EXPERIMENTAL ASCARIDIA GALLI INFECTION:

EFFECTS ON EGG - TYPE PULLETS

By

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DEDICATION

This thesis is dedicated to my parents
Mr. O.E. Ntekim and Mrs. A.E. Ntekim
for fulfilling their role as parents
by providing a good future for me.

DECLARATION

This is to certify that I carried out the work reported in this thesis in the Department of Veterinary Parasitology and Entomology, Ahmadu Bello University, Zaria. The project was supervised by Drs. R.A. Ogunsusi and O.O. Akerejola both of the Faculty of Veterinary Medicine and Dr. S.O. Ogundipe of the Institute of Agricultural Research.

The work of other investigators in related field is acknowledged and referred to. No part of this thesis has been submitted elsewhere for a degree or diploma.

_____

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ABSTRACT

In an experiment designed to study the effect of Ascaridia galli on the performance of egg-type pullets, 90 Harco chickens, divided into 3 groups of 30 each were used. Groups I and II were given a total of 10,000 and 50,000 infective Ascaridia eggs respectively in 10 instalmental doses and group III served as uninfected control.

Chickens in group I started shedding Ascaridia eggs in their faeces on day 91 while those in group II started shedding eggs on day 85 after initial infection.

Within the first 2 weeks, slight depression in feed intake was observed among the infected chickens but during the growing and laying phases, the infected chickens consumed more feed than the control chickens.

During the growing phase, feed conversion was significantly higher in the control group than the group which received 50,000 infective eggs.

Similarly, feed efficiency was significantly higher in the control group than the infected chickens between the third and fourth week in lay. Thereafter, the differences were not significant.

The control chickens initially gained more weight than the infected birds especially within the first 2 weeks after initial infection but the gain was only significant between the control and the group that received 50,000 infective eggs. During the growing and laying phases, differences in body weight gain between the 3 groups were not significant.

The control chickens started laying eggs 10 days ahead of the infected chickens and their rate of egg production was significantly higher between the third and fourth weeks in lay. Thereafter differences between groups were not significant. The control chickens attained 50% level of egg production ahead of the infected chickens.

Chicken mortality due to infection was insignificant and the numbers of worms recovered at necropsy were very low.

TABLE OF CONTENTS

	Page
INTRODUCTION	1
Justification for the research ...	2
LITERATURE REVIEW	4
Life cycle of *Ascaridia galli*	4
The preparasitic phase	4
Effect of temperature on egg development	4
Effect of sunlight on egg development	5
Effect of oxygen on egg development ...	6
Effect of dessication on egg development	6
The parasitic phase	7
Prepatent period of *Ascaridia galli* ...	8
Effect of *Ascaridia galli* infection on feed consumption and feed efficiency	10
Effect of *Ascaridia galli* infection on body weight	12
Effect of *Ascaridia galli* infection on egg production	16
Mortality due to *Ascaridia galli* infection ...	18
Ascaridia galli burden in the intestine of the host	20
Pathological changes due to *Ascaridia galli* infection	22
Resistance to infection with *Ascaridia galli* ...	27

Page

Age resistance 28

Acquired resistance 30

Resistance due to deficient diet ... 33

Vitamins and minerals 33

Proteins and amino acids ... 36

Resistance due to blood loss ... 38

Resistance due to breed of the host ... 38

Management systems and the predisposition of chickens to helminth infections ... 39

Risk of infection in the different management systems 41

MATERIALS AND METHODS 44

Selection of experimental chickens ... 44

Feeding 46

Preparation of infective Ascaridia galli eggs 47

Infection of chickens 48

Data collection 49

Worm recovery 50

Statistical analysis 51

RESULTS 58

Prepatency period 58

Feed consumption 58

Page

Feed conversion and feed efficiency ... 60

Changes in body weight 61

Egg production 62

Egg weight 62

Worm recovery 63

Feed analysis 64

DISCUSSION 76

CONCLUSIONS AND RECOMMENDATIONS 86

REFERENCES 88

APPENDIX

I The periodic (bi-weekly) mean values and standard errors for feed consumption, cumulative feed conversion, feed efficiency, changes in body weight, hen-day egg production and egg weight per group of the experimental chickens 97

II Analysis of variance (ANOVA) for feed consumption, cumulative feed conversion, feed efficiency, changes in body weight, hen-day egg production, hen-housed egg production, fifty percent egg production, and egg weight in the three groups of experimental chickens ... 107

LIST OF TABLES

Table		Page
1.	Percentage composition of poultry feed used in the experiment as indicated by Pfizer Livestock feeds	55
2.	Chemical analysis of the 3 feed types used for the experiment	65

LIST OF FIGURES

Figure		Page
1.	Sketch of the poultry house showing the arrangement of chicken groups in the cage cells	54
2.	Mean feed consumption of experimental chickens per fortnight during the growing phase	66
3.	Mean feed consumption of experimental chickens per fortnight during the laying phase	67
4.	Cumulative feed conversion for experimental chickens per fortnight during the growing phase	68
5.	Feed efficiency of experimental chickens per fortnight during the laying phase ...	69
6.	Changes in body weight of experimental chickens per fortnight during the growing phase	70
7.	Changes in body weight of experimental chickens per fortnight during the laying phase	71
8.	Hen-day egg production of experimental chickens per fortnight during the laying phase	72

Figure Page

9. Hen-housed egg production of experimental chickens per fortnight during the laying phase 73

10. Mean egg weight of experimental chickens per fortnight during the laying phase ... 74

LIST OF PLATES

Plate		Page
I	The poultry rearing house used for housing the experimental chickens after brooding	56
II	Cage cells with common feeders and drinkers for experimental chickens ...	56
III	Fresh Ascaridia egg	57
IV	Infective Ascaridia eggs as obtained from a 21 day old culture	57
V	Worms at a cut end of an intestine from one of the infected chickens	75

INTRODUCTION

In commercial poultry farming, birds are usually managed under artificial confitions involving large numbers. The main objective of every poultry farmer is to achieve maximum production efficiency. That objective can only be achieved when broiler birds reach slaughter weight with the least amount of feed consumed and maximum egg production in the laying period obtained from pullets with high feed efficiency. If a bird requires a longer period to reach slaughter weight, that bird consumes more feed and that may reduce or eliminate the farmer's profit. Such reduction in profit may also be encountered by a farmer if the feed efficiency of laying pullets is low.

The effect of intestinal worm infections in poultry flocks have often been underestimated. The significance of intestinal worms infection tc overall flock health and profits have not been highly regarded by many poultrymen. Intestinal parasites of domestic fowls bring about losses directly and indirectly (Dearstyne et al., 1929). The direct loss takes place through mortality, retarded growth and drop in egg production. The indirect loss is incurred by the fact that mechanical injury brought about by the parasites predisposes the fowls to other diseases.

This present study involves the large roundworm of chickens, Ascaridia galli, which is normally parasitic in the small intestine of the host. It belongs to the Phylum Nemathelminthes, Class Nematoda, Order Ascaridida and Family Ascarididae.

The purpose of the study was to determine the effect of experimental infection on feed consumption, feed conversion, feed efficiency, growth rates, age at onset of egg production, rate of egg production, egg weight and mortality in chickens infected orally with repeated doses of infective eggs of Ascaridia galli. The prepatency of the infection and intestinal worm load in the chicken were also determined.

Justification for the research

In Nigeria more and better quality food is one of the urgent needs of the people. Animal protein is one of the nutrients currently lacking in many Nigerian diets and the poultry industry has the greatest potential to bridge the protein deficiency gap in the shortest possible time (David-West, 1979).

It is becoming more apparent that the expansion of cattle population cannot progress at a satisfactory rate to cope with the increasing demand for meat. As such many Nigerians are taking to poultry production because it yields quick and

adequate returns. Many Nigerians keep poultry (Chickens) for one or more of the following reasons:

- to supply eggs and meat for family use;
- as an important enterprise on the farm to supplement the annual income;
- as a commercial enterprise providing the bulk of the family income;
- as a hobby.

In Nigeria the commonly practised systems of poultry maintenance are the free range, semi-intensive, deep litter, and the cage systems. Most commercial poultry flocks in Nigeria are still managed under the deep litter system. Some of these systems despite their advantages predispose chickens to infection from Ascaridia galli and many other intestinal worms. Unfortunately, Ascaridia galli is not given the same degree of attention as is given to other causes of ill health in poultry flock, so losses due to ascaridiasis is not well documented.

The infection due to Ascaridia galli could be insidious or obvious. It is highly essential to draw the attention of our farmers to the loss which could be incurred in monetary terms as well as efforts as a result of Ascaridia galli infection. This is the principal aim of this study.

LITERATURE REVIEW

Life cycle of Ascaridia galli

The Preparasitic Phase

Ascaridia galli eggs are passed out in the faeces of chicken and develop in the outside environment. The preparasitic stages are known to be fairly resistant to adverse weather conditions with the embryonated eggs being more resistant than the earlier stages of development. The rate of development of the eggs to the second stage infective larvae is influenced by the temperature, sunlight and moisture content of the environment.

Effect of temperature on egg development

Itagaki (1927), observed in an experimental study that eggs of A. galli survived five minutes exposure between 50°C and 53°C but not at 54°C. Ackert (1931), reported that at 30°C, eggs of A. galli became infective in 16 days but at 33°C in 10 days. Twelve hours exposure at 43°C was lethal for eggs in all stages of development. He also observed that 10°C was the lowest temperature at which development could take place.

Roberts (1936a), in an experimental study reported that the infective stage (L_2) could be reached in nine days at 30°C and seven days at 33°C. He also observed that the optimum temperature range for development of eggs was 30°C to 33°C. High temperature as shown by the results from exposure to 45°C to 46.5°C was fatal to all the eggs within 24 hours. Prolonged exposure to low temperatures was fatal. At -7.5°C to -3°C, the eggs could not survive beyond 16 days.

In another experiment, Reid (1958) reported that the minimum period required for eggs to reach infective stage after being voided by the bird was six days at 30°C and five days at 34°C and above, normal development ceased. Development, according to him, continued at a slower rate as the temperature was reduced until the minimum of 19°C was reached, below which development was arrested.

Effect of sunlight on egg development

Hartman (1924), reported that eggs of A. galli on the surface and down to a depth of six inches in unshaded soil were killed in the hot season while eggs in shady spots maintained their viability for 100 days. Roberts (1936a), observed that eggs in droppings exposed to sunlight and in the presence of moisture maintained their viability for about 28 days while eggs in droppings under dry conditions survived only 14 days. Since the two experiments were carried out concurrently, Roberts concluded that the condition of dessication was responsible for the decreased survival period.

Effect of oxygen on egg development

The amount of oxygen in the environment also influence the rate of development of the eggs. Itagaki (1927), reported that fresh Ascaridia eggs kept in culture medium free from oxygen failed to develop and died within a few days. Fairbairn (1970), reported that Ascaris eggs are obligate aerobes, as such embryonation should be carried out where there is adequate amount of oxygen.

Effect of dessication on egg development

The influence of dryness on the egg has been investigated by Itagaki (1927), who noted that fresh eggs of A. galli maintained in a dessicator for four days did not develop completely and subsequently died. On the other hand, embryonated eggs containing the infective larvae were still alive up to 60 days.

Mc-Rae (1935), demonstrated that at 22°C a minimum relative humidity of 82% to 86% was necessary for complete development of A. galli eggs. With relative humidity of 40% to 50% all the eggs died after eight days. Eggs kept in an atmosphere saturated with water, at 30°C developed exactly as did those in water at the same temperature.

Roberts (1936a), observed that both fresh and embryonated eggs of A. galli which were dried out on glass slides, survived twenty-five but not thirty days. He also observed

that embryonated eggs appeared more resistant to dessication than eggs in the earlier stages of development.

The Parasitic Phase

The life cycle of A. galli is direct. Infection takes place by ingestion of the eggs with food or water. Earthworms may ingest the eggs and may, when they are ingested, transmit infection mechanically (Scott, 1913). Other mechanical transmitters include grasshoppers (Jacob et al., 1970), housefly (Schwartz, 1975). The infective eggs when picked up by chicken hatch mainly in the duodenum (Roberts, 1936b). A few eggs may hatch either in the crop or gizzard (Itagaki, 1927).

It was previously thought that the tissue phase in the life cycle of A. galli, which is the phase in which the young worms bury their anterior ends in the mucosa of the small intestine, began from the seventh to the seventeenth day after infection (Ackert, 1931), from the tenth to the seventeenth day after infection (Roberts, 1936b) and from the sixth to the fourteenth day after infection (Khouri and Pande, 1970). Tugwell and Ackert (1952), and Horton-Smith et al. (1968) however reported that the tissue phase began on the first day of parasitism by a few Ascaridia larvae. Tugwell and Ackert (1952) stated that this continued at least to the twenty sixth day but that the great majority of the larvae made their entry into the intestinal mucosa from

about the seventeenth day after hatching. During this period a few Ascaridia may also occur freely in the intestinal lumen. They noted that the growth rates of the tissue and lumen larvae were about the same until the seventeenth day of parasitism, after which the lumen worms made normal growth but the tissue larvae little growth. The failure of the tissue larvae to make growth comparable with that of the lumen larvae, according to Tugwell and Ackert (1952),explained the occasional presence of diminutive larvae in the lumen flushings. Migration of the larvae outside the intestinal tissue is very rare (Moran and Mizelle,1956). Of the four moults characteristic of A. galli, the first occurs in the egg within eight days of development (Roberts, 1936b). The second, third and fourth moults occur between six to eight days, fourteen to fifteen days and eighteen to twenty two days after infection respectively (Tugwell and Ackert,1952). The young adults emerge from the mucosa twenty two days after infection and remain in the lumen till they reach sexual maturity. However, Tugwell and Ackert (1952), stated that the third and the fourth moults may be delayed if the larvae spend too long a time in the mucosa.

Prepatent Period of Ascaridia galli

Itagaki (1927), reported that the worms reached maturity from 37 days after infection but the number of eggs fed to the chicken and age of the chicken were not mentioned. Ackert

(1930), infected one month old white Leghorn chickens with a single dose of 50 A. galli eggs and observed that the worms did not mature earlier than 50 days. Roberts (1936b), working with four week old birds observed that when a single dose of 100 eggs of A. galli were fed to chickens, the minimum period of maturity was 27 days.

In another experiment, Kerr (1955) reported that when chickens up to and including 32 days of age were infected with eggs of A. galli, viable eggs were consistently found in faeces 30 to 33 days after infection. Chickens, 62 days of age, passed out eggs consistently from 35 days after infection. In chickens infected at 90 days of age, no mature eggs were found until after 50 days of infection.

Soulsby (1968), reported that the infective larvae of A. galli may reach sexual maturity between six and eight weeks while chickens over three months or more are more resistant to infection and prepatent periods may be prolonged.

Ikeme (1971a), infected three groups of chickens with 10 (group 1), 100 (group 2) and 1,000 (group 3) eggs of A. galli, daily for six weeks. Birds in each of the groups were divided into two subgroups. Sub-group A received 10% protein diet with vitamin supplement and mineral salts while sub-group B received 10% protein diet without vitamin supplement but with mineral salts. The chickens were infected

at 17 days of age. He observed that the prepatent period for birds in the group that received 10 eggs daily was 36 days irrespective of dietary deficiency. The prepatent period for birds in subgroups 3A and 4B was 42 days. Birds in subgroup 4A passed no eggs throughout the 11 week period of the experiment. From the results obtained, he stated that at low dose level, a substantial proportion of worms reached adult stage quickly. At high dose levels development to the adult stage was significantly delayed and larval populations persisted in high proportions.

Macchoini et al. (1971), infected three groups 1, 2, 3, of guinea fowls with 350, 1,000 and 3,000 eggs of Ascaridia numidae respectively. The birds were infected at 20 days of age. They observed that the prepatent period in the three groups was 27 days.

Effect of Ascaridia galli infection on feed consumption and feed efficiency

One of the ways in which helminths affect animals is through depression of feed intake and this is thought to be the major reason for weight loss in helminth infections.

Ackert (1930), summarising the results of his investigation showed that chicks 2 to 7 weeks old to which doses of 1,000 to 2,000 eggs of A. galli were given lost their appetite within

ten days. By the twelfth day they were visibly affected with depressed humped-up appearance. The appetite of the birds that survived after the seventeenth day became voracious and recovery was rapid.

Costa (1970), in the bid to determine the effect of A. galli infections on feed efficiency of egg-type pullets used three groups of 60 chickens each. Chickens in group A were each inoculated with 150 infective eggs of A. galli at 30 days of age and those in group B were given the same number of egg at 60 days of age. Chickens in group C were used as uninfected controls. At 150 days of age, he observed that feed efficiency in groups A and B were lower than in group C.

In another experiment, Dimitrov (1971), studied the effect of A. galli infection on broiler production and concluded that feed intake of the infected birds was 4 to 6% more than that of the control chickens 54 days after infection. But increase in body weight of infected chickens was 6 - 17% less than that of the control chickens. The feed effeciency of chickens dosed with 100, 500 and 1,000 infective eggs was 4.34, 4.44, 4.47 respectively as compared with 4.20 for the uninfected control chickens.

Another member of the superfamily Ascariodea which brings about loss of appetite in infected animals is Ascaris

suum. Chubis (1964), reported loss of appetite in pigs infected with Ascaris suum.

Effect of Ascaridia galli infection on body weight

Several studies have shown that an important aspect of pathogenicity in A. galli infection is the effect on weight of infected birds. Herrick (1924), reported significant differences in the weight gain between infected and control chickens, especially during the first two weeks of infection. Infected chicks he said may eventually throw off their worms and recover to a great extent if not entirely.

Stunted growth, and, or loss of weight have been reported in chickens naturally and experimentally infected with A. galli (Ackert and Herrick, 1928; Ackert, 1930; Brewer and Edgar, 1971; Dimitrov, 1971; Pavlicek and Dykova, 1975; Amer et al., 1976; Katara and Rai, 1980; Matta and Ahluwalia, 1980).

Ackert and Herrick (1928) attributed loss of weight to injury caused to the intestinal mucosa by the parasite, with the resultant loss of blood and probable secondary bacterial infection. Ackert and Wisseman (1946), infected chickens 23 days of age with 200 infective eggs of Ascaridia galli and discovered that the differences between the mean weight gains made by the control chickens as compared with

the parasitized chickens were negligible two and four weeks after infection.

Other parasitic helminths which can bring about loss of weight or stunted growth in chickens include Capillaria caudiflata (Greevaerts, 1962; Willomitzer and Lebduska 1963), Heterakis gallinarum (Stoimenov, 1972). Kulkarni and Bradley (1972) recorded weight losses in chickens infected with mixed infections of Ascaridia galli and Eimeria tenella; Heterakis gallinarum and Eimeria tenella; Ascaridia galli and Heterakis gallinarum. They noted that the combinations of Eimeria tenella and Heterakis gallinarum were the most pathogenic.

Weight loss or stunted growth due to ascaridiasis has been reported in hosts other than chickens. Macchioni et al. (1971), infected three groups of guinea fowls, 20 days of age with a single dose of 350, 1,000 and 3,000 infective eggs of Ascaridia numidae respectively. The reduction in growth rates began 21 days after infection and those infected with 3,000 eggs lost weight between 31 and 37 days after infection Weight loss due to Ascaridia numidae infection has also been reported by Macchioni et al. (1978).

These experiments from which information on weight of infected chickens were obtained are mostly short experiments based on single infections. There are relatively few reports on loss of weight with infections when repeated doses are used to observe a long term effect on weight of

chickens. Roberts (1936c), in one of such studies used three groups of five chickens each. Chickens in the first group were dosed with 100 eggs of Ascaridia galli from 30 days of age. The doses were increased to 300 eggs daily when the chickens were 85 days old. Chickens in the second group were dosed with 300 eggs daily from 58 days of age. The doses were increased to 1,000 eggs when the chickens were 118 days old. Chickens in the third group were infected with 500 eggs from 79 days of age and the doses increased to 1,000 eggs per day when the chickens were 119 days of age. Stunted growth was observed in all the groups.

In another experiment, Sadun (1948) used seven chickens which at five days of age were dosed with 7,000 eggs of Ascaridia galli. Twenty three days after the first dose, each chicken received 40,000 eggs and twenty two days after the second dose, they received 1,250 eggs. Eighteen days after the third dose each chicken received another dose of 8,000 eggs. Weight loss was noted, though the experiment was designed to study resistance to infection with A. galli.

Ikeme (1971b), studied the comparative growth rates of three groups of chickens dosed daily with 10, 100 and 1,000 infective eggs respectively of Ascaridia galli under different nutritional levels. The experiment was commenced when the chickens were 17 days old and daily doses at the appropriate levels continued for six weeks. From the results obtained he

stated that when chickens received repeated doses of eggs, there was a significant depression in growth rates.

Depression in growth rates or loss of weight is also influenced by the size of the infecting dose. Ikeme (1971b), in the same experiment reported that the most severely affected chickens were those that received 1,000 eggs daily and the least affected were those that received 10 eggs daily for six weeks. Macchioni et al. (1971, 1978), also observed that ascaridiasis adversely affected the growth rates of guinea fowls only in heavily infected birds (1,000 and 3,000 eggs). In another experiment, Matta and Ahluwalia (1980), reported that chicks infected with single doses of 500 and 1,000 infective eggs of A. galli showed an average depression of 17% and 34% respectively in weight gains compared to the uninfected control chickens.

It is on record that there is a correlation between adult worm burden in the host and depression in weight gain. Todd and Hansen (1951), in a series of experiments with 566 two-week old chicks each infected with 50 eggs of A. galli, found out that chickens with fewer worms gained less weight than chickens with many worms, three weeks after infection. From the results obtained, they developed a hypothesis that birds which expended energy in throwing off infections gained less weight than those which tolerated the infection.

These experimental results are, however, contrary to the generally accepted view of parasitism, that worm damage is in part directly dependent upon worm number.

In another experiment involving 338 three-week old chicks exposed to 300 infective eggs of A. galli, Reid and Carmon (1958), found that the chickens had significant decreases in body weight as the number of adult worms increased.

Effect of Ascaridia galli infection on egg production

Though a number of investigators have reported the effect of A. galli infection on growth rates of chickens, only a few reports of the effect of infection on egg production are available.

It is generally conceeded that Ascaridia infection has a marked effect on egg production. But Ackert and Herrick (1928), did not find any marked difference in egg production of hens suffering from ascaridiasis and uninfected flock. They reported that chicks given several doses of infective eggs sufficiently high to produce marked symptoms, may eventually recover and that no effect of the infection on egg production may be visible several months later when birds become mature.

Costa (1970), inoculated two groups of 60 chickens each with 150 infective eggs of A. galli. Chickens in group A were infected at the age of 30 days while chickens in group B were infected at 60 days of age.
There were 60 controls. He observed that the first eggs were dropped at 135 days of age by the uninfected control chickens and the infected birds started laying at 146 days of age. Fifty percent production was attained by the control chickens at 170 days of age and by chickens in group B and A at 172 and 183 days of age respectively. The rate of egg production was lower in the parasitized as compared with control chickens.

Sadzikova (1975), observed that in a natural A. galli infection, egg laying was delayed and reduced. It was also observed that the eggs were fewer and smaller (49.9 grams) compared with eggs of uninfected flock which had an average weight of 54.3 grams.

In another experiment, Matta (1981), used 22 white Leghorn pullets at three months of age. He divided them into two groups such that there were 16 chickens in group A and 6 chickens in group B. Chickens in group A were each infected with 1,000 eggs of A. galli and chickens in group B served as controls. The average production of eggs per infected hen for a period of 12 weeks was 36.5 while average egg production of the uninfected control chickens for the same period was 50.2 eggs.

Greevaerts (1962), reported that heavy infection of chickens with species of Capillaria resulted in decreased egg production. In their report, Belonosov and Kravtsov (1976), stated that mixed infection of laying pullets with A. galli and Heterakis gallinarum resulted in lowered egg production and that proteins were less well digested in infected chickens.

Mortality due to Ascaridia galli infection

Guberlet (1924), found that a single dose of 750 infective eggs of A. galli given to chickens one month of age caused droopiness and death on the ninth and tenth day after infection. Heavy infection were noted to be fatal as early as the eighth day. The effects were considered by Guberlet to be due to intestinal irritation and toxemia.

Ackert and Herrick (1928), reported increased mortality when chicks four to eleven weeks of age were infected with 500 to 2,000 infective eggs of A. galli. They found out that the most deleterous period in the life cycle of the worms was about the fourteenth day after infection. The symptoms of weakness, droopiness, loss of appetite exhibited by heavily infected chicks were most marked between the tenth and the seventeenth day after infection, thus, suggesting that the majority of the worms were in the mucosa during that period.

In another experiment, Roberts (1936c), infected three chickens 43 days of age with a single dose of 10,000, 10,000 and 15,000 eggs respectively. As early as three days after infection the birds were depressed, humped up, with ruffled feathers and drooping wings. By the eleventh day the birds were extremely emaciated and were voiding blood streaked faeces. One of the birds that received 10,000 ova died on the twelfth day and the bird that received 15,000 ova died on the thirteenth day. The second bird that received 10,000 ova was still alive on the sixteenth day when it was killed.

Horton-Smith et al (1968), reported that when large numbers of Ascaridia dissimilis eggs were given to 2 one-week old turkey poults, death occurred on the tenth and the eleventh day respectively. Large numbers of larvae were recovered from the small intestine. This, they said, indicated that Ascaridia dissimilis like Ascaridia galli was capable of killing poults by massive invasion of the tissue by the larvae.

Ikeme (1971b), infected three groups of chickens with 10 (group I), 100 (group II) and 1,000 (group III), eggs of A. galli daily for six weeks. Each of the groups was subdivided into two. Chickens in the first subgroup received 10% protein diet with vitamin supplement and mineral salts. The second sub-group received 10% protein and only mineral salts. Mortalities were recorded in all the subgroups. The highest

mortalities occurred in the subgroups that had 10% protein diet with no vitamin supplement. In all subgroups, mortality was directly related to egg dosage size; the highest deaths occurred in the 1,000 egg per day dosage group.

Hamsley (1971), recorded mortalities ranging from 8% to 20% in flocks of trukey naturally infected with Ascaridia species. Pavlicek and Dykova (1975), fed five groups of cockerels aged 5 to 48 days with varying doses of between 1,500 and 3,000 infective eggs of A. galli. The effect was greatest in birds five to six days old in which mortality was between 46% to 100%. In another experiment, Belonosov and Kravtsov (1976), recorded a lower survival rate in laying hens infected concurrently with A. galli, and Heterakis gallinarum as compared with control chickens.

Ascaridia galli burden in the intestine of the host

It is on record that in certain helminth infections the proportion of large doses of eggs which became established is often smaller than in small doses. Among these are the observations of Clapham (1934), with Heterakis gallinae and Norton and Joyner (1965), with Capillaria obsignata in chickens.

Several studies have shown that in Ascaridia infection, a good number of larvae do not develop to maturity. Reid and Carmon (1958), infected 388 three-week old chicks each

with 300 infective eggs and three weeks after infection they recovered a total of 1,957 worms. Of the 388 infected birds, 58 birds contained no worms, and the majority contained less than 10 worms. Macchioni et al.(1971), infected three groups of guinea fowls, 20 days of age with a single dose of 350, 1,000 and 3,000 infective eggs of Ascaridia numidae respectively. They observed that larval development to the adult stage varied between 24 and 34 percent.

In another experiment, Dimitrov (1971), infected three groups of 120 chicks with 100 (group I), 500 (group II) and 1,000 (group III) eggs of Ascaridia galli. At 15, 30, and 54 days after infection, the mean number of worms per bird were 12, 11, 6 respectively for group I; 16, 10, 7 in group II and 21, 15, 9 in group III. The proportion of larvae recovered at the different periods became less with increasing dose.

Ikeme (1971a), tried to find out the difference in development of infection acquired at the rates of 10 eggs and 1,000 eggs per day for six weeks. In the low dose group, out of 60 birds, 46 carried infection and contained a total of 370 adult worms at necropsy. The high dose group according to him, did not produce adult worms until the tenth week and only nine out of the whole group had adult worms (20 worms). From the results obtained, he stated that substantial numbers of worms reach maturity in the birds with

small infections whereas only a very small number of worms in heavy infections reach the adult stage. The results prompted him to study changes that might take place in larval population with a single dose of 1,000 eggs. The chickens were given a single dose of 1,000 eggs of A. galli, and slaughtered weekly for worm counts. The average weekly worm populations were as follows: 867 larvae in the first week, 500 larvae in the second week and 133 larvae in the third week. In the fourth week there was an average of 33.3 larvae and 5.2 adult worms per chicken.

Matta and Ahluwalia (1980), recovered a maximum of 82 adult worms per bird, 20 weeks after infecting 2-week old chicks with a single dose of 1,000 eggs of A. galli. In another experiment,Matta (1981) recovered an average of 20 worms per bird, 20 weeks after infecting three month old pullets with 1,000 eggs of A. galli.

Pathological changes due to Ascaridia galli infection

Itagaki (1927), reported in Tokyo, Japan that the larvae of Ascaridia galli caused formation of nodules in the intestinal walls but he made no other comments on the pathogenicity of Ascaridia galli. Nodule formation due to A. galli infection have been reported by Deorani (1968) and Matta and Ahluwalia (1979). Deorani (1968), suggested that the nodule formation was indicative of irritation due to the larvae and their metabolic products. The lesions were aggravated by occasional entry of adult worms.

Backer et al., (1929), reported that in experimentally produced ascaridiasis, there was a noticeable enlargement of the liver. He also reported that there was increased tendency to the formation of neoplasm in chickens that survived acute infections. Similar nodules have been reported in the gizzard of chickens due to Acuaria infection (Boch, 1955; Nath and Pande, 1963).

In another experiment Ackert (1930), infected 2 to 7 week old chicks with doses of 1,000 and 2,000 Ascaridia galli eggs. He observed that the invasion of the intestinal wall by the larvae caused conspicuous haemorrhages and the larvae destroyed the intestinal glands. The thymus gland was greatly reduced in size and the blood lost a significant portion of its sugar. There was marked retardation of muscular and bone development and this was accompanied by excessive deposition of urates in the ureters. He attributed these effects to injury to intestinal mucosa by the larvae, loss of blood from intestinal haemorrhage, bacterial infection, partial starvation through loss of appetite and absorption of metabolic waste of the worms.

Roberts (1936c), observed that in the early stages of worm development, there were minute haemorrhagic spots, areas of congestion and thickened walls in those portions where larvae were present. Larval attack according to him resulted in superficial erosion of the mucous membrane. The epithelial

lining of the intestine became entirely destroyed permitting leakage of blood into the lumen. The heart was enlarged and flabby, with the presence of fluid in the pericardial and peritoneal cavity. The liver was paler in colour and enlarged. The kidney was enlarged with distended ureters. There were local aggragation of histiocytes in the intestine, liver and kidney. He concluded that such a condition was the result of toxemia caused by the absorption of waste products of worms and their distribution throughout the body, through the blood stream.

Sadun (1950), reported that when young chickens were infected with 14,000 eggs of Ascaridia galli, they developed mild anaemia and increased number of leukocytes in the blood 10 days after infection. Intestinal and renal damage were observed. Twenty days after infection, the intestinal mucosa had undergone almost complete regeneration but renal condition was still present. The liver and spleen were enlarged.

Nath and Pande (1963), observed extensive damage of the intestinal mucosa in chickens naturally infected with A. galli. They attributed the damage to the tissue phase larvae. In cases where A. galli coexisted with Raillietina echinobothrida, sections of the intestine showed nodules with Ascaridia larvae encapsulated by fibrous tissues in the mesenteric wall.

In some other cases where the larvae co-existed with Amoebotaenia sphenoides and Hymenolepis contaniana, the sections revealed Ascaridia larvae surrounded by fibrous tissues in the surface of the duodenum.

Leutskaya (1963), observed that there was a reduction in vitamin A content of the liver of chickens infected with Ascaridia galli as compared with control chickens. He suggested that the cause of the reduction in vitamin A was its participation in biochemical processes related to the development of immunity. It was observed that the higher the antibody titre of infected chickens the lower the vitamin A content of the liver and vice versa. Reduction in vitamin A content of the liver due to Ascaridia infection has also been reported by Sadzikova (1975).

Hamsley (1971), reported 3 outbreaks of enteritis in turkeys 8-16 weeks of age which were associated with Ascaridia spp. The lesions in the intestine were fibrinous clots 3-5mm in diameter. He reported that when these clots were torn from the mucosa, bleeding areas were left. Kozlowicz (1971), reported chronic enteritis in pigs infected naturally with Ascaris suum. The mucosa was damaged and this condition he said facilitated perforation of the intestine and caused poor absorption of nutrients.

Ikeme (1971c), infected three groups of chickens with repeated doses of infective Ascaridia galli eggs. He observed that the albumin-globulin ratio, packed cell volume and haemoglobin concentration in the blood of infected chickens did not vary significantly from those of uninfected control chickens. But on histological examination of the intestine, he observed macroscopic lesions which were visible to the naked eyes as haemorrhagic patches intermixed with mucus and larvae. These were prominent in chickens killed three weeks after infection. In chronic cases, the intestinal walls assumed a flabby appearance when viewed in situ. Examination of the epithelium revealed extensive destruction and erosion of the glandular epithelium by migrating larvae. There was proliferation of mucus secreting cells and this resulted in adhesion of the villi. Adults when in great numbers caused obstruction. The most severely affected chickens were those on the least nutritive diet and sustaining the highest level of continuing challenge.

Litvishko and Pilipenko (1972), reported a reduction from normal, in the size of the thymus glands of 16 white Leghorn chickens naturally infected with A. galli compared with uninfected chickens. Sadzikova (1975), observed that the weight of the liver, stomach and ovary and the length of the intestine was reduced in chicken infected with A. galli as compared with the control chickens. The kidney, liver and

spleen were partially atrophied and the area of the intestine occupied by the larvae were thickened.

Dubinsky et al.(1976), reported that when chickens were experimentally infected with A. galli and placed on a cereal diet, there was a decrease in serum protein, lipid and glucose level and a reduced albumin-globulin ratio of the blood as compared with uninfected chickens. In another experiment, Dubinsky and Rybos (1977), reported that due to A. galli infection, the level of free amino acid and the activity of aspartate and alanine amino transferase (enzyme) in the serum was lowered. But they said that at low worm burden and when chickens were kept on a full diet in hygienic conditions, the chickens were able to compensate for decreases and were better able to maintain their body weight.

Resistance to infection with Ascaridia galli

A review of the observations recorded by previous investigators on the development of resistance by chickens to infection with Ascaridia galli show that resistance has been demonstrated under conditions involving age, pre-infection (Acquired resistance), diet, blood picture and breed of host.

Age resistance

Though chickens of all ages can be affected by worms, young chickens are generally more susceptible to infection and suffer more severely from heavy worm burden than older chickens. This has been recorded for cestodes and nematodes.

Chickens develop a marked resistance to infection with Ascaridia galli with increasing age. Herrick (1926), demonstrated that the growth and chances of survival of Ascaridia was inversely proportional to the age of the host. He infected chickens 5 to 240 days of age with equal number of infective eggs of Ascaridia galli and ten days after infection observed that the average length of larvae in chicks 5 days of age was 5.3mm; in chickens 60 days old, 2.6mm; 90 days old, .4mm; 103 days of age, .1mm respectively. He concluded from the results obtained that birds 240 days old were no more resistant to worms than did 103 day old chickens. The number of birds containing worms decreased from 87% among chicks five days old to 29% in chicks 147 days old and over.

Ackert (1930), reported experiments in which chickens of various ages were infected with 1,000 eggs of A. galli. Using two months old chickens, he observed that uninfected birds (controls) made greater weight gains than the infected birds and at autopsy all the infected chickens except one had worms. With chickens 10 to 12 weeks old, there was no

such difference in weight and in each group only one chicken contained worms. In another experiment 24 chickens were reared in a worm free condition till three months old and were placed with infected birds in contaminated soil. At the end of six weeks all the 24 birds were thrifty in appearance and on examination only four had worms. The maximum number of worms was eight. Ackert concluded that if chickens were kept worm free for the first three months of life, they may then be reared on infested ground with little danger of subsequent ill-effect.

Roberts (1936d), from a series of experimental studies with Ascaridia galli in chickens observed that as the age of chickens increased, fewer chickens became infected, fewer worms became established and the growth and survival rate of these worms became significantly reduced. He also observed that when chickens were maintained in worm free conditions for four months, the age resistance which was responsible for the decreasing percentage of larval survival and the increasing influence of growth inhibitory factor, became developed to such an extent that very few larvae could succeed in establishing themselves. He also observed a gradual decrease in the number of larvae during the first nine days after infection. He attributed the gradual elimination to natural resistance existing in the host.

Deo and Srivastava (1962), reported that the first sign of resistance to Ascaridia galli infection in chickens was at 90 days of age. At 121 days of age there was complete resistance to infection. Soulsby (1968), also stated that chickens over three months of age are more resistant to infection than younger chickens and that this may be associated with a marked increase, in goblet cells in the gut mucosa at about this time.

Acquired resistance

Active acquired resistance is developed by a host as a result of infection and its influence tends to become more marked under the stimulus of repeated infection.

Sadun (1948), working with Ascaridia galli, tried to find out the effect that primary infections with egg doses of different sizes would have on the number of worms that developed from a second infection. He used five groups of six day old chickens. Chickens in groups I to IV were infected with 100 (group I), 500 (group II), 1,000 (group III) and 2,000 (group IV) eggs of A. galli respectively. Chickens in group five acted as uninfected controls. Twenty two days after the initial infection, all the chickens in the five groups were infected with 3,000 eggs of A. galli. The chickens were slaughtered 25 days after the second infection. Chicks in group V had 0 to 74 worms with an average of

30.2 worms. Chickens in groups I and III contained no worms. One chicken each in group II and IV had worms and all were from the second infection. The stricking reduction in number of worms from the first and second infection compared with the control chickens made him to conclude that a secondary heavy infection given 22 days after first infection, brought a host reaction with the consequent elimination of some or all of the worms of the primary infection. In another experiment, he demonstrated that when chickens were given repeated heavy doses of eggs, they were able to eliminate practically all the worms of the test infection within eight days.

Ikeme (1970), worked with two groups of 60 chickens each. At 14 days of age he dosed one group with 10 eggs and the other 1,000 infective Ascaridia galli eggs daily for six weeks. In the low challenge group, adults were present from the third week after infection while in the high challenge group there was retarded development of larvae and this resulted in the persistence of the third stage larvae up till the tenth week. This, he said, was as a result of inhibition due to host resistance. There was occasional appearance of adult worms in the high challenge group after the tenth week and he said it was probably due to a decline in resistance or retardation of the immune mechanism of the host. He infected a third group of chickens with 1,000 eggs and observed that at the fourth week while some worms had reached maturity, some were still at third larval stage (L_3).

He concluded that inhibition of larval development depended mainly on the size of the dosage, irrespective of whether it is given as a single or continuing challenge. But the duration of inhibition was longer with repeated doses.

It has been demonstrated by several authors that extracts from larvae and adult Ascaridia and serum from previously infected chickens can confer passive immunity on chickens infected with Ascaridia galli. Rebrassier and McCrory (1931), tried to establish if extracts obtained from larvae and adult Ascaridia galli could confer passive immunity in chickens. They used three groups of chickens, which were infected at 46, 97 and 131 days of age respectively. Each group was then divided into three subgroups. Subgroup I was given 500 infective eggs only; subgroup II, 500 eggs, plus injections of Ascaridia extract, whilst subgroup III was retained as uninfected controls. In the 46 day old group, the subgroup that received eggs plus extract made a greater gain in weight than that given eggs only. Similar results were obtained with the 97 day old group. It was also observed that in younger birds, the addition of the extracts protected them against the enteritis that accompanied infection. In another experiment, Brewer and Edgar (1971) reported that the injection of whole worm and embryonated egg proteins intraperitoneally made chickens significantly immuned to a challenge of 500 eggs of Ascaridia galli. This immunity partially protected the chicks against the developing adults

and the effect of worms on weight gains. They reported that circulating antibodies began seven days after the injection of serum and lasted two months with peak production between the eighth and seventeenth days.

Sadun (1949), in a series of experiment using sera from chickens infected with Ascaridia galli, demonstrated antibody reaction in vitro with metabolic products of living larvae. Precipitates were observed at the oral and excretory pores. Eggs and adult worms incubated in immune sera showed no visible reaction. He stated that the formation of precipitates was due to antigen-antibody reaction. In another experiment, passive transfer of serum from resistant to susceptible chickens conferred considerable degree of resistance to the development and growth of worms. He therefore, concluded that this type of protection indicated that antibodies played a role in the mechanism of immunity in chickens.

Effect of diet on resistance

Vitamins and Minerals

Zimmerman et al. (1926), reviewed by Sadun et al. (1950), first demonstrated that natural resistance of chickens to infections of Ascaridia galli could be lowered by nutritional deficiencies in the diet of the host. It was shown that chickens on vitamin B deficient diet contained more worms,

which were significantly longer than worms recovered from chickens on an adequate diet. Since then, other investigators have shown that a deficiency of vitamin B complex (Ackert and Nolf 1931), vitamin B_{12} and pteroylglutamic acid (Sadun et al., 1950; Broody, 1954); pyridoxine (Broody, 1954); vitamin A (Ackert and Nolf 1931; Pande and Krishnamurty, 1959; Babos, 1962; Leutskaya, 1963 and Aruo, 1978); vitamin D (Ackert and Spindler, 1929), likewise lowered resistance to Ascaridia galli infection. Deo and Srivastava (1963), reported lowered resistance in chickens infected with Hetarakis gallinae when fed on vitamin A deficient diet. In all cases more worms were able to survive and also grew rapidly in infected chickens on deficient diet than in those chickens receiving adequate diets.

Several attempts have been made by some of the authors mentioned above to explain the causes of lowered resistance in infected chickens on deficient diet. Sadun et al. (1950), observed a degeneration of the thymus gland, anaemia and reduction in the white blood cells of chickens on pteroylglutamic acid deficient diet. They suggested that this condition was partially responsible for reduced degree of resistance through their adverse effects on antibody forming mechanism. Ackert and Nolf (1931), suggested that the effect of vitamin A deficiency probably resulted from lower rate of peristalsis of the intestine. Deo and Srivastava (1963), also made similar suggestion from results obtained in chickens infected with

Heterakis gallinae. Pande and Krishnamurty (1959), suggested the existence of a vicious circle in the relationship between vitamin A deficiency and A. galli. Hypovitaminosis A according to them favoured infection with Ascaridia galli and Ascaridia galli in its turn by damaging the intestinal mucosa set a condition of vitamin A deficiency. They further stated that if rations containing the recommended level of vitamin A as contained in 56.7 grams of greens were fed to each bird daily, all year round, it would be possible to keep birds in good health and production on grounds contaminated with eggs of Ascaridia galli. Babos (1962), reported that deficiency of vitamin A promoted invasion of the intestine by Ascaridia and that enteritis caused by the infection impaired utilization of vitamin A.

It is on record that the absence of certain minerals in the diet of chickens infected with A. galli can result in significantly fewer and shorter worms compared with control chickens on normal diet. One of such studies is that of Gaafer and Ackert (1950), with phosphorous and calcium. They attributed the fewer and smaller worms in the group of chickens fed calcium and phosphorous deficient diet to the worms requirement for these minerals, since according to them phosphorous and calcium are among the most important mineral constituents of ascarid parasites. Manganese appeared to have a little or no effect in ascaridiasis of fowl.

The chickens thrived normally without addition of manganese to their ration. The lack of effect of manganese on the development of Ascaridia galli was attributed to the lack of manganese in the ascarid body. Deo and Srivastava (1963), also reported decrease in length of worms when calcium deficient diet was fed to chickens infected with Heterakis gallinae.

Protein and Amino acid

Ackert and Beach (1933), investigating the effect of dietary supplement upon resistance of chickens to Ascaridia galli, reported that the type of amino acid could influence the course of infection. They reported that infected chickens placed on a diet deficient in proteins of animal origin were less resistant to infection by Ascaridia galli than birds fed a vegetable diet supplemented with either skimmed milk or meat. Reidel and Ackert (1950), made similar observations. They were of the opinion that the beneficial effect of skimmed milk might be due to the ease of digestibility of milk casein or to the high concentration of lysine and tryptophane present in the skimmed milk. However, Reidel (1950), could not demonstrate that high levels of lysine in the diet, increased resistance of chickens to Ascaridia galli.

In another experiment, Reidel (1954), reported that a diet inadequate in glycine retarded growth of New Hampshire

chickens but the resistance of chickens to infection of Ascaridia galli was not increased when glycine supplemented diet was fed to chickens. Reidel (1955), also showed that the quantity of leucine in the diet did not alter resistance of the chicken host to Ascaridia galli. The criteria used in all the experiments were growth and general appearance of the host, number and length of worms harboured. Chickens fed both the leucine and glycine deficient diets harboured the same number of worms with those fed diets adequate in leucine and glycine content.

Reidel and Vest (1956), reported that chickens fed rations containing supplemented phenylalanine had a total mean weight gain slightly higher than birds fed low phenylalanine ration, but the weight increase was not significant. He suggested that the phenylalanine supplemented diet did not protect the host from the effect of parasitism to the extent that significant increased growth could result.

Gibson (1963), reported that the presence of parasites in an animal host affects nutrition of the host by decreasing appetite and reducing the digestibility and utilization of food. Proteins, he said, are particularly important since adequate amount of proteins in the diet increases resistance to both infection and the effects of infection. However, he stated that well fed animals

harbouring many parasites without ill effects could be of importance in the spread of the disease.

Effect of blood loss on resistance

The effect of bleeding on growth and survival of Ascaridia galli has been investigated by Ackert and Porter (1931). They used 300 two-week old chickens. The chickens were divided into two groups of 150 each. Group A was bled weekly and the amount taken was gradually increased from 0.5ml at week 1 to 5ml at week 9. Group B was not bled at all. At six weeks of age the groups were subdivided into two. Each subgroup was given a single dose of worm eggs varying from 25 to 500 in number. Those birds given 300 or 500 eggs and bled contained larger worms three weeks after infection than those given a similar dose and not bled. The interpretation of the results was that bleeding affected growth inhibitory mechanism, lowering the resistance of the chickens to the worms.

Resistance due to breed of the host

The degree of resistance to infection of Ascaridia galli is influenced by the breed of chickens exposed to infection. Ackert et al. (1935), used a total of 1,351 chickens composed of six different breeds, namely, White Leghorns, Buff Orpingtons, White Plymouth Rocks, Barred Plymouth Rocks, Rhode Island Reds and White Minorcas. Two varieties of White

Minorcas were used. Groups of birds belonging to each of the breeds were infected with single doses of 50 eggs at the age of 12, 21, 33 and 44 days respectively. The number and average length of worms present in each group 21 days after infection were used for interpreting the results. They observed that the heavier breeds (i.e. Rhode Island Reds, White Plymouth Rocks and Barred Plymouth Rocks) were significantly more resistant to infection than the lighter breeds (White Leghorns, Buff Orpingtons, and White Minorcas). Of the two strains of White Minorcas, the heavy strain was more resistant than the lighter strains. The greater susceptibility of the lighter breeds was considered to be due to the fact that these breeds were markedly more active and nervous than the heavier breeds, and as such utilized more energy. They suggested that this utilization of energy might be unfavourable to the development of inhibitory factors.

Management systems and the predisposition of chickens to helminth infections

There are two main types of poultry management systems - the extensive system and the intensive system. The extensive system permits fullest exposure of chickens to sunlight and pasture while the intensive or total confinement system practically minimises or precludes this exposure. The semi-intensive system is intermediate between the two and provides some degree of exposure.

The most commonly practised systems in Nigeria are the free range (extensive), semi-intensive or restricted free range, deep litter, and the cage systems.

In the free range system, the chickens are referred to as the village flock in Nigeria. They are not confined but wander about in towns and villages. Their diets include green feed (growing plants), white ants and other insects, slugs and earthworms. The chickens are able to feed on waste grains in the field (Ayivor and Hellins 1956), and also have access to refuse dumps. Suitable shelters are lacking for such birds and supplemented feeding is minimal. The flock size is up to 25 chickens. Owners get eggs at minimal cost, though many of the eggs are usually destroyed before they are discovered.

The semi-intensive system involves making runs, which are areas of land enclosed by fences of wire-netting on wooden posts. The birds are free to wander about the runs during the day and at night they are kept in the poultry houses which are simple fixed buildings. The level of management is intermediate. Feed is supplemented and there is better health care. Most backyard poultry farmers in Nigeria practise this system of housing and the flock size is about 30-150 birds. It was almost the only form of intensive husbandry practised for the laying hens in Nigeria before the evolution of the deep litter and cage systems (Ogundipe, 1976).

The deep litter system was developed during the second world war in America due to shortage of labour for regular removal of litter (Feltwell, 1958; Oluyemi and Roberts 1979), so that fresh litter was added on the old whenever necessary. The buildings are usually rectangular in plan and open sided though in Northern Nigeria some are of round design. Litter materials include saw dust, rice husks, wood shavings. Hanging feeders are used and drinkers placed on raised platforms . It has been reported (Faltwell, 1958; Oluyemi and Roberts, 1979), that droppings are broken down by bacterial and microbial activities and the final result is usually dry, friable, litter with high absorptive power.

In the cage system, chickens spend the whole of their productive life on wire floored cages with food and water in front of the cages. Eggs roll down the slopping floors to the front of the cages. The droppings are collected on either individual removable trays, mechanically cleared with scrappers or belts running beneath the wire floors. The cages are made up in blocks called batteries. There might be colony cages that take up to 10-20 chickens. There are however, many modifications of the cage system.

Risk of infection in the different management systems

Apart from exposure to extreme weather conditions, the free range and the semi-intensive systems predispose chickens to infection from parasitic worms. Since they feed

as scavengers, they can either pick up the infective eggs of the worms, their intermediate or transport hosts. Ascaridia galli has a direct life cycle, so chickens may pick up infective eggs from the soil while feeding. In the process of scratching the soil for food, chickens may pick up earthworms (Soulsby, 1968), grasshoppers (Jacob et al., 1970), which act as mechanical transmitters. Other parasitic nematodes which may be picked up by range chickens include members of the following genera: Heterakis, Syngamus, Capillaria and Acuaria. Though direct transmission through embryonated eggs are possible, infective stages of Capillaria (Wehr, 1952), Syngamus (Soulsby, 1968), can be transported by earthworms. Acuaria species can be transported by beetles, and grasshoppers (Logliger, 1956). Lund (1960), reported that soils a few centimeters from the surface could act as source for Heterakis eggs and that these eggs are probably brought to the surface by earthworms and insect larvae. All the tapeworms of chickens need intermediate host which are either insects or molluscs and chickens acquire infection by ingesting the intermediate hosts in the runs.

One aspect of confinement is the attendant high stocking density. This means that there is greater contact among the birds and hence a greater risk of the spread of disease. But if well managed infection by parasitic worms can be brought to a minimal level.

In the deep litter system, the eggs of Ascaridia galli and other parasitic worms may develop on wet spots. This is usually near the entrance to the house and around water troughs. Reid (1958), reported that near optimum temperatures for development of A. galli eggs prevail in poultry houses where confinement rearing is practised. This according to him is due to body heat from the birds when hovering or resting on the litter which produces sufficient heat to induce rapid larval development within the eggs. Chickens can also acquire Ascaridia galli infection by ingesting flies which act as mechanical vectors or by drinking egg-contaminated water. In such a confinement, a large number of chickens can come down with infection if the litter is highly contaminated by infective eggs of worms.

In the cage system, layers may not have access to manure or their droppings where the infective eggs of Ascaridia galli may be present but it has been reported (Schwartz, 1975), that flies while laying their eggs in the manure may pick up the infective eggs in their feet and contaminate feed or water for hens. An outbreak of Choanotaenia infundibulum (tapeworm) infection was attributed by Abrams (1976), to heavy ingestion of large number of housefly, Musca domestica by the chickens.

MATERIALS AND METHODS

One hundred and thirty one Harco pullet chicks were purchased from Mitchel farms, Lagos and brooded on concrete floor using a wooden brooder box. Each bird was allowed a .038 sq. m. brooder space. Heat was supplied into the brooding chamber using electric bulbs while hurricane lanterns were used in cases of power failure. Light was provided for 24 hours daily until chicks were six weeks of age.

Two feeding trays each measuring 18 cm in diameter and two metal feeding troughs each measuring 50 cm long were used to supply feed until the chickens were 38 days of age. Four plastic chick drinkers were used for providing water.

A poultry rearing house measuring 13.4 sq.m. was constructed for housing the chickens after brooding (plate 1). Colony cages comprising of 15 cage cells were constructed and fixed to the walls of the house. The cages were raised 61 cm from the floor. Each cage cell measured 90 cm long by 63 cm wide by 59 cm high.

Selection of experimental chickens

The chickens were raised helminth free, on wood shavings litter until they attained the age of 38 days. The best 90 of the chicks based on body weight were selected and alloted into six 50 gram weight groups. Chicks from each

weight group were randomly selected and distributed into each of the 15 cage cells, each cage cell taking six chickens. The 15 cage cells were arranged in four rows and four columns and were divided into 3 treatment groups (I, II & III). Arrangements were such that each row contained representatives of each of the three treatment groups (fig 1). This arrangement provided as uniform an environment as possible for birds in the three treatment groups, in order to distribute the effect of environmental factors equally on all the treatment groups. Common feeders and drinkers were provided for chicks in each cage cell (plate II).

The chickens were vaccinated against New Castle disease at six weeks and at 17 weeks of age with the Lasota and Komarov strains of the New Castle disease vaccines respectively. They were also vaccinated against Gumboro disease at three weeks of age and fowl pox at six weeks of age. The New Castle disease and fowl pox vaccines were administered intramuscularly while the Gumboro vaccine was administered in drinking water. All the chickens were debeaked at the age of 223 days when pecking became a problem in some cage cells.

Drinkers were washed daily and feeders were washed weekly. The floor of the poultry house was cleaned and washed daily. Any sick chickens were removed from the cages and isolated separately and all the

parameters monitored for the chickens in the cages were also recorded for the isolated sick chickens. Chickens in all the treatment groups accidentally contracted coccidiosis at the age of 194 days and were promptly treated with Amprolium. A chicken in group II sustained injury on the head and was placed on penicillin injection for four days.

Feeding

Pfizer* poultry feed was used throughout the duration of the experiment. The amount of feed given to chickens in each cage cell was weighed daily and the left-over at the end of a fortnight was collected and weighed in order to determine the total amount of feed consumed per chicken per fortnight. Water was provided freely. From one day old up to 54 days of age, chicks were fed with chick mash. Growers mash was fed from 55 to 140 days of age and layers from 141 to 252 days of age. The percentage composition of the 3 types of feed used as indicated by the manufacturers are shown in table 1. Feed samples were taken from each of the three feed types used in the experiment and analysed for calcium, phosphorus, amino acids, fibre, fat and Nitrogen. The amino acid analysis was carried out with the Technicon Sequential Multiple amino

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*Livestock feeds Limited, Kaduna.

acid analyser (TSM-1), while the analysis for Nitrogen, fibre and fat were carried out as described in "Official Methods of analysis of the Association of Analytical Chemists, (AOAC, 1975)". The crude protein was determined by multiplying the Nitrogen content by the factor 6.25.

Preparation of infective Ascaridia galli eggs

Adult Ascaridia galli were obtained from freshly slaughtered chickens in the Sabon Gari slaughter slab, Zaria and placed in 0.9% saline solution. In the laboratory, female worms were selected and rinsed in distilled water. They were cut into two horizontally and their internal organs pressed out into a petri dish. The uteri were collected and agitated in 0.5N sodium hydroxide solution for 30 minutes in order to dissolve the sticky albuminous layer of the eggs and allow for uniform sampling (Fairbairn, 1970). The eggs liberated were concentrated using the Gallenkamp laboratory centrifuge Model CFD-400, at 1,500 r.p.m. for three minutes. The eggs were washed thrice in distilled water and further washed thrice in an embryonating fluid - 0.1N sulphuric acid (Ikeme, 1971a). Finally, equal aliquots of the fluid containing the eggs were distributed into petri dishes measuring 90 cm in diameter by 14 cm deep to a level of about 3 mm from the bottom of the dishes. The dishes were covered and incubated at temperatures between 30 - 31^{O}C for 21 days. Plate III shows a fresh Ascaridia egg before culturing.

The eggs were aerated daily by removing the cover of the petri dishes and gently rotating the dishes (Elliot, 1954). Egg cultures were replenished with embryonating fluid whenever evaporation took place. The cultures were examined periodically for embryonation and larval movement.

Infective eggs were obtained from the cultures after 21 days. These were pooled together and stored at a temperature of about 29°C. Plate IV shows infective eggs obtained from a 21 day old culture.

Infection of chickens

The embryonated eggs were washed in distilled water and suspended in 1.25 molar solution of sucrose (Hansen et al., 1954), to prevent the rapid settling of eggs and to produce a uniformly dispersed egg suspension. The amount of Ascaridia galli eggs at the time of infection was obtained by adopting the proceedure of Elliot (1954). After thorough agitation of the egg suspension, drops from the syringe were placed on glass slides and counted using a light microscope. The mean count of 6 drops was calculated. From this, the amount of suspension to be given to the experimental chickens in order to obtain the desired infection level was determined.

At 39 days of age, the chickens were given the first infective dose using the crop intubation method (Macy et al., 1971). This was carried out by gently squirting the desired volume of egg suspension into the crop through a needle 3.5cm long with a catheter 8 cm long fitted onto it.

Chickens in treatment groups I and II were given daily dose of 1,000 and 5,000 infective eggs respectively for 10 days. Group III served as uninfected control.

Data Collection

All the chickens in each cage cell were weighed fortnightly. There was an initial difficulty in obtaining a single suitable scale for all the weighings as a result of which the following three scale types with different sensitivities were used. From 39 days to 98 days of age, a Salter scale, model number 115 was used. This was calibrated in 5 grams. Hansen utility scale, model 2060 was used from 112 to 125 days of age and was calibrated in 57 grams. Gallenkamp rotating balance, model 7 was used from 126 to 252 days of age. This was calibrated in 0.5 grams.

From 24 days after initial infection, daily faecal analysis of the chickens was conducted using zinc sulphate solution with specific gravity of 1.2, to verify the establishment of infection through egg elimination. The faecal examinations were discontinued as soon as helminth eggs were detected in faecal samples of the chickens in groups I and II daily for one week.

The age at which the chickens started laying eggs in each cage cell was recorded. Eggs from each cage cell were collected daily and weighed. Bi-weekly mean egg weights were determined

for each cage cell from the weights of all the eggs collected for that particular cage. The age at which 50% level of egg production was attained was recorded for all the cage cells. The rate of egg production was expressed as hen-day and hen-housed egg production. The calculations were done from the third week (period 2) of the laying phase because during period 1 eggs were not recovered from many of the cages. The formulae for hen-day and hen-housed egg production are shown below:-

$$\text{Hen-day egg production} = \frac{\text{Total number of eggs produced per period}}{\text{number of birds alive}} \times \frac{100}{1}$$

$$\text{Hen-housed egg production} = \frac{\text{Total number of eggs produced per period}}{\text{number of birds at the beginning of the laying phase}} \times \frac{100}{1}$$

Mortality was recorded as they occurred. The intestines of dead chickens were removed, cut open and worms in them recovered and counted. In all, 4 chickens died during the experiment. One from group I and the other 3 from group II.

Worm recovery

At the termination of the experiment, all the chickens in the three treatment groups were slaughtered. The group which received a total of 10,000 infective eggs first, followed by the group which received 50,000 eggs and then the control group.

Each intestine was slit open longitudinally in a tray and the intestinal contents were removed and preserved in normal saline with 10% formalin.

The unwashed intestines were cut into small pieces and immersed in 150 ml of digesting fluid in 250 ml Beatson glass beakers. The digesting fluid was made up of 5g of pepsin powder, 7 ml of concentrated hydrochloric acid and 993 ml of distilled water. Each digest was incubated for three hours at 37°C with periodic shaking of the beaker. The digest process was stopped after 3 hours and the materials were preserved by adding a few drops of 10% formalin. Adult and macroscopic worms were removed and the rest of the intestinal content was examined in 3 ml aliquots. The search for worms was made in petri dishes using a dissecting microscope - Spencer, Model HIT (American Optical Company Instruments Division, N.Y.), at a magnification of 25. Adult and larval worms recovered were preserved in 5% formalin. The total number of worms recovered from birds in each cage cell was counted and recorded.

Statistical Analysis

All data were subjected to statistical analysis using a computer - CDC Machine, Cyber 72. The analysis of the results obtained was carried out in phases. Phase 1 covered the period between 1-16 days after administration of the first infective dose, when the chicks were still on chick mash.

Phase 2 covered the period when the birds were fed growers mash (17-100 days after initial infection) and phase 3 covered the period when the chickens were on layers mash (101-212 days after initial infection). Different parameters were measured during the 3 different phases. In phase 1, cage averages were used for analysing the changes in body weight and feed consumption. In phase 2, cage averages were used for analysing feed consumption, cumulative feed conversion and change in body weight.

Cumulative feed conversion was calculated using the following formula:

$$\text{Cumulative feed conversion} = \frac{\text{Cumulative feed consumed (g)}}{\text{Cumulative weight gain (g)}}$$

During phase 3, cage averages were used for analysing feed consumption, feed efficiency, change in body weight, Hen-day egg production, hen-housed egg production, egg weight and the age at which 50% level of egg production was attained. Feed efficiency was calculated from periods 2 to 8 using the following formula:

$$\text{Feed efficiency} = \frac{\text{Feed consumed (kg)}}{\text{Dozens of eggs produced}}$$

Feed efficiency for period 1 was not determined. Results obtained in each of the 3 phases are computed in periods, each period representing a 14 days duration.

The analysis which was common to all the parameters was the analysis of variance (ANOVA) using the Genstat Package (Genstat Reference Manual, Vols. I and II; developed in the Rothampted Experimental Station, Harpenden). The analysis of variance for all the phases employed a factorial design. The sources of variation or factors for phase 1 were Treatment and cage. The sources of variation for phases 2 and 3 were:

Period	(Per)
Group	(Gp)
Cage	(Cage)
Period x Group	(PerxGp)
Period x Cage	(PerxCage)
Group x Cage	(GpxCage)

The group means and corresponding standard errors were computed.

Further analysis beyond ANOVA for all the phases employed the least significant difference test (LSD) as described by Clarke (1962), in order to determine the specific differences between means for significant factors. All the factors were tested at the .05, .01 and .001 levels of probability.

Fig. 1: Sketch of the poultry house showing the arrangement of chicken groups in the cage cells.

GP = Group

GP 1 = Received 1,000 eggs daily for 10 days

GP 2 = Received 5,000 eggs daily for 10 days

GP 3 = Uninfected control

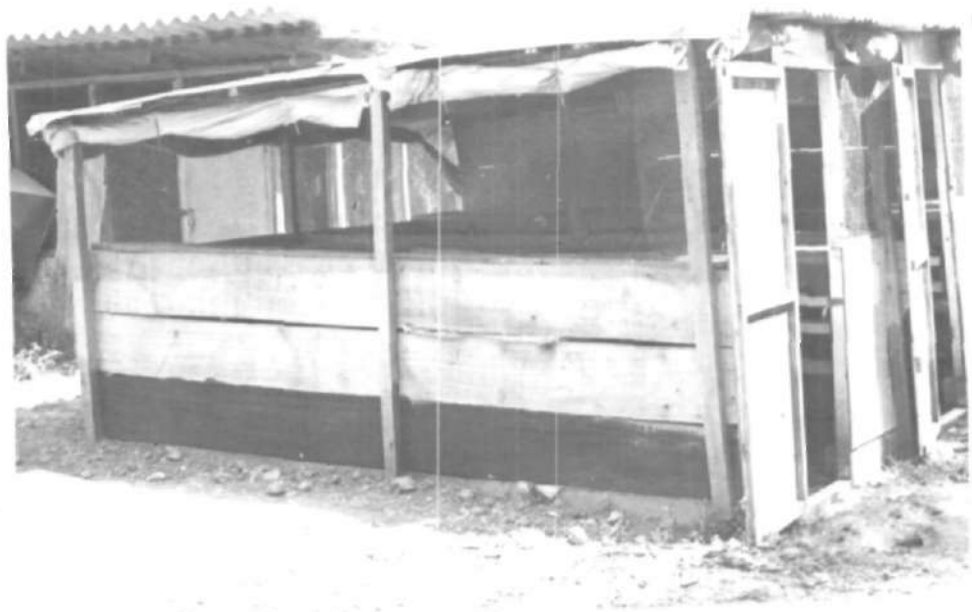
GP II	GP III	GP I	GP II
GP I	GP III	GP II	GP III
BLANK	GP I	GP III	GP II
GP III	GP I	GP II	GP I

Table 1: Percentage composition of poultry feed used in the experiment as indicated by Pfizer Livestock Feeds.

Feed Type	Protein	Fat	Fibre
Chick Mash	20.0% min	3.5% min	5.0% max
Growers Mash	16.0% min	3.5% min	7.5% max
Layers Mash	16.5% min	3.7% min	6.5% max

Plate I: The poultry rearing house used for housing the experimental chickens after brooding.

Plate II: Cage cells with common feeders and drinkers for experimental chickens.



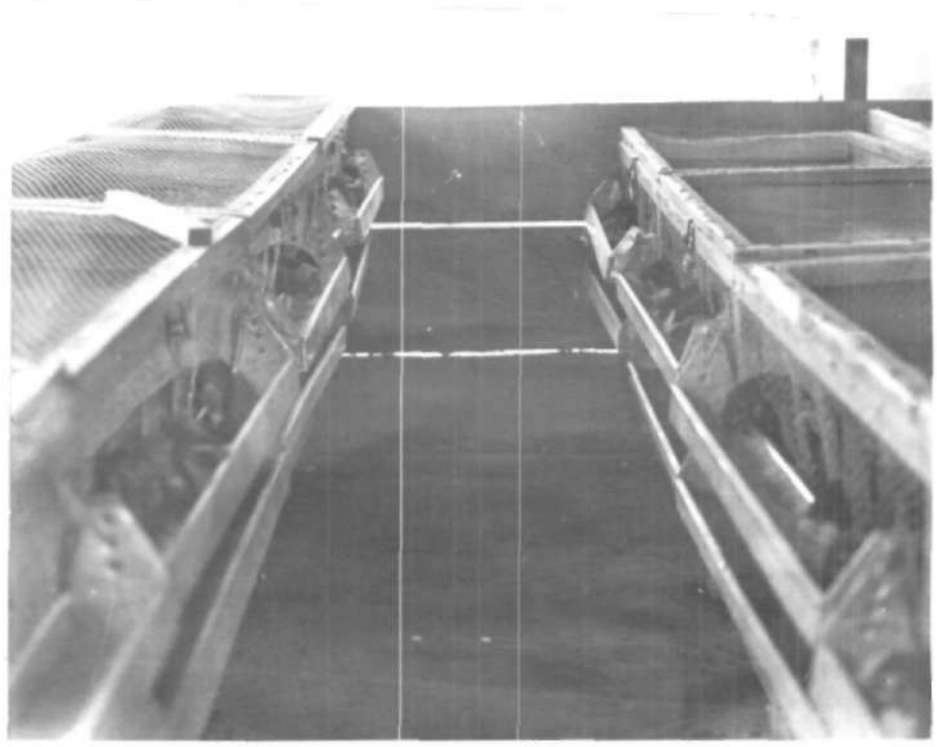


Plate III: Fresh *Ascaridia* egg

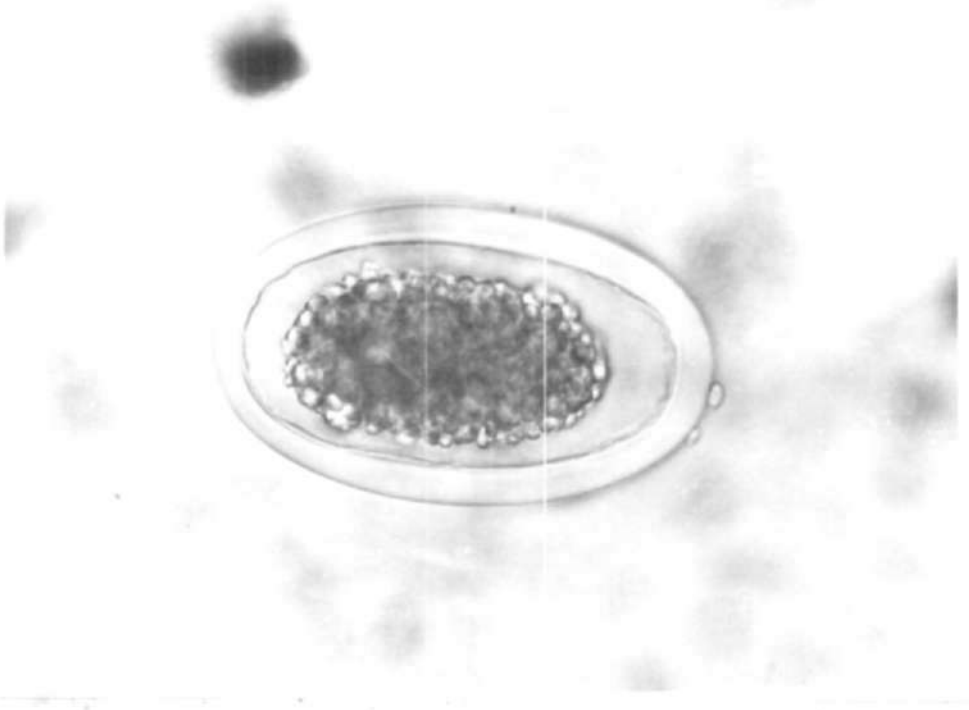
x 400

Plate IV: Infective *Ascaridia* eggs as obtained from a 21 day old culture

x 200







RESULTS

The results obtained in this experiment are presented in table 2 and figures 2 to 10. The periodic (bi-weekly) mean vulues for each treatment group of chickens for feed consumption feed conversion, feed efficiency, changes in body weight, hen-day egg production, hen-housed egg production and egg weight are presented in Appendix 1. Analysis of variance (ANOVA) tables for all the parameters listed above are presented in Appendix 2.

Prepatency

Eggs of Ascaridia galli were detected in the faeces of the group of chickens which received a total of 10,000 infective eggs (group I) 91 days after initial infection. In the group which received 50,000 infective eggs, (group II) prepatency was 85 days. Ascaridia eggs were detected in most of the cages after the chickens had started laying eggs. The control group (group III) did not shed Ascaridia eggs.

Feed Consumption

The amount of feed consumed were recorded separately for the 3 phases in the experiment.

During phase 1 calculations were based only on 14 out of the 16 days which constitute the phase. This was because accurate measurement of feed consumed during the first 2 days after initial infection was not determined. The mean weight of feed consumed per bird in groups I, II and III was 783.70 grams, 773.00 grams and 795.50 grams respectively. The differences between the 3 groups in this phase were not statistically significant.

In phase 2, which covered a duration of 12 weeks broken into 6 bi-weekly periods, the total mean weight of feed consumed per bird was 1363.40 grams, 1358.00 grams and 1317.60 grams for groups I, II and III respectively. The amount of feed consumed by chickens in groups I and II were significantly higher ($P<0.001$) than that of group III. Pattern of the periodic mean is presented in figure 2. The birds in group II consumed more feed than those in group I within the first 2 periods. Thereafter the birds in group II consumed less. The differences between groups within the individual periods were not statistically significant.

Phase 3 consisted of 16 weeks which was broken into 8 bi-weekly periods. Chickens in groups I, II and III consumed a total mean of 1829.20 grams, 1828.40 grams and 1767.10 grams respectively. The birds in groups I and II consumed significantly more feed ($P<.01$) than group III. The periodic

mean of feed consumption of the 3 groups of chickens during the phase is presented in figure 3. Differences between groups within the individual periods were not statistically significant. Chickens in group I consumed slightly more feed than those in group II within the first three periods. There was a drop in the amount of feed consumed by the three groups during periods 6 and 7.

Feed Conversion and Feed Efficiency

These parameters were not determined for phase 1. The results obtained on cumulative feed conversion, for the bi-weekly periods in phase 2 are presented in figure 4. During periods 1 and 2 the birds in group III had better feed conversion than those in groups I and II. However differences between the 3 groups over individual periods were not significant. The mean values for cumulative feed conversion during phase 2 were 6.057, 6.225 and 6.065 for the birds in groups I, II and III respectively. Feed conversion was significantly better ($P<.01$) for groups I and III than for group II.

During phase 3 differences were observed in mean feed efficiency between groups during the individual periods (figure 5) but it was only in period I that differences were significant ($P<.01$). The mean feed efficiency for egg production was 2.675, 2.494 and 2.419 for groups I, II and III respectively. The values for groups II and III were significantly

higher ($P<.05$) than for group I. Some drop in efficiency was observed during the last 2 periods.

Changes in Body Weight

In phase 1, the mean body weight gains were 181.50 grams for group 1, 175.30 grams for group II and 194.50 grams for group III. A statistically significant difference ($P<.05$) occurred between groups II and III. The difference between groups I and III was not significant.

During phase 2, chickens in group I gained 178.00 grams, those in group II gained 165.60 grams and those in group III gained 168.90 grams. Figure 6 represents the periodic weight changes during the phase. Differences between groups were not significant. Some drop in body weight was observed in period 5.

In phase 3, the mean weight gained during the 8 periods by groups I, II and III are presented in figure 7. Some differences were recorded between the groups but these differences were not significant. The mean body weight gain for group I was 32.6 grams. Group II gained 35.70 grams and group III gained 32.90 grams. Weight loss was recorded in the 3 groups during periods 3, 4 and 6.

Egg production

Chickens in group III (controls) started dropping eggs at the age of 132 days while those in groups I and II dropped their first eggs at 142 and 143 days respectively. Mean values for hed-day egg production for the laying phase were 61.69, 65.36 and 64.49 percent for groups I, II and III respectively. Differences between groups were not significant. Periodic differences between the groups were significant ($P<.01$). During periods 2 and 3, chickens in group III laid more eggs than those in groups I and II, significant differences being observed in period 2 (figure 8). But during the remaining 5 periods, the differences were not statistically significant. A drop was observed between periods 5 and 7. Results of the analysis on hen-housed egg production followed similar pattern (figure 9) as those for hen-day egg production.

Group III chickens attained 50% level of egg production at 151 days of age. Groups II and I attained that percentage at 155 and 159 days of age respectively. The difference between groups I and III was significant ($P<.05$).

Egg weight

There were slight variations in the mean egg weight of the 3 groups of chickens over the periods (figure 10) but the differences were not statistically significant. The mean egg

weight for chickens in group I was 54.18 grams, it was 53.96 grams for group II and it was 53.51 grams for those in group III.

Worm Recovery

Eighteen larvae of A. galli were recovered from the chicken which died on the forty-ninth day of initial infection in group I. Among the 3 deaths in group II, 41 larvae were recovered from the intestine of the first one which died 25 days after initial infection; 5 larvae from the second one which died of oviduct prolapse 171 days after initial infection and 13 larvae from the third one which died 214 days after initial infection.

One chicken in group I passed out 2 adult A. galli worms on day 207 after initial infection and one chicken from group II passed out 11 adult worms on day 201 after initial infection.

At the termination of the experiment, a total of 268 worms which included 4[illegible] larvae were recovered from the chickens in group I. One chicken from the group contained no worms at all and 18 chickens contained less than 10 worms. The range of worms recovered was 0 to 32 with a chicken average of 8.80 worms. In group II, a total of 538 worms were recovered. This number included 87 larvae. One chicken contained no worms and 11 contained less than 10 worms. The range of recovery was 0 to 66 worms with a chicken average of 17.90 worms. Plate V shows worms at a cut end of an intestine. There were no worms recovered from group III chickens.

Feed Analysis

Results of the feed analysis of the 3 types of mash used in the experiment are summarized in table 2. The percentages of crude protein were 20.69, 18.73 and 17.76 in the chick mash, growers mash and layers mash respectively. The fat contents were a little above 3% in chick and growers mash while it was less than 3% in layers mash. Crude fibre was highest in growers mash in which it was found to be 8.97% whereas in the chick and layers mash, the percentage of crude fibre was 6.57 and 5.52 respectively. The amino acid components were as presented in table 2.

Table 2: Chemical analysis of the 3 feed types used for the experiment

Feed Type	Crude protein %	Fat %	Fibre %	Expressed in gram percent air dry matter (gram %)															
				LYS	HIS	ARG	ASP	THR	SER	GLU	PRO	GLY	ALA	VAL	MET	ILEU	LEU	TYR	PHE	CYS
Chick Mash	20.69	3.33	6.57	0.87	0.67	1.39	1.59	0.70	0.88	3.21	1.11	1.09	1.06	0.94	0.29	0.71	1.46	0.64	0.85	0.48
Growers Mash	18.73	3.47	6.97	0.79	0.67	1.50	1.50	0.66	0.90	3.53	1.10	1.02	1.00	0.90	0.25	0.67	1.40	0.67	0.87	0.48
Layers Mash	17.76	2.29	5.52	0.79	0.67	1.68	1.63	0.64	0.92	3.43	1.08	1.01	0.99	0.83	0.26	0.65	1.41	0.68	0.88	0.47

Fig. 2: Mean feed consumption of experimental chickens per fortnight during the growing phase.

Δ ----------Δ = Group I 10,000 eggs

o ---------- o = Group II 50,000 eggs

▱ — .—. —..— ▱ = Group III control

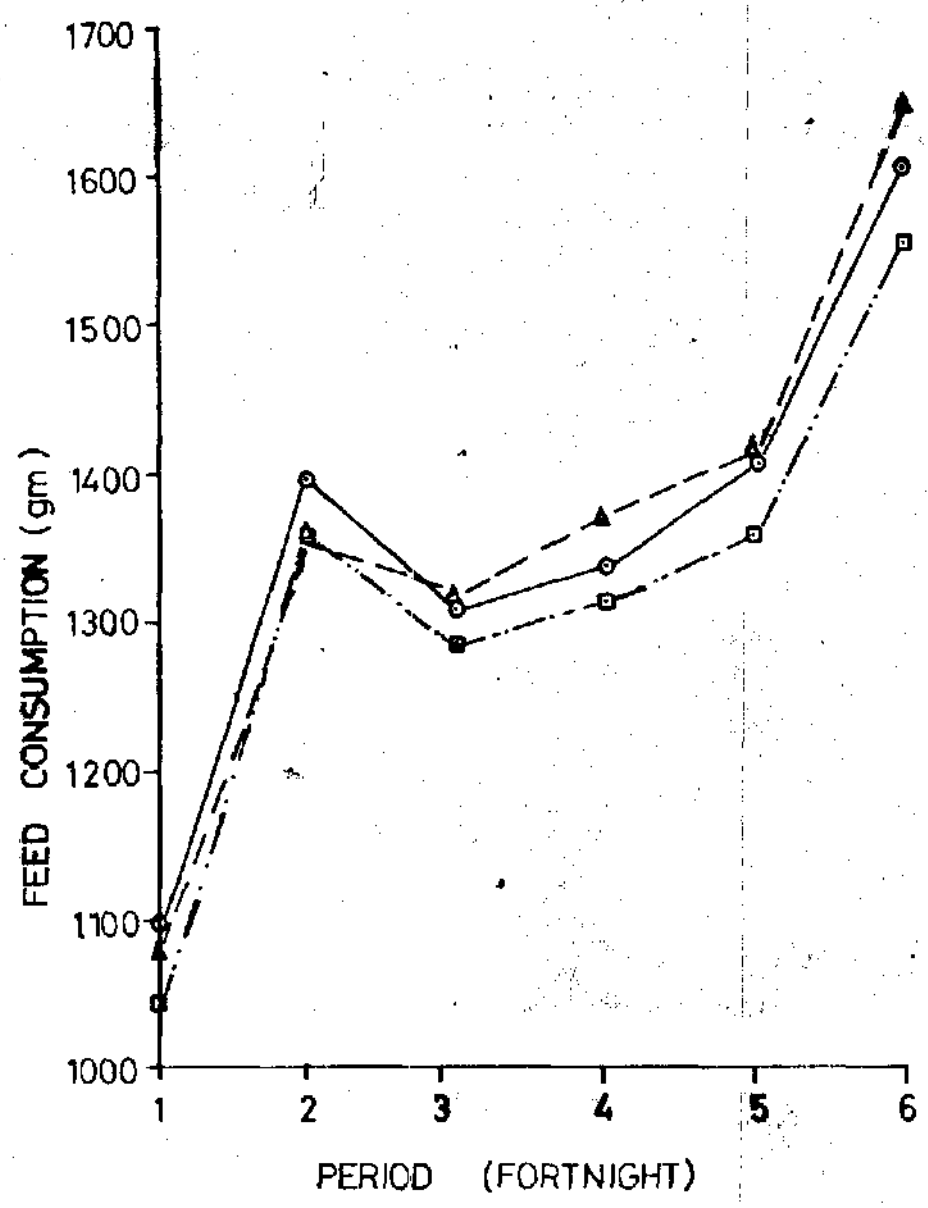

1700
1600
1500
1400
1300
1200
1100
1000
FEED CONSUMPTION (gm)
1
2
3
4
5
6
PERIOD (FORTNIGHT)

Fig. 3: Mean feed consumption of experimental chickens per fortnight during the laying phase.

Δ ---------- Δ = Group I 10,000 eggs

o ———————— o = Group II 50,000 eggs

/ / — · — · — · — / / = Group III control

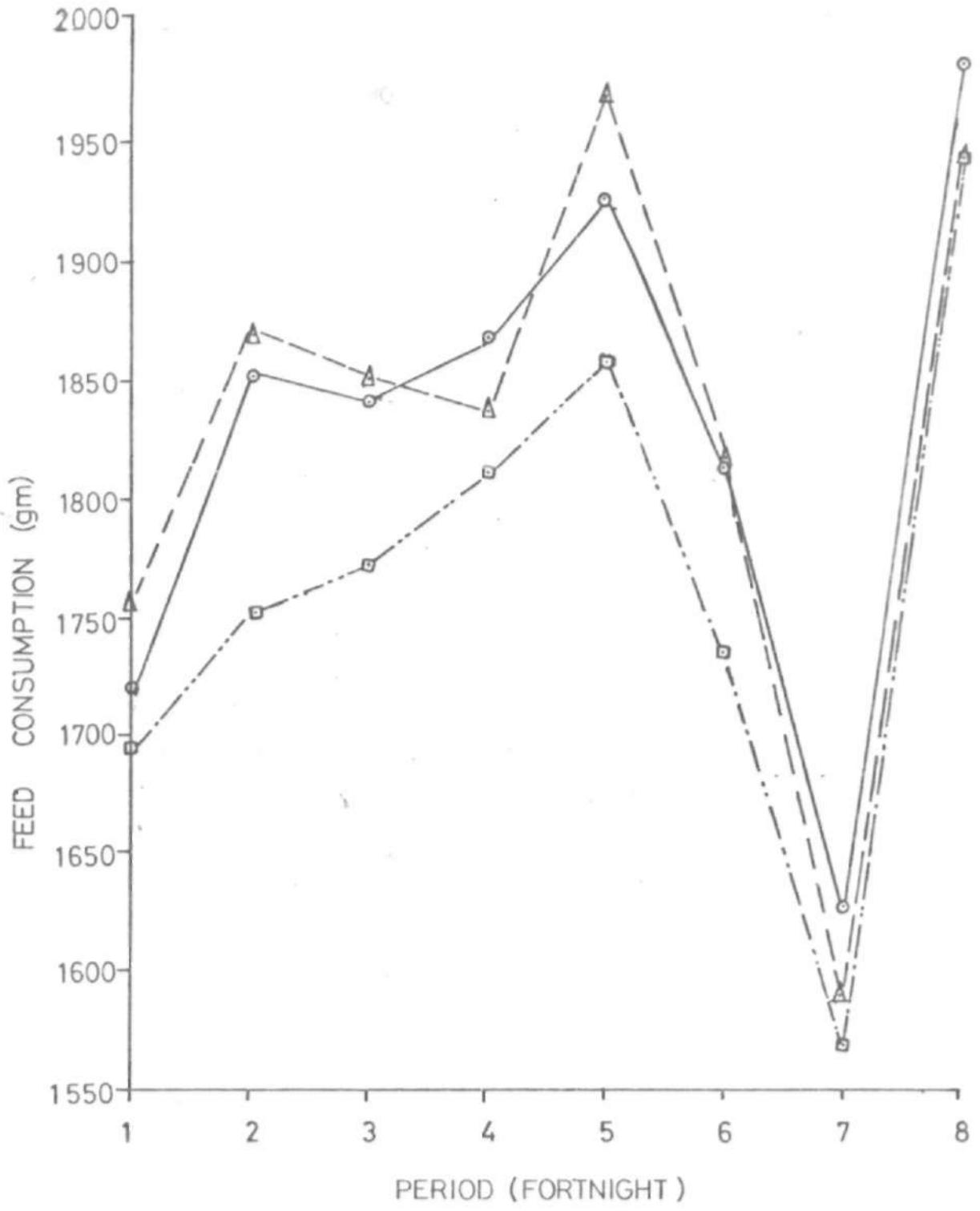

2000
1950
1900
1850
1800
1750
1700
1650
1600
1550
FEED CONSUMPTION (gm)
1
2
3
4
5
6
7
8
PERIOD (FORTNIGHT)

Fig. 4: Cumulative feed conversion for experimental chickens per fortnight during the growing phase.

Δ ---------- Δ = Group I 10,000 eggs

o ———————— o = Group II 50,000 eggs

/_/ — · · — · ·—/_/ = Group III control

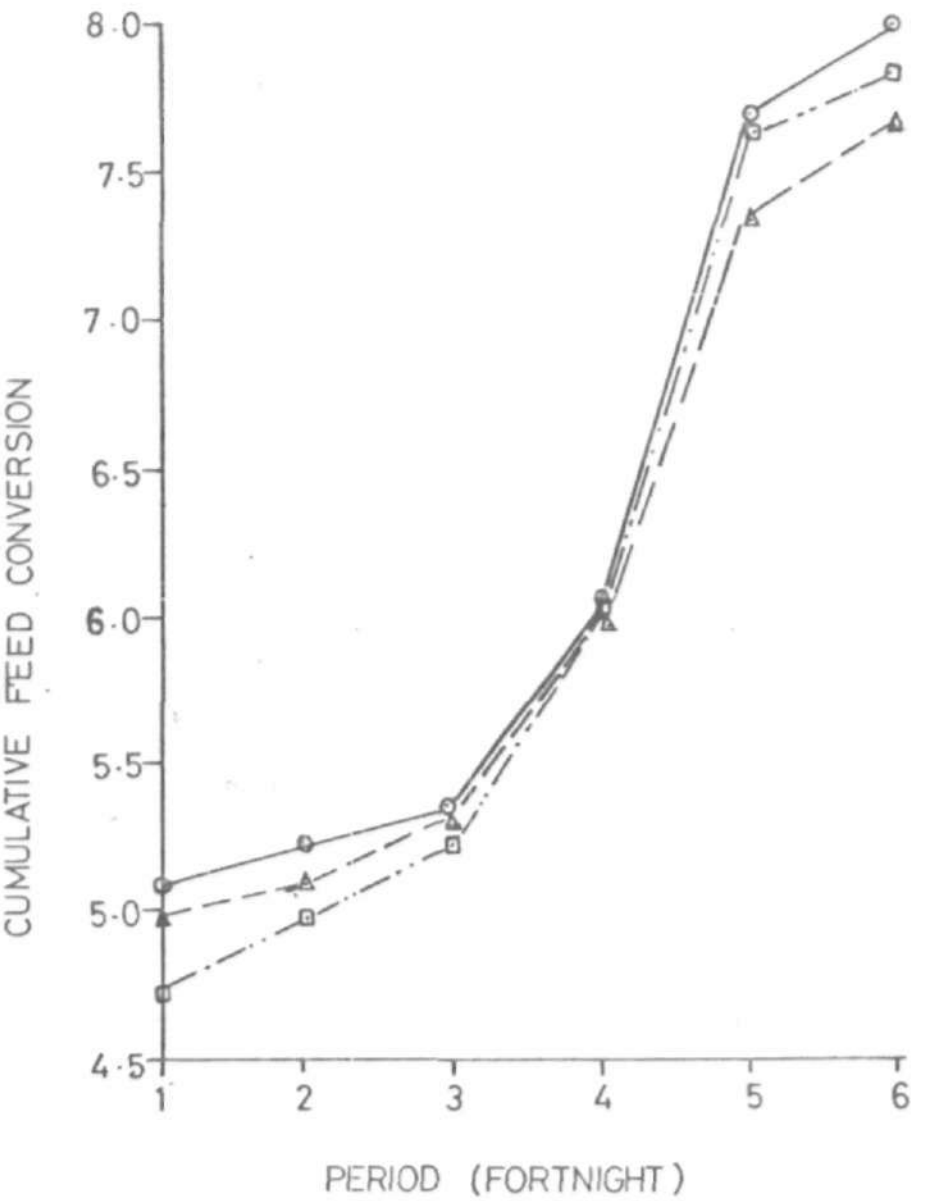

CUMULATIVE FEED CONVERSION
8·0
7·5
7·0
6·5
6·0
5·5
5·0
4·5
1
2
3
4
5
6
PERIOD (FORTNIGHT)

Fig. 5: Feed efficiency of experimental chickens per fortnight during the laying phase.

Δ ---------- Δ = Group I 10,000 eggs

o ———————— o = Group II 50,000 eggs

□ —·—·—·— □ = Group III control

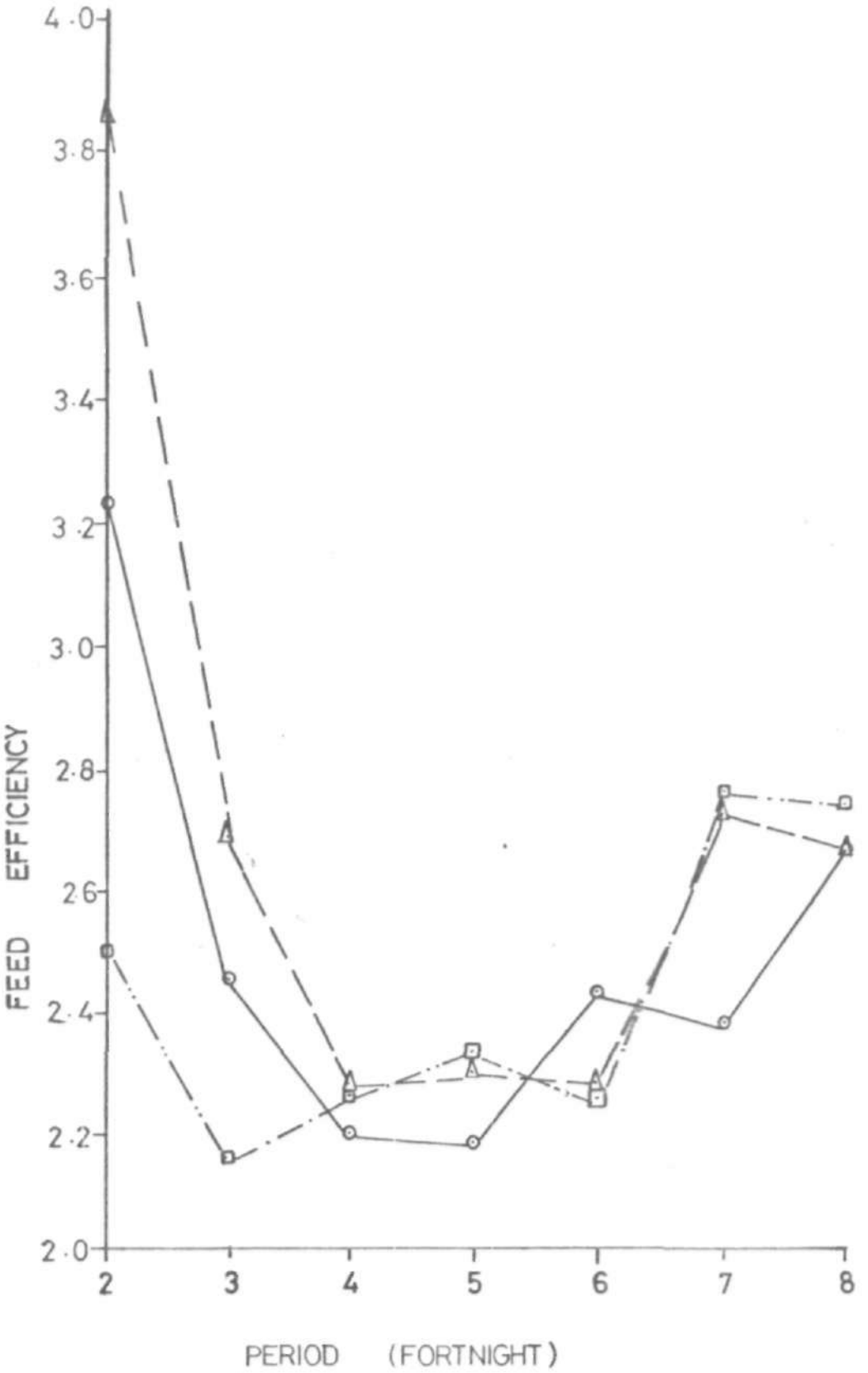
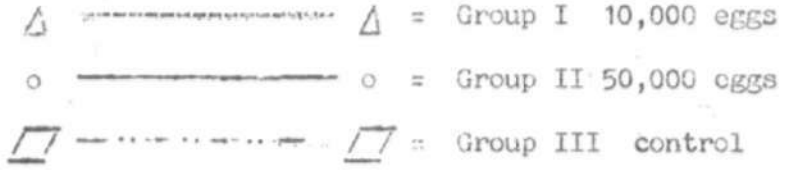

4·0
3·8
3·6
3·4
3·2
3·0
2·8
2·6
2·4
2·2
2·0
FEED EFFICIENCY
2
3
4
5
6
7
8
PERIOD (FORTNIGHT)

Fig. 6: Changes in body weight of experimental chickens per fortnight during the growing phase.



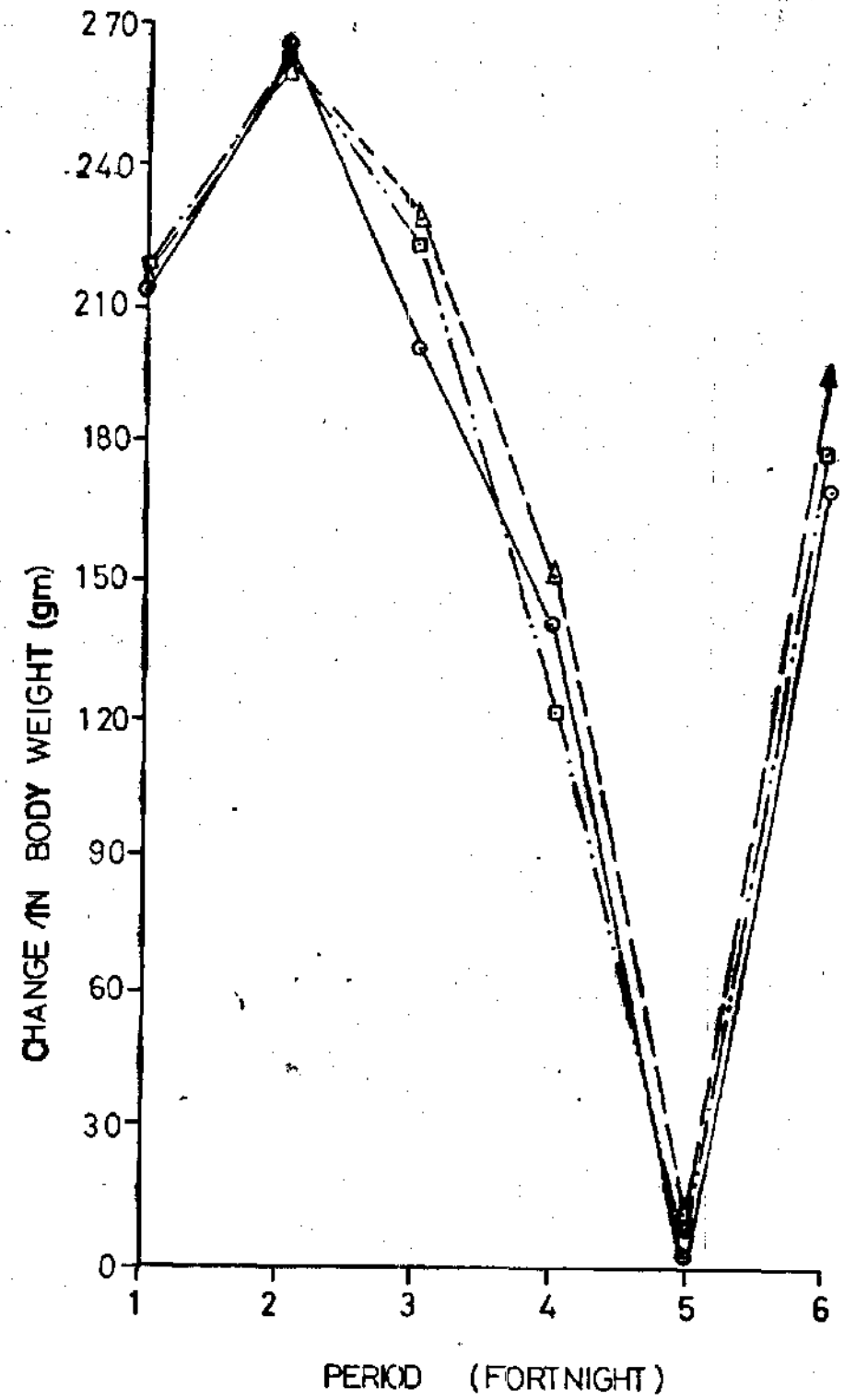
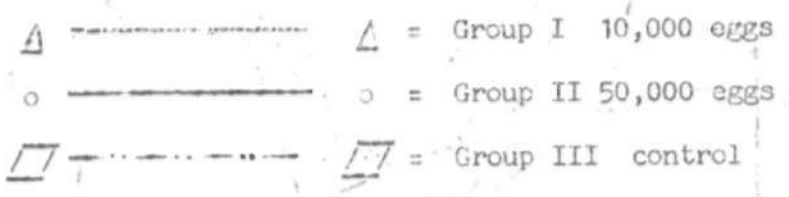

270
240
210
180
150
120
90
60
30
0
CHANGE IN BODY WEIGHT (gm)
1
2
3
4
5
6
PERIOD (FORTNIGHT)

Fig. 7: Changes in body weight of experimental chicken per fortnight during the laying phase.



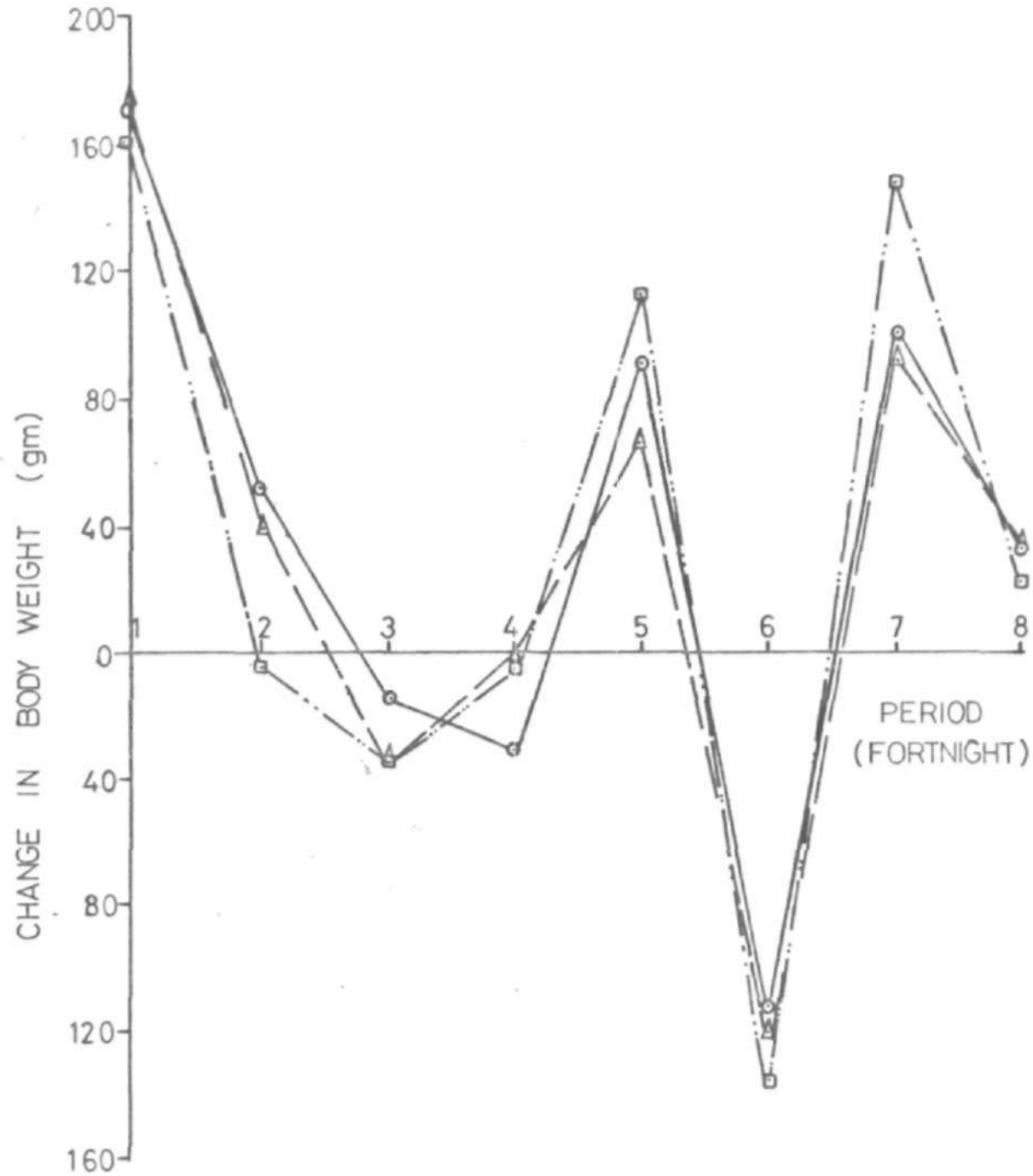
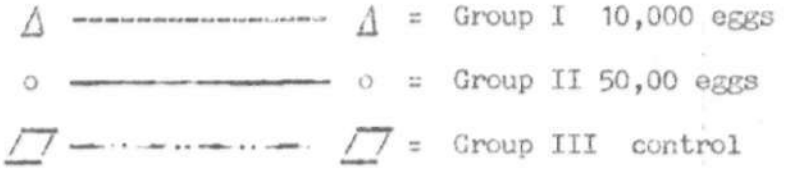

200
160
120
80
40
0
40
80
120
160
CHANGE IN BODY WEIGHT (gm)
1
2
3
4
5
6
7
8
PERIOD
(FORTNIGHT)

Fig. 8: Hen-day egg production of experimental chickens per fortnight during the laying phase.



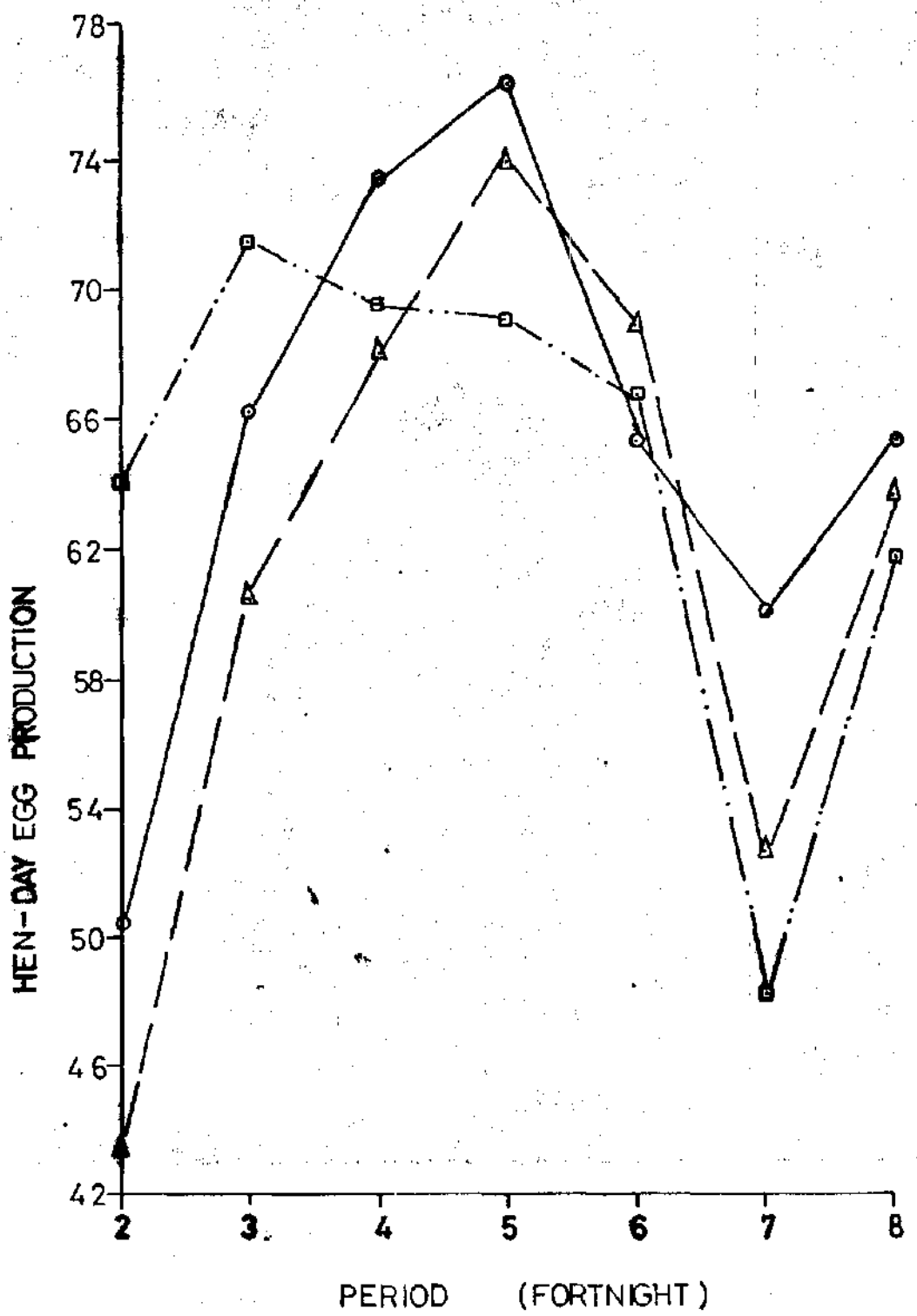

HEN-DAY EGG PRODUCTION
78
74
70
66
62
58
54
50
46
42
2
3
4
5
6
7
8
PERIOD (FORTNIGHT)

Fig. 9: Hen-housed egg production of experimental chickens per fortnight during the laying phase.

Δ ---------- Δ = Group I 10,000 eggs

o ———————— o = Group II 50,000 eggs

▱ — · — · — · — ▱ = Group III control

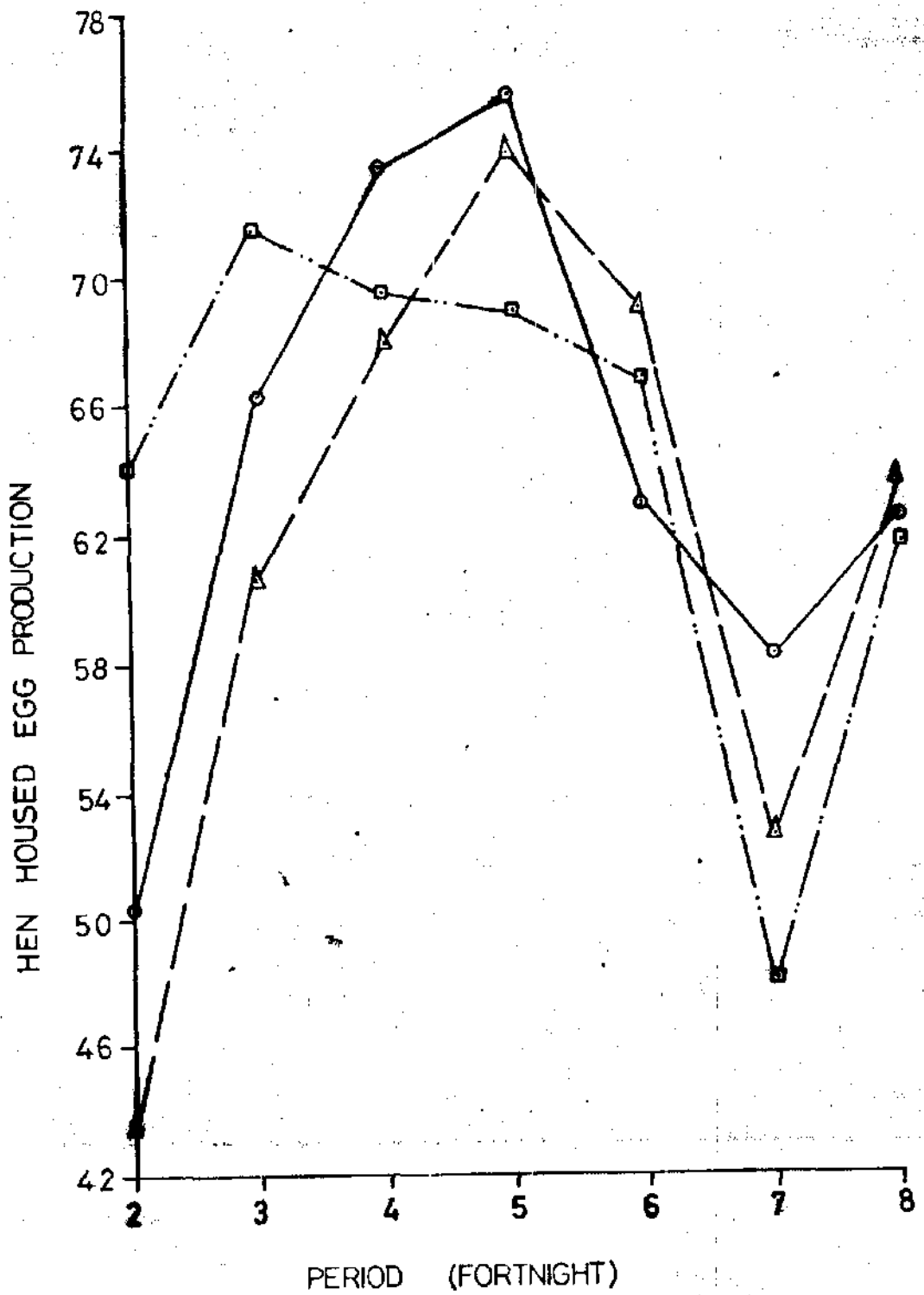

78
74
70
66
62
58
54
50
46
42
HEN HOUSED EGG PRODUCTION
2
3
4
5
6
7
8
PERIOD (FORTNIGHT)

Fig. 10: Mean egg weight of experimental chickens per fortnight during the laying phase.

Δ ---------- Δ = Group I 10,000 eggs

o ———————— o = Group II 50,000 eggs

□ —·—·—·— □ = Group III control

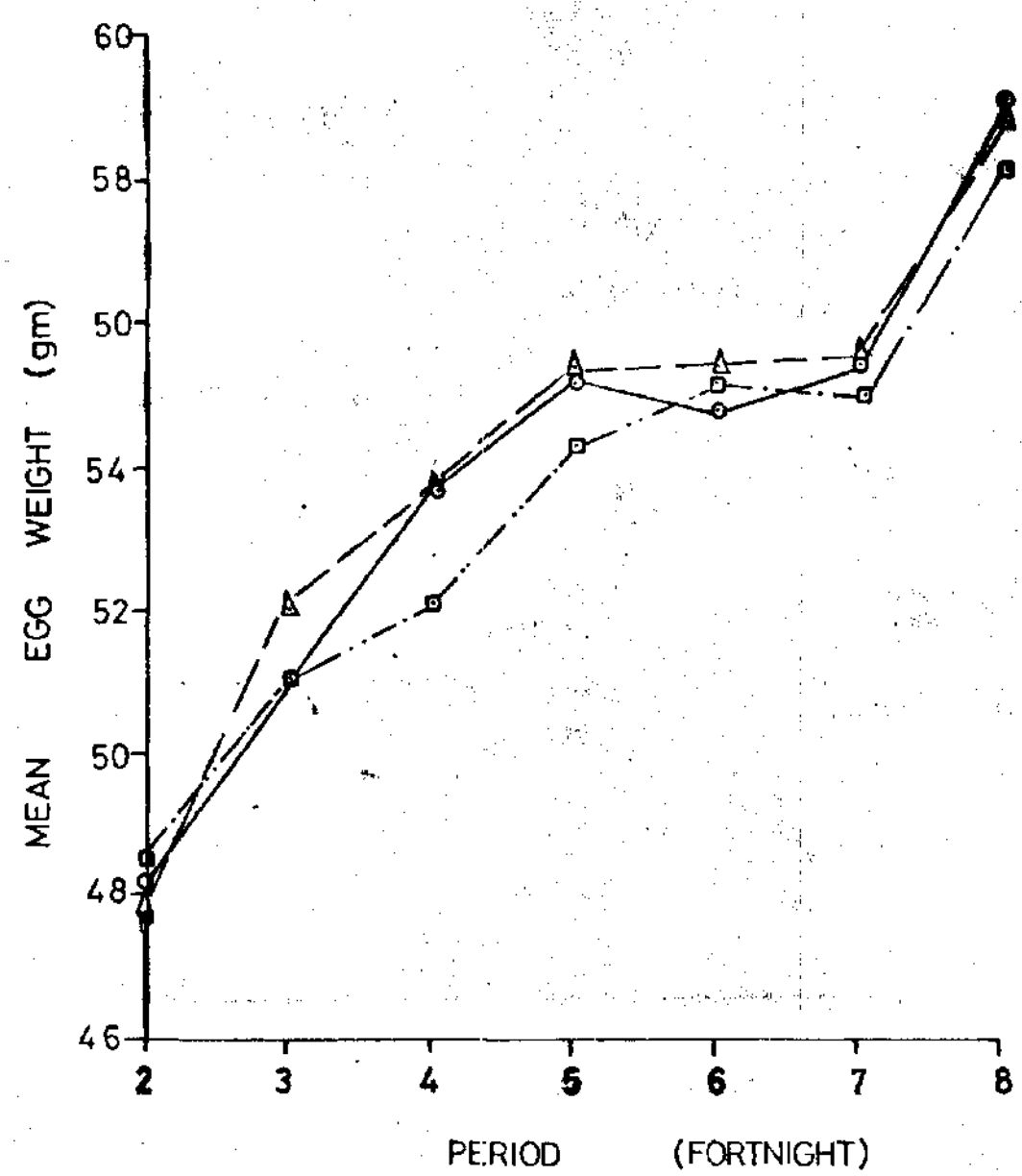

MEAN EGG WEIGHT (gm)
60
58
50
54
52
50
48
46
2
3
4
5
6
7
8
PERIOD (FORTNIGHT)

Plate V: Worms at a cut end of an intestine from one of the infected chickens.


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DISCUSSION

Some differences exist in the results obtained from the present investigation and results of previous investigators on feed consumption, feed efficiency, changes in body weight, mortality and egg production of chickens and prepatency of worms. Under the conditions of this study one or more of several reasons may be responsible for these differences.

The breed of chickens used by most of the previous investigators were Leghorn chickens which according to Ackert et al. (1935) are about the most susceptible breeds of chickens to Ascaridia galli infection, whereas Harco pullets which are a heavier breed (Oluyemi and Roberts, 1979) were used for the present study. This probably affected the results obtained from the present study.

Another possible reason may be the age of the chickens used. Young chickens are generally more susceptible to infection than older ones. Apart from a few cases, chickens used for the present study were of more advanced age (39 days old) than those used by previous investigators. It appears however that the breed of chickens is a more probable reason for the results obtained. Ackert (1930), observed that the weight of chickens infected at 7 weeks of age were adversely affected. Matta (1981), reported a drop in egg production

after infecting 12 week-old birds with Ascaridia galli eggs. Ackert and Matta used white Leghorns in their experiment.

The size of the infective dose may be another possible reason. It is on record that the proportion of large doses and repeated doses of eggs which get established are often smaller than that of small doses. The total number of eggs used by some of the previous investigators were smaller than the number used in the present study and were mostly single doses. Ikeme (1971b,c) using repeated doses observed that chickens were adversely affected, but he utilized Leghorn chicks which are lighter breeds and the chickens were only 17 days old at the time of infection.

Another probable reason for the results obtained may be the system of management. With the cage system of housing, there was practically no contact between the chickens and their droppings. The chances of reinfection with A. galli eggs or possible infection with eggs of other helminths were eliminated. The floor of the house was cleaned and disinfected daily to prevent development of eggs within the poultry house. The results would probably have been different if the chickens were raised on deep litter. The presence of litter and faecal droppings with their moisture content usually provide suitable environment for rapid development of A. galli eggs and thereby increasing the chances of

reinfection and exposes the chickens to other parasitic helminths which in conjunction with A. galli would have produced more marked effects on the parameters monitored in the present study.

The outbreak of coccidiosis among the chickens might have been due to contamination of their feed or water by oocysts. The oocysts were detected at period 4 of the laying phase. It is not very clear whether the infection did affect production, but a drop in body weight was observed between periods 3 and 4 of the laying phase in all the three treatment groups.

It is evident from the results obtained in the present study that there was a delay in the development to adult stage of established larvae. This was seen in the prolonged prepatent periods. Similar observations were made by Ikeme (1970, 1971a) in chickens infected daily for six weeks with 1,000 eggs of Ascaridia galli. He attributed this to inhibition due to host resistance, stating that emergence of adult worms 10 weeks after the last dose must have been due to a decline in resistance or a relaxation of the immune mechanism of the host. This probably would explain why in the present study, worm eggs were detected from most of the cage cells only after the chickens had started laying eggs. Since egg laying brings stress on the host. In the present study, the repeated doses and adequate nutrition might have contributed to the prolonged prepatency of the worms.

Apart from prolonged prepatent periods, the size of worms recovered from chickens that died before the termination of the experiment also indicated that there was delayed development of the larvae. Forty nine days after initial infection, the chicken which died from group I contained 18 larval worms, while the two from group II which died at 171 and 214 days had 5 and 13 larvae respectively. Roberts (1936a), obtained prepatency of 27 days for Ascaridia while Kerr (1955) obtained prepatency of 30 to 33 days. Based on these findings, the recovery of larvae from chickens in this experiment at 41, 171 and 214 days after initial infection must have been due to delayed development especially as the chickens were not exposed to any other source of infection.

Within 16 days of initial infection control chickens consumed more feed than those in groups I and II though differences were not significant. Ikeme (1971b) observed marked inappetance in chickens infected with A. galli between 2 to 5 weeks of infection. But he used 17 day old Leghorn chickens whereas in the present study 39 day old Harco pullets were used. The age and breed of chickens might have modified the effects of the worms on feed consumption. Group II chickens which had a heavier worm load consumed less feed than those in group I.

Throughout the growing and laying phases, it was observed that mean feed intake of birds in groups I and II were significantly higher than those of group III. It is rather difficult to explain why uninfected control chickens were consuming less feed. Ackert (1930), reported that the appetite of chickens that survived after the seventeenth day of infection became voracious. This might serve to explain the discrepancy in feed intake. It was also observed that during the growing phase chickens in group II with a heavier worm burden consumed less feed than those in group I.

During the growing phase, feed conversion of groups I and III were better than that of group II. This agrees with the results of Dimitrov (1971) who observed poorer feed conversion by infected chickens. Feed conversion was poorer in group II which sustained the heavier worm load than group I which had a lighter worm load. Similar observations were made by Dimitrov (1971).

Feed efficiency was better for control chickens within the first two period of the laying phase than those of groups I and II. Poorer feed efficiency has been reported by Costa (1970) in chickens infected with A. galli, compared with control chickens. Throughout the laying phase feed efficiency was better for group II than I. It is difficult to explain why this should be so.

Control chickens gained more weight than those in groups I and II within 16 days of infection. This conforms with the results of Herrick (1924) who reported that infected birds lost weight within 2 weeks of infection. The significant differences between group II and III must have been due to the heavy worm load. Chickens in group II with a heavier worm load, gained less weight than those in group I. Ackert and Herrick (1928) using single doses observed that this is the period when most of the larvae are in the intestinal mucosa causing the disturbances observed.

During the growing and laying phases no significant differences were observed in body weight gained by the 3 groups. This might be due to the fact that infected chickens consumed more food than control chickens during the two phases and were able to tolerate infection to such an extent that their performances were not affected. It is also possible that chickens at this time were recovering from the effect of the worms after the re-entry of worms into the intestinal lumen. The drop in weight gained in the 3 groups during period 5 of the growing phase was probably due to the change in the scale previously used for weighing to another one with more sensitive calibration.

The drop in weight gain at periods 6 of the laying phase coincided with the time when chickens were debeaked due to pecking in some cage cells.

Chickens in group III started laying eggs much earlier than the infected ones in groups I and II. Similar observations have been made by Sadzikova (1975).

The rate of egg production was only significantly higher for group III than groups I and II at the first period. This might be due to the fact that groups I and II chickens started laying eggs late or due to the effect of maturing worms at that period.

The drop in production between periods 4 and 6 was probably due to the effect of debeaking around that time..

Control chickens attained 50% level of egg production earlier than infected chickens. The significant delay between groups I and III might have been due to the effect of the worms.

During the laying phase differences in mean egg weight of the 3 groups were not significant. This is in contrast to the results obtained by Sadzikova (1975) who reported that eggs laid by infected chickens weighed less compared with those from uninfected chickens. Some of the factors which determine egg size include the breed, age of chickens, composition of the feed and total amount of feed consumed. Since the chickens used in the present study were of the same age, breed and were fed on the same diet, it is probable that

the insignificant difference in egg weight was due to the fact that infected chickens consumed more feed at this phase than control chickens and were able to compensate for the effects of the worms.

Mortality due to infection was very low. This is in contrast to the results obtained by Ackert and Herrick (1928) and Hamsley (1971) who observed high mortality in infected chickens. Out of 4 deaths only two could be attributed to ascaridiasis. These were the chickens that died 25 and 49 days after initial infection. Haemorrhagic spots were observed along the intestinal wall. Similar observations were made by Ikeme (1971c). Guberlet (1924) attributed early death to intestinal irritation and toxemia.

The number of worms recovered from chickens at necropsy was very low. This might be due to elimination as had been observed by Roberts (1936b). He discovered a gradual decrease in the number of worms during the first 9 days of infection and suggested that elimination of larvae was due to natural resistance that existed in the host.

Elimination of adult worms by infected chickens is indicative of self-cure due to host resistance. Self-cure has been described by Macchioni et al. (1978) when guinea fowls were subjected to heavy doses of Ascaridia numidae.

The presence of larvae in chickens up to the time of slaughter might be due to delayed development. Ikeme (1971a) infected 17 day old chickens with 1,000 eggs of A. galli daily for 6 weeks. Larvae were still recovered 13 weeks after the last dose. He attributed this to a significant delay in the moult from the third (L_3) to the fourth (L_4) stage larvae.

The composition of the feed utilized in the present study might have contributed to the results obtained on prepatency of worms and mortality of chickens. Natural resistance of chickens to A. galli infection could be lowered by nutritional deficiencies especially proteins and vitamins. Ikeme (1971a) reported that prepatent period was more prolonged in the group that received 15% protein diet than those that received 10 or 12.5% protein diet. In another experiment, Ikeme (1971b) reported highest mortality in the group that received the poorest diet. The high level of protein in the feed utilized in the present study might have contributed to the prolonged prepatent period and low mortality observed. The proportions of protein in the diet fed to chickens in the present study were within the range recommended by the National Research Council (NRC 1971). The NRC figures were 20%, 16% and 15% protein for chick, growers and layers mash, while the percentage of proteins in the feed used for the present study were 20.69%, 18.73% and 17.76% for chick, growers and layers mash respectively.

Gibson (1963), is of the opinion that adequate amount of proteins in the diet increased resistance to both infection and the effects of infection.

CONCLUSION AND RECOMMENDATIONS

Given the conditions in which the present study was carried out, the following conclusions can be drawn based on the results:

- Feed intake of chickens infected with *A. galli* is depressed within 16 days of infection, although the difference between infected and control chickens is not statistically significant. The infected chickens thereafter consumed more feed than uninfected control chickens.
- *Ascaridia* infection lowers feed conversion of heavily infected chickens during the growing phase.
- Feed efficiency of infected chickens is significantly lower than that of the controls between 3 to 4 weeks in lay.
- Uninfected chickens gained significantly more weight than infected chickens within 16 days of infection. Thereafter infected chickens recover and no differences in body weight gain may be observed between control and infected chickens.
- Age at onset of egg laying is more prolonged in infected than uninfected chickens.
- *Ascaridia* infection lowers significantly the rate of egg production of infected chickens between 3 to 4 weeks in lay.

- There is delay in the attainment of 50% level of egg production in infected flocks.
- The weight of eggs laid by infected chickens is comparable to those produced by uninfected chickens.
- Mortality is low even when birds are heavily infected.
- Worm recovery at necropsy in Ascaridia infection is low.

Ascaridia infection can have adverse effects on the body weight gain and rate of egg production of chickens. Sanitary measures should be inaugurated to prevent infections as far as possible rather than spend money on worming drugs.

With the deep litter system, it is advisable to feed chickens from raised or hanging hoppers to prevent contamination of feed with droppings. Water troughs should also be raised from the floor to prevent contamination and water spillage must be avoided so that the surrounding soil will not constantly be in a state of dampness. Special attention should be given to drinkers and feeders in the cage system.

It is necessary to remove the droppings at regular intervals of 5 days in the cage system. This is not practicable with the deep litter system. But daily turning of litter with rake can be of help in maintaining dry litter.

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APPENDIX I

The periodic (bi-weekly) mean values and standard errors for feed consumption, cumulative feed conversion, feed efficiency, changes in body weight, hen-day egg production, hen-housed egg production and egg weight per group of the experimental chickens.

Table 3: Mean feed in grams, consumed by each chicken per 14 - day period during the growing phase (phase 2)

PERIOD	GROUP I	GROUP II	GROUP III
1	1075.9 ± 7.34	1093.1 ± 16.72	1041.3 ± 29.99
2	1353.5 ± 19.14	1398.8 ± 41.4	1359.7 ± 15.35
3	1317.8 ± 26.19	1309.9 ± 32.59	1202.2 ± 8.44
4	1371.4 ± 43.42	1338.2 ± 7.07	1313.2 ± 10.76
5	1412.5 ± 49.20	1405.8 ± 34.33	1354.0 ± 10.60
6	1649.3 ± 45.51	1602.2 ± 39.12	1555.4 ± 29.38
$\bar{x}$	1363.4 ± 75.07	1358.0 ± 67.45	1317.6 ± 66.69

$\bar{x}$ = Mean of means.

Table 4: Mean feed in grams consumed by each chicken per 14 - day period during the laying phase (phase 3)

PERIOD	GROUP I	GROUP II	GROUP III
1	1756.2 ± 38.08	1717.1 ± 27.74	1693.8 ± 31.16
2	1872.0 ± 19.78	1853.5 ± 27.20	1751.8 ± 53.46
3	1852.1 ± 22.05	1841.4 ± 29.95	1772.7 ± 45.97
4	1837.1 ± 54.81	1867.7 ± 45.88	1810.6 ± 33.19
5	1968.8 ± 65.28	1926.4 ± 39.77	1859.6 ± 40.20
6	1817.1 ± 38.35	1814.4 ± 20.92	1734.6 ± 32.45
7.	1587.6 ± 53.65	1625.1 ± 49.98	1568.2 ± 60.79
8	1942.5 ± 43.82	1981.3 ± 52.71	1945.8 ± 73.49
$\bar{x}$	1829.2 ± 41.96	1328.4 ± 39.92	1767.1 ± 39.78

$\bar{x}$ = Mean of means.

Table 5: Cumulutive feed conversion of each chicken per 14 - day period during the growing phase (phase 2)

PERIOD	GROUP I	GROUP II	GROUP III
1	4.990 ± .06	5.092 ± .06	4.738 ± .16
2	5.084 ± .06	5.212 ± .13	4.976 ± .08
3	5.302 ± .06	5.328 ± .12	5.220 ± .05
4	5.964 ± .11	6.048 ± .08	6.034 ± .04
5	7.344 ± .13	7.688 ± .07	7.612 ± .08
6	7.656 ± .14	7.980 ± .06	7.810 ± .10
$\bar{x}$	6.057 ± .48	6.225 ± .53	6.065 ± .55

$\bar{x}$ = mean of means

Table 6: Mean feed efficiency of each chicken per 14 - day period during the laying phase (phase 3)

PERIOD	GROUP I	GROUP II	GROUP III
1	3.848 ± .39	3.222 ± .44	2.490 ± .13
2	2.682 ± .22	2.436 ± .19	2.148 ± .14
3	2.270 ± .07	2.188 ± .05	2.252 ± .12
4	2.288 ± .06	2.172 ± .05	2.320 ± .12
5	2.276 ± .13	2.420 ± .17	2.242 ± .12
6	2.714 ± .34	2.362 ± .19	2.764 ± .22
7	2.648 ± .12	2.658 ± .23	2.718 ± .10
$\bar{x}$	2.675 ± .21	2.494 ± .14	2.419 ± .09

$\bar{x}$ = Mean of means.

Table 7: Changes in grams, in body weight of each chicken per 14 - day period during the growing phase (phase 2)

PERIOD	GROUP I	GROUP II	GROUP III
1	215.8 ± 2.33	214.8 ± 3.79	219.8 ± 3.12
2	262.2 ± 2.03	265.6 ± 5.58	263.0 ± 4.63
3	229.0 ± 3.24	200.1 ± 24.95	223.1 ± 5.55
4	152.3 ± 17.24	141.4 ± 4.48	122.4 ± 4.37
5	11.5 ± 20.17	1.1 ± 10.96	6.3 ± 5.30
6	197.1 ± 12.41	170.5 ± 7.72	179.0 ± 16.55
$\bar{x}$	178.0 ± 36.45	165.6 ± 37.09	168.9 ± 37.9

$\bar{x}$ = Mean of means.

Table 8: Changes in grams in body weight of each chicken per 14-day period during the laying phase (phase 3)

PERIOD	GROUP I	GROUP II	GROUP III
1	175.3 ± 32.38	171.0 ± 7.65	161.7 ± 36.56
2	41.8 ± 37.75	52.1 ± 9.5	-4.3 ± 35.88
3	-35.0 ± 22.29	-15.3 ± 20.59	-34.1 ± 29.66
4	-0.4 ± 29.28	-32.4 ± 21.37	-6.7 ± 10.92
5	67.8 ± 10.26	91.3 ± 14.1	112.3 ± 14.10
6	-120.8 ± 9.64	-114.5 ± 22.91	-137 ± 11.93
7	93.7 ± 12.07	100.1 ± 11.02	149.4 ± 20.25
8	35.5 ± 8.44	33.0 ± 15.76	22.2 ± 27.67
$\bar{\bar{x}}$	32.6 ± 30.27	35.7 ± 31.52	32.9 ± 36.06

$\bar{\bar{x}}$ = Mean of means.

Table 9: Mean hen-day egg production of each chicken per 14-day period during the laying phase (phase 3)

PERIOD	GROUP I	GROUP II	GROUP III
1	43.48 ± 4.39	50.38 ± 6.31	64.05 ± 2.54
2	60.67 ± 4.81	66.24 ± 4.87	71.67 ± 3.88
3	68.05 ± 3.33	73.43 ± 2.82	69.53 ± 3.25
4	74.05 ± 3.64	76.44 ± 1.54	69.29 ± 2.78
5	69.10 ± 3.32	65.43 ± 4.20	66.91 ± 2.65
6	52.81 ± 5.79	60.29 ± 4.58	48.10 ± 4.63
7	63.71 ± 4.12	65.31 ± 4.47	61.90 ± 4.00
$\bar{x}$	61.69 ± 3.98	65.36 ± 3.23	64.49 ± 3.01

$\bar{x}$ = Mean of means.

Table 10: Mean hen-housed egg production of each chicken per 14-day period during the laying phase (phase 3)

PERIOD	GROUP I	GROUP II	GROUP III
1	43.48 ± 4.39	50.38 ± 6.31	64.05 ± 2.54
2	60.67 ± 4.81	66.24 ± 4.87	71.67 ± 3.88
3	68.05 ± 3.33	73.43 ± 2.82	69.53 ± 3.25
4	74.05 ± 3.64	75.67 ± 1.42	69.29 ± 2.78
5	69.10 ± 3.32	63.05 ± 4.02	66.91 ± 2.65
6	52.81 ± 5.79	58.33 ± 5.15	48.10 ± 4.63
7	63.71 ± 4.12	62.62 ± 3.54	61.90 ± 4.00
$\bar{x}$	61.69 ± 3.98	64.25 ± 3.27	64.49 ± 3.01

$\bar{x}$ = Mean of means.

Table 11: Mean egg weight in grams of each chicken per 14-day period during the laying phase (phase 3)

PERIOD	GROUP I	GROUP II	GROUP III
1	47.61 ± .58	48.09 ± .56	48.46 ± .73
2	52.15 ± .60	51.02 ± .30	51.04 ± .67
3	53.78 ± .38	53.73 ± .38	52.11 ± .64
4	55.44 ± .61	55.32 ± .34	54.39 ± .62
5	55.54 ± .55	54.89 ± .25	55.26 ± .55
6	55.65 ± .83	55.51 ± .66	55.05 ± .58
7	58.80 ± .40	59.14 ± .48	58.24 ± .55
$\bar{x}$	54.18 ± 1.31	53.96 ± 1.34	53.51 ± 1.22

$\bar{x}$ = Mean of means.

APPENDIX II

Analysis of variance (ANOVA) for feed consumption, cumulative feed conversion, feed efficiency, changes in body weight, hed-day egg production, hen-housed egg production, fifty percent egg production and egg weight in the 3 groups of experimental chickens.

Table 12: Analysis of variance for feed consumption by experimental chickens during the period they were on chick mash (phase 1)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	1278.0	639.0	2.291
Cage	4	794.0	198.5	0.712
Residual	8	2231.4	278.9	
Total	14	4303.4	307.4	

Table 13: Analysis of variance for feed consumption by experimental chickens during the growing phase (phase 2)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	37538	18769	10.859###
Cage	4	62077	15519	8.979###
Period	5	2193707	438741	253.832###
Group x Cage	8	151952	18981	10.982###
Group x Period	10	19724	1972	1.141
Cage x Period	20	28879	1444	0.835
Residual	40	69139	1728	
Total	89	2562914	28797	

Significant at .001 level of probability.

Table 14: Analysis of variance for feed consumption by experimental chickens during the laying phase (phase 3)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	101307	50653	11.576**
Cage	4	40090	10022	2.291
Period	7	1339910	191416	43.746***
Group x Cage	3	479054	59882	13.685***
Group x Period	14	42507	3036	0.694
Cage x Period	28	165444	5909	1.350
Residual	56	245036	4376	
Total	119	2413347	20280	

** Significant at .01 level of probability.

*** Significant at .001 level of probability

Table 15: Analysis of variance for cumulative feed conversion by experimental chickens during the growing phase (phase 2)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	0.53787	0.26893	7.648**
Cage	4	0.23842	0.05960	1.695
Period	5	121.14162	24.22832	688.998***
Group x Cage	8	1.15250	0.14406	4.097**
Group x Period	10	0.57490	0.05750	1.635
Cage x Period	20	0.41786	0.02089	0.594
Residual	40	1.40658	0.03516	
Total	89	125.46983	1.40977	

** Significant at .01 level of probability

*** Significant at .001 level of probability.

Table 16: Analysis of variance for feed efficiency of experimental chickens during the laying phase (phase 3)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	1.2126	0.6064	4.985*
Cage	4	1.4614	0.3653	3.003*
Period	6	10.1539	1.6923	13.913***
Group x Cage	8	1.8417	0.2302	1.893
Group x Period	12	4.7842	0.3987	3.278**
Cage x Period	24	7.7084	0.3212	2.640**
Residual	48	5.8387	0.1216	
Total	104	33.0010	0.3173	

* Significant at .05 level of probability.

** Significant at .01 level of probability.

*** Significant at .001 level of probability.

Table 17: Analysis of variance for body weight changes in experimental chickens during the period they were on chick mash (phase 1)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	949.73	474.86	5.521*
Cage	4	228.73	57.18	0.665
Residual	8	688.10	86.01	
Total	14	1866.55	133.32	

* Significant at .05 level of probability

Table 18: Analysis of variance for body weight changes in experimental chickens during the growing phase (phase 2)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	2459.6	1229.8	2.055
Cage	4	3586.4	896.6	1.498
Period	5	617059.3	123411.9	206.256***
Group x Cage	8	3829.6	478.7	0.800
Group x Period	10	4369.9	437.0	0.730
Cage x Period	20	12299.9	615.0	1.028
Residual	40	23933.7	598.3	
Total	89	667538.5	7500.4	

*** Significant at .001 level of probability

Table 20: Analysis of variance for hen-day egg production of experimental chickens during the laying phase (phase 3)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Period	6	5633.64	938.94	19.665***
Group	2	256.37	128.18	2.685
Cage	4	642.21	160.55	3.363*
Period x Group	12	1785.96	148.83	3.117**
Period x Cage	24	2453.08	102.21	2.141*
Group x Cage	8	1514.53	189.32	3.965**
Residual	48	2291.79	47.75	
Total	104	14577.58	140.17	

* Significant at .05 level of probability.

** Significant at .01 level of probability

*** Significant at .001 level of probability.

Table 21: Analysis of variance for hen-housed egg production of experimental chickens during the laying phase (phase 3)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Period	6	5692.59	948.76	20.379***
Group	2	167.83	83.91	1.802
Cage	4	934.55	233.64	5.018**
Period x Group	12	1782.35	148.53	3.190**
Period x Cage	24	2057.78	85.74	1.842*
Group x Cage	8	1598.47	199.81	4.292***
Residual	48	2234.67	46.56	
Total	104	14468.23	139.12	

* Significant at .05 level of probability.

** Significant at .01 level of probability.

*** Significant at .001 level of probability.

Table 22: Analysis of variance for 50% level of egg production of experimental chickens during the laying phase (phase 3)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	3.2748	1.6374	8.293*
Cage	4	2.5768	0.6442	3.259
Residual	8	1.5814	0.1977	
Total	14	7.4330	0.5309	

* Significant at .05 level of probability.

Table 23: Analysis of variance for egg weights of experimental chickens

Source of variation	Degrees of freedom	Sum of squares	Mean square	F statistics
Group	2	8.204	4.102	2.619
Period	6	1034.716	172.453	110.088***
Cage	4	4.539	1.135	0.724
Group x Period	12	13.653	1.138	0.726
Group x Cage	8	31.319	3.519	2.499*
Period x Cage	24	20.346	0.848	0.541
Residual	48	75.192	1.566	
Total	104	1187.970	11.423	

* Significant at .05 level of probability.

** Significant at .01 level of probability.

*** Significant at .001 level of probability.