves short-term control of feeding and the other one that controls long-term regulation of feed intake. Control of feed intake in the short-term (i.e. meal to meal) involves hormonal and neural signals that originate primarily in the gut but also in the pancreas and liver. These satiety signals are generated in response to nutrient content and physical presence of feed or specific feed components in the gut.

Short-term regulation of feed consumption with satiety signals originating in the gut are transmitted to the brainstem via the activation of neural or vagal afferent pathways (Denbow, 1994; Woods *et al.*, 1998). Long-term regulation of feed intake and energy expenditure results in homeostasis of body energy stores. In addition to meeting immediate energy demands, feed intake can be adjusted to ensure that energy and nutrients are stored in anticipation of period of high demand or period of feed shortage (McMinn *et al.*, 2000; Blevins *et al.*, 2002; Richards, 2003).

The hypothalamus contains peptidergic neuronal pathways that are involved in the regulation of feed intake and energy homeostasis. The pathways are divided into anabolic and catabolic pathways (Woods *et al.*, 1998). Stimulation of anabolic pathway mediates a

net increase in energy intake and storage, whereas stimulation of catabolic pathway results in a net decrease in energy intake and storage (Hillgartner *et al.,* 1995; Jensen, 2001). Short-term and long-term regulation of feed intake and energy balance is also controlled by the external (e.g. environmental cues, sensory cues and diet composition) and internal (e.g. metabolic and hormonal) factors (Berhoud, 2002; Richards, 2003). However, only metabolic and hormonal factors will be discussed because of their relevance to the present study.

Circulating levels of metabolites (Glucose, triglycerides, free fatty acids and amino acids) might serve as signals for energy or nutritional status to the brain. Metabolic pathways and metabolites produced by them would be integrated into the regulatory scheme for feed intake and energy metabolism. When energy is consumed in excess of the quantity needed to meet the requirements, it is generally stored in the form of triglycerides which are products of the lipogenic metabolic pathway. In birds, the major site of lipogenesis (i.e., the de novo synthesis of triglycerides from glucose) is the liver (Hillgartner *et al.*, 1995; Richards, 2003), with adipose tissue serving primarily as a repository for accumulated triglycerides.

Energy is sequestered in the form of high energy phosphate bonds of adenosine triphosphate (ATP) that are subsequently used in energy requiring reactions in metabolism. In most non-ruminants there is evidence that blood glucose is negatively related to feed intake over short term, and that hunger contractions of stomach are more pronounced when blood glucose is low (Pond *et al.,* 1995; Simon *et al.,* 2000). Glucose is

normally the main metabolic fuel of the brain, and falls in blood glucose or blockade of neuronal glucose utilization stimulate feeding. It has long been recognized that specific parts of the central nervous system (CNS) contain neurons that can detect changes in ambient glucose level, but their place in the hierarchy of the CNS systems that regulate feeding is uncertain. However, it is only now becoming clear that glucose-sensing neurons may communicate extensively with other appetite-regulating neuronal systems (Williams *et al.,* 2002).

Hormones play an important role in appetite and satiety. Cholecystokinin (CCK) is probably one of the most important hormones affecting satiety (Lawrence and Fowler, 1998). Cholecystokinin is a peptide that is found throughout the brain and in neurons and endocrine cells of the gastrointestinal tract. Cholecystokinin acts as a neurotransmitter or neuromodulator within two different brain regions to produce satiety: one region that includes the nucleus tractus solitarius in the hindbrain, and another more distributed region within the medial-basal hypothalamus. This conclusion is supported by studies showing that food intake releases hypothalamic CCK (De Fanti *et al.,* 1998), site-specific injections of CCK into the medial-basal hypothalamus and the caudal brain stem inhibit food intake (Blevins *et al.,* 2000), and brain injections of CCK antisera and CCK receptor antagonists stimulate food intake (Reidelberger *et al.,* 2003). Cholecystokinin may be involved in the control of food intake and in regulating energy balance (Lawrence and Fowler, 1998). The release of CCK in the gut is stimulated by protein and fat. Cholecystokinin slows gastric emptying and reduces food intake in both animals and humans by terminating the feeding episode. According to Richards (2003), CCK has been reported as satiety signal in poultry species. Vagotomy blocks the effect of CCK on feed

intake, indicating that gastrointestinal CCK regulates food intake primarily through vagal afferent signals to the brain rather than through endocrine system. Cholecystokenin may interact with long term signals of energy balance such as estrogen, leptin and insulin (Considine, 2002).

The primary function of the pancreatic hormone insulin is the regulation of glucose homeostasis. The fact that pancreas secretes this hormone in response to feeding, also places it in a position to signal energy intake to the central nervous system. Insulin is one of the main metabolic hormones and is significantly involved in the regulation of energy processes. Insulin enhances glucose disposal, storage and oxidation in the muscles. Insulin also stimulates leptin secretion; on the other hand administration of leptin reduces plasma insulin levels (Macajova *et al.,* 2003). Secretion of insulin is stimulated by glucose and amino acids but not dietary fat. Insulin receptors are found in many brain areas and are localized in the hypothalamus nuclei, regulating feeding behavior. Circulating insulin levels are proportional to the amount of body fat, therefore, insulin not only signals nutrient intake but also acts as a measure of energy stores in the body (Considine, 2002). Insulin is transported into the brain where it exerts a catabolic influence, in part by decreasing the expression of NPY mRNA in the arcuate nucleus (Schwartz *et al.*, 2000). Adenosine triphosphate-sensitive potassium (K_{ATP}) channels are expressed in the hypothalamus and can be activated by insulin and leptin in selective hypothalamic neurons. Central stimulation of K_{ATP} channels lowers blood glucose by inhibiting glucose production (Pocai *et al.*, 2005).

Both adipose tissue and liver are believed to be important peripheral sites involved in food intake, body weight and lipid regulation in birds (Lamosova *et al.,* 2004). The signal between the peripheral lipid stores and the CNS is exerted by the hormone leptin. It circulates in the blood at levels correlated with the body fat mass and controls food intake and energy homeostasis (Ashwell *et al.,* 1999).

Leptin is a hormone produced mainly by adipocyte cells and plays an important role as a signal of the body fat content to the brain, where it regulates food intake and energy expenditure (Macajova *et al.,* 2003). Leptin interacts with several central neuroendocrine systems including neuropeptide Y, leading to inhibition of food intake (Trayhurn and Beattie, 2001). Leptin exerts catabolic effects in the brain by up-regulating the activity of POMC/CART neurons and down regulating the activity of NPY/ACRP neurons (Jobst *et al.,* 2004). Leptin is not only important in regulation of food intake and energy balance, but it appears increasingly as a general metabolic hormone involved in many physiological processes including inhibition of insulin secretion by β-cells of pancreas, stimulation of glucose utilization and stimulation of lipolysis in the adipocytes (Macajova *et al.,* 2003).

2.3 Methods used to control feed intake

Several approaches, both qualitative and quantitative have been employed to restrict nutrient or caloric intake in broiler chickens in order to reduce cost of feeding, improve feed efficiency and reduce excessive abdominal fat deposition and carcass fat among other problems associated with ad libitum feeding (Oyedeji and Atteh, 2005). Qualitative and quantitative feed restrictions are procedures that can be applied to manipulate the

feeding strategies of poultry in order to decrease fat deposition (Urdaneta and Leeson, 2002). Some quantitative feed restriction procedures used include physical feed restriction and lighting while qualitative feed restriction includes diet dilution, nutrient density of the diets and chemical methods.

2.3.1 Physical feed restriction

Physical feed restriction is a procedure that limits growth rate by limiting amount of feed chickens receive per day. This feed restriction procedure is aimed at avoiding rapid growth and high body weights which are associated with pathological conditions, such as ascites, lameness, and mortality (Mench, 2002). However, severe quantitative feed restriction frequently results in abnormal behaviours such as overdrinking, stereotypic pecking at non-food objects, and increased pacing (Savory and Maros, 1993; Hocking *et al.,* 1997). Such behaviours are as a result of frustration due to unfulfilled feeding motivation (Savory *et al.,* 1996). This procedure causes a welfare problem for chickens reared under this system. In a study conducted by Dozier *et al.* (2003), broiler chickens subjected to early skip a day feed removal had decreased final live weight and feed consumption and improved feed conversion ratio when compared with birds fed *ad libitum*. In comparison with birds fed *ad libitum*, early skip a day feed removal suppressed body weight at the end of last feed removal period by 46% for males and 43% for females (Dozier *et al.,* 2003). Deaton (1995) restricted birds to 90, 75 or 60% of the previous 24-hour feed consumption of full-fed controls from 7 to 14 days and showed significant improvement in feed conversion in restricted birds.

Quantitative feed restriction has been observed to reduce mortality and culling (Fontana e*t al.*, 1992; Robinson *et al.,* 1992), improve feed conversion ratio (Fontana *et al.,* 1992; Deaton, 1995; Plavnik and Hurwitz, 1988; Lee and Lesson, 2001) and allow a complete recovery of body weight if the degree of restriction was not too severe and slaughter ages were extended beyond 6 weeks (Deaton, 1995; Plavnik and Hurtwiz, 1988). Dozier *et al.* (2003), referred to feed restriction programs of yielding inconsistent results in the literature and that variation maybe partially attributed to differences in bird management, lighting, strain and ventilation. Benyi and Habi (1998), with a 30% food restriction, reported less abdominal fat deposition than when there was a 15% food reduction, reduction of feeding time by 2 days per week or *ad libitum* feeding. Tumova *et al.* (2002) reported an accelerated growth rate on the previously restricted birds at the age of 21 days resulting in a similar daily weight gain with full-fed cockerel, and from the age of 35 days daily weight gain of the previously restricted birds was higher at about 15% than full-fed broiler chickens. No significant differences were observed with regard to feed conversion ratio and total carcass fat, although the restricted birds showed a tendency towards a higher abdominal fat content. Rosebrough and McMurtry (1993) suggested that even feed-restricted broiler chickens are still over-eating and that the level of feed intake may control de novo lipogenesis. A controversial aspect of feed restriction programs has been the inconsistent carcass fat deposition. Summers *et al*. (1990) and Jones and Farrell (1992) did not find changes in carcass composition of birds after feed restriction conditions; however, Plavnick and Hurwitz (1985, 1989) and Plavnick *et al.* (1986) reported a decrease in fat pad in broiler chickens restricted from 6 to 12 days of age,

without adverse effects on growth. Lee and Leeson (2001), Leeson *et al*. (1991), Saleh *et al*. (2004, 2005) and Urdaneta and Leeson (2002) were not able to show a clear effect.

2.3.2 Lighting

Broiler chickens grow fastest when fed *ad libitum* and under continuous light. Lighting is a powerful exogenous factor in control of many physiological and behavioral processes. Light allows the birds to establish rhythmicity and synchronize many essential functions, including body temperature and various metabolic steps that facilitate feeding and digestion (Olanrenwaju *et al.,* 2006). Light intensity, colour, and the photoperiodic regime can affect the physical activity of broiler chickens (Lewis and Morris, 1998). Altering lighting schedules by reducing the hours of light or developing intermittent schedules improves feed utilization (Blair *et al.,* 1993; Wilson *et al.,* 1984; Alpedoorn *et al.,* 1999). Light manipulation is used in broiler chicken production to control growth, improve feed efficiency, minimize mortality and reduce electricity costs. Broiler chickens under different reduced lighting programs, therefore, will reduce their feed intake. Olanrewaju *et al.* (2006) hypothesized that short photoperiods early in life will reduce feed intake and limit growth. Buyse *et al.* (1994) observed lower cumulative feed intake and significantly improved feed conversion ratio in chickens under an intermittent lighting program (1 Light-hour: 23 Dark-hour from 8 to 49 days) compared with those under a continuous light schedule (23.5 Light-hour: 0.5 Dark-hour or 23 Light-hour: 1 Dark-hour). Classen (2004), compared 12 light-hours versus 12 dark-hours, 16 light-hours versus 8 dark-hours and 20 light-hours versus 4 dark-hours and demonstrated clearly that longer periods of darkness prevent regular access to feed and consequently

reduce feed intake and limit growth. However, chickens can learn to eat in the dark (Perry, 1981), but their feed intake in the dark is much reduced (Buyse and Decuypere, 1988). They can also learn to increase feed intake during the light period in anticipation of the dark period but are limited by their crop size (Perry, 1981).

2.3.3 Diet dilution

Diet dilution has been used as an alternative method of nutrient restriction because of the advantage of attaining a more consistent growth pattern within a flock. Diets are mixed with non-digestible ingredients such as fibre, so that they are of reduced nutrient density. Leeson *et al.* (1991) and Jones and Farrell (1992) used 50 to 65 % diet dilution with rice hulls in order to retard early growth and reported a complete compensatory growth at either 42 or 48 days of age. Zubair and Leeson (1994) also reported no differences in body weight at either 42 or 49 days when birds were fed a 50 % oat-hull diluted diet for six days during the starter stage. Leeson *et al.* (1992) offered broiler chickens a conventional finisher diet diluted up to 50 % with a 50:50 mixture of sand: oat hulls from 35 to 49 days of age, and showed no significant difference in body weight at 49 days or breast weight at 42 or 49 days of age. Cabel and Waldroup (1990) observed that diluting the starter diet with sand from 5 to 11 days of age moderately restricted growth, which was completely recovered by 49 days of age. Griffiths *et al.* (1977) lowered the energy of a broiler chicken diet to 2233 kcal ME/kg DM from 3087 kcal ME/kg DM of feed by substituting ground yellow corn with oat meal as the main ingredient. Chickens fed the low energy diet consumed significantly more feed than those fed the high energy diet. When fed the low energy diet from 0 to 3 weeks of age, the chicks were not significantly

different in body weight or in abdominal fat pad development from the *ad libitum* birds at 4 weeks of age.

2.3.4 Nutrient density of the diets

The use of low energy and low protein diets is another way of achieving reduced growth rate. Broiler chickens require 220, 200 and 180 g crude protein per kg diet during the starting, growing and finishing periods, respectively, and 3200 kcal ME/kg diet for optimal growth (NRC, 1994). When broiler chickens are fed with diets low in nutrients they will increase their feed intake in an attempt to maintain nutrient intake levels (Leeson and Summers, 1997). Diets with higher energy concentration will have lower feed intake and those with lower energy concentration will have higher feed intake (Macleod, 1991; Leeson, 1996). Holsheimer and Veerkamp (1992) and Yolcin *et al*. (1990) reported that high energy diets significantly increased absolute carcass weight and yield of abdominal fat, however, carcass part weights were not influenced by dietary energy. Also, relative abdominal fat weight increased linearly with increments in dietary energy.

Coon *et al*. (1981) compared the performance of male and female broiler chickens fed low or high energy rations for 56 days and found a significant improvement in the feed conversion ratio with high energy diet. Sizemore and Siegel (1993) tested the effects of early energy restriction, while keeping protein and other nutrients constant, on different female broiler chicken crosses. They observed significant differences in the response of female broiler chickens to energy restriction. They concluded that the reason the results on early feed restriction are often contradictory is that the genetic makeup of the broiler

chicken may interact with its response to the nutritive content of the diet and change the final result. Fisher (1984) reported that the broiler chickens tend to increase their feed intake to make up for deficiencies when fed diets that are marginally deficient in crude protein. Babu *et al.* (1986) reported comparable feed intake, weight gain and feed coversion ratio for broiler chickens subjected to low crude protein diets compared with those on higher crude protein diets. In contrast, Plavnik and Hurwitz (1990) reported that broilers fed *ad libitum* with a 9 % crude protein diet from 8 to 14 days markedly reduced their feed intake and weight gain by 63 % and 88 %, respectively and did not recover the body weight as measured at 56 days of age. This reduction in feed intake may be due to an imposition of a protein/amino acid deficiency, since other nutrients were at normal levels. Morris (1971) also reported 25 % growth retardation by feeding low crude protein diets.

Feeding broiler chickens with combinations of high density diets (21.2 to 25.9 % protein, 13.9 to 14.3 MJ ME/kg diet) produces greater live weights and carcass meat components until 50 days of age, but the rate of gain was lower when compared with combinations of low-high density diets (19.1 to 22.5 % protein, 12.7 to 13.1 MJ ME/kg diet) (Walker *et al.*, 1995). Growth depression observed in birds fed diets low in energy could be overcome with an increased feed intake and longer length of time to attain the desirable body weight (Urdaneta and Leeson, 2000).

2.3.5 Chemical methods of feed restriction

Chemicals have been used as another method to depress feed intake in chickens. Restriction of feed intake of broiler chickens by chemical means was suggested by

Francher and Jensen (1988). Pinchasov and Elmaliah (1994) incorporated 3 % propionic acid in the diet as an anorectic agent. Reduction in feed intake was achieved, but this depression in feed intake was lower than that obtained when using physical feed restriction. This effect might be due to an adaptation to the propionic acid by birds when used for a long period (Oyawoye and Kreuger, 1990). In addition, Pinchasov and Elmaliah (1994) showed that 1 to 3% of acetic and propionic acids included in the diet act as appetite suppressors, and decrease body weight gain in broiler chickens. Savory *et al.* (1996) used 50g calcium propionate per kg diet as an appetite suppressor and found that weight gains of chemically restricted birds were close to those obtained under a recommended program of quantitative feed restriction for female broiler breeders between 2 to 6 weeks of age. The findings indicate that quantitative and qualitative feed restriction can yield similar effects on the productivity of broiler chickens. Oyawoye and Krueger (1990) showed that 400 and 300 mg of phenylpropanolamine hydrochloride or monensin sodium per kg diet, respectively, significantly depressed body weight of broiler chickens at 4 weeks of age, due to a significant reduction in feed consumption. However, the use of phenylpropanolamine to reduce feed intake is not appropriate for older birds because of tolerance developed to the drug (Oyawoye and Kreuger, 1990).

McLean and Luo (2004) isolated P57 from *Hoodia gordonii*, defined in medical terms as a steroidal glycoside with anorectic activity in animals (causes animals to decrease their appetite). They administered intracerebroventricular injections of the purified P57, but found that in rats it did not bind or alter the activity of known receptors or proteins relating to cardiac glycosides. However, the trial revealed that the compound increases ATP by 50-150% in neurons located in the hypothalamus. The injection of P57 also

reduced food intake for the next 24 hours by 40-60%. In related studies, rats were fed a low calorie diet for 4 days. The ATP content found in the hypothalamus of the control animal group fell by 30-50%. This may support the belief that ATP may be an energy sensor, and in turn provokes the appropriate neural, endocrinal and appetitive responses, similar to other "fundamental hypothalamic homeostatic centers for temperature and osmolarity". Thus, the P57 that was injected cause an increase in the amount of ATP in the hypothalamus. The P57 from *Hoodia gordonii* mimics ATP in the hypothalamus to cause a satiety feeling in rats (McLean and Luo, 2004).

In other studies, Heerden *et al.* (2007) tested *Hoodia gordonii* plant for its appetite suppressant properties in rats by oral gavage at 6.25-50 mg /kg and reported that all doses resulted in a decrease in food consumption over an eight day period and a body mass decrease when compared to the control group sample receiving only the vehicle. Tulp *et al.* (2001) showed that the animals that received *Hoodia* had rapid onset of decreased food intake, which was sustained over a period of weeks. This resulted in major degrees of weight loss in the rats. It was also found that body fat was reduced by a factor of 50% in both lean and obese rats, compared to similar animals on a conventional diet that did not include Hoodia. In the clinical trial which was performed on humans, where a double-blind, placebo-controlled group of healthy volunteers were involved, it was found that a significant calorie reduction resulted after *Hoodia gordonii* was taken by the subjects. The two-week study revealed that on the 15^{th} day, the subject caloric intakes decreased by about 1,000 calories per day (FAQ, 2001). No similar studies have been found in chicken experiments.

2.4 Genetic and sex effect on feed intake

The response of broiler chickens to various feed restriction programs will depend on the genotype and sex of the chickens. Gous *et al*. (1999) suggested that genetic potential influences the broiler chicken's growth response because it affects its nutritional requirements. Havenstein *et al*. (1994) pointed out that genetic potential rather than nutritional requirements has a greater effect on broiler chicken body composition. Hence, the discrepancies in the results concerning the response of broiler chickens subjected to various feeding programs has been credited to differences in genetics of birds used (Fontana *et al.,* 1992; Schiedeler and Baughman, 1993).

There have been differing growth responses between the sexes of broiler chickens, but the findings have not always been consistent. Sexual dimorphism has been reported in terms of feed consumption and growth responses (Emmerson, 1997). The difference in responses between the sexes is likely the result of the higher innate rate of growth of male chickens in comparison to female chickens (Zubair and Leeson, 1996). Nir *et al.* (1987) suggested that feed consumption may be influenced by the capacity of gastrointestinal system. Generally, male broiler chickens have a higher feed intake, growth rate and leaner body composition than do female broiler chickens (Hurwitz *et al*., 1978; Han and Baker, 1994; Nahashon *et al.,* 2004). However, other workers found no responses in feed intake and growth responses between sexes of chickens (Leeson *et al.,* 1991; Lippens *et al.,* 2000).

2.5 Fat deposition

The success of poultry meat production has been strongly related to improvements in growth and carcass yield, mainly by increasing breast proportion and reducing abdominal fat (Zerehdaran *et al.*, 2004). Rapid growth rate is accompanied by a number of negative consequences, including an increase in fat deposition (Griffin, 1996). In chickens, lipogenic activity in the liver is much greater than that in the adipose tissue. Most of fat accumulated in the adipose tissues results from incorporation of triglycerides from plasma lipoproteins in particular very low density lipoproteins that are either synthesized in the liver or provided from dietary fats (Hermier, 1997; Kobayashi *et al.*, 2006). Triglycerides are major lipids in poultry diets. Fats are usually added to poultry diets as sources of energy and linoleic acid, an essential fatty acid for poultry. The fat in poultry tissues contains higher quantities of unsaturated fatty acids than are found in most of domestic animals. All tissues of the body store triglycerides. Adipose tissues are the most notable storage sites. Adipose tissue is capable of synthesizing fat from carbohydrates. Energy in excess of current needs of the animal result in a net deposition of triglycerides and energy intake less than current needs (as in fasting) results in a net loss of triglycerides. Triglycerides of fat have fatty acid composition characteristics for each animal species. In non ruminants, however, the fatty acid composition of fat depots resembles that of the diet. Liver, mammary gland and adipose tissues are the three major sites of biosynthesis of fatty acids and triglycerides.

The regulatory processes of energy balance maintain stability in body energy stores usually body fat. This homeostatic process is critical to the long term survival of the animal. According to Boswell (2005), birds are capable of detecting loss of body mass

and are able to compensate by making precise adjustments to short-term and long-term intake. However, excessive fatness in broiler chickens is widely recognized as a problem in the poultry industry. Abdominal and subcutaneous fat are regarded as the main sources of waste in the slaughterhouse. Abdominal fat is highly correlated (0.6 to 0.9) with total carcass lipid and it is used as the main criterion reflecting excessive fat deposition in broilers (Chambers, 1990). Fat content in broiler chickens at day 42 of age accounts for 10 to 15 % of total carcass weight. Most results obtained intended to diminish carcass fat content in broiler chickens have been inconsistent. This inconsistency may be due to different strategies of feed restriction applied, conditions of re-alimentation, age of imposition, strain and sex of the birds, all of which may affect bird's response. Reduction in abdominal fat content due to the application of a feed restriction regimen was achieved by some workers (Plavnik and Hurwitz, 1985, 1991; Jones and Farrell, 1992; Santoso *et al.,* 1995). However, this desirable response has not been shown by other workers (Summers *et al.,* 1990; Fontana *et al.,* 1992; Deaton, 1995).

The activity of the enzymes associated with hepatic lipogenesis, namely fatty acid synthetase, isocitrate dehydrogenase, and malic enzyme are depressed during the nutrient restriction period, but after re-feeding their activity is increased (Rosebrough *et al.,* 1986; and McMurtry *et al.,* 1988). Rosebrough and McMurtry (1993) suggested that after a short period of feed restriction broiler chickens exhibited an increased *de novo* lipogenesis, which was related to the quantity of feed given. Leeson (1996) reported that feed restricted broiler chickens had the same percentage of fat content as an *ad-libitum* group did. Zhong *et al.* (1995) fed restricted broiler chickens from 7 to 12 days of age

and showed no difference in the adipocyte numbers from the abdominal fat pads at either 28 or 42 days of age for restricted and *ad libitum* birds.

2.6 Summary

Poultry breeders have intensively selected meat-type birds over many generations with specific emphasis on increasing growth rate and meat production. Increased body size in commercial chicken lines has been accompanied by changes such as increased voluntary feed intake and increased fat deposition. Several approaches, both qualitative and quantitative have been employed to restrict nutrient or caloric intake in broilers in order to reduce cost of feeding, improve feed efficiency and reduce excessive abdominal fat deposition and carcass fat among other problems associated with ad *libitum* feeding. Regulation of feed intake and energy balance occurs in the central nervous system (CNS), primarily in the hypothalamus. There is evidence that P 57 from *Hoodia gordonii* suppresses intake in rats and human beings. However, the effect of *Hoodia gordonii* meal supplementation on food intake and carcass fat content of broiler chickens remains to be established. It is, therefore, important to ascertain the potential of improving carcass characteristics of broiler chickens through the use of *Hoodia gordonii* meal as a feed supplement.

CHAPTER 3

MATERIALS AND METHODS

3.1 Study site

This study was conducted in an open-sided broiler chicken house with curtains at the University of Limpopo Experimental farm. The farm is situated 10 km north-west of the Turfloop campus of the University of Limpopo. The ambient temperatures around this area are above 32 °C during summer and around 25 °C or below during winter seasons. The mean annual rainfall is between 446.8 and 468.4 mm.

3.2 Preparation of the house

The experimental house was cleaned properly with water and a disinfectant, and then fumigated with formalin from NTK, Polokwane. The house was left empty for two weeks after cleaning to break the life cycle of any disease-causing organisms that were not killed by the disinfectant. After proper drying, the experimental house was divided into 72 floor pens of 2 m^2 each. Fresh saw dust and wood shavings were placed on the floor making a 7 cm thickness from the floor. The drinkers and feeders were thoroughly cleaned and disinfected.

3.3 Acquisition of materials and birds

A total of 800 day-old male and female Ross 308 chicks were purchased from the SA Chicks Hatchery in Pretoria for this study. *Hoodia gordonii* meal a feed supplement used in this study was purchased from Hoodiabushman in Cape Town. Commercial starter and

grower diets were purchased from NTK in Polokwane. A grower diet was used for both experiments one and two. The nutrient composition of the grower diet used in this study is presented in Table 3.1. The diet had 200 g crude protein /kg DM and 16.9 MJ energy /kg DM feed.

Table 3.1. Nutrient composition of the grower diet (the units are in g/kg for dry matter, g/kg DM for protein, lysine, fat, calcium, phosphorous and crude fibre, and MJ/kg DM for energy).

	Nutrients							
Diet	Dry Matter	Energy	Protein	Lysine	Fat	Calcium	Phosphorus	Fibre
Grower	880	16.9	200.0	11.5	25	10	5.5	60

3.4 Experimental procedure, dietary treatments and design.

The first experiment determined the effect of level of *Hoodia gordonii* meal supplementation at finisher stage on productivity and carcass characteristics of Ross 308 broiler chickens. A total of 360 male and female chickens were used for this experiment. The chickens were raised for 29 days before the experiment commenced on the 30th day. The experiment was terminated when the chickens were 42 days old. Feed and water were offered *ad libitum* throughout the experiment. Levels of *Hoodia gordonii* were orally administered in a paste form. The design of the experiment was a 2 (male and female chickens) x 6 (levels of *Hoodia gordonii* meal) factorial arrangement in a completely randomized (SAS, 2003). Therefore, the experiment had 12 treatments replicated 3 times, resulting in a total of 36 floor pens with 10 birds in each. At 30 days

of age, chickens were randomly allocated to the 12 treatments. The treatments were as follows:

MH_0: Male broiler chickens fed a grower diet without *Hoodia gordonii* meal supplementation

MH_1: Male broiler chickens fed a grower diet supplemented with 100 mg of *Hoodia gordonii* meal / bird/ day

MH_2: Male broiler chickens fed a grower diet supplemented with 200 mg of *Hoodia gordonii* meal / bird/ day

MH_3: Male broiler chickens fed a grower diet supplemented with 300 mg of *Hoodia gordonii* meal / bird/ day

MH_4: Male broiler chickens fed a grower diet supplemented with 400 mg of *Hoodia gordonii* meal / bird/ day

MH_5: Male broiler chickens fed a grower diet supplemented with 500 mg of *Hoodia gordonii* meal / bird/ day

FH_0: Female broiler chickens fed a grower diet without *Hoodia gordonii* meal supplementation

FH_1: Female broiler chickens fed a grower diet supplemented with 100 mg of *Hoodia gordonii* meal / bird/ day

$FH_{2:}$ Female broiler chickens fed a grower diet supplemented with 200 mg of *Hoodia gordonii* meal / bird/ day

FH_3: Female broiler chickens fed a grower diet supplemented with 300 mg of *Hoodia gordonii* meal / bird/ day

FH_4: Female broiler chickens fed a grower diet supplemented with 400 mg of *Hoodia gordonii* meal / bird/ day

FH_5: Female broiler chickens fed a grower diet supplemented with 500 mg of *Hoodia gordonii* meal / bird/ day

The second experiment determined the effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on productivity and carcass characteristics of Ross 308 broiler chickens. A total of 360 male and female chickens were used in this experiment. The chickens were raised for 29 days before the experiment commenced. The experiment was terminated when the chickens were 42 days old. Feed and water were provided *ad-libitum* throughout the experiment. *Hoodia gordonii* meal was orally administered. The design for the experiment was a 2 (male and female chickens) x 3 (dose intervals) factorial arrangement in a completely randomized design (SAS, 2003). The level of *Hoodia gordonii* was decided after the first experiment. The experiment had 6 treatments replicated 6 times, resulting in a total of 36 floor pens of 10 birds each. At Day 30, chickens were randomly allocated to 6 treatments. The treatments were as follows:

H_0MD_0 : Male broiler chickens fed a grower diet without supplementation of *Hoodia gordonii* meal .

$H_{300}MD_6$: Male broiler chickens fed a grower diet supplemented with 300 mg of *Hoodia gordonii* meal / bird/ day on Days 30 and 36.

$H_{300}MD_{12}$: Male broiler chickens fed a grower diet supplemented with 300 mg of *Hoodia gordonii* meal / bird everyday for 12 days.

H_0FD_0 : Female broiler chickens fed a grower diet without supplementation of *Hoodia gordonii* meal.

$H_{300}FD_6$: Female broiler chickens fed a grower diet supplemented with 300 mg of *Hoodia gordonii* meal / bird/ day on Days 30 and 36.

$H_{300}FD_{12}$: Female broiler chickens fed a grower diet supplemented with 300 mg of *Hoodia gordonii* meal / bird everyday for 12 days.

3.5 Data collection

Daily feed was measured between 30 and 42 days of age by subtracting the weight of the feed residuals from that of the feed offered per day, and the difference was divided by total number of the birds in the pen.

The initial live weight of 1226 +_ 2 g was measured at 30 days of age when the experiment commenced. Thereafter, average live weight was measured daily by weighing all the birds in each pen. These live weights were used to calculate growth rates. Feed conversion ratio was calculated as the total amount of feed consumed divided by the weight gain of live birds plus the weight gain of dead or culled birds in the pen.

Apparent digestibility of the diets was carried out when the birds were between 39 and 42 days old. This was done in metabolic cages. The excreta was collected from each replicate and stored at -15 ^{0}C during the collection period. Feed offered and refusals were weighed. Apparent digestibility (AD) of the nutrients was calculated according to procedures of McDonald *et al.* (1992) as follows:

$$AD\ (\%) = \frac{\text{Amount of nutrient ingested} - \text{amount of nutrient excreted}}{\text{Amount of nutrient ingested}} \times 100$$

Apparent metabolizable energy (AME) of the diet was calculated as follows (AOAC, 1984):

AME = Energy in feed consumed – energy in excreta

Nitrogen retention was calculated by subtracting daily nitrogen amount in faeces from daily nitrogen intake.

Mortality rate of the chickens was calculated as the total number of deaths divided by total number of chickens in the cage.

All remaining chickens were slaughtered at 42 days of age. Before slaughtering each chicken was weighed by using an electronic weighing scale. After slaughtering, carcass weight of each chicken was measured. Dressing percentage was determined by dividing carcass weight by live weight and then multiplied by 100. Breast, fat pad, thigh and drumstick weights were measured by using an electronic weighing scale.

3.6 Chemical analyses

Dry matter (DM) contents of feeds, feed refusals, excreta and meat were determined by drying the samples at 105 ^{0}C for 24 hours. Feeds, feed refusals and excreta samples were also analyzed for ash by placing the samples in the furnace at 300 ^{0}C for 48 hours (AOAC, 1984). The nitrogen content was determined using LECO FP 2000® Protein Analyser (University of Limpopo laboratory, Polokwane). The bomb calorimeter was used to measure gross energy values for feeds and feaces (University of Kwazulu-Natal laboratory, Durban). Nutritional analysis of *Hoodia gordonii* meal revealed that it contained 5.9 % mean protein content and 6.5 % Ca, 5.1 % K, 0.3 % P, 1 % Mg, 1.9 % Na, and 0.7 % S, with smaller amounts of micro and trace minerals (Tulp *et al.*, 2001). Intergratedbiomolecule Corporation (2005) analytical report has shown that 430 mg of *Hoodia gordonii* meal contains 0.006 mg of P 57- isoberberine alkaloid.

3.7 Statistical analysis

General linear model (GLM) procedures of the Statistical Analysis System (SAS, 2003) package were used to analyze data on feed intake, digestibility, live weight, carcass characteristics, feed conversion ratio and mortality in Experiments 1 and 2. The effects of interactions were not included in the model because earlier analyses including all the interactions showed that they were not important. Tukey test was used to test the significance of differences between treatment means at 5% significance level ($P < 0.05$).

CHAPTER 4

RESULTS

Hoodia gordonii meal contained 920 g dry matter/kg and 59 g crude protein per kg DM. Four hundred and thirty milligram of *Hoodia gordonii* meal contained 0.006 mg of P 57-isoberberine alkaloid.

Results of the effect of level of *Hoodia gordonii* meal supplementation at finisher stage on dry matter intake, feed conversion ratio, growth rate, live weight, intake as percentage of live weight and mortality rate of Ross 308 broiler chickens between 30 and 42 days of age are presented in Table 4.1. Level of *Hoodia gordonii* meal supplementation had no effect ($P>0.05$) on dry matter intake, feed conversion ratio, growth rate, live weight, intake as percentage of live weight and mortality rate of Ross 308 broiler chickens. However, male chickens had a higher ($P<0.05$) dry matter intake than female chickens.

Level of *Hoodia gordonii* meal supplementation had no effect ($P>0.05$) on apparent diet dry matter and nitrogen digestibilities, nitrogen retention and metabolizable energy of Ross 308 broiler chickens (Table 4.2). Similarly, sex of Ross 308 broiler chickens had no effect ($P>0.05$) on diet dry matter and nitrogen digestibilities, nitrogen retention and metabolizable energy.

Level of *Hoodia gordonii* meal supplementation had no effect ($P>0.05$) on carcass weight, dressing percentage, and carcass parts of Ross 308 broiler chickens except fat pads (Table 4.3). Chickens given a daily supplement of 300 mg of *Hoodia gordonii* meal had lower ($P<0.05$) fat pad weights than unsupplemented ones. Male chickens had heavier ($P<0.05$) drum sticks than female chickens.

Level of *Hoodia gordonii* meal supplementation had no effect ($P>0.05$) on nitrogen content of breast meat samples of Ross 308 broiler chickens at 42 days of age (Table 4.4). Similarly, male and female Ross 308 broiler chicken breast meat samples had similar ($P>0.05$) nitrogen content.

Table 4.1. Effect of level of *Hoodia gordonii* meal supplementation at finisher stage on dry matter intake (g/bird/day), feed conversion ratio, growth rate (g/bird/day), live weight (g/bird at 42 days old), intake as % of live weight, and mortality (%) of Ross 308 broiler chickens between 30 and 42 days of age.

Treatment	Variables					
	DM intake	FCR	Growth rate	Live weight	Intake as % of Live weight	Mortality
Males						
MH_0	153.3	2.2	71	2063	10.3	0
MH_1	148.4	2.5	61	2117	11.3	0
MH_2	148.7	2.3	66	2067	10.0	0
MH_3	148.8	2.2	68	1977	11.2	0
MH_4	142.0	2.3	64	1937	10.6	0
MH_5	139.1	2.5	61	2007	10.0	0
SE	5.40	0.30	7.80	88.30	0.50	0
Females						
FH_0	138.0	2.0	68	1992	11.7	0
FH_1	133.6	2.4	56	1862	11.5	0
FH_2	136.9	2.2	61	1969	11.4	0
FH_3	136.5	2.1	65	1999	10.7	0
FH_4	138.4	2.2	65	1982	10.7	0
FH_5	145.7	2.4	62	1956	10.3	0
SE	3.00	0.10	3.50	37.80	0.40	0
Sex						
Males	146.7 [a]	2.3	65	2028	10.9	0
Females	138.2 [b]	2.2	63	1960	11.2	0
SE	1.70	0.10	2.30	24.30	0.20	0

[a,b] : Means in the same column not sharing a common superscript are significantly different ($P<0.05$).

SE: Standard error

Table 4.2. Effect of level of *Hoodia gordonii* meal supplementation at finisher stage on apparent diet dry matter and nitrogen digestibilities (decimal), nitrogen retention (g/bird/day) and metabolizable energy (ME) (MJ/kg DM) of Ross 308 broiler chickens between 39 and 42 days of age.

Treatment	Variables			
	Dry matter digestibility	Nitrogen digestibility	Nitrogen retention	ME
Males				
MH_0	0.42	0.76	3.23	13.96
MH_1	0.50	0.80	3.23	14.55
MH_2	0.59	0.81	3.14	14.63
MH_3	0.41	0.78	2.84	14.35
MH_4	0.47	0.79	3.00	14.31
MH_5	0.36	0.74	2.84	13.64
SE	0.070	0.030	0.170	0.330
Females				
FH_0	0.41	0.81	3.08	14.45
FH_1	0.48	0.78	2.91	14.03
FH_2	0.51	0.78	2.93	14.04
FH_3	0.47	0.72	2.62	13.92
FH_4	0.44	0.79	3.01	14.35
FH_5	0.40	0.73	2.71	14.39
SE	0.060	0.060	0.180	0.290
Sex				
Males	0.46	0.78	3.05	14.24
Females	0.45	0.77	2.88	14.20
SE	0.030	0.020	0.070	0.130

SE: Standard error

Table 4.3. Effect of level of *Hoodia gordonii* meal supplementation at finisher stage on carcass weight (g/bird), dressing percentage (%) and carcass parts expressed as percentage of carcass weight Ross 308 broiler chickens at 42 days of age.

	Variables								
Diet	Carcass	Dressing %	Thigh	Drum sticks	Wings	Breast	Fat pad	Gizzard	Liver
Males									
MH_0	1589	76	15	12.8	12	32	1.5 [a]	2.5	2.7
MH_1	1712	74	13	13.0	12	33	1.3 [ab]	2.4	2.8
MH_2	1573	72	14	12.9	13	31	1.1 [ab]	2.3	3.0
MH_3	1602	73	14	13.1	12	31	0.9 [b]	2.3	2.8
MH_4	1501	71	14	13.3	12	30	1.2 [ab]	2.4	3.3
MH_5	1545	71	15	12.7	12	31	1.2 [ab]	2.4	3.4
SE	101.6	3.04	0.56	0.26	0.47	0.31	0.12	0.15	0.33
Females									
FH_0	1551	74	16	12.6	12	31	1.4 [a]	2.4	2.8
FH_1	1421	72	15	12.2	12	31	1.2 [ab]	2.6	2.7
FH_2	1591	76	15	12.8	11	31	1.2 [ab]	2.7	2.7
FH_3	1559	73	15	12.3	12	31	0.8 [b]	2.8	2.7
FH_4	1578	74	15	12.6	12	31	1.3 [ab]	2.2	2.8
FH_5	1501	70	15	12.5	12	30	1.2 [ab]	2.3	3.1
SE	57.13	1.22	0.35	0.14	0.41	0.79	0.12	0.19	0.19
Sex									
Males	1587	73	14	13.0 [a]	12	31	1.2	2.4	3.0
	1533	73	14	12.5 [b]	12	31	1.2	2.5	2.8
Females									
SE	23.32	0.87	0.19	0.08	0.18	0.30	0.05	0.07	0.11

[a,b] : Means in the same column not sharing a common superscript are significantly different ($P<0.05$).

SE: Standard error

Table 4.4. Effect of level of *Hoodia gordonii* meal supplementation on nitrogen content (%) of Ross 308 broiler chicken breast meat samples at 42 days of age.

Treatment	Nitrogen Content
Males	
MH_0	44.1
MH_1	44.1
MH_2	44.7
MH_3	44.2
MH_4	45.2
MH_5	44.1
SE	0.94
Females	
FH_0	45.2
FH_1	44.5
FH_2	44.2
FH_3	45.0
FH_4	44.3
FH_5	44.5
SE	0.53
Sex	
Males	44.4
Females	44.6
SE	0.31

SE: Standard error

Hoodia gordonii meal dose interval had no effect (P>0.05) on dry matter intake, feed conversion ratio, growth rate, live weight, intake as percentage of live weight and mortality rate of Ross 308 broiler chickens between 30 and 42 days of age (Table 4.5). However, male broiler chickens had higher (P<0.05) dry matter intake and live weight values than female broiler chickens.

Results of the effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on apparent diet dry matter and nitrogen digestibilities, nitrogen retention and metabolizable energy of Ross 308 broiler chickens between 38 and 42 days of age are presented in Table 4.6. *Hoodia gordonii* meal dose interval had no effect (P>0.05) on apparent diet dry matter and nitrogen digestibilities, nitrogen retention and metabolizable energy of Ross 308 broiler chickens. However, male Ross 308 broiler chickens had higher (P<0.05) nitrogen digestibility and nitrogen retention values than those of female Ross broiler chickens.

Hoodia gordonii meal dose interval had no effect (P>0.05) on carcass weight, dressing percentage and other carcass parts of Ross 308 broiler chickens at 42 days of age (Table 4.7). However, *Hoodia gordonii* dose interval had an effect (P<0.05) on abdominal fat pad of broiler chickens. Broiler chickens given a daily dose supplement of 300 mg of *Hoodia gordonii* meal had lower (P<0.05) fat pad weights than unsupplemented ones. Male Ross 308 broiler chickens had higher (P<0.05) carcass weights than female chickens.

Hoodia gordonii meal dose interval had no effect (P>0.05) on nitrogen content of breast meat of Ross 308 broiler chickens at 42 days of age (Table 4.8). Similarly, sex of Ross 308 broiler chickens had no effect (P>0.05) on nitrogen content of Ross 308 broiler chicken breast meat.

Table 4.5. Effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on dry matter intake (g/bird/day), feed conversion ratio, growth rate (g/bird/day), live weight (g/bird at 42 days old), intake as % of live weight, and mortality (%) of Ross 308 broiler chickens between 30 and 42 days of age.

	Variables					
Treatment	DM intake	FCR	Growth rate	Live weight	Intake as% of Live weight	Mortality
Dose interval						
$H_{300}D_{12}$	144.0	2.16	67.1	1974	10.8	0
$H_{300}D_{6}$	149.3	2.05	73.0	2065	10.8	0
$H_{0}D_{0}$	146.0	2.16	68.3	2004	11.0	0
SE	2.41	0.06	2.40	41.2	0.32	0
Sex						
Males	154.9 [a]	2.2	72	2073 [a]	10.5	0
Females	138.0 [b]	2.1	67	1956 [b]	10.7	0
SE	2.10	0.05	1.90	36.10	0.68	0

[a,b] : Means in the same column not sharing a common superscript are significantly different ($P<0.05$).

$H_{300}D_{12}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird everyday for 12 days.

$H_{300}D_{6}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird/ day on days 30 and 36.

$H_{0}D_{0}$: Male and female broiler chickens fed a grower diet without supplementation of *Hoodia gordonii* meal.

SE : Standard error

Table 4.6. Effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on diet dry matter and nitrogen digestibilities (decimal), nitrogen retention (g/bird/day) and metabolizable energy (ME) (MJ/kg DM) of Ross 308 broiler chickens between 39 and 42 days of age.

Treatment	Variables			
	Dry Matter digestibility	Nitrogen digestibility	Nitrogen retention	ME
Dose interval				
$H_{300}D_{12}$	0.41	0.78	2.91	14.23
H_{300} D_6	0.49	0.79	2.93	14.42
H_0D_0	0.41	0.77	2.83	14.44
SE	0.030	0.010	0.080	0.140
Sex				
Males	0.43	0.80 [a]	3.01 [a]	14.31
Females	0.44	0.75 [b]	2.77 [b]	14.41
SE	0.020	0.020	0.070	0.110

[a,b] : Means in the same column not sharing a common superscript are significantly different ($P<0.05$).

$H_{300}D_{12}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird everyday for 12 days.

$H_{300}D_6$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird/ day on days 30 and 36.

H_0D_0 : Male and female broiler chickens fed a grower diet without supplementation of *Hoodia gordonii* meal.

SE : Standard error

Table 4.7. Effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on carcass weight (g/bird), dressing percentage (%) and carcass parts expressed as percentage of carcass weight of Ross 308 broiler chickens at 42 days of age.

	Variables								
Treatment	Carcass	Dressing %	Thigh	Drum stick	Wings	Breast	Fat pad	Gizzard	Liver
Dose interval									
$H_{300}D_{12}$	1558	76.4	14.6	12.9	12.1	32.1	0.9 [b]	2.5	2.8
$H_{300}D_{6}$	1584	77.7	15.4	13.6	12.2	32.6	1.2 [a]	2.6	2.7
$H_{0}D_{0}$	1595	78.2	15.1	13.1	12.2	33.3	1.1 [a]	2.6	2.8
SE	37.6	1.84	0.49	0.37	0.23	1.02	0.09	0.09	0.07
Sex									
Males	1647 [a]	76	15	12.9	11.6	31.3	0.94	2.50	2.68
Females	1511 [b]	75	14	12.4	11.8	31.3	1..07	2.40	2.67
SE	32.09	0.47	0.24	0.21	0.10	0.39	0.07	0.06	0.07

[a,b] : Means in the same column not sharing a common superscript are significantly different ($P<0.05$).

$H_{300}D_{12}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird everyday for 12 days.

$H_{300}D_{6}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird/ day on days 30 and 36.

$H_{0}D_{0}$: Male and female broiler chickens fed a grower diet without supplementation of *Hoodia gordonii* meal.

SE : Standard error

Table 4.8. Effect of *Hoodia gordonii* meal dose interval and sex at finisher stage on nitrogen content (%) of Ross 308 broiler chicken breast meat samples at 42 days of age.

Treatment	Nitrogen Content
Dose Interval	
$H_{300}D_{12}$	44.9
$H_{300}D_{6}$	44.5
$H_{0}D_{0}$	44.5
SE	0.44
Sex	
Males	44.7
Females	44.6
SE	0.34

$H_{300}D_{12}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird everyday for 12 days.

$H_{300}D_{6}$: Male and female broiler chickens fed a grower diet supplemented with 300 mg *Hoodia gordonii* meal / bird/ day on days 30 and 36.

$H_{0}D_{0}$: Male and female broiler chickens fed a grower diet without supplementation of *Hoodia gordonii* meal.

SE : Standard error

CHAPTER 5

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1 Discussion

The present study showed that *Hoodia gordonii* meal supplementation has no effect on feed intake and intake as percentage of live weight of broiler chickens at 42 days of age. These findings are contrary to the findings of FAQ (2001), McLean and Luo (2004) and Heerden *et al.* (2007) who found that *Hoodia gordonii* caused satiety in rats and human beings, thus, reducing appetite. McLean and Luo (2004) showed that the active chemical P57 AS 3 in *Hoodia gordonii* meal mimics adenosine triphosphate (ATP) in the hypothalamus to cause satiety in rats, thus, reducing intake.

Hoodia gordonii meal supplementation had no effect on growth rate, live weight, feed conversion ratio and mortality of broiler chickens. These results may be explained in terms of similar feed intakes and digestibilities in the chickens, irrespective of the treatment. Contrary to the present findings, Heerden *et al.* (2007) showed that the oral dosage of *Hoodia gordonii* meal reduced growth rate and live weight of rats.

Hoodia gordonii meal supplementation had an effect on fat pad of broiler chickens. Daily dosing with 300 mg of *Hoodia gordonii* meal reduced fat pad weights in broiler chickens by 42 and 18 percentage points in the first and second experiment, respectively. This was achieved without any significant reduction in feed intake and digestibility. The physiological explanation for this effect is not clear and merits further investigation. However, it is known that *Hoodia gordonii* meal intake reduces caloric intake of the diet (FAQ, 2001) and increases ATP content in the hypothalamus, thus reducing blood glucose. Pocai (2005), Richards (2003) and Rosebrough and McMurtry (1993)

suggested that when blood glucose drops, the body releases the fat destroying hormones (growth hormones, glucagon and cholecystokinin) and suppresses energy storing insulin. Such transient changes in plasma glucose level do not appear to alter feed intake in chickens (Simon *et al.*, 2000). As such, fat pad deposition in the chickens may have been reduced irrespective of feed intake. Similar results were reported by Tulp *et al.* (2001). These authors found *Hoodia gordonii* meal reduced body fat in obese rats by 50 %. No similar studies in chickens were found.

Hoodia gordonii meal supplementation had no effect on dressing percentage, other carcass parts and nitrogen contents of breast meat of broiler chickens at 42 days of age. These results could be explained in terms of similar feed intakes, digestibilities and nitrogen retention (Ferket and Gernat, 2006; Richards, 2003).

Male broiler chickens consumed more feed than female broiler chickens. The present findings are consistent with earlier studies of Hurwitz *et al.* (1978) and Nahashon *et al.* (2004). Similarly, Dozier *et al.* (2003) found that male broiler chickens had higher feed intake than female chickens when both sexes were fed *ad libitum*. The differences were explained in terms of female chickens requiring on average 13 % less feed for maintenance per kg metabolic body weight than males. However, Gous *et al.* (1999) suggested that genetic potential influences broiler chicken growth responses because it affects their nutritional requirements. Thus, male broiler chickens have pronounced genetic advantage on feed intake compared to female broiler chickens. However, in the present study male and female chickens had similar feed intake values when intake was expressed as a percentage of live weight. The differences in intake did not reflect true sex

effect but weight differences associated with sex. This is similar to the findings of Leeson *et al.* (1991) and Lippens *et al.* (2000).

Male Ross 308 broiler chickens had heavier live weights than females. These differences can be explained in terms of higher feed intake in males than females. These differences may also be due to higher nitrogen digestibilities and nitrogen retentions in male chickens than in female ones. These findings are similar to those of Han and Baker (1994) who reported that male broiler chickens had heavier live weights than female broiler chickens. The differences were explained in terms of higher feed intake in male than female chickens. However, Havenstein *et al.* (1994) and Emmerson (1997) suggested higher early postnatal growth of male chickens as the cause of the differences in live weights. The present results are contrary to the findings of Scheilder and Baughman (1993) and Deaton (1995) who found no differences in live weights between male and female broiler chickens.

In the present study, male broiler chickens yielded larger portions of drum sticks than female broiler chickens. These differences may be explained in terms of differences in live weights between the sexes. These results are similar to the findings of Young *et al.* (2001) and Lippens *et al.* (2000) who reported that female broiler chickens yielded smaller portions of drum sticks than male broiler chickens. However, Leeson *et al*. (1991) found no differences in weights of carcass parts between male and female broiler chickens. In the present study, male and female broiler chickens had similar dressing percentage, breast meat, and nitrogen content of breast meat at 42 days of age. These

results may be explained in terms of similarities in diets and intakes. These results are similar to the findings of Pescatore *et al*. (1992) and Acar *et al.* (1993) who reported that sex had similar effect on carcass parts and breast meat yield of broiler chickens. However, the present results are different from the findings of Becker *et al.* (1981), Dozier *et al.* (2003), Rondelli *et al.* (2003) and Santos *et al.* (2005) who reported that sex had an effect on carcass parts and nitrogen content of breast meat of broiler chickens. These authors reported a better performance on carcass parts and nitrogen content of breast meat of male chickens when compared with those of female chickens. It was suggested that the differences between sexes probably arise from metabolic differences and also from the differences in the onset of fattening of broiler chickens.

5.2 Conclusions and recommendations

Hoodia gordonii meal supplementation at finisher stage had no effect on feed intake, digestibility, live weight, growth rate, mortality and breast meat yield of Ross 308 broiler chickens. However, chickens given a daily supplement of 300 mg of *Hoodia gordonii* meal had lower fat pad weights than unsupplemented ones. This could not be explained in terms of differences in feed intake, digestibility, or growth rate. More research is required to explore biochemical reasons for a reduction in chicken fat pad weights following *Hoodia gordonii* meal supplementation. There is also a need to study the effects of *Hoodia gordonii* meal supplementation on adipocyte cell development in young chickens.

CHAPTER 6

REFERENCES

6.0 References

ACAR, N., MORAN, E.T., and D.R, MULVANEY. 1993. Breast muscle development of commercial broiler from hatching to twelve weeks of age. Poultry Science 72:317-325.

APELDOORN, E.J., SCHRAMA, J.W., MASHALY, M.M., and H.K, PARMENTIER. 1999. Effects of melatonin and lighting schedule on energy metabolism in broiler chickens. Poultry Science 78: 223-227.

AOAC. 1984. Official Methods of Analysis. 14th Edition. Washington.

ASHWELL, M.C., CZERWINSKI, S.M., BRONCHT, D.M., and J.P, McMURTRY. 1999. Hormonal regulation of Leptin expression in broiler chickens. American Journal of Physiology 276:226–232.

BABU, M., SANDARANRAN, V., and S, KOTHANDARAM. 1986. Studies on energy and protein requirements of broilers chickens. Indian Journal of Poultry Science 26(2): 275-279.

BBC. 2003. Hoodia cactus – Trip to Africa by BBC (May, 2003). http://hoodiagordoniiguide.com/hoodia-cactus.htm.

BECKER, W.A., SPENCER, J.V., MIROSH, L.W., and J.A, VERSTRATE. 1981. Specific gravity, carcass fat, abdominal and carcass fat, and yield data in broiler chickens. Poultry Science 60: 2045-2052.

BERTHOUD, H.R. 2002. Multiple neural systems controlling food intake and body weight. Neuroscience and Biobehavioral Reviews 23: 393-428.

BENYI, K., and H, HABI. 1998. Effects of food restriction during then finishing period on the performance of broiler chickens. British Poultry Science 39:432-425.

BLAIR, R., NEWBERRY, R. C., and E.E, GARDINE. 1993. Effects of lighting pattern and dietary tryptophan supplementation on growth and mortality of broilers Poultry Science 72:495–502.

BLEVINS, J.E., SCHWARTZ, M.W., and D.G, BASKIN. 2002. Peptide signals regulating food intake and energy homeostasis. Canadian Journal of Physiology and Pharmacology 80:396-406.

BOSWELL, T. 2005. Regulation of energy balance in birds by neuroendocrine hypothalamus. Journal of Poultry Science 42:161-181.

BUYSE, J., DECUYPERE, E., and H, MICHELS. 1994. Intermittent lighting and broiler production. 1. Effect on female performance. Acrhive fur Gelugelkundc 58: 69-74.

BUYSE, J., and E, DECUYPERE. 1988. The influence of intermittent light on broiler performance and on patterns of food intake. In: Leanness in mestic birds. Genetic, Metabolic and Hormonal Aspects. B. Leclerc and C.C. Whitehead, Butterworths. London. Pages 133-134.

CABEL, M.C., and P.W, WALDROUP. 1990. Effect of different nutrient-restriction programs early in life on broiler performance and abdominal fat content. Poultry Science 69:652-660.

CHAMBERS, J.R. 1990. Genetics of growth and meat production in chickens. Qualitative Genetics and Selection. R.D, Crawford, ed. Poultry Breeding and Genetics. Elsevier. Amsterdam. Pages 599–643.

CLASSEN, H.L. 2004. Day length affects performance, health and condemnations in broiler chickens. Proc. of the Australian Poultry Science Society, University of Sydney, Sydney, NSW.

CONSODINE, R.V. 2002. Regulation of energy intake. http://www.endotext.org/obesity/obesityframe.html.

COON, C.N., BECKER, W.A., and J.V, SPENCER. 1981. The effect of feeding high energy diets containing supplemental fat on broiler weight gain, feed efficiency, and carcass composition. Poultry Science 60:1264-1271.

DEATON, J.W. 1995. The effect of early feed restriction on broiler chickens performance. Poultry Science 74: 1280-1286.

De FANTI, B.A., BACKUS, R.C., HAMILTON, J.S., GIETZEN, D.W., and B.A, HURWITZ. 1998. Lean (Fa/Fa) but not obese (fa/fa) Zucker rats release cholecystokinin at PVN after a gavaged meal. American Journal of Physiology 275: 1–5.

DENBOW, D.M. 1994. Peripheral regulation of food intake in poultry. Journal of Nutrition 124:1349-1354.

DOZIER, W.A., LEIN, R.J., HESS, J.B., and S.F, BILGILI. 2003. Influence of early skip-a-day feed removal on live performance and carcass yield of broilers of different sexes and strain sources. Journal of Applied Poultry Research 12: 439-448.

EMMERSON, D.A. 1997. Commercial approaches to genetic selection for growth and feed conversion in domestic poultry. Poultry Science 76:1121-1125.

ENGLER, L. 2007. What Is *Hoodia Gordonii*?http://www.healthmad.com/weight-loss/what is *Hoodia gordonii*. 16682.

FAQ. 2001. Proof of principle clinical study of P57 for obesity-successful completion of second stage (phase 2). www.phytopharm.co.uk/hoodia_faq.html#6

FERKET, P.R and A.G, GERNAT. 2006. Factors that affect feed intake of meat birds: A review. International Journal of Poultry Science 5(10): 905-911.

FISHER, C. 1984. Fat deposition in broilers. In: Fats in animal nutrition. (Ed: J. Wiseman). Easter School in Agricultural Science, University of Nottingham (37th), Butterworth, London, U.K. Pages 437-470.

FONTANA, E.A., WEAVER, W.D., WATTKINS, Jr., B.A., and D.M, DENBOW. 1992. Effect of early feed restriction on growth, feed conversion, and mortality in the broiler chickens. Poultry Science 71: 1296–1305.

FRANCHER, B.I., and L.S, JESNSEN. 1988. Induction of voluntary feed intake restriction in broiler chicks by dietary glycolic acid supplementation. Poultry Science 67: 1469-1482.

GOUS, R.M., MORAN, E.T, Jr., STILBORN, H.R., BRADFORD, G.D., and G.C, EMMANS. 1999. Evaluation of the parameters needed to describe the overall growth, the chemical growth, and the growth of feathers and breast muscles of broilers. Poultry Science 78:812-821.

GRIFFIN, H.D. 1996. Understanding Genetic Variation in Fatness in Chickens. Annual Report. Roslin Institute, Edinburgh.

GRIFFITHS, L., LEESON, S. and J.D, SUMMERS. 1977. Fat deposition in broilers: Effect of dietary energy to protein balance and early life caloric restriction on productive performance and abdominal fat pad size. Poultry Science 56: 638-646.

HAN, Y., and D.H, BAKER. 1994. Digestible lysine requirements of male and female broiler chicks during the period three to six weeks post hatching. Poultry Science 73: 1739-1745

HAVENSTEIN, G.B., FERKET, P.R., SCHIEDELER, S.E., and D.V, RIVES. 1994. Carcass composition and yield of 1991 versus 1957 broilers when fed ‘typical’ 1957 and 1991 broiler diets. Poultry Science 73:1795-1804.

HEERDEN, F.R., HORAK M.R., MAHARAJ, V.J., VLEGGAAR, R., SENABE, J.V., and P.J, GUNNING. 2007. An appetite suppressant from Hoodia species. Phytochemistry 68: 2545-2553.

HERMIER, D. 1997. Lipoprotein metabolism and fattening in poultry. Journal of Nutrition 127: 805-808.

HILLGARTNER, F.B., SALATI, L.M., and A.G, GOODRIDGE. 1995. Physiological and molecular mechanisms involved in nutritional regulation of fatty acid synthesis. Physiological Reviews 75:47-76.

HOCKING, P.M., HUGHES, B.O., and S, KEERKEER. 1997. Comparison of food intake, rate of consumption, pecking activity and behaviour in layer and broiler breeder males. British Poultry Science 38:237-240.

HOLT, S.M.D. 2005. Natural Products Industry INSIDER "The Supreme Qualities of Hoodia Gordonii".http:// www.cellhealthmakeover.com/hoodia-trim-fast-pill.html.

HOLSHEIMER, J.P., and C.H, VEERKAMP. 1992. Effect of dietary energy, protein and lysine content on performance and yield of two strains of male broiler chicks. Poultry Science 71:872-879.

HURWITZ, S., SKLAN, D., and I, BARTOV. 1978. New formal approaches for determination of energy and amino acid requirements of chicks. Poultry Science 57: 197-205.

INTERGRATEDBIOMOLECULE CORPORATION. 2005. Analytical report. www.biomoleculecorporation.com.

JENSEN, J. 2001. Regulatory peptides and control of food intake in non-mammalian vertebrates. Comparative Biochemistry and Physiology 128:471-479.

JOBST, E.E., ENROIRI, P.J., and M.A, COWLEY. 2004. The electrophysiology of feeding circuits. Trends in Endocrinology and Metabolism 15:488-499.

JONES, G.P.D., and D.J, FARRELL. 1992. Early life food restriction of the chicken. I. Methods of application, amino acid supplementation and the age at which restriction should commence. British Poultry Science 33:579-587.

KOBAYASHI, S., TERASHIMA, Y., and H, ITOH. 2006. Effects of dietary chitosan and glucosamine HCL on liver lipid concentrations and fat deposition in broiler chickens. Journal of Poultry Science 43: 156-161.

LAMOSOVA, D., MACAJOVA, M., and M, ZEMAN. 2004. Effects of short – term fasting on selected physiological functions in adult male and female Japanese quail. Acta Veterinary Brunesis 73:9–16.

LAWRENCE, T.LJ., and V.R, FOWLER. 1998. Growth of Farm Animals, CAB international, UK. Pages 104-05, 213-215.

LEE, K.H., and S, LEESON. 2001. Performance of broilers fed limited quantities of feed or nutrients during seven to fourteen days of age. Poultry Science 80: 446-454.

LEESON, S., SUMMERS, J.D., and L.J, CASTON. 1991. Diet dilution and compensatory growth in broilers. Poultry Science 70:867-873.

LEESON, S. 1996. Nutrition and Broiler Carcass Quality. Hubbard Farms Technical Report.

LEESON, S. 2000. Is Feed Efficiency Still a Useful Measure of Broiler Performance? University Books, Quelph, Ontario.

LEESON, S., and J.D, SUMMERS. 1997. Feeding programs for broilers. Commercial Poultry Nutrition. 2nd ed. University Books, Guelph. Ontario. Pages 207-254.

LEESON, S., SUMMERS, J.D., and L.J, CASTON. 1992. Response of broilers to feed restriction or diet dilution in the finisher period. Poultry Science 71:2056-2064.

LEWIS, P.D., and T.R, MORRIS. 1998. Responses of domestic poultry to various light sources. World's Poultry Science Journal 54: 72-75.

LICHTENSTEIN, A.H. 1999. Nutrtional diseases in Human beings. Nutrition Review 57:11–14.

LIPPENS, M., ROOM, G., De GROOTE, G., and E, DECUYPERE. 2000. Early and temporary quantitative food restriction of broiler chickens. 1. Effects on performance characteristics, mortality and meat quality. British Poultry Science 41:343-345.

MACAJOVA, M.D., LAMOSOVA, D., and E, ZEMAN. 2003. Physiological effects of leptin, insulin and triamcinolon on adult male Japanese quail. Acta Veterinary Brunesis 72:515–522.

MACDONALD, P., EDWARDS, R.A., and J.F.D, GREENHALGH. 1992. Evaluation of Foods (A) Digestibility. In: Animal Nutrition, 4th Edition. Longman Scientific and Technical publishers. New York. U.S.A. Pages 200–216.

MACLEAN, D.B., and L.G, LUO. 2004. Increased ATP content/ production in the hypothalamus may be a signal for energy- sensing of satiety: Studies of the anorectic mechanism of plant steroidal glycoside. Brain Research 1020(1–2): 1–11.

MACLEOD, M.G. 1991. Fat deposition and heat production as response to surplus dietary energy in fowls given a wide range of metabolisable energy:protein. British Poultry Science 32:1097-1108.

MCMINN, J.E., BASKIN, D.G., and M.W, SCHAWRTZ. 2000. Neuroendocrine mechanisms regulating food intake and body weight. Obesity Reviews 1:37-46.

MCMURTRY, J. P., ROSEBROUGH, R.W., PLAVNIK, I. and A.I, CARTWRIGHT. 1988. Influence of early plane of nutrition on enzyme systems and subsequent tissue deposition. In: Biomechanisms Regulating Growth and Development (G. L. Steffens and T. S. Rumsey, ed). Betsville Symposia on Agricultural Research [12] Klumer Academic Publishers, Dordrecht, the Netherlands. Pages 329-341.

MENCH, J.A. 2002. Broiler breeders: Feed restriction and welfare. World Poultry Science Journal 58:20-29.

MORRIS, T.R. 1971. The effects of ahemeral light and dark cycles on egg production in the fowl. Poultry Science 52(2):423-445.

NATIONAL RESEARCH COUNCIL. 1994. Nutrient Requirements of Poultry. 9th Revised edition, National Academy Press, Washington, DC.

NAHASHON, S.N., BARTLETT, J., and J.E, SMITH. 2004. Effects of late feathering genotypes on performance and carcass traits of broiler chickens. Livestock Production Science 91: 83-91.

NIR, I., NITSAN, Z., DUNNINGTON, E.A., and P.B, SIEGEL. 1987. Growth associated traits parenteral and F1 populations of chickens under different feeding programs.2. Ad libitum and intermittent feeding. Poultry Science 66: 10 - 22.

OYEDEJI, J.O., and J.O, ATTEH. 2005. Response of broilers to feeding manipulations. International Journal of Poultry Science 4(2): 91-95.

OYAWOYE, E.O., and W.F, KRUEGER. 1990. Potential of chemical regulation of food intake and body weight of broiler breeder chick. British Poultry Science 31:735-742.

OLANREWAJU, H.A., THAXTON, J.P., DIZIER, W.P., PURSEUL, J., ROUSH, W.B., and S.L, BRANTON. 2006. A review of lighting programs for broiler production. International Journal of Poultry Science 5(4):301-3078.

PASTERNAK, H., and B.A, SHALEV. 1983. Genetic-economic evaluation of traits in broiler enterprise: reduction of food intake due to increased growth rate. British Poultry Science 24:531-536.

PESCATORE, A.J., CANTOR, A.H., and H, XIANGBAI. 1992. Processing yield of eight commercial strain crosses of broilers. Poultry Science 1:71-76.

PERRY, C.C. 1981. Growth and food intake of broilers under various lighting regimes. British Poultry Science 22:219-225.

PINCHASOV, Y., and S, ELMALIAH. 1994. Broiler chick responses to anorectic agents: 1. dietary acetic and propionic acids and the digestive system. Pharmacology Biochemistry and Behavior 48:371-376.

PLAVNIK, I., and S, HURWITZ. 1985. The performance of broiler chicks during and following a severe feed restriction at an early age. Poultry Science 64:348–355.

PLAVNIK, I., and S, HURWITZ. 1988. Early feed restriction in chicks: Effects of age, duration and Sex. Poultry Science 67: 1407-1413.

PLAVNIK, I., MCMURTRY, J.P., and R.W, ROSEBROUGH. 1986. Effect of early feed restriction in broilers. In. Growth performance and carcass composition. Growth 50:68–76.

PLAVNIK, I., and S, HURWITZ. 1989. Effect of dietary protein, energy and feed pelleting on the response of chicks to early feed restriction. Poultry Science 68:1118-1125.

PLAVNIK, I., and S, HURWITZ. 1990. Performance of broiler chickens and turkeys poults subjected to feed restriction or to feeding of low sodium diets at an early age. Poultry Science 69(4):945-952.

PLAVNIK, I., and S, HURWITZ. 1991. Response of broiler chickens and turkey poults to food restriction of varied severity during early life. British Poultry Science 32:343–352.

POCAI, A., LAM, T.K.T., JUAREZ, R.G., OBICI, S., SCHWARTZ, G.T., BRYAN, L.A., and L, ROSSETTI. 2005. Hypothalamic K_{ATP} channels control hepatic glucose production. Nature 434:1026–1031.

POND, W.G., CHURCH, D.C., and K.R, POND. 1995. Basic Animal Nutrition and Feeding. 4th Edition. U.S.A. Pages 273-275, 495-498.

REIDELBERGER, R.D., HERNANDEZ, J., FRITZSCH, B., and M, HULCE. 2003. Abdominal vagal mediation of satiety effects of CCK in rats. American Journal of Physiology 286:1005–1012.

RICHARDS, M.P. 2003. Genetic regulation of feed intake and energy balance in poultry. Poultry Science 82:907–916.

ROBERT, E.T. 1992. Poultry and egg products. In: Paul, C (Ed). Scientific Farm Animal Production: An Introduction to Animal Science, Fourth Edition. MacMillan, publishing company. New York. U.S.A. Pages 11–25.

RONDELLI, S., MARTINEZ, O., and P.T, GARCIA. 2003. Sex effect on productive parameters, carcass and body fat composition of two commercial broiler lines. Brazilian Journal of Poultry Science 5(3): 169-173.

ROSEBROUGH, R.W., and J.P, MCMURTRY. 1993. Energy repletion and lipid metabolism during compensatory gain in broiler chickens. Growth Development and Aging 57:73-83.

ROSEBROUGH, R.W., STEELE, N.C., MCMURTRY, J.P., and I, PLAVNICK. 1986. Effects of early feed restriction in broilers. II. Lipid metabolism. Growth 50:217-227.

SACKS, F.M. 2002. The role of high density lipoprotein (HDL) cholesterol in the prevention and treatment of coronary heart disease. American Journal of Cardiology 15: 139–143.

SALEH, E.A., WATKINS, S.E., WALDROUP, A.L., AND P.W, WALDROUP. 2005. Effects of early quantitative feed restriction on live performance and carcass composition of male broiler grown for further processing. Journal of Applied Poultry Research 14:87-93.

SALEH, E.A., WATKINS, S.E., WALDROUP, A.L., and P.W, WALDROUP. 2004. Comparison of energy feeding programs and early feed restriction on live performance and carcass quality of large male broilers grown for further processing at 9 to 12 weeks of age. Journal of Applied Poultry Research 3(1):61-69.

SANTOS, A.L., SAKOMURA, N.K., FREITAS, E.R., FORTES, C.M.S., and E.N.V.M, CARRILHO. 2005. Comparison of free range broiler chicken strain raised in confined or semi confined systems. Brazilian Journal of Poultry Science 7(2): 85-92.

SANTOSO, U., TANAKA, K., and S, OHTANI. 1995. Early skip-a-day feeding of female broiler chicks fed high-protein realimentation diets. Performance and body composition. Poultry Science 74:494-501.

SAS. 2003. Statistical Analysis Systems User's Guide: Statistics, 9th edition. SAS Institute, Inc. Raleigh, North Caroline, USA.

SAVORY, C.J., and K, MAROS. 1993. Influence of degree of food restriction, age and time of the day on behaviour of broiler breeder chickens. Behaviour Process 29:179-190.

SAVORY, C.J., HOCKING, P.M., MANN, J.S., and M.H, MAXWELL. 1996. Is broiler breeder welfare improved by using quantitative food restriction to limit growth rate? Animal Welfare 5:105-127.

SCHEIDELER, S.E, and G.R, BAUGHMAN. 1993. Computerised early feed restriction Programs for various strains of broilers. Poultry Science 72: 236-242.

SCHWARTZ, M.W., WOODS, S.C., PORTE, D, JR., SEELEY, R.J., and D.G, BASKIN. 2000. Central nervous system control of food intake. Nature 404:661-671.

SIMON, J., DEROUET, M., and C, GESPACH. 2000. An anti-insulin serum but not a glucagon antagonist, alters glycemia in fed chickens. Hormonal and Metabolic Research 32: 139-141.

SIZEMORE, F. G., and H.S. SIEGEL. 1993. Growth, feed conversion, and carcass composition in females of four broiler crosses fed starter diets with different energy levels and energy to protein ratios. Poultry Science 72:2216–2228.

STAHL, L. 2004. *Hoodia gordonii*. November 21. 2004 CBS 60 minutes report. http://www.hoodiagordoniiplus.com.

SUMMERS, J.D., SPRATT, D. and J.L, ATKINSON. 1990. Restricted feeding and compensatory growth for broilers. Poultry Science 69:1855-1861.

TULP, O.L., HARBI, N.A., and A, DERMARDEROSIAN. 2001. Effect of Hoodia plant on food intake and body weight in lean and obese LA/Ntul//cp-rats. FASEB Journal 15(4):404.

TUMOVA, E., SKRIVAN, M., SKRIVANOVA, V., and L, KACEROVSKA. 2002. Effect of early feed restriction on growth in broiler chickens, turkeys and rabbits. Czech Journal of Animal Science 47(10):418-428.

TRAYHURN, P., and J.H, BEATTIE. 2001. Physiological role of adipose white tissue as an endocrine and secretory organ. Proceedings of the Nutrition Society 60:329-339.

URDANETA, M.R., and S, LEESON. 2000. Mild Feed Restriction and Compensatory Growth in the Broiler Chickens. University of Guelph. National Library of Canada, Canada.

URDANETA, M.R., and S, LEESON. 2002. Quantitative and Qualitative feed restriction on growth characteristics of male broiler chickens. Poultry Science 81:679-688.

WALKER, A.W., WISEMAN, N.J.L., and D.R, CHARLSE. 1995. Recent finding on the effects of nutrition on the growth of specific broiler carcass components. In: Recent advances in animal nutrition. Nottingham University Press. Nottingham. England.

WILSON, L.J., WEAVER, JR, W.D., BEANE, W.L., and A, CHERRY. 1984. Effects of light and feeding space on leg abnormalities in broilers. Poultry Science 63:565-567.

WILLIAMS, G., BING, C., CAI, X.J., HARROLD, J.A., KING, P.J., AND X.H, LUI. 2002. The hypothalamus and the control of energy homeostasis. Physiology and Behavior 74(4–5): 683–701.

WOODS, S.C., SEELEY, C.R., PORTE, JR, D., and M.W, SCHWARTZ. 1998. Signals that regulate food intake and energy homeostasis. Science 280:1378–1383.

YAMI, A. 1995. Poultry production in Ethiopia. World Poultry Science Journal 51:197-202.

YOLCIN, S., OZOKAN, S., ACIKGOZ, Z., and K, OZAN. 1990. Influence of dietary energy on performance, carcass parts yield and nutrients composition of broilers reared at natural optimum and summer temperatures. British Poultry Science 39:633-638.

YOUNG, L.L., NORTHCUTT, J.K., BUHR, R.J, LYON, C.E., and G.O, WARE. 2001. Effects of age, sex, and duration of postmorterm aging on percentage yield of parts from broiler chicken carcasses. Poultry Science 80: 376-379.

ZHONG, C., NAKAUE, H.S., HU, C.Y., and W, MIROSH. 1995. Effect of full feed and early feed restriction on broiler performance, abdominal fat level, cellularity, and fat metabolism in broiler chickens. Poultry Science 74:1636-1643.

ZUBAIR, A.K., and S, LEESON. 1994. Effect of varying period of early nutrient restriction on growth compensation and carcass characteristics of male broilers. Poultry Science 73:129–136.

ZUBAIR, A.K., and S, LEESON. 1996. Compensatory growth in the broiler chicken: a review. World's Poultry Science 52:189-201.

ZEREHDARAN, S., VEREIJKEN, A.L.J., ARENDONK, J.A.M., and E.H. VAN DER WAAIJ. 2004. Estimation of genetic parameters for fat deposition and carcass traits in broilers. Poultry Science 23:521-525.

CHAPTER 7

APPENDICES

APPENDIX 7.1: VACCINATION PROGRAM

The vaccination program of the study was as indicated below.

WEEK ONE:

Day one on arrival:

1. Chicks were vaccinated against Newcastle disease from the hatchery using Clone 30. Secondly, Vita stress was added in the drinking water immediately on arrival for the first two days to calm down the chicks due to stress they might have experienced through transportation and handling.

Day three:

1. Tylo Tad was added in the drinking water for prevention of *Escheria coli* bacteria and other disease causing microorganisms

Day seven:

Chicks were vaccinated against Infectious Bronchitis using “IBH 120”.

WEEK TWO:

Day twelve:

Chicks were vaccinated against Gombora using D78 through drinking water.

WEEK THREE:

Day eighteen:

Chicks were vaccinated against Gumbora using D78 through drinking water.

Day twenty one:

Tylo Tad was added in the drinking water. The withdrawal period was 15 days.

WEEK FOUR:

Day twenty three:

Chickens were vaccinated against Newcastle disease using Clone 30.