EFFECTS OF VARYING LEVELS OF HAEMONCHUS INFECTION ON SOME HAEMATOLOGICAL AND BIOCHEMICAL PARAMETERS OF SHEEP DURING THE DRY SEASON

BY

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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 Dr, R. A. Ogunsusi, Prof. D. I. Saror, both of the Faculty of Veterinary Medicine and Dr. E. O. Oyedipe of the National Animal Production Research Institute.

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

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THESIS APPROVAL

This thesis by Ngozi Chiezey meets the regulations governing the award of the degree of Master of Science of Ahmadu Bello University and is approved for its contribution to scientific knowledge and literary presentation.

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DEDICATION

Dedicated to Emmanuel and Victoria Ekwonu who started it all, and to Uche, Chizoba and Obiageli who were all very much a part of it.

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ABSTRACT

The effects of varying levels of *Haemonchus* spp on haematological and some serum parameters of sheep were investigated, Forty-seven experimental animals with ages ranging from 6 - 18 months, which had previously been exposed to infection were used. They were divided into four groups, I, II, III and IV each consisting of 12 animals with the exception of group IV with 11 animals. Groups I, II and III were innoculated with 35,000, 14,000 and 2,8000 *Haemonchus* infective larvae respectively while Group IV served as uninfected controls. Each animal was dosed in seven instalments spreading over seven days. The results of the experiment were evaluated on haematological, serum protein and serum electrolyte changes, faecal egg output and worm recovery over a 21 - week period.

The sheep showed some resistance to experimental infections of up to 35,000 larvae - as was shown by their faecal helminth egg output and worm recovery data. All animals reacted in a similar pattern irrespective of dose given. Average nematode egg counts of each group did not reflect the level of initial larvae given and was below 4,000 eggs per gram in all infected animals. Animals with the lightest infections had the highest egg counts, cummulative mean values being 626, 982 and 1173 for Groups I, II and III respectively.

Packed cell volume, haemoglobin concentrations erythrocyte and leucocyte counts as well as serum iron values did not show marked changes as a result of infection but the controls had higher mean haemoglobin concentrations and total mean serum iron values significantly higher than those of the infected groups. Differential white cell counts did not show significant changes but there was slight eosirohilia in infected animals.

No abnormal changes in the total serum protein and serum protein fractions were detected as a result of the infection. However, values for the controls were higher than those of the infected but these differences were not statistically significant. Albumin values for Group I were significantly ($P<0.05$) lower than all other groups, values being 46.9 ± 6.6, 49.1 ± 4.9, 49.9 ± 6.0 and 49.5 ± 5.2 percent for Groups I, II, III and IV respectively. Group IV had significantly lower globulin values than any of the infected groups.

Serum sodium and Potassium values did not show any discernable patterns but Group IV had lower sodium values than any of the infected groups while Potassium did not differ significantly between the groups.

At necropsy, the worm burdens did not reflect the doses given and less than two percent of helminths given to each animal was recovered. Mean numbers of worms recovered in Groups I, II and III were 357, 175 and 48 respectively. No arrested larvae were recovered in all the infected groups.

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INTRODUCTION

Sheep, like all domestic animals, harbour helminth parasites in varying numbers within a few days of birth. The extent of the infection and the species of helminth involved determine their general state of health and productivity.

Sheep in Nigeria are not only a source of protein, but constitute an important economic commodity for a large percentage of rural households particularly during religious and traditional festivals. Despite this importance, intensive production of sheep as has been done with poultry has been limited. This was attributed to poor husbandary methods employed as well as heavy internal parasitic burdens particularly _Haemonchus contortus_ (Food and Agrácultural Organisation report 1966).

H. contortus causes a debilitating disease in sheep characterised by progressive anaemia, unthriftiness, poor weight gain, submandibular oedema, severe hemorragic gastritis and death in acute and severe cases (Soulsby 1965). Once infection occurs, clinical manifestations develop rapidly and mortalities may occur within a few weeks striking even the healthiest sheep regardless of age and plane of nutrition. This is particularly true during the early rainy season when climatic conditions favour the development of worms eggs and larvae, and animals are still debilitated from malnutrition and environmental stress from the previous dry season.

This classical form of haemonchosis presents little problem in diagnosis as the symptoms are easily recognised. Allonby and Urquhart (1975) pointed out that one of the significant features of haemonchosis was not necessarily the acute syndrome characteristically associated with the disease, but the fact that a few hundred worms persisting over a long period could produce chronic anaemia and ultimately loss of condition and death in sheep, especially if they are grazing low quality pasture. This insidious effect of chronic haemonchosis has important implications for the attainment of maximum productivity by adult sheep. Its diagnosis also presents problems since morbidity may be high and flock lose weight and general condition without showing obvious signs of the disease. Even when sheep die, diagnosis is often complicated by the fact that moribund animals often lose their worm burdens shortly before death and may also show a terminal haemoconcentration.

It is frequently advocated that adult sheep which have had a previous helminth exposure be used to graze fields that have been contaminated by other stock in order to reduce the level of helminth infection available to lambs with no experience of infection (Michel 1963). This assumes that these older sheep have acquired some degree of resistance to all helminths, from the previous infection, and so have a large threshold of tolerance or resistance to reinfection.

This study was therefore designed to study the effects which graded doses of Haemonchus species would have on haematological and certain biochemical parameters of sheep that had previously been exposed to a natural infection, as well as follow the course of the infection by faecal egg counts and evaluation of worm burden on termination of the experiment.

Studies of Haemonchus infections in sheep in Nigeria have been mostly as outbreaks or as mixed infections with other parasites (Kuil 1972, Fabiyi 1973, Schilhorn 1973, Anosa 1977, Schilhorn 1978, Ogunsusi 1978). There is need however to separate the parasitic gastroenteritis complex into specific disease entities based on observations of pure infestations of the different causative nematode species. This will provide some information on the roles played by each member in producing the disease complex. The outcome may lead to improvement in our control strategies.

LITERATURE REVIEW.

Historical Background

Haemonchus contortus was described by Rudolphi (1803) but was included in the genus *Strongylus*. It was only towards the end of that century that Cobb (1898) removed *Strongylus contortus* and established it as an independent genus *Haemonchus*. The life history of the parasite was first worked out experimentally by Ransom (1906). Nine years later Veglia (1915) made a classical study of the pathogenesis of the parasite and Fourie (1931) made the first detailed study of the pathology and haemotology of *H. contortus* infection in sheep.

The parasite has long been recognised as a major problem in small ruminants in Nigeria. Henderson (1928) reported on gastro-intestinal helminthiasis in sheep caused by *Haemonchus*. Other early reports of the parasite in ruminants include those of Beaton (1937), Lee (1955) and Lindquist (1958).

Biology of Haemonchus spp

Life cycle

H. contortus is a parasite of the abomasum of ruminants. They are slender worms of which the males are 10 - 20 mm and females 18 - 30 mm in length (Soulsby 1965). The morophology of the parasite has been adequately described by Veglia (1915). It has a direct life cycle. Fecundity is very high, females producing between 5,000 and 10,000 eggs per day. *Haemonchus* egg is ovoid with a thin transparent shell and it is excreted with the faeces of the host. At about 26^{o}C the first stage larvae hatch out of the eggs in about 24 hours. The hatched larvae moult twice, developing into second and third stage larvae respectively. Under optimal conditions, the third stage or infective larvae stage is reached within four to five days after the eggs are passed out of the host. The infective larvae can only continue their development inside a suitable host. Infections of the host occur when the infective larvae are ingested during grazing. Inside the host, infective larvae moult twice before they grow into mature male and female worms on the mucosa of the abomasum of the host; this process takes about nineteen days in susceptible animals.

Effect of Temperature on Egg Development

According to Shorb (1943, 1944) no development of Haemonchus eggs takes place below 13°C while constant temperatures of about 36.6° are deleterious to the eggs. Dinaburg (1944) reported that eggs of Haemonchus did not develop to infective stage at constant maximum temperatures of even 18.3°C regardless of rainfall. However, Gordon (1948) produced evidence that development of Haemonchus could take place below 18.3°C, suggesting that Dinaburg's maximum temperature was too high for universal application. In Kenya, Dinnik and Dinnik (1958) found that larvae of Haemonchus reached the infective stage in eight to twelve days, only when the diurnal fluctuation of air temperature was between a mean maximum of 24°C and 12.3°C.

Silverman and Campbell (1959) studied the relationship between temperature and development of the pre-parasitic larvae of Haemonchus. They found that the first infective larvae appeared in five days at a constant temperature of 21.7°C, in nine days at 14.4°C and in at least fifteen days at 11°C. At temperatures lower than 7.2°C, development was inhibited and the pre-hatch stage of the eggs were the most resistant to low temperatures. They concluded that development of H. contortus egg require about two weeks in summer and considerably longer during cooler seasons.

Effect of Moisture on Egg Development

Eggs and larvae of Haemonchus also need moist conditions for development, as dessication kills the eggs rapidly especially in the pre-hatch stage. Dinnik and Dinnik (1958) observed that in the Kenyan highlands, a monthly rainfall of over two inches was essential for H. contortus larvae to develop to infective stage in large numbers. Similarly, Okon and Enyenihi (1977) working in Nigeria found that for eggs to develop to infective stage, there must be a mean daily rainfall of 3 mm or more.

Dinnik and Dinnik (1961) further observed that 10% of larvae in a shade which were protected from drying out survived for ten to thirty days depending on the thickness of the grass cover.

It has also been observed by Veglia (1915) and Monnig (1930) that development of H. contortus eggs and larvae failed to take place in wet or immersed faeces, as the eggs were killed unless water was removed in less than seven days.

Effect of Oxygen on Egg Development

When free circulation of air is prevented in cultures by sealing of culture jars, development of *H. contortus* eggs does not take place; normal development and metabolic process only take place in the presence of oxygen (Blood and Henderson 1963, Soulsby 1965). Oxygen however, would not be a limiting factor to developing eggs and larvae on pasture, but may be to those in faeces dropped into ponds or bodies of water.

Clinico-Pathologic Aspects of Haemonchosis

Clinical symptoms

The most general features of haemonchosis are: progressive loss of condition, unthriftiness, loss of weight and anaemia. In more advanced cases, there may be a "pot-belly" appearance and a swelling underneath the jaw (bottle jaw). In severely infected animals, the loss of condition may reach emaciation, the fleece is broken and lacks lustre, the animal becomes extremely weak, then recumbent and later dies (Soulsby 1965).

Dargie and Allonby (1975) gave the three main features of haemonchosis as anaemia, hypoproteinaemia and poor weight gain. They attributed these features directly to the blood sucking activities of the parasite and on host factors such as age, nutritional status and the rate and size of larval challenge. This, they said, produced a complete spectrum of clinical effects which could be categorised as hyperacute, acute and chronic. The hyperacute type is uncommon, lasts from zero to seven days with a severe haemorrhagic gastritis and rapidly fatal severe anaemia and sudden death in previously healthy sheep with no diarrhoea. The acute type is more common and lasts from one to six weeks and is due to a prevailing burden of adult parasites with continued re-infection. The outcome is often agalactia leading to death of lambs or weakness and loss of condition. The third type of haemonchosis according to Dargie and Allonby, is the chronic type which is more widespread and lasts for up to six months and is caused by a relatively low burden of adult worms. It is characterised by chronic gastritis with chronic blood loss and abomasal dysfunction leading to progressive weight loss and stunting of growth. The outcome of this depends largely on nutritional status of the host and death only occurs in extended spells of dry weather with concomitant poor pasture conditions.

Morbidity and mortality

There is considerable variation in the pathogenicity of _H. contortus_ infections reported by different authors. Fourie (1931) artificially infected 38 worm-free sheep with varying doses of _H. contortus_ larvae given as single or multiple doses. One animal developed a fatal progressive anaemia after it had received 20,600 larvae in multiple doses, while six developed anaemia after receiving doses of either

3,000, 10,000 or 20,000 but later recovered. The rest of the animals did not show any visible sings of haemonchosis.

Andrews (1942) administered doses ranging from 2,000 to 181,000 infective larvae of _H. contortus_ to lambs in single and multiple doses. Thirty five thousand larvae given as a single dose to a three-month old lamb proved fatal, as th animal died 45 days later. Another three-month old lamb which received 100,000 larvae died in 23 days while an eight month old lamb given a dose of 45,000 survived the infection. Some animals which received up to 70,000 larvae in multiple doses survived, and Andrews attributed this to development of immunity in these animals.

Stewart (1953) found that in single and multiple infections of lambs with 3,000 _H. contortus_ larvae, followed by a challenge dose of 5,000 (after 29 weeks), the animals which received multiple doses were more resistant to the infection than those which received the infection in a single dose.

Charleston (1964) using four and a half month-old lambs, gave four of them 10,000 _H. contortus_ larvae daily for up to 53 days. Of the four, one died on the 27th day and another on the 29th day after the start of the dosing, having received 270,000 and 290,000 larvae respectively. At post mortem, totals of 8,960 and 42,860 worms respectively were recovered. Two other lambs survived a total of 530,000 larvae given in this way and then killed 56 days later; 100

and 4,080 helminths were recovered from them respectively.

Brambel, Charleston and Tothill (1964) gave six worm-free sheep 100,000 infective H. contortus larvae in two doses of 50,000 each at three hourly intervals. The animals showed age resistance in that the infection became patent in only one of the infected animals. Number of worms recovered at post-mortem were 400, 10, 360, 0, 640 and 8,550 respectively.

Christie, Brambel and Charleston (1964) studied worm population in seven-month old sheep dosed daily with 10,000 larvae of H. contortus. They started slaughter of these animals as from the fifth day of infection and on till day twenty-six. They found that some of the animals were anaemic at slaughter and some were resistant to the infection. The number of worms recovered was proportional to the degree of anaemia. In the sheep that were resistant, not only were there few adult worms but there were proportionally fewer adolescent worms of sizes corresponding to nine to thirteen days growth- Christie et al (1964) concluded that developing worms encounter some particular hazard about the eight day of its life in sheep that had recently become resistant to Haemonchus.

Silverman, Mansfield and Scott (1970) found that two-month old lambs on a high protein diet survived exposure to as many as 10,000 H. contortus larvae. Administration of 25,000 larvae caused death of two out of five lambs and a dose of 50,000 larvae caused death of 4 out of five lambs. On treatment and subsequent challenge with 10,000 ;arvae, the animals which survived the 25,000 primary innoculation proved to be the most resistant to re-infection. They concluded that the 50,000 larvae used as a primary dose may have paralysed the immune mechanisms in the animals that received it.

Christie (1970) investigated the fate of very large doses of Haemonchus larvae in sheep and reported that a dose of three million infective larvae produced only two cases of relatively milk haemonchosis out of five adult sheep of varied worm history. A dose of one million infective larvae induced self-cure of an established population of adult worms, supporting Christie's hypothesis that very large doses of Haemonchus produce changes in the abomasum unfavourable to development and persistence of the worms.

Misra and Ruprah (1972) followed the course of H. contortus infection in ten week old lambs. After experimental infection with five doses of 500 larvae given every alternate day, half of the lambs developed diarrhoea ten days after the first dose and eleven of these died within three weeks. No blood in the faeces or rise in body temperature were noticed but there was a decrease in body weight in infected animals.

Haematology

H. contortus has been shown to produce disease primarily by extraction of the host's blood.

Fourie (1931) reported that in fatal cases, there was progressive decrease in red blood cell count, percentage haemoglobin and percentage volume of red cells. The red cells remained normal until the count fell from 11.4 to 5.9 million/cumm. Thereafter, anisocytosis, poikilocystosis, polychromasia punctuate basophilia were noticed. The white cell count started at 4200 thousand/cumm. and dropped to 1,600 thousand/cumm. and thereafter rose. Neutrophilia was seen towards the end of the disease, eosinophilia was only apparent in the early stages. In less severe cases, the progressive decrease in the number of red blood cells was not always reflected in the value of the haemoglobin and packed cell volume curves. This was probably due to an increase in the size of the cells.

Andrews (1942) found that in fatal cases of experimental pure H. contortus infection in sheep (when death sometimes occurred before the end of the prepatent period), there was a fairly sharp and steady downward trend in the packed cell volume, red cell count and percentage haemoglobin. There was a corresponding steady increase in the amount of blood elements recovered from the faeces. Andrews (1942) also found reticulocytes in the blood, with a higher number in the more heavily infected animals. He took this as evidence that haematopoiesis was not interfered with in haemonchosis.

Delaune and Maylew (1943) gave a description of the blood picture of calves dosed with Haemonchus. The erythrocyte and haemoglobin values began to decrease five to seven days after the infection and continued to the end of the prepatent period. The total leucocyte count however, showed an initial increase at this period. After the infection became patent, the red cell counts, haemoglobin concentration and leucocyte counts gradually increased and in some cases exceeded the pre-infection levels. They therefore emphasised the importance of the prepatent period in *Haemonchus* infections.

Silverman and Patterson (1960) reported that there was no response of sheep to the third and fifth histotrophic phases of larvae in either resistant or susceptible hosts until the ninth day following infection, when the young adults burrowed into the mucosa to suck blood.

Charleston (1964) compared the haematology and bone marrow cytology of sheep experimentally infected with *H. contortus* with those of worm-free sheep bled to simulate the blood loss of *Haemonchus* infection. He found the decline in packed cell volume to be comparable in both groups. However, in haemonchosis, the anisocytosis was less marked and there was a smaller over-all increase in mean cell volume and a higher proportion of immature cells which were deficient in haemoglobin. In heavy parasitic burdens, normoblasts with abnormal morphology were produced. Charleston (1964) also found that total leucocyte counts failed to follow any trend that could be related to the experimental procedures while the number of neutrophils showed a decline in all groups.

An eosinophilia of recovery was seen in parasitised sheep which survived the infection.

From his studies on haemonchosis in goats, Fabiyi (1968) concluded that a population of less than 250 adult worms had no effect on the packed cell volume and haemoglobin concentration of goats. With increased number of helminths however, packed cell volume and haemoglobin values became lower.

Harness, Fitzsimmons and Sellwood (1970) infected three groups of calves with either 50,000, 125,000 or 500,000 Haemonchus placei larvae and the blood pictures compared with those of a control group. There was little deviation from normal in the calves infected with 50,000 larvae. Calves infected with 125,000 or 500,000 showed transient oligocythaemia which generally did not vary greatly in it's severity, but recovery was more protracted in the calves with the heavier infection. In the calves with a higher level of infection, there was a reduction in lymphocytes and neutrophils and also a fluctuation in the number of monocytes, though the number never exceeded ten percent of total leucocyte count.

Kuil (1972) carried out studies of gastrointestinal nematodes of sheep in Northern Nigeria and found that values of haemoglobin concentration, packed cell volume and serum protein concentration always depreciated according to the level of infection. The depreciations were especially significant in animals with worm burdens over 5,000.

Misra and Ruprah (1972) found a decrease in all blood values in lambs with experimental Haemonchus infection. There was however an increase in the percentage of monocytes, neutrophils and to a lesser extent eosinophils.

Pradhan and Johnstone (1972) subjected one group of lambs to 3,500 H. contortus larvae at weekly intervals and another group to 500 larvae each day. The dosage was decreased to 1,400 and 200 larvae respectively after eight weeks. They observed that the daily infection with a small number of larvae induced a severe anaemic condition with the picture falling to 7% packed cell volume, 2% haemoglobin concentration and $1.6 \times 10^6/mm^3$ red cell count in 78 days. The anaemia with the weekly dosed group was less severe with values of 10 - 20% packed cell volume, 4% haemoglobin concentration and $3.6 \times 10^6/mm^3$ red cell count. They suggested that haematological values of sheep seven to fifteen days after infection might be indicative of their genetic ability to resist Haemonchus infection.

Allonby and Urquhart (1975) studied the epizootiology of H. controtus infection in ewes and lambs in an endemic area of haemonchosis in East Africa. They observed that mean packed cell volume and haemoglobin values dropped progressively with increase in adult worm burden. They also noted that there was a uniformity of faecal egg counts in both ewes and lambs over long periods, though necropsies revealed that their worm burdens were markedly different.

They pointed out that one of the most significant features of haemonchosis was not necessarily the acute syndrome characteristically associated with the disease, but the fact that a moderate infection of a few hundred worms persisting over a long period could produce chronic anaemia and ultimately loss of condition and death in ewes and lambs grazing low quality pasture.

In two to four month-old sheep given a single infection of H. contortus larvae, Coadwell and Ward (1975) observed two types of reaction to the infection. In one group, the packed cell volume fell sharply from ten days after infection and the sheep eventually died. In the second group, the fall in packed cell volume was less marked and returned to normal after about 40 days. They also compared the rate of increase of parasite body length and dry weight in both groups. These results showed that the first group of sheep had a heavier infection but the parasites were small in size probably due to a greater interaction chemically and physically between parasites. This would have led to a more severe blood leakage and alteration in abomasal PH and osmotic concentration, creating a less favourable environment for the growth of the parasites. The second group of sheep on the other hand, had a smaller parasitic burden and the parasites were larger due to a more stable environment.

Other reports of depreciation of blood values in sheep with chronic nematodiasis of which Haemonchus was the dominant specie include those of Oduye (1976) Saror and Schilhorn (1977) and Ogunsusi (1978). Ogunsusi (1978) showed that with increase in egg output, there was a rapid decline in values of packed cell volume, haemoglobin concentration and red cell count. The white blood cell count did not show any descernable pattern but there was a progressive increase in neutrophils and fall in number of lymphocytes. In those animals with a chronic infection, helminth egg output was unsteady for a long time and there was a more gradual decline in the values of packed cell volume and haemoglobin concentration.

Anaemia

The discovery by Fourie (1931) that larval and adult stages of H. contortus could suck blood, led various groups of workers to study the amount of blood lost and its relation to the anaemia of haemonchosis. Evidence that the anaemia of haemonchosis was due primarily to blood ingestion by the parasite has been given by many authors.

Martin and Ross (1934) estimated the amount of blood lost daily in Haemonchus infections. They calculated the quantities of phosphorus in the eggs laid by the parasite and on the assumption that this phosphorus came wholly from the blood of the host, estimated that about 0.05 ml of blood per worm per day is removed by the parasite. This finding was confirmed by Baker et al (1959) using chromium labelled erythrocytes to study the iron reserves in Haemonchus-infected lambs.

Boughton and Hardy (1935) were able to observe the behaviour of H. contortus through fistulas made in the abomasum of infected sheep. They found that the parasite attached themselves by a peculiar striking motion of the head and neck; that they remained attached for about 12 minutes and sucked blood, which could be followed through the intestines of the nematode almost to the anus. Minute haemorrhages which continued for about seven minutes occurred as soon as the worms detached themselves from the stomach wall. Andrews (1942) was also able to show that the anaemia associated with H. contortus infection in sheep was due to gastric hemorrhage alone, by a study of the icteric index, reticulocytes in the blood of anaemic lambs and the gross pathology and histology of affected lambs. Clark, Keisel and Goby (1962), Brambel, et al (1964) also studied blood loss in haemonchosis using chromium-labelled erythrocytes. Both groups of investigators showed that the anaemia was due to blood letting and ingestion by the immature as well as mature stages of the parasite.

There are other reports which indicate that anaemia in relation to haemonchosis may not be due to blood loss alone. Neklyudor, Bolkhovitinov and Sominski (1956) infected three to ten month-old lambs with single or repeated doses of larvae ranging from 4,000 to 106,000. They reported that the progressive anaemia which developed was aplastic and not hemolytic. They therefore suggested that the anaemia may have resulted from a disturbance of the neuro-humoral regulation of the haemotopoietic system.

Oshio (1952, 1953) believed that the anaemia of haemonchosis was due to an anaemia-producing substance eliminated by the parasite. He was able to produce severe anaemia in rabbits by injecting serum from infected sheep and goats into rabbits. Serum from non-infected animals did not produce anaemia. The weakness of this hypothesis however is the lack of direct experimentation on the hosts.

Charleston (1964) also studied the type of anaemia in *Haemonchus* infection and reported a normochromic macrocytic anaemia in both bled and parasitised sheep. He however, presented evidence of interference with normoblastic maturation and haemoglobination in the latter, and thereforee concluded that the anaemia of haemonchosis differed from that of phlebotomy.

From a measurement of mean cell volume and mean cell haemoglobin of infected lambs, Misra and Ruprah (1972) concluded that the anaemia of haemonchosis was microcytic and hyprochromic. Holmes and Dargie (1975) observed a hypochromic but macrocytic anaemia in heavy *Haemonchus* infections. Higher infections initially resulted in a normocytic normochromic type which later also developed into a macrocytic hypochromic anaemia.

Most of these data concerning the anaemia produced by *H. contortus* have been derived from studies on peripherial blood with little attention paid to the concurrent serum iron alterations. Allonby and Dargie (1974) observed that sheep infected with only 8,000 H. contortus would be losing about one billion red cell containing 15 gm of haemoglobin and 50 mg iron into its abomasum every day. An infected animal therefore has to become 'hyperkinetic' in order to replace this daily loss as well as maintain normal haematological indices, and so is under constant strain. If not treated, this will develop to the stage where exhaustion of it's synthetic machinery sets in.

Scott, Silverman, Mansfield and Levine (1971) studied active and passive immunity in sheep given oral iron supplement. They found that unvaccinated and otherwise untreated lambs fed iron ad libitum survived oral innoculation of 50,000 H. contortus larvae that killed 8 - 100% of lambs not fed oral iron. Lambs fed oral iron until their growth was attained were resistant to innoculations of 70,000 larvae. They concluded that resistance of young rapidly growing lambs to initial innoculation of H. contortus larvae depended on the capacity of the haemopoietic system of the host to compensate for the loss of blood.

Dargie and Allonby (1975) studied iron metabolism in single and challenge infections with H. contortus. During the initial period, there was some depletion in the level of serum iron (from 131±23 ug% to 100±30 ug%). With the challenge infection, animals varied in individual response. Animals with the "self-cure and protection" response, had improved serum iron levels which did not return to pre-infection values. On the other hand, sheep which succumbed to the challenge infection had very low levels of serum iron. These sheep they said, were already depleted of iron as a result of the blood loss incurred from initial infection and so, were unable to meet the level of red cell production required to keep pace with haemorrhage resulting from subsequent infection.

Serum Proteins

Following helminth infection, major changes in the serum protein concentrations can occur. These changes depend on the host and parasite considered and may be manifested singly or in various combinations in all of the major serum protein components (Leland 1961). Where serum protein changes have occurred as a result of experimental helminth infection, the general picture has been a reduction in albumin accompained by an increase in one or more of the globulins.

Shumard and Eveleth (1955) as well as Shumard, Bolin and Eveleth (1957) observed a depression in the total protein and the albumin: globulin ratio in lambs experimentally infected with equal numbers of <u>H. contortus</u>, <u>Nematodirus spathiger</u> and <u>Trichostrongylus colubriformis</u> larvae.

Ross, Lee and Armour (1959) described a genetic line of zebu cattle that exhibited resistance to haemonchosis. The resistant cattle tended to show an increase in the faster-moving gamma-1 globulin while the low resistance cattle showed the increase in the slower moving gamma-2 globulin. The resistant animals also showed a higher immunological response and Ross et al (1959) suggested that complement-fixing antibody may be present in the gamma-1 fraction of the serum proteins. Ross and Armour (1960) further suggested that serum albumin levels and packed cell volume could be useful measures of pathogenicity when considered in conjuction with a series of differential worm counts.

Ross, Armour and Hart (1962) made daily serum collections from calves infected with 40,000 larvae of H. placei and noticed the maximum fall in serum albumin levels 19 days after infection and nine days before patency, In addition, a sharp transient drop occurred in serum albumin levels 96 hours after infection. Anderson et al (1960) on the hand, did not observe any marked changes in serum protein in calves experimentally infected with 12,000 and 165,000 H. placei larvae of an antelope strain.

Kutler and Marble (1960) recorded the serum protein changes in lambs with naturally acquired clinical, moderate and light infections of mixed nematodes. In addition, the serum proteins of lightly parasitised animals with anaemia which had been induced by periodic bleeding was measured, to ascertain that the anaemia often associated with clinical parasitisms was actually responsible for the serum protein changes. Significant changes in the serum proteins occurred with moderate to clinical parasitism. In these lambs, the total serum protein and albumin fraction were decreased, while the alpha-1, alpha-2, beta and gamma globulin fractions were increased. This confirmed earlier observations by Leland _et al_ (1958) who reported extensive and prolonged hypoprotenamia with hypoalbunaemia and an increase in the alpha-2 globulin in calves experimentally infected with _Trichostrongylus axei_. They suggested that liver function and hence synthesis of plasma proteins, might be altered by malnutrition or by toxins from the parasites.

Cornelius _et al_ (1962) found that hypoalbuminaemia in cattle heavily parasitised by trichostrongylids was caused by reduced synthesis of albumin; only in terminal cases was increased catabolism observed. Wilson and Turner (1965) observed high albumin: globulin ratios and lower gamma globulin percentages in lambs raised parasite free in comparison to those moderately or heavily parasitised. They postulated that increase in gamma globulin in parasitic infections may be one of the factors in the increase in resistance to the infection.

Leland, Drudge and Dillard (1966) were able to show that even with a 16% crude protein supplement, s superimposed nematode infection was still able to produce hypoproteinaemia and serial sequence of changes in the serum proteins of infected calves. Serum protein changes consisted of an increase in alpha-2 globulin that lasted for several weeks and then declined. With this decrease, the beta-globulin increased moderately, the gamma globulin increased markedly while the albumin decreased markedly. These changes were more significant in those animals that had succumbed to parasitism, and this was interpreted as a reaction of the host to injury or stress caused directly or indirectly by the helminth parasites. Akerejola (1976) on the other hand did not observe any significant serum protein changes in a mixed breed of cattle fed varying protein diets and given a mixed nematode infection of Haemonchus, Trichostrongylus and Oesophagostomum species. Decrease in albumin and an increase in serum globulin was only observed during the last week of the study when faecal egg count was highest.

Serum minerals

Little attention has been paid to the effects produced by helminth infection on the blood or serum mineral elements of ruminants. Most studies have been on effects of mineral supplements on the course of artificial and pasture infestation (Weir et al, 1948, Richard et al, 1954, Shumard et al, 1956). These investigations showed that although additional mineral might in some instances enable the host to withstand parasitic attack better, such supplements might also have the effect of increasing the fecundity and anaemia-producing properties of the parasites.

Ross and Gordon (1933) found difficulty in establishing H. contortus infections in sheep on a diet poor in protein, calcium and phosphorus. When the protein was reduced further but calcium and phosphorus increased, heavy infestation resulted. Shearer and Steward (1933) observed interference with calcium and phosphorus metabolism but no change in the metabolism of sodium and potassium in lambs heavily infected with a mixture of intestinal nematodes. Similar results were obtained by Franklin et al (1946) who experimentally infected lambs with T. colubriformis and found a lower rate of live weight increase, lower protein digestion and poor utilization of calcium and phosphorus in the infected animals. Blood analysis revealed low calcium and phosphorus levels.

Shumard and Herrick (1954) showed that H. contortus larvae cultured from eggs excreted by cobalt-supplemented lambs were of greater length than those cultured from eggs excreted by non-supplemented lambs. Threlkeld, Price and Lincous (1956) produced further evidence of the susceptibility of H. contortus to the host's cobalt status.

Evans, Blunt and Southcott (1963) described an anaemia of different haemoglobin types caused by H. contortus, characterised by a marked rise in the concentration of Potassium (K^+) in the erythrocytes and a fall in the concentration of sodium (Na^+), The total ($Na^+ + K^+$) remained constant. Increased potassium and decreased sodium levels were associated with a fall in haemoglobin and packed cell volume as well as a reduced appetite and slow growth rate. They explained the rise in potassium as being a result of an influx into the circulation of young cells containing higher concentrations of potassium. Cells of this type have been demonstrated in blood loss anaemias produced experimentally (Blunt and Evans 1963).

Pradhan and Johnstone (1972) in their studies of haematological changes in lambs during prolonged exposure to infective larvae of H. contortus also found alterations in blood electrolyte values. At first, the potassium concentration increased and the sodium decreased but both subsequently began to increase. They also attributed these changes to influx of young erythrocytes into the circulation.

These findings on helminth and host mineral relationships indicate that the helminths require and/or interfere with metabolism of some host minerals in order that their (the helminths') development and reproduction may attain maximum potential.

Resistance to Haemonchus infection

Factors that have been found to influence the resistance of the host to H. contortus infection include: genetic constitution, host diet, age of host, intercurrent infection and debility from other causes (Culbertson, 1941).

Genetic Resistance

The difference in natural resistance against a given parasite manifested by two different races or breeds within a single species of host is best explained through genetic constitution of individuals.

Cheviot sheep have been found to be more tolerant to infection with H. contortus than lowland breeds of sheep (Cameron, 1939).

Ross, Lee and Armour (1959) during an investigation into poor post-weaning performance in zebu cattle in Nigeria, found evidence of a genetically controlled resistance to haemonchosis.

Blunt and Evans (1963) showed that sheep with haemoglobin type HbA were more resistant to H. contortus infection than those with type HbAB. Sheep with both haemoglobin types were infected, and the trend in faecal worm egg counts and worm burdens at necropsy suggested that the HbA sheep were more resistant to the establishment of the infection although there were no significant differences in haematological indices of the two haemoglobin types. Later, Evans and Whitlock (1964) demonstrated a significant association between the seasonal maximum haematocrit of grazing sheep and haemoglobin type. They suggested that this might be related to a varying degree of resistance to natural infection with H. contortus.

Jilek and Bradley (1969) as well as Radhakrishnan, Bradley and Hoggins (1972) in a study of the relationship of haemoglobin type in Florida Native sheep to naturally acquired and experimental H. contortus infections, concluded that there was no definitive data to support the theory that haemoglobin types in Florida sheep were indicators of resistance to haemonchosis, or that HbA sheep generally had higher packed cell volume values and underwent self-cure more readily. However, Allonby and Urquhart (1976) produced evidence that merino sheep with HbA type showed self-cure more frequently and effectively than those with HbB and HbAB types.

Examination of the packed cell volume values and total body weights of merino ewes showed that the HbA types had the highest haematological parameters and the heaviest body weights while the HbB types were consitantly lower.

Altif and Dargie (1978) examined strain and breed differences in the response of Scottish Blackface and Finn Dorest sheep to primary infection with H. contortus. They found that both types of sheep with HbAA type had lower worm burdens and faecal egg outputs, and suffered less severe clinical and pathophysiological disturbances than those with haemoglobin type HbBB. In addition, Scottish Blackface sheep displayed similar advantages over Finn Dorsets with the same type, and variations in the severity of the disease as judged by a variety of pathophysiological indices correlated with parasite numbers. They therefore concluded that genetic resistance operated primarily against worm establishment and that baring the unlikely involvement of non-specific physiological factors, this was controlled by the immune response elucidated.

In Northern Nigeria, Schilhorn and Folaranmi (1976) investigated the importance of haemonchosis in the natural selection of haemoglobin type in the area. They found that almost all sheep were of the homozygotic HbBB type which had been reported to be less resistant to haemonchosis. They concluded that with sheep under the dry conditions of the North, the selection pressure for HbBB type which is more adapted to a dry environment is stronger than that for HbA type which is more resistant to haemonchosis.

Effect of diet on resistance

The nutritional status of the host animal can exert a profound influence on it's natural and acquired immunities to nematode parasites (Chandler 1953).

Several observations on controlled experimental infections have shown that sheep which receive diets inadequate in some form to support their full growth potential invariably harbour more worms than adequately fed animals. Fraser and Robertson (1933) determined the influence of nutritional levels (pasture and grain supplement versus pasture) on infections of H. contortus in lambs. Although infections acquired were very low, well fed animals acquired fewer worms than poorly fed lambs. Ross and Gordon (1933) reported that acquired resistance of sheep could be broken down by feeding a diet markedly deficient in protein and minerals. Taylor (1934) found heavier mixed infections including Haemonchus in sheep given straw only or hay only, than in sheep given hay and concentrate. In further experimental work, Taylor (1943) divided his lambs into three groups and exposed them to the same infection of Trichostrongyles. Group 1 which received hay and concentrate showed an average egg count of 3,000, group II on hay alone showed 9,000 while group III on straw alone showed 14,000 eggs per gram of faeces. He subsequently divided group III into two groups. Those receiving hay and concentrate showed a reduction in their egg counts to one ninth in seven months while those on hay alone showed an increase up to five times the original, despite the absence of continued re-infection.

The effect of alfa-alfa hay and grain was compared by Lucker and Neumayer (1947) with that of a diet of timothy hay alone on Bumostomum trignocephalum. The timothy hay diet was poor in protein, iron, calcium and some vitamins compared with the good diet. There were more worms and a greater worm egg output from lambs on the poor diet. Gordon (1948) reported the effect of two levels of nutrition on naturally infected sheep brought into two pens, and on their resistance to re-infection with H. contortus. Those on the better ration lost their worms more rapidly and resisted development of a challenge dose more successfully than those on the poor ration.

An apparent exception to the above reports that sheep on good feed have fewer H. contortus than sheep similarly exposed to infection, but on a poorer quality feed, is the report of Whitlock (1949).He reported that supplement-fed lambs on pasture had more H. contortus than pasture fed lambs and that dietary differences were responsible for larger numbers of this parasite in the group on the better feed. Earlier however, Whitlock et al (1943) had reported that an improved diet consisting of alfa-alfa hay and corn was responsible for lessening egg counts and markedly reducing the number of Haemonchus present in sheep that had been on pasture. This has been supported by the work of later authors.

Laurence et al (1951) found that naturally infected Merino lambs succumbed to infections of H. contortus and Oesophagostomum venulosum when fed on a standard sheep ration with no maize supplement. Another group of the same lambs showed a drop in worm egg count and improvement in condition when given lucerne hay and maize in generous amounts. Further experiments then led them to conclude that lambs kept on a high plane of nutrition with liberal amounts of lucerne hay and yellow maize in addition to veld hay, had fewer parasites than lambs kept on a low plane with less lucerne hay and maize. Vegors et al (1955) found that calves on fescue pasture with low nutritive value had more gastro-intestinal nematodes at autopsy than similar calves on temporary winter or crimson clover pasture. They also noted that calves on each type of pasture which received corn supplement generally had fewer worms than calves on the same type of pasture which did not receive corn supplement. Bilkovich (1962) similarly observed a decreased egg production when corn was substituted for other grain in the ration of calves harbouring established infections of H. placei. Only half as many worms were recovered at necropsy from the corn-fed animals, although those that survived were longer. He concluded that corn per se had produced a degree of parasite expulsion as compared with the other group of infected calves that received a good quality mixed grain ration without corn.

In another experiment by the same author, (Bilkovich, 1967) the effect of an all- corn supplement on established Haemonchus infections was again evaluated. Once again, the faecal egg count level decreased markedly as soon as corn was added to the diet. Egg production by parasites infecting calves on the mixed grain diet was unchanged. Again significantly fewer worms were recovered at necropsy from calves fed corn. Although he was unable to explain, Bilkovich speculated that the effect of corn on the parasites may be due to a nutritional deficiency or simply due to the mechanical effect of corn on the parasite.

Goldberg (1965) studied the relation of diet to gastrointestinal parasitism in cattle and reported that animals on the lower quality feeding level harboured 10% more worms and passed out 11% more eggs than those on the higher feeding level. Kates and Wilson (1962) also compared the effects of two diets, namely: chopped lucerne hay and a pelleted feed containing lucerne meal, maize and mollases on experimental haemonchosis in lambs. They observed that lambs given the well balanced pelleted feed were more resistant to single experimental infections of Haemonchus and to the clinical effects thereof, than lambs fed chopped lucerne hay. The number of worms recovered from the lucerne-fed animals also averaged about twice the number recovered from three pelleted feed lambs. They deduced that a high carbohydrate diet in the form of a grain supplement was beneficial in reducing the severity of anaemia and other ill effects associated with worm parasitism in sheep.

Age Resistance

Helminth infections are generally more severe in young animals than in older ones. According to Gibson (1965) a distinction should be drawn between resistance to infection and resistance to the effects of infection. He is of the opinion that under field conditions, it is not possible to separate the effects of age from the effects of previous infections.

In the case of Haemonchus, age resistance has been reported by some authors. Manton et al (1962) using the Dorset Horn and Dorset Down breeds of sheep were able to demonstrate that lambs aged two to four months which had been given a preliminary infection of 3,000 H. contortus larvae, did not resist a later challenge (after 64 days) of 5,000 larvae as well as did another group of sheep aged 10 - 12 months which were challenged with 15,000 larvae. They concluded that this was good evidence that age was an important factor in the acquisition of resistance to haemonchosis. Similarly, Brambell, Charleston and Tothill (1964) encountered age resistance in five of six nine-month old sheep infected with 100,000 H. contortus larvae in one day. The infection became patent only in one animal.

The greater susceptibility of young animals to parasites is thought to be a reflection of their poor capacity to manifest a specific immune response after infection. The capacity of the specific antibody-forming mechanisms of young animals is known to be poor compared to that of adults. In such young animals, the parasite will be able to establish itself and cause an intense infection whereas in animals able to respond with specific antagonistic antibodies, the infection may largely be inhibited and qucikly eliminated.

Acquired resistance and the self-cure phenomenon

Acquired resistance refers to immunity acquired by the host when it is naturally infected by a parasite or artifically acquired when the host is given an infection by man. According to Soulsby (1958) acquired immunity expresses itself by resistance to further infection by the parasite, by elimination of the parasite, by the inhibition of the growth of larval stages of the parasite or by the inhibition of the reproductive capacity of the adult parasites.

Resistance to further infection by Haemonchus is stimulated by previous infection and has been shown by a number of experiments using experimental innoculation of sheep with patent or attenuated larvae. Stoll (1958) reported a high degree of immunity to re-infection when sheep were immunized by subcutaneous or intraperitoneal injection of large numbers of living exsheathed third stage H. contortus larvae. Jarret et al (1959, 1961) published results obtained from the experimental vaccination of sheep using a single dose of x-irradiated larvae. They suggested that the adult stage is not needed for the production of immunity. This was confirmed by Holman and Patterson

(1960) who showed that it was the fourth and early fifth stages which were the most antigenic and therefore the most susceptible to antibodies. They claimed that the injection of several thousand fresh or formaline treated fourth and early fifth stage larvae could give 70% protection against re-infection. They observed that in resistant sheep, the majority of worms failed to develop beyond the fourth and fifth stages and that these were thrown off ten to fifteen days after infection.

According to Mulligan, Gordon, Stewart and Wagland (1961) two doses of irradiated larvae given at an interval of one month reduced the infection which resulted from a challenge with 10,000 larvae by 98%. However, Jarret *et al* (1961) were unable to produce such an immunity in younger sheep similarly vaccinated at two to three months of age. They suggested that immunological immaturity, interference from colostrally derived antibodies or immunological unresponsiveness produced by excessive amounts of antigen were responsible for their results.

Dineen and Wagland (1966a) suggested that immature stages of *H. contortus* were more immunogenic than the adults. They produced evidence (Dinnen and Wagland, 1966b) which showed that persistent administration of sensitizing infections were capable of producing a state of immunological exhaustion in sheep. Later, Wagland and Dinnen (1967) demonstrated that in sheep experimentally infected with *H. contortus* and then treated with an anthelminthic, there was an immunologically latent period. After this period, there was a rise and then a fall of immune response. The maximum regeneration of immune response occurred four to eight weeks after removal of the sensitizing infections.

Christie _et al_ (1964) attempted to confer immunity to seven month-old sheep by giving them ten daily doses of 10,000 _H. contortus_ larvae followed by Thiabendazole treatment on the eleventh day. There was evidence of resistance to a challenge infection given on the twenty second day and must have been produced by larval stages removed by the drug. Adams and Beh (1981) assessed the immunity acquired by sheep from a primary infection with _H. contortus_ not terminated by anthelminthic treatment. After challenge, they concluded that primary infection confered significant immunity but in some cases this was not sufficiently protective to prevent the development of clinical haemonchosis. They suggested that an adaptation in the host-parasite relationship which promotes the longevity of primary infection with _H. contortus_ may be operational in the induction of acquired immunity.

These experiments show that sheep, acquire some resistance to _Haemonchus_ infection but the immunity involved appears to be a variable and unreliable phenomenon. The best known phenomenon of acquired immunity to haemonchosis is the 'self-cure' response. The term was first used by Stoll (1928) and describes an abrupt rejection of established adult worms when an intake of infective larvae triggers a hypersensitivity reaction (Stewart 1953, 1955). This response is associated with antigenic substances liberated by living larvae during third ecdysis (Soulsby and Stewart, 1960) and occurs independent of age or experience of infection (Lopez and Urquhart, 1968). The self-cure reaction is not solely any experimental phenomenon and has been found to be an important mechanism for terminating natural gastro-intestinal parasites in sheep.

Other aspects of acquired resistance have been demonstrated more vividly in other helminth infections. Michel (1953) showed that rabbits become resistant to establishment of Trichostrongylus raetortifirmis after prolonged exposure to infection. Foster (1935) as well as Otto and Kerr (1939) gave repeated dose of Ancylostoma caninum to dogs and found that dogs immunized in this way, when given a large dose of larvae and were killed 12-35 days later, harboured fewers worms than did susceptible control dogs. Similarly, Sheldon (1937) showed that fewer Strongyloides ratti became established in rats which had had previous experience of infection. Culbertson (±941) showed that in rats which had been previously infected with Trichinella spiralis, fewer worms became established in the intestine when they were re-infected. Chandler (1935) demonstrated that fewer Nippostrongylus muris were established in rats if these had previously experienced infection and Sarles and Taliaferro (1936) showed that this resistance acted largely against the worms during their migration, a few being destroyed in the skin and many more in the lungs. A similar phenomenon was described by Kerr (1938) whos showed that when guinea pigs which had been infected with *Ascaris lumbricoides* were re-infected many larvae were destroyed in the liver and lungs.

It has also been demonstrated that worms which establish and develop in resistant hosts, do not become as prolific as those in susceptible hosts. This has been shown in infections with many species among which are *T. colubriformis* in sheep (Stewart and Gordon, 1958), *H. placei* in calves (Roberts, 1957) *Ostertagia ostertagi* in calves (Michel, 1963) *T. spiralis* in rats (Semrad and Coors, 1951) and *N. Muris* in rats (Schwartz, Alicata and Lucker 1931). In each case, the phenomenon appears to be a failure of worms which develop late in an infection, to become as fertile as those which developed earlier, rather than the suppression of ovulation in worms which have once been fertile.

Experiments on the experience of infection which is required to produce resistance suggests that it is not only the total number of larvae, the number of occasions in which they are administered or the time over which the infections are administered that determine whether resistance to the establishment of infection or inhibition of ovulation will result. The pattern of dosing schedule is also important, increasing doses being more effective than equally spaced doses and doses increasing at increasing rates being still more effective.

MATERIALS AND METHODS

Experimental Animals

Forty-seven Yankasa sheep aged between six and eighteen months, purchased locally were used for the experiment. The animals were maintained on concrete floor pens under conditions designed to prevent accidental helminth infections. They were not allowed to graze throughout the period of the experiment and were fed hay *ad libitum* and a ration consisting of maize, wheat offal, groundnut cake, bone meal, salt and a vitamin mineral premix (Table 1). They also had access to water and trace mineralised salt blocks.

On acquisition of the animals their helminth status was checked and this showed that all animals were infected with different combinations of Strongyle spp, Coccidia and *Moniezia spp*. Egg counts ranged between 200 to 8,000 eggs per gram of faeces. They were treated with Panacur (Fenbendazole, Hoechst AG) 75 mg per 15kg body weight, Amprol (Amprolium. Meck Sharp and Dohme) 35 mg/kg as well as Terramycin Long acting (Oxtetracycline. Pfizer) 200 mg per 10 kg body weight. All animals showed zero Strongyle egg count at the beginning of the experiment.

Culture of *Haemonchus* infective larvae

For the initial cultures, worms were collected from sheep with natural *Haemonchus* infection slaughtered at a local abattoir. The adult female worms were collected from freshly killed sheep and cleaned of mucus and debris. The eggs were removed from the worms by gently crushing of the worms in a porcelain mortar. The eggs were cultured in helminthologically sterile faeces using 500 ml wide-mouthed jars as described by Roberts and O'Sullivan (1950). The larvae recovered from the cultures were cleaned and given orally to three donor animals. When these began to pass eggs of Haemonchus, the faeces were cultured and the harvested larvae cleaned and suspended in isotonic saline solution.

Experimental Plan

The forty-seven animals were alloted into four groups of twelve animals each, with the control group consisting of eleven animals. Allocation of animals was based on liveweight. Each group was balanced as much as possible with the different weight ranges of animals. Liveweight of the animals ranged from 9-30 kg. Each animal in the groups was to receive the following dose of infective third stage larvae.Group I sheep, 5,000 larvae daily for seven days, a total of 35,000 larvae per animal. Group II sheep, 2,000 larvae daily for seven days to give a total of 14,000 larvae per animal. Group III sheep received 400 larvae daily for seven days, making a total of 2,800 larvae per animal. Group IV was to serve as the uninfected controls.

Infection of experimental animals

The number of vigorously active larvae per ml was estimated by standard dilution counts. The larvae-water suspension was diluted to 200 ml in a graduated cylinder. After proper mixing, 0.1 ml of the suspension was spread evenly on a microscope slide. All the active larvae on the slide were counted and the process repeated several times after which the total number of larvae was summed, and divided by the number of counts made, to find the average. The average number obtained was then multiplied by ten to obtain the number of larvae per ml. From this, the quantity of fluid that would contain the desired number of larvae in each case was calculated.

The larval suspension was administered orally using plastic disposable syringes. The suspension was expelled as far down the oesophagus as possible. This was followed by two washings of distilled water through the syringe to flush down the larvae.

Helminth egg counts

Faecal samples were collected from each animal weekly beginning one week after the last larval innoculation. This was done for five weeks post infection and thereafter every two weeks until the end of the experiment. Faecal helminth egg counts per gram (e.p.g.) were determined using the modified Mcmaster technique (Whitlock, 1948).

Haematological and serological analysis

Blood samples were collected from the jugular vein of each animal weekly, for five weeks and thereafter every two weeks until the end of the experiment. Coagulation was prevented by the addition of disodium salt of ethylene diamine tetracetic acid (EDTA) to each collection bottle before the blood was taken.

Blood smears were prepared immediately from whole blood for white cell differential count. Packed cell volume (PCV) was estimated using a Hawksley[1] microhaematocrit centrifuge. Haemoglobin concentration (Hb conc.) was estimated as cyanmethaemoglobin in a coulter haemoglobinometer[2]. Red blood cell counts (RBC) and white blood cell count (WBC) were made using a Coulter blood cell counter model ZB1. Differential WBC count was based on an evaluation of 200 cells.

To obtain serum, blood samples were collected in plastic tubes to minimise glass contamination with iron. The blood was allowed to clot at room temperature, centrifuged at 2,500 g for 15 minutes, the serum aspirated and stored at -20°C untill analysed.

Sodium and Potassium concentration in serum were estimated in a Flame photometer[3].

Serum iron

Serum iron was estimated colorimetrically according to the method recommended by the Iron Panel of the International Committee for Standardization in Haematology (Bothwell et al 1979).

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(1) Microhaematocrit centrifuge. Hawksley, England.

(2) Coulter Electronics Ltd. Harpendon, England.

(3) Evans Electroselenium Ltd. Halstead, England.

Precautions were taken to prevent glass contamination of samples for serum iron analysis by collecting the serum in plastic tubes. To make glassware used iron free, they were first washed in detergent, soaked overnight in 50% nitric acid and then rinsed in iron free water. The procedure for iron determination was as follows: one ml of serum was pipetted into an iron-free test tube and one ml of protein precipitant (10% Trichloroacetic acid, one ml hydrochloric acid and 3% thioglycolic acid) solution added. This was then mixed thoroughly in a vortex mixer and allowed to stand for five minutes. It was then centrifuged at 1500g for 20 minutes. One ml of the optically clear supernatant was transfered to a separate iron-free test tube. One ml of a chromogen solution (Ferrozine) was added mixed well, and allowed to stand for five minutes. The optical density of the solution was then measured in a spectrophotometer at 562 nm wavelength. The same procedure was carried out for the working iron standard solution and a reagent blank solution. Serum iron concentration (u/m1) was calculated as follows:

$$\frac{\text{Optical Density (O.D) unknwon serum} - \text{O.D. blank}}{\text{O.D. Iron standard} - \text{O.D. blank}} \times 35.8$$

Serum protein determination

Serum total protein was determined by the Biuret method (Coles, 1968). Electrophoresis of all serum samples was carried out according to the procedure outlined for a micro-zone electrophoresis system (Anon,1975). The Albumin, Alpha-1 Alpha-2,Beta and Gamma globulin fractions were run on cellulose acetate strips using Beckman B-2 buffer at PH 8.6 and μ = 0.075. The electrophoretic strips were fed into a Beckman RI 10 densitometer and estimates of each protein fraction obtained based on the total serum protein obtained.

Worm recovery

All animals that died were necropsied and the worms present in them recovered and counted. The rest of the animals were slaughtered at the end of the experiment to recover the helminths in the abomasum. The abomasum was ligatured at both ends and removed. It was then slit along the greater curvature over a 0.42 nm sieve and the contents washed in a fine spray of water. The mucosa was scrubbed thoroughly to remove any adhering worms, after which it was scraped with an object slide and digested overnight in 1% Pepsin-hydrochloric acid solution. The digesting process was stopped and the samples fixed in 10% formalin.

Immature stages were searched for under the dissecting microscope while adult Haemonchus were counted using a Picker x-ray lamp. In each case, the abomasal contents or digest was made up to three liters with water. This was agitated vigorously and three samples of 30 ml were withdrawn. Small quantities of this were then placed in a glass petri dish having parallel lines marked on it and the number of worms present counted. The total number of worms counted in the 30 ml samples was multiplied by 100 to give the total number of worms present. Where worm burdens were less than 100, all were counted individually.

Statistical Analysis

Data were analysed using a statistical package (SPSS*) comprising an analysis of variance (ANOVA), and Duncan's multiple range test. A simple correlation analysis (Steel and Torrie, 1960) was done to check the relationship between different parameters.

* As developed by Vogelback Computing Centre, North Western University. April, 1976.

Table 1: Percentage composition of concentrate fed to the animals which were used in the experiment

INGREDIENTS	%
Maize	63.4
Wheat offal	25
Groundnut cake	5
Dry brewers grain	5
Bone meal	1.5
Vitamin mineral Premix	0.1
Total	100
Crude Protein	12.7
Total Digestible Nitrogen	71.7

RESULTS

Results obtained are presented in Figures I to 18 and Table 2. Raw data on the different groups are presented in appendices I to IV.

Mortality

Mortality in the experimental animals was complicated by an undiagnosed cause which set in during the first week of infection. In Group I, (35,000 larvae) seven animals died within the first two weeks of the infection. In Group II, (14,000 larvae) five animals died before the end of the experiment and four of these deaths were within the first four weeks of the infection. Group III had the highest mortality with eight animals dead. Six of these deaths occured between the third and eleventh weeks of infection. In Group IV, six animals died before the end of the experiment but only one of these occured within the first two weeks of the experiment, while the rest died during the last six weeks of the experiment.

Animals that died within the first four weeks of the experiment were mostly young animals between 6 - 10 months of age which had body weights of 10 - 15kg at the beginning of the experiment. Number of worms recovered from these animals (Tables 22 - 24) ranged from 19 to 1205. Older animals among all the groups responded to prompt antibiotic treatment and no other deaths took place except at the last six weeks of the experiment when control animals also suffered some mortalities.

Helminth Egg Production

Group mean helminth egg counts are summarized in Figure I. Prepatency as used in this experiment, is the time between initial innoculation to initial detection of parasite eggs in the faeces of the animals. Prepatent periods ranged from 14 days in Groups II and III animals to 22 days in Group I. One animal each from Groups II and III had positive egg counts by day 14 and by day 22 all animals in these two groups were positive for Haemonchus eggs. On the other hand, no animal in Group I was positive until day 22. Uptill the fifth week of infection, two animals in Group I had no helminth eggs in their faeces but all animals were positive by the seventh week.

There was no significant difference between the total mean egg counts of all infected groups but Group III animals had the highest mean egg count (e.p.g.) (1172 e.p.g.) as compared with 626 and 982 e.p.g. for Group I and II respectively. It is important to note that Group I had the highest egg count by the second week of the experiment but this dropped by 92% by the third week, and did not pick up again till the seventh week. Group III recorded the highest mean egg count of all the infected groups and this was on the seventh week of infection. Groups I and III reached peak egg outputs by week seven while Group II had its peak egg output on week nine.

Haematological Values

Figures 2 to 9 summarize data obtained for haematological values of the experimental animals. The different levels of infective Haemonchus larvae did not have distinctive effects on the hemogram of infected animals. The changes that occured had virtually the same pattern in all animals irrespective of amount of larvae given.

Packed Cell Volume

Packed cell volume (PCV) values are shown in Figure 2. Groups I and IV had total mean PCV ~~values~~ significantly higher ($P<0.05$) than that of Group II but not from the other infected groups. There was a significant drop of PCV values in Group II animals on the third week of infection and a decrease in all groups on the seventh and fifteenth weeks of infection. By week 21, PCV values for Group I had dropped by 29%, Group II by 32%, Group III by 31% and Group IV by 18%. There was a negative correlation between PCV and egg count ($r = -.29$) and this was very significant.

Haemoglobin

Haemoglobin values are shown in Figure 3. Group IV had total mean haemoglobin values significantly higher ($P<0.05$) than those of all the infected groups. There were fluctuations in haemoglobin values of all groups but by week 21, values for Group IV had declined by 17%, while Groups I, II and III declined by 31, 33 and 34% of pre-infection levels respectively. There was a negative correlation between haemoglobin concentration and gamma globulin and this was very significant ($P<0.01$)

Erythrocyte counts

Figure 4 shows the changes in erythrocyte count (RBC) in the experimental animals. Group II showed a significant decrease in erythrocyte counts by the fourth week of infection but marked changes were not observed in all other groups until week seven. Erythrocyte counts also showed marked fluctuations between the eleventh and seventeenth weeks of infection but this occurred in all groups. By the end of the experiment, total mean values (± standard deviation) for the respective groups were 6.7 ± 0.8, 5.6 ± 0.9, 6.3 ± 0.7 and 7.1 ± 0.2 RBC x 10^6/µl for Groups I, II, III and IV and these represented decreases of 30, 33, 30 and 17% respectively from original values.

Leucocytes

Infection with Haemonchus spp did not markedly affect the total leucocyte values (WBC) of the infected groups as far as was indicated by the averages of their counts when compared with the controls. Total leucocyte counts of all groups fluctuated irregularly showing a significant difference between the groups only on the seventeenth week of infection. Differential counts indicated that Group II had total mean eosinophil counts significantly higher ($P<0.05$) (5.6 ± 4.4) than Groups I, II, and IV which had values of 4.3 ± 2.4, 4.4 ± 4.5 and 3.9 ± 2.4 percent respectively. Groups I and II had significantly higher eosinophil counts than Groups II and IV, on the third and fifteenth weeks of infection and all the infected groups showed significantly higher eosinophil counts than the controls by the nineteenth week of infection. Eosinophil counts showed different patterns in the various groups (Figure 7). In Group I, it increased by the third week of infection, showed the highest value by week thirteen and thereafter decreased. In Group II, eosinophil counts increased by the second week of infection, reached a peak by week nine and never returned to pre-infection levels. In Group II eosinophil counts did not increase until the fourth week of inection and remained high until the fifteenth week of the experiment; when it feel abruptly from 8.3 to 1.0%. Group IV animals did not show any significant changes all through and the highest value recorded for the group represented only a two fold increase over pre-infection values.

Groups II and III had total mean lymphocyte values significantly lower ($P<0.05$) than that of Group I but not significantly different from Group IV; values being 33.8 ± 11.0, 33.6 ± 10.7, 37.7 ± 11.8 and 36.2 ± 11.5 percent respectively in the order the groups are mentioned. Lymphocyte values of all infected groups dropped on the first week of infection but all recovered by the fifth week. Lymphocyte values of all groups decreased on the seventh week of infection and in Groups II, III and IV this decrease was followed by increase to normal levels by the end of the experiment while Group I showed progressive increases which preceeded pre-infection levels by the seventeenth week of infection. There was significant difference in lymphocyte values between groups only on the eleventh week of infection.

There was a negative correlation between lymphocytes and total leucocytes and this was significant ($P<0.05$).

Neutrophil values are shown in Figure 6. Group III had Neutrophil values significantly higher ($P<0.05$) (59.7 ± 9.7) than values for the control 56.4 ± 12.9 percent, but not significantly different from values of the other infected groups. Neutrophil values of all infected animals rose on the first week of infection and at week 21, values for Groups I and III exceeded pre-infection levels. There was a very significant negative correlation ($r = -.82$, $P<.001$) between Neutrophils and lymphocytes.

There was no significant differences between Monocyte values of infected and control groups. All groups fluctuated irregularly with no discernible pattern and showed marked decreases at weeks 17 to 21 with Group II having no monocyte counts by week 21. Monocyte counts were highly correlated with total leucocyte counts.

Serum Analysis

Serum iron

Serum iron values for Group IV were significantly higher ($P<0.05$) than those of the infected groups, but there was no significant difference between any of the infected groups. Values were 21.7 ± 2.9, 21.3 ± 3.6, 22.1 ± 3.8 and 23.0 ± 2.9 for Groups I, II, III and IV respectively. Serum iron values showed a decline in all groups on week seven but this was not significant in any group. Values for Group II dropped significantly lower than that of the control on weeks four and 21 while those of Group III decreased significantly below those of the controls on weeks 11 and 17. By the end of the experiment, serum iron values for Group I had decreased by 24% of pre-infection levels and Groups II to IV had decreased by 31, 29 and 13% respectively.

Serum protein

Total serum protein did not show marked changes in both infected and control animals but the controls had significantly higher overall mean Total Protein values than the infected groups. There were significant differences between the groups on weeks 4, 11, 17 and 21 but this was not consistent for any group. (Figure II)

Fractionated serum protein values showed that Albumin was significantly lower ($P<0.05$) in Group I than in all ther groups. Percentage Albumin values showed wide fluctuations in all groups with Group I showing the widest fluctuations. Albumin values in Group I decreased significantly to values lower than the controls on week five and lower than any other group by week 21. Group I also showed a progressive decrease as from the second to fifth weeks of infection but picked up thereafter until week fifteen when values of all other groups also showed significant decreases. There was a very significant negative correlation between Albumin and all the globulins and a significant but positive correlation between Albumin and haematological values. There was also a negative correlation between Albumin and egg counts but this was not significant.

Alpha I globulin

Alpha I globulin values are shown in Figure 13. Group III had total mean alpha-1 globulin values significantly higher ($P<0.05$) than those of all other groups, values being 4.3 ± 1.9, 4.3 ± 1.9, 4.8 ± 2.0 and 4.1 ± 1.2 percent for Groups I to IV respectively. Significant differences were seen between the groups only on weeks three and nine. Values for Group I and IV were significantly lower than those of Groups II and III on week three. Values for the control were also significantly lower than those of all infected groups on the ninth week of infection.

Alpha-2 globulin

Total mean percentage values of 12.1 ± 3.2, 11.6 ± 2.5, and 11.3 ± 2.0 were recorded for Groups I to IV respectively. Values for Groups I and III were significantly higher than those of Groups II and IV. There was a drop in alpha-2 globulin values for all groups on the second week of the experiment but this was followed by immediate recovery by the following week. Thereafter, only the infected groups showed marked increases on the ninth and fifteenth weeks of infection followed by a decrease which in Group I was significant from all other groups.

Beta globulin

Groups I and III had Beta globulin values significantly higher ($P<0.05$) than those of Groups II and IV. (Figure 5). Total mean percentage values were 7.8 ± 2.3 for group I and 6.9 ± 2.2, 7.7 ± 2.6 and 7.8 ± 2.3 for Groups II, II and IV respectively. The infected groups showed marked changes from the controls only on the first and third weeks of infection when values for Groups I and III were significantly higher than that of the controls.

Gamma globulin

Gamma globulin percentages are shown in Figure 16. Groups I and II had total mean Gamma globulin values higher than Groups III and IV, these values being 28.0 ± 7.2, 27.1 ± 4.4, 25.1 ± 4.7 and 25.6 ± 4.7 percent respectively. In Group I, Gamma globulin values increased markedly on the second week of infection and remained high until the end of the experiment, the highest values being recorded in this group on the fifth and fifteenth weeks of infection. For Group II, significant increases in Gamma globulin occured on weeks 15 and 21 while for Group III the increases were on the fifth and twenty first weeks of infection. Group IV did not show any significant increases but showed decreases from their initial values. Highest increases in gamma globulin values of infected group I, II, III represented a

41, 27 and 24% change from the pre-infection values respectively. Gamma globulin showed a negative correlation with all other parameters except Egg count, Total Protein and Sodium.

Serum electrolytes

Serum sodium values are shown in Figure 17. The controls had significantly lower sodium levels than Groups I and III but not from Group II. Total mean values obtained were 136.9 ± 15.2, 130.9 ± 14.4, 142.8 ± 10.1 and 128.0 ± 17.8 um/1 for Groups I, II, III and IV respectively. Serum sodium values for the infected groups were consistently higher than those of the control at the various sampling periods.

Serum Potassium values are shown in Figure 18. The control group had lower total mean Potassium values than any of the infected groups. Overall serum Potassium values were 4.5 ± 0.6, 4.6 ± 0.5, 4.8 ± 0.6 and 4.4 ± 0.8 um/1 for groups I to IV respectively. The control group also showed serum Potassium values that were consistently lower than those of the infected groups during the first seven weeks of infection, thereafter the changes were not significant. There was a significant ($P<0.01$) negative correlation ($r = -.21$) between Potassium and Egg count.

Worm Recovery

Summary of worms recovered at necropsy is presented in Table 2. A total of 4,285 Haemonchus adults were recovered from Group I animals with a mean worm burden of 357 per animal and a range of 22 to 1205 worms. Patent infection established was 1%. For Group II, a total of 2103 adult worms were recovered, with a mean worm burden of 175, range of 30 - 750 and a patent infection of 1.3%. For Group III, a total of 535 worms were recovered, with a mean worm burden of 48 per animal and a patent infection of 1.7%. No parasite was recovered from one animal in Group III. No parasite was recovered from Group IV and no larvae was found in any group.

TABLE 2: Mean worm recovery from the Four experimental groups of sheep infected with varying levels of <u>Haemonchus</u> larvae

GROUP	MEAN NO OF L3 GIVEN	MEAN WORM BURDEN	PATENT INFECTION (PERCENT)
I	35,000	357	1.01
II	14,000	175	1.25
III	2,800	44	1.70
IV	0	0	0

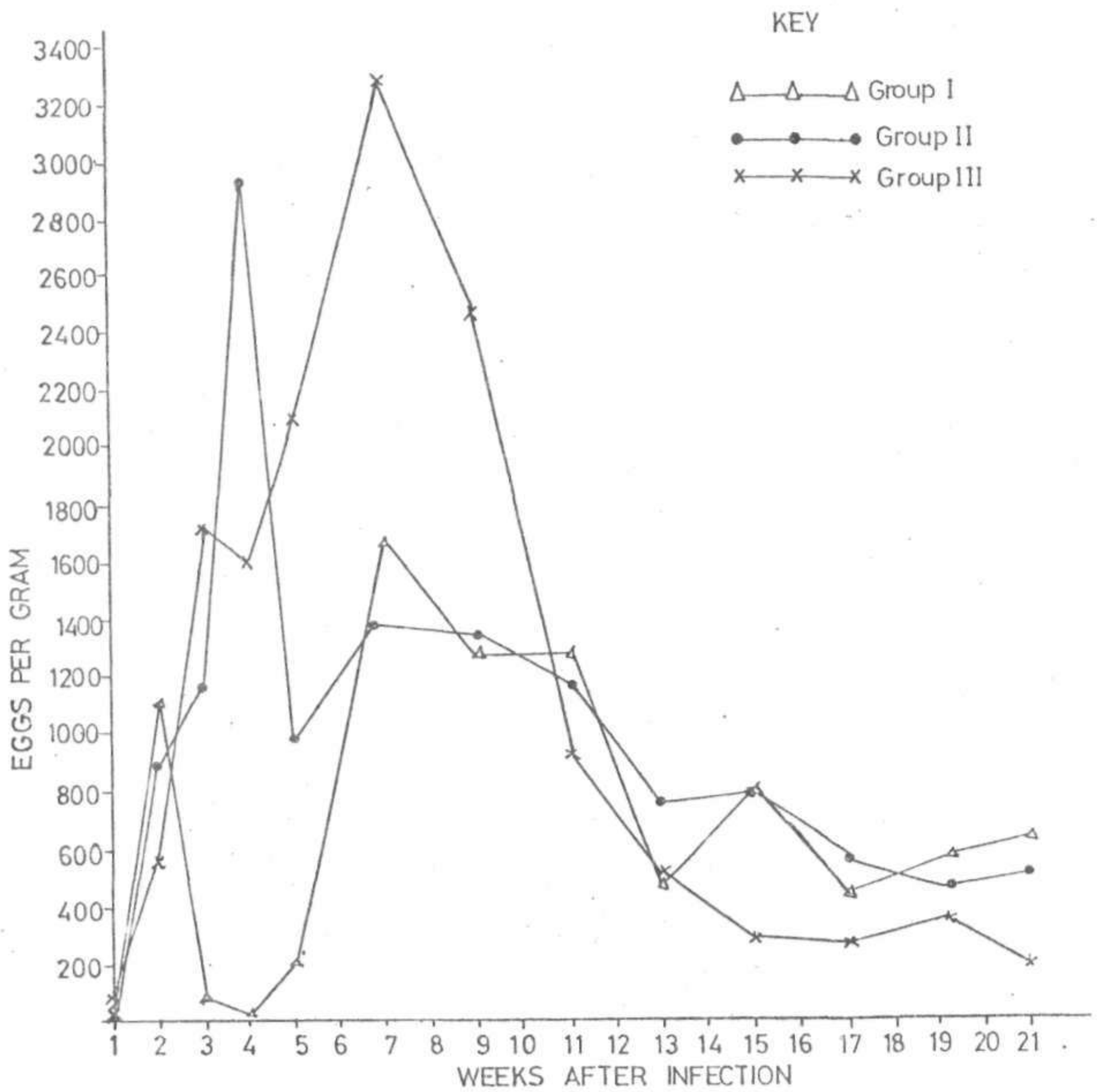

Fig I
Group mean faecal egg count of sheep infected with varying levels of *Haemonchus* larvae

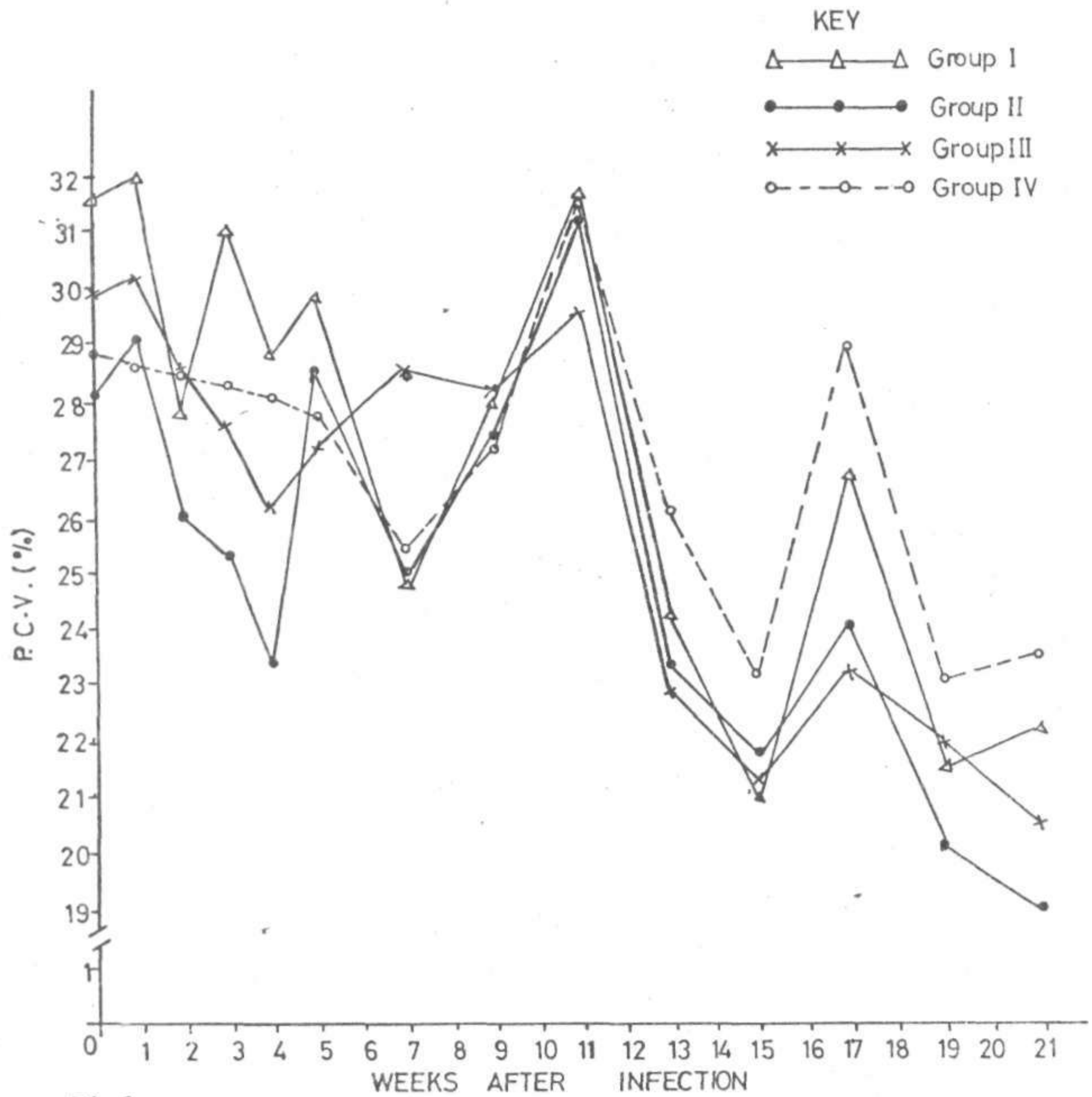

Fig 2
Group mean packed cell volume (PCV) of sheep infected with varying levels of *Haemonchus* larvae

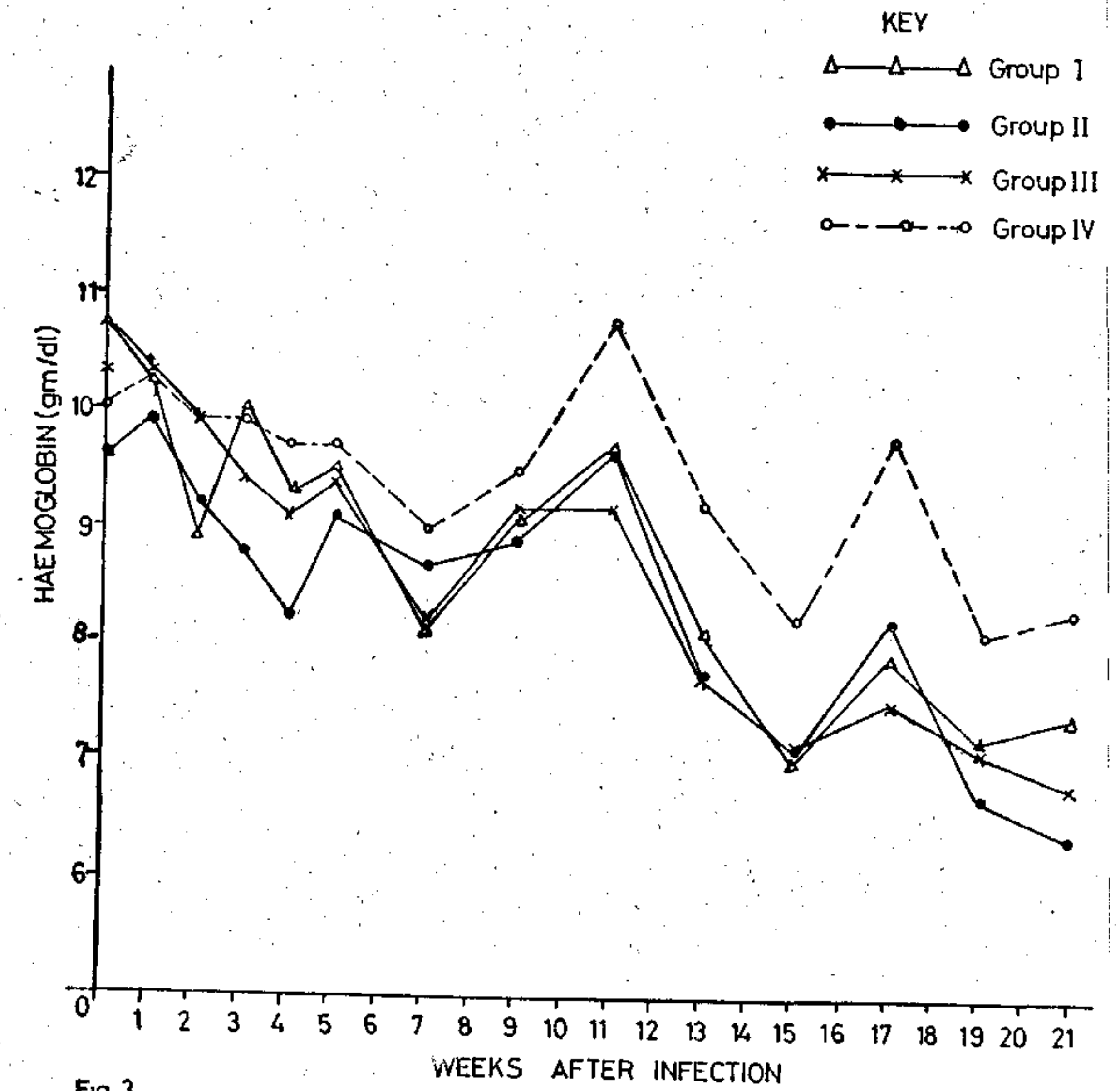

Fig 3.
Group mean Haemoglobin concentration of sheep infected with varying levels of <u>Haemonchus</u> larvae

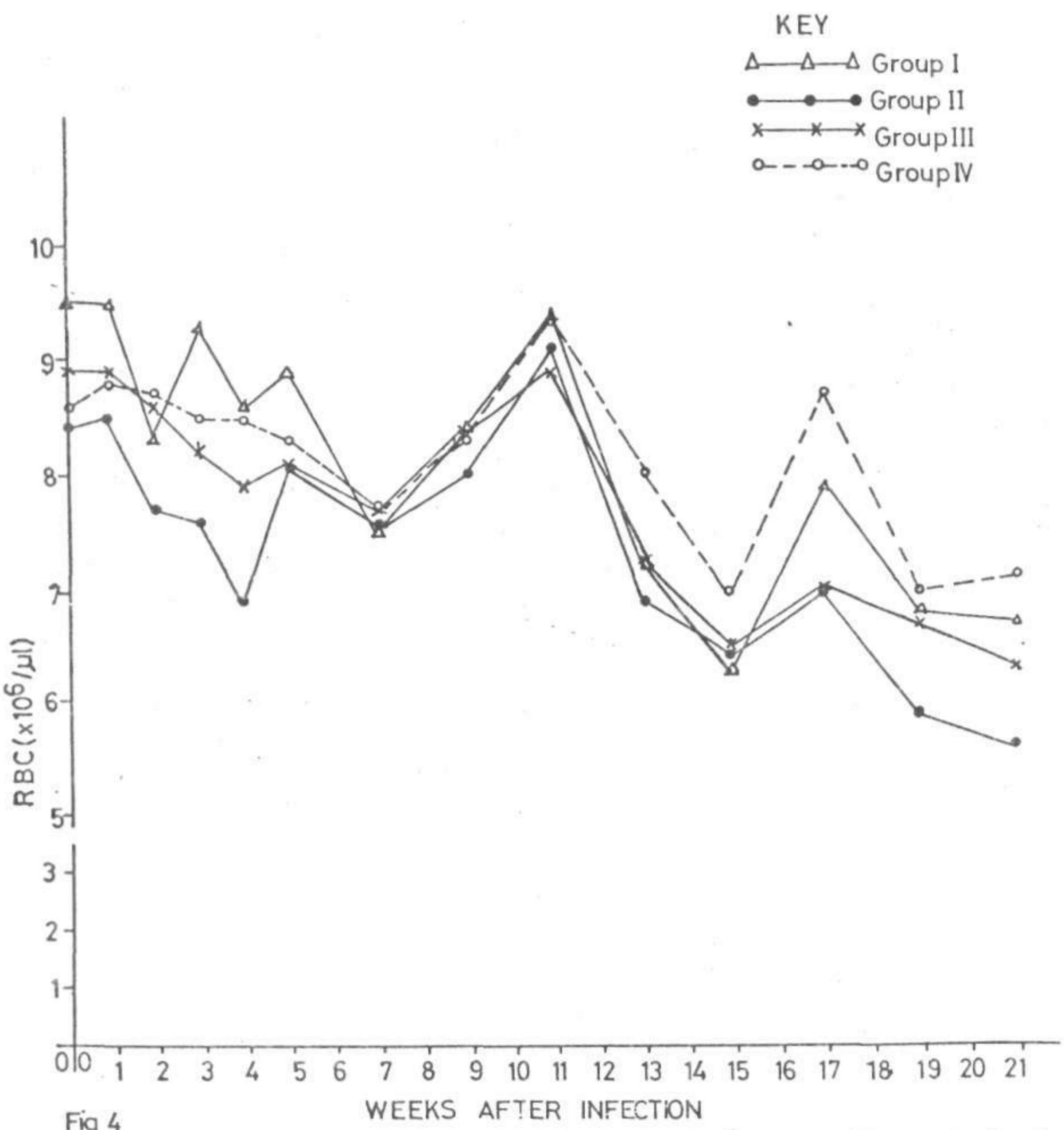

Fig 4
Group mean Red Blood Cell (RBC) counts of sheep infected with varying levels of Haemonchus spp larvae

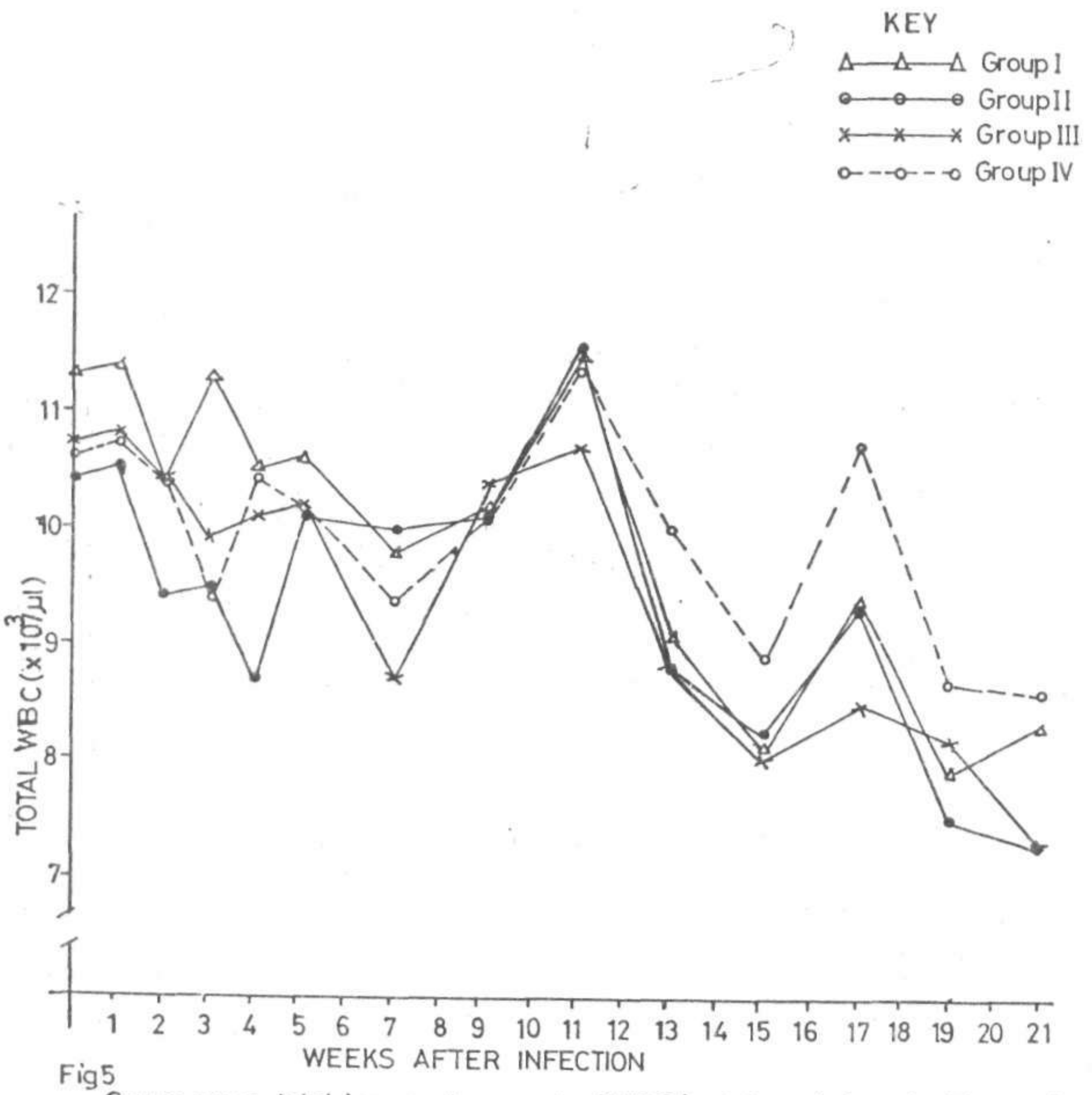

Fig 5
Group mean total Leucocyte counts (WBC) of sheep infected with varying levels of Haemonchus larvae

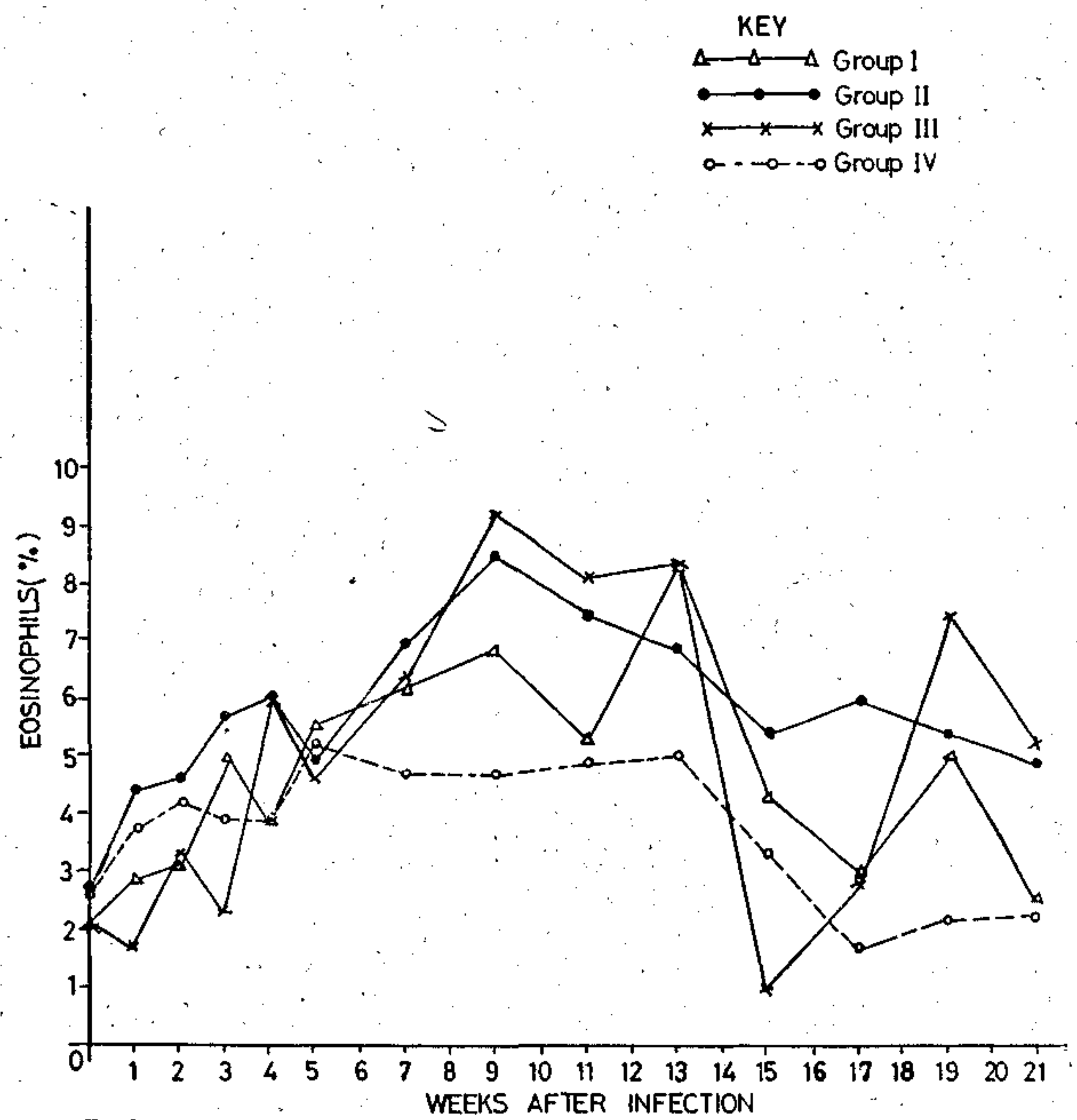

Fig 6
Group mean Eosinophil values of sheep infected with varying levels of Haemonchus larvae.

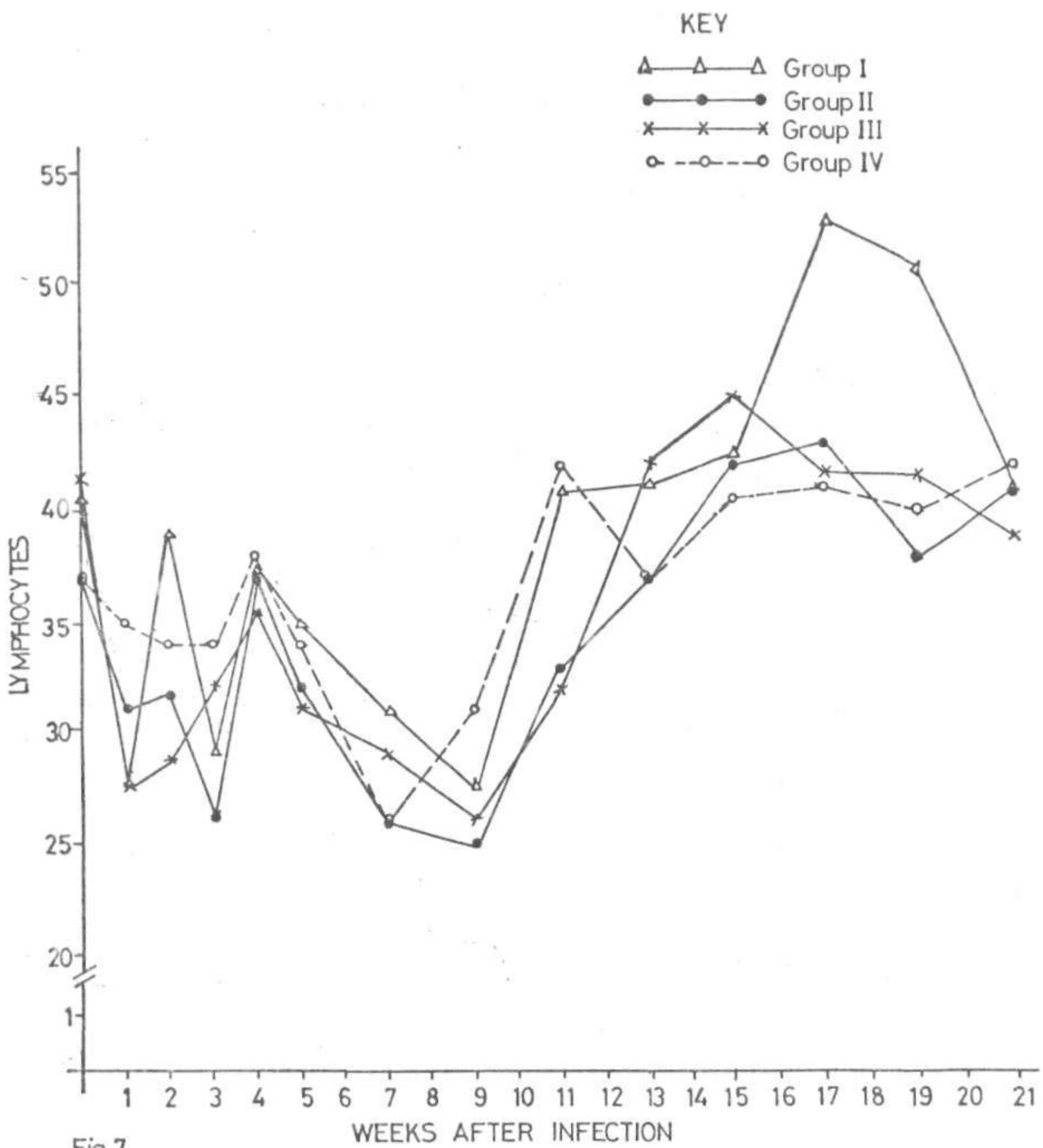

Fig 7
Group mean Lymphocyte values of sheep infected with varying levels of <u>Haemonchus</u> larvae

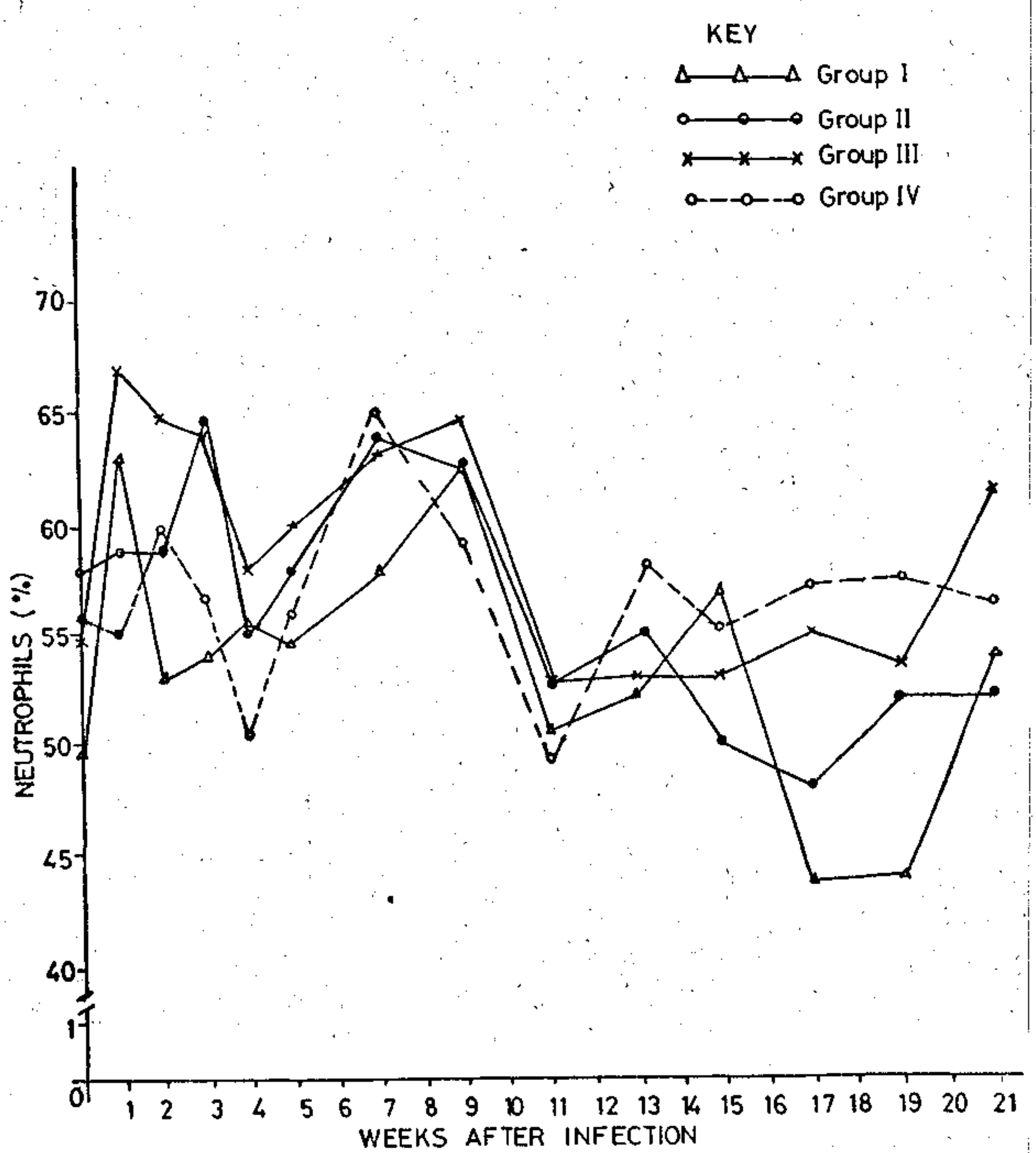

Fig 8
Group mean Neutrophil values of sheep infected with varying levels of <u>Haemonchus</u> larvae

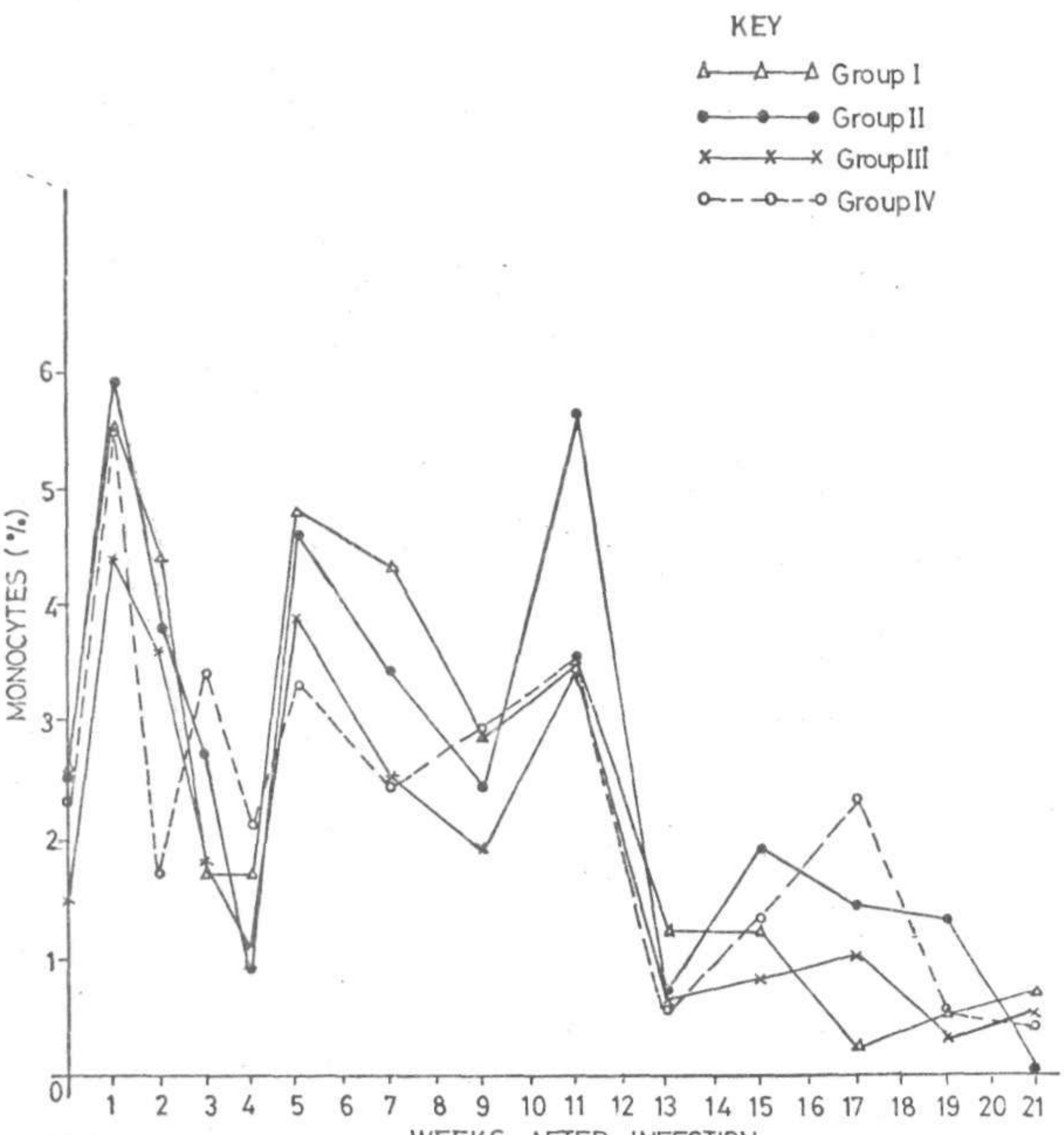

Fig 9
Group mean monocyte values of sheep infected with varying levels of
Haemonchus larvae

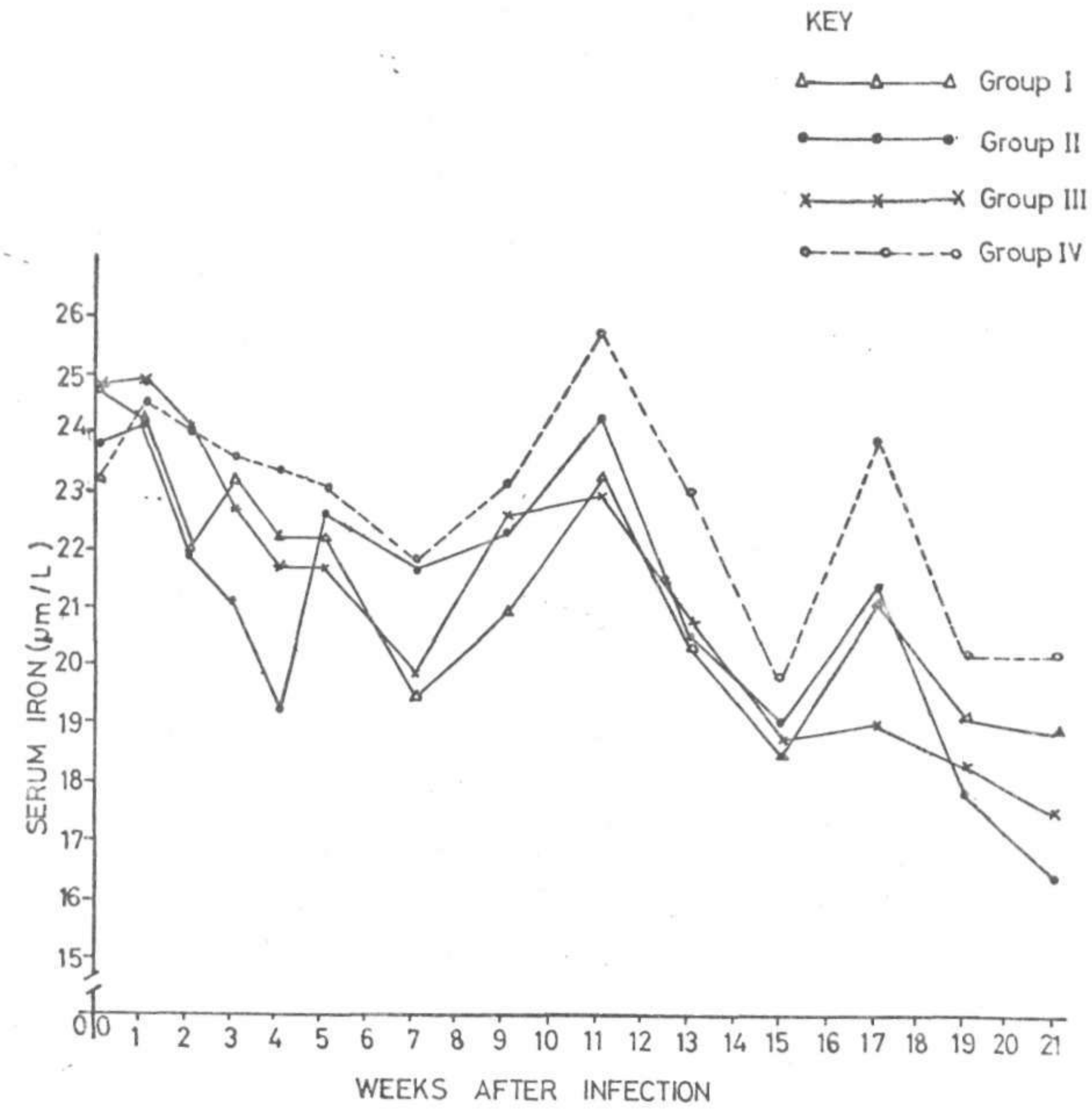

Fig. 10

Group mean serum iron concentration of sheep infected with varying levels of Haemonchus larvae

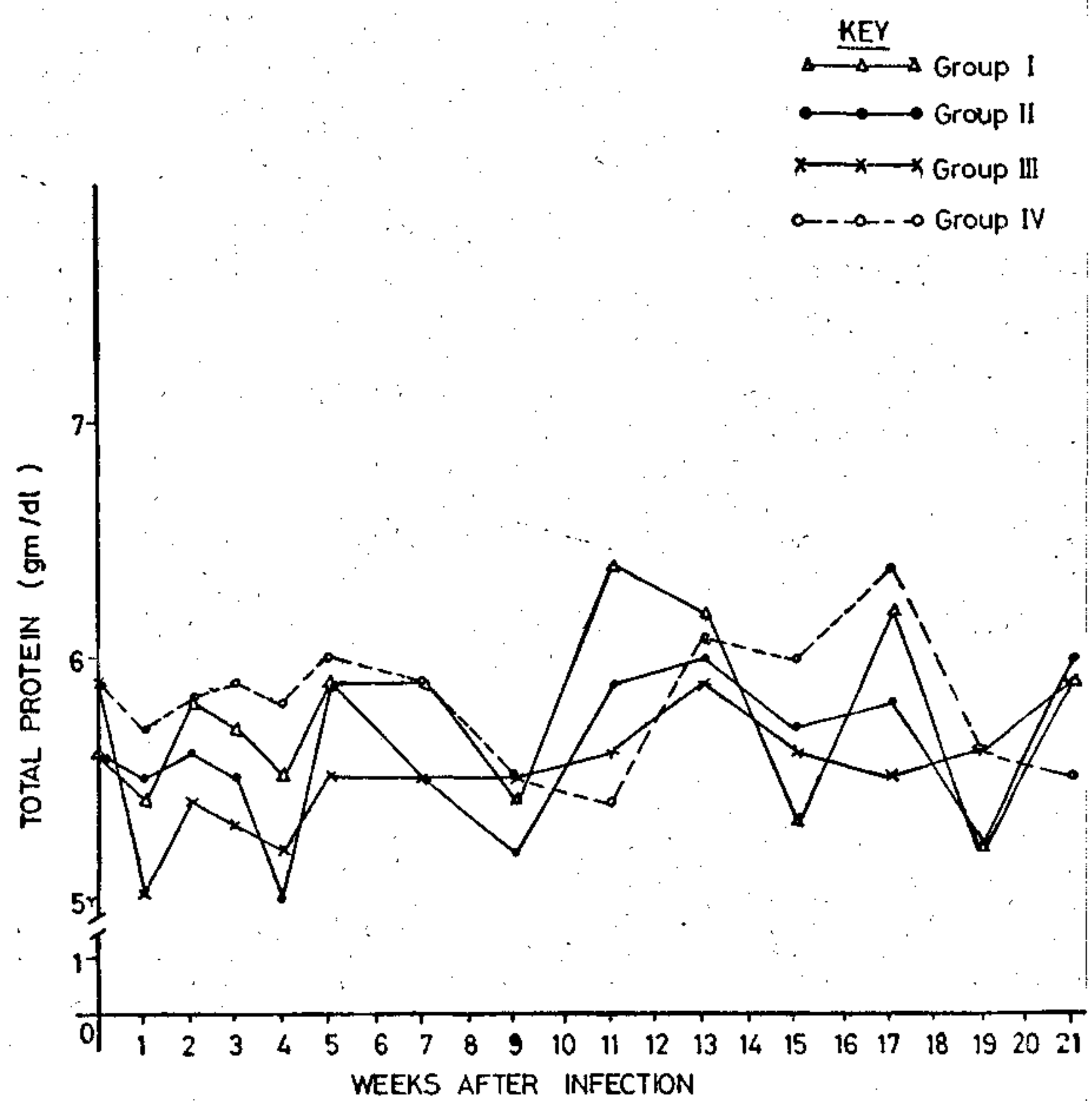

Fig.11 Mean serum protein concentration of sheep infected with varying levels of Haemonchus larvae

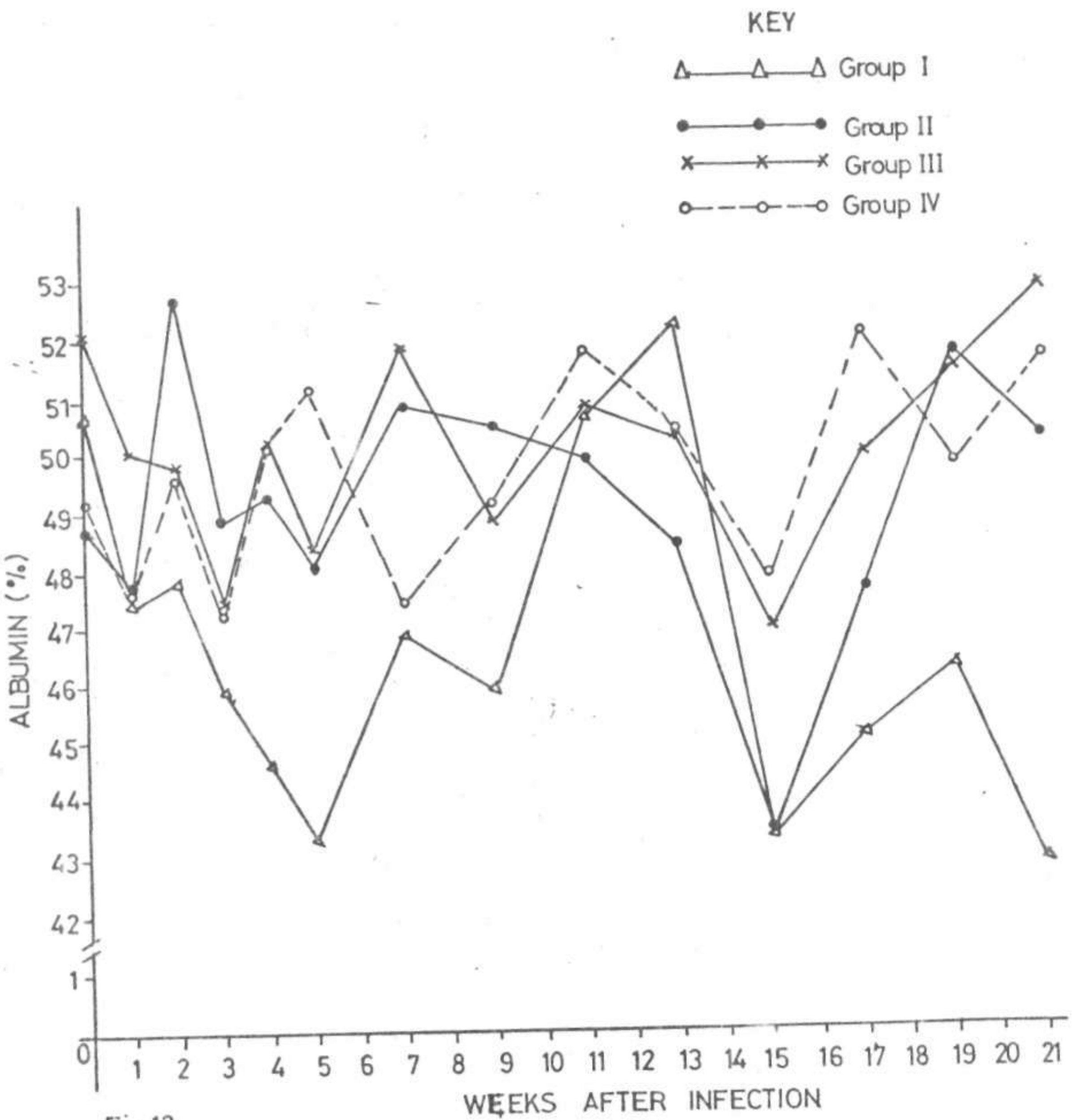

Fig 12
Group mean albumin concentration of sheep infected with varying levels *Haemonchus* larvae

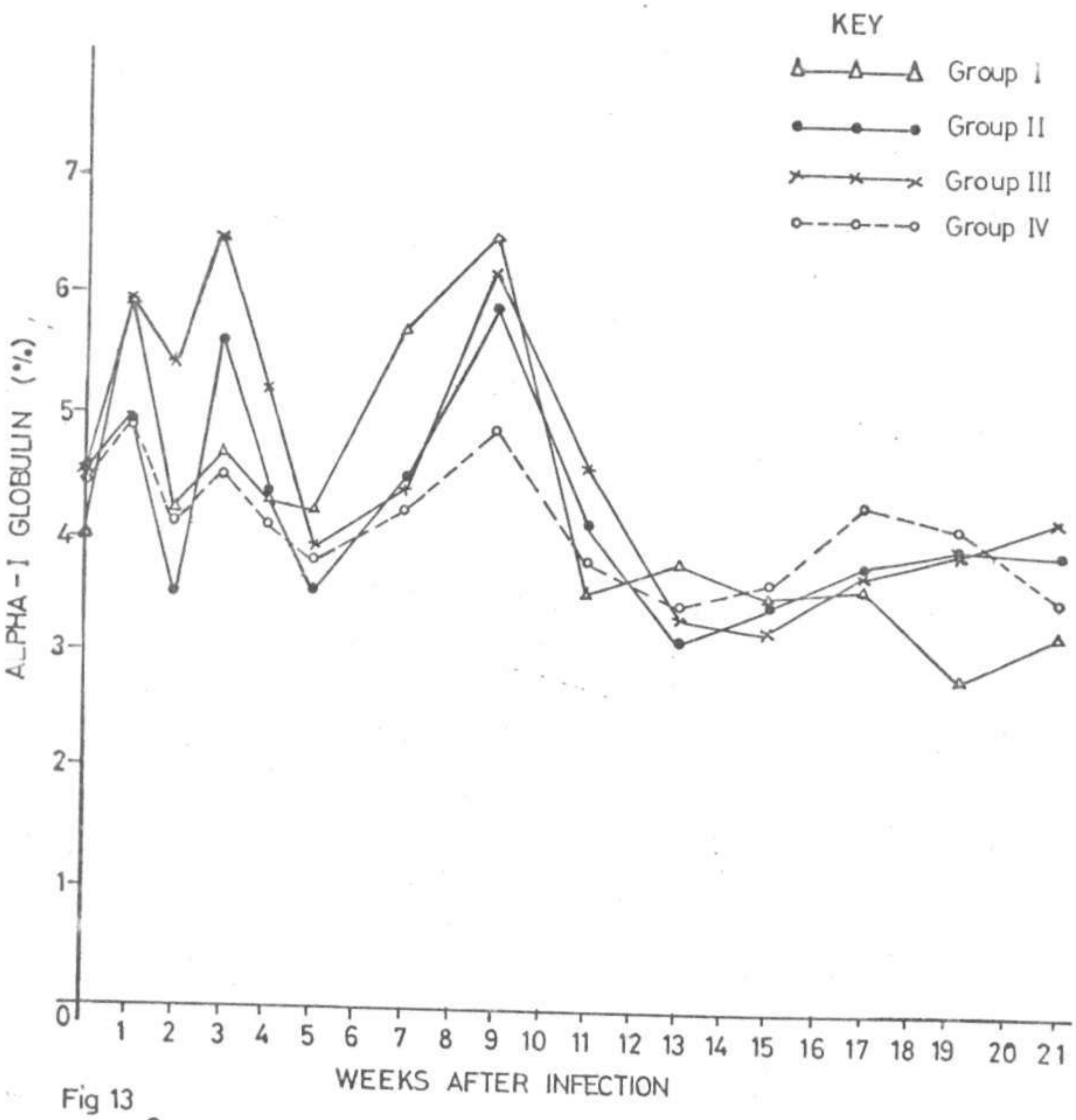

Fig 13

Group mean Alpha-I globulin concentration of sheep infected with varying levels of Haemonchus larvae

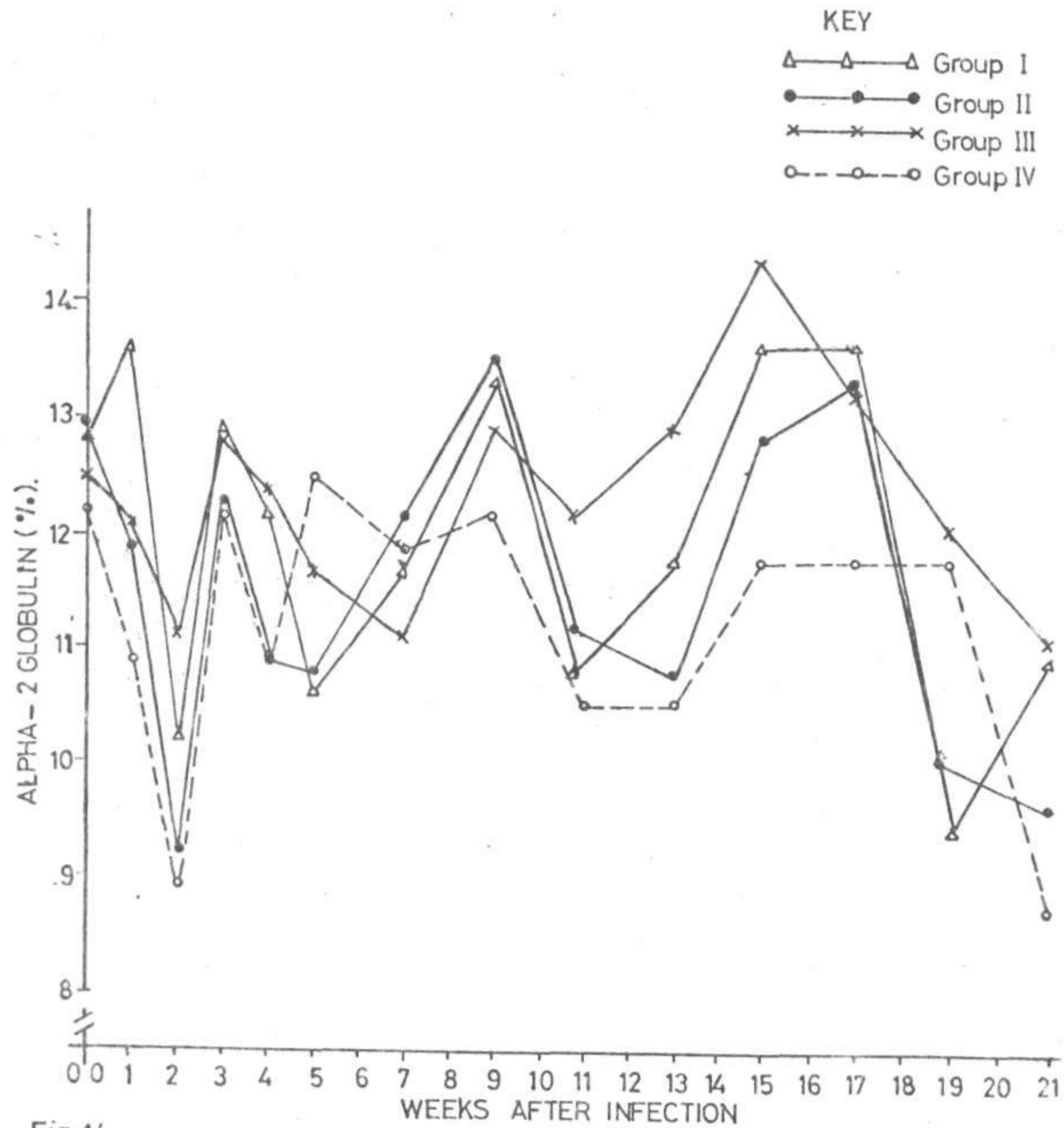

Fig. 14
Group mean Alpha-2 globulin concentration of sheep infected with varying levels of Haemonchus larvae

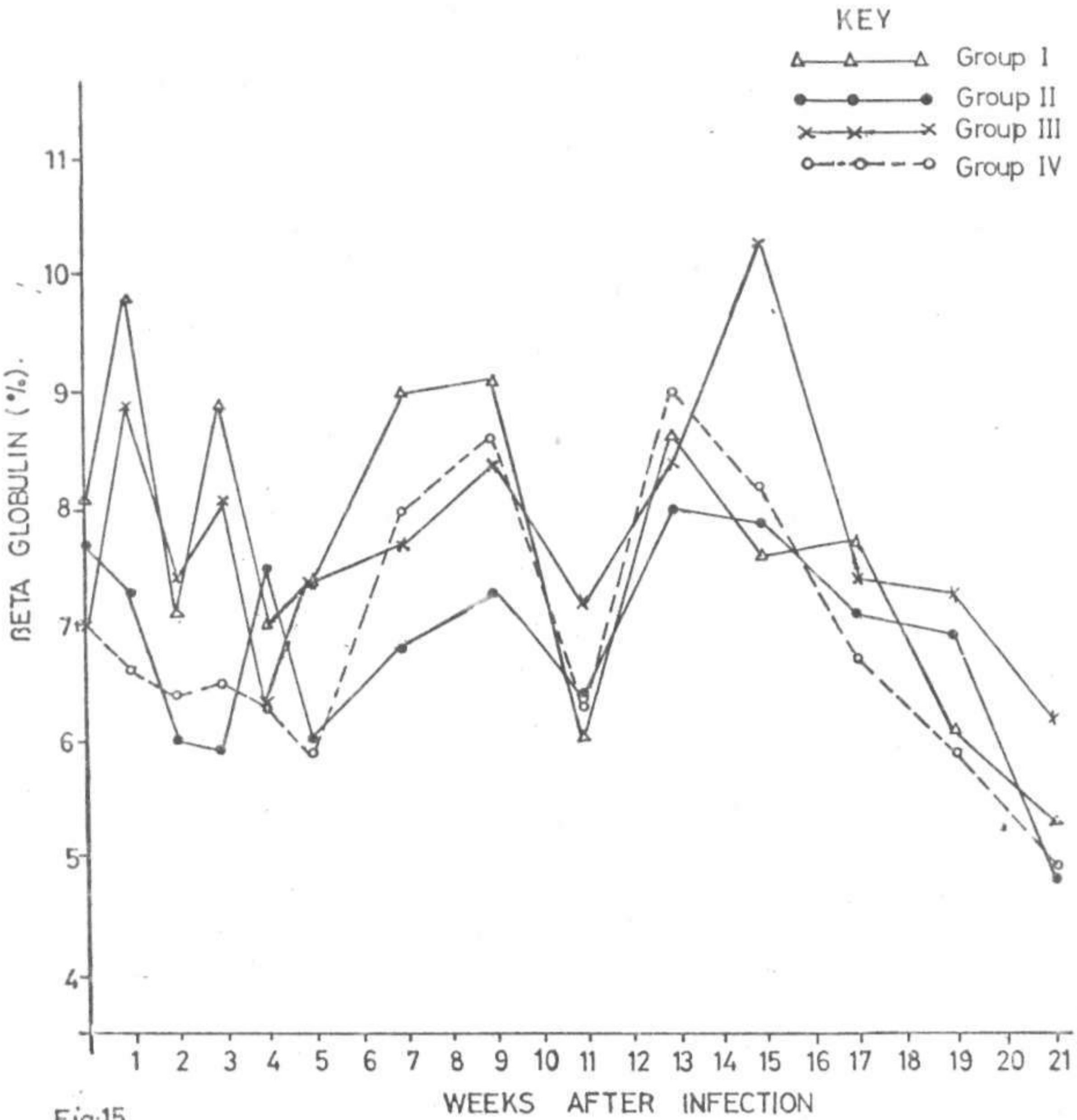

Fig 15
Group mean Beta globulin concentration of sheep infected with varying levels *Haemonchus* larvae

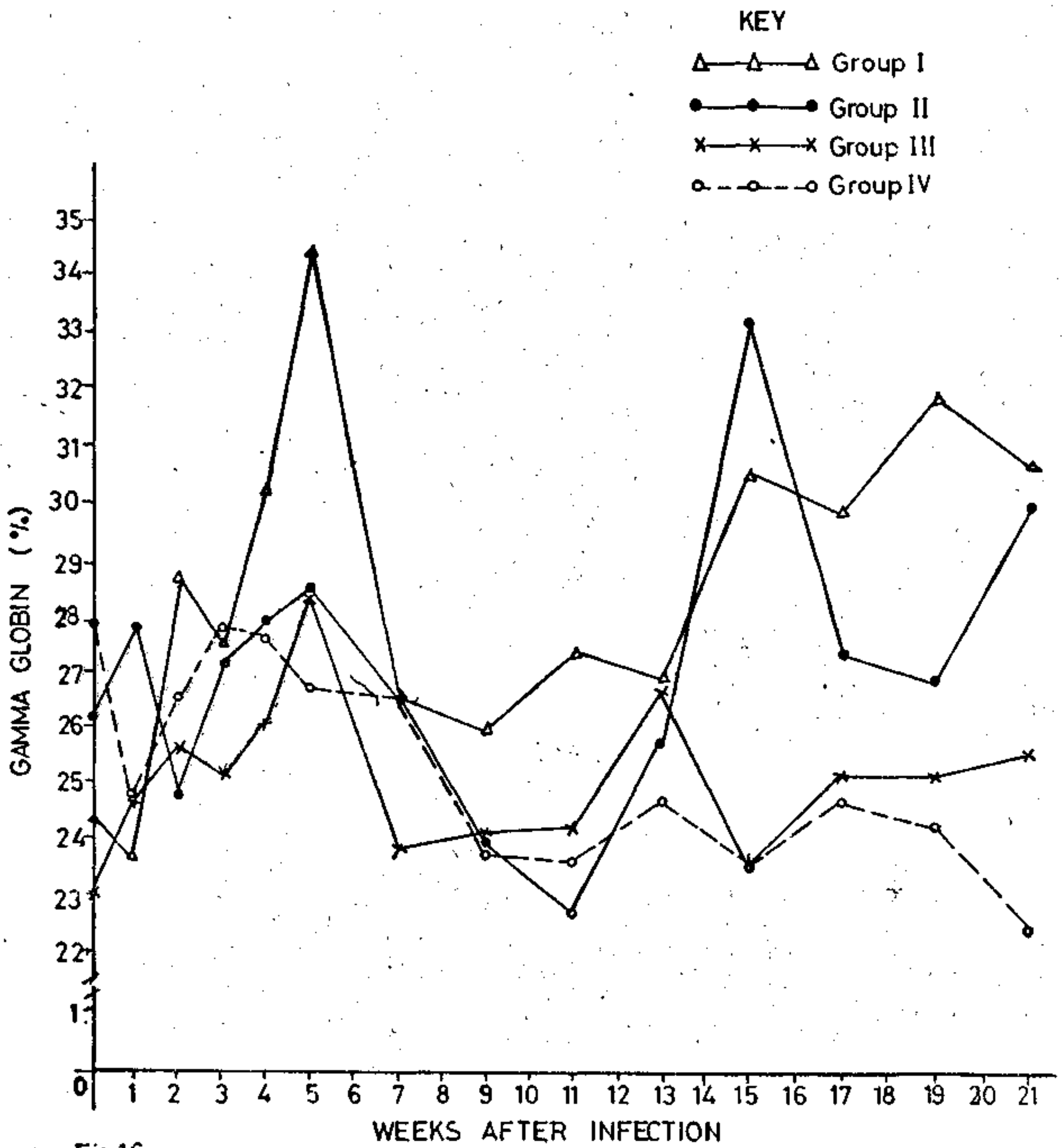

Fig.16

Group Gamma globulin concentration of sheep infected with varying levels of Haemonchus larvae

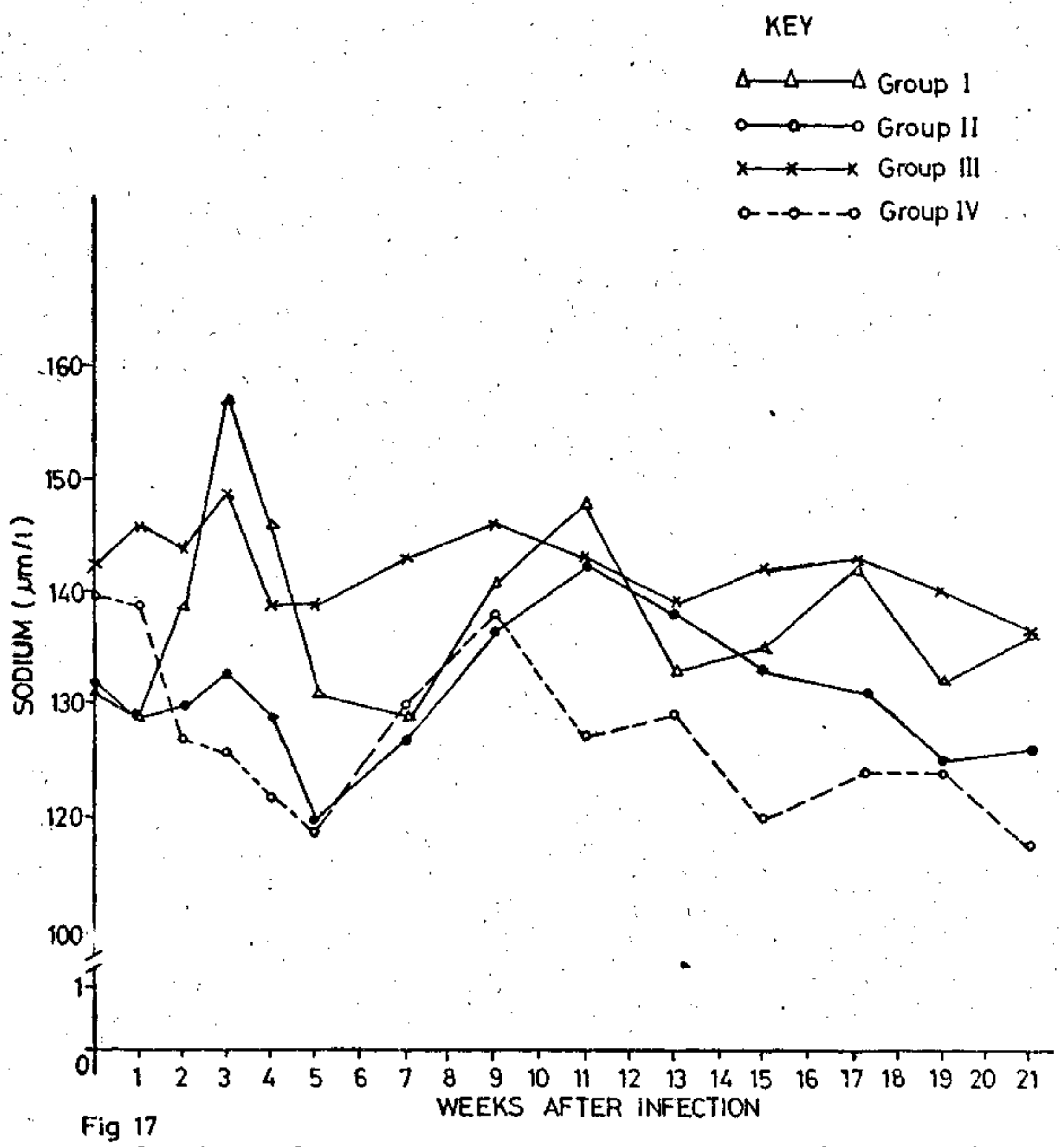

Fig 17

Group mean Sodium concentration of sheep infected with varying levels of Haemonchus larvae

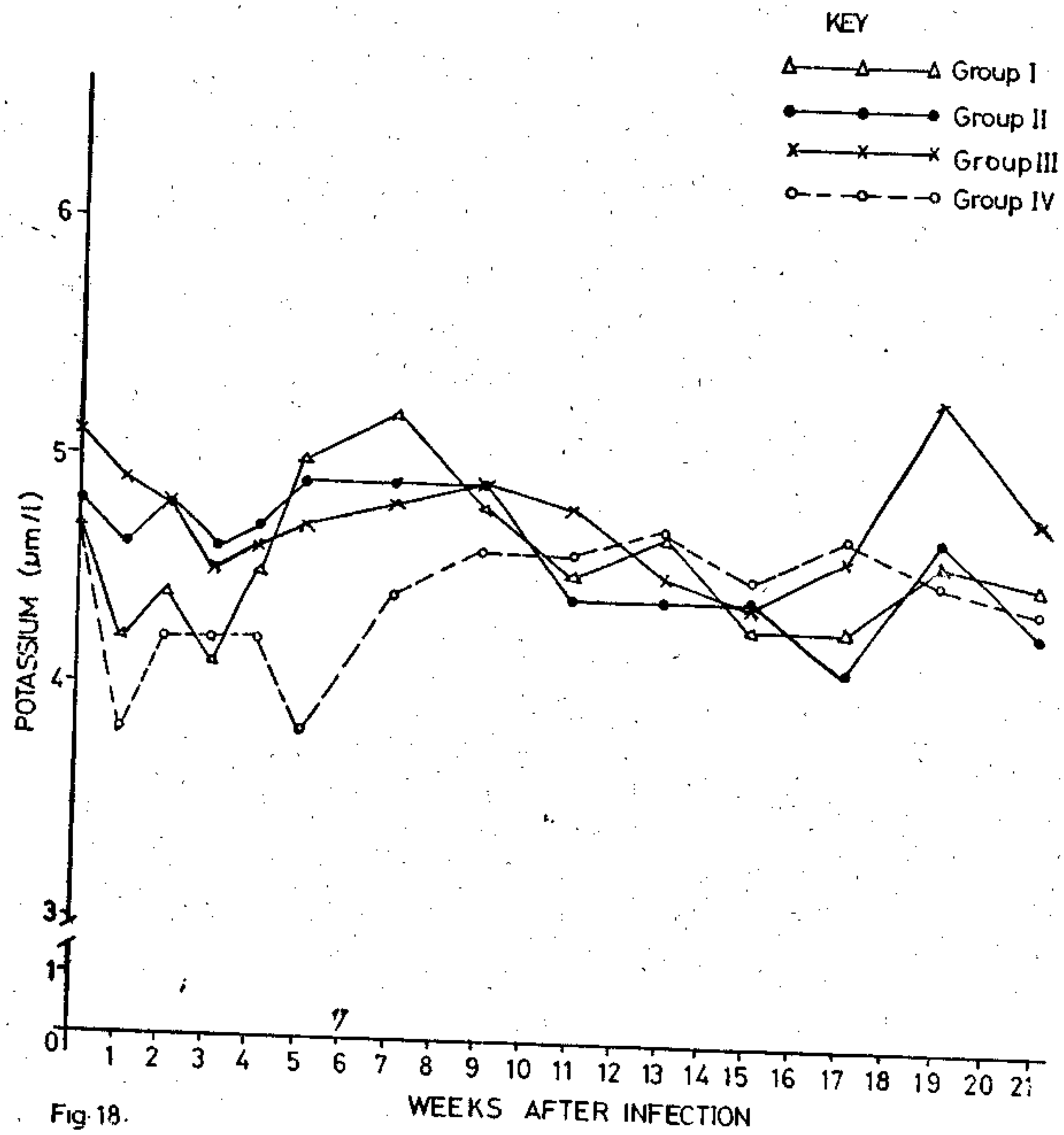

Fig. 18.

Group mean Potassium concentration of sheep infected with varying levels of Haemonchus larvae

DISCUSSION

Low helminth egg production in all infected groups and prolonged prepatent period in Group I animals suggested that helminth establishment and fecundity were affected. Further evidence of this was obtained by correlating mean egg counts with mean number of females present. Considering Gordon's (1950) estimate that one female Haemonchus produced about 5 to 10 epg of faeces in adult sheep, and a 1:1 male: female ratio, the number of eggs produc-d per female (EPF) can be calculated. By taking mean EPG counts for the sheep on the day of slaughter and correlating it with the estimated number of females present on slaughter,EPF can be calculated. For Group I EPF was 3.5 (620 epg/178 females); for Group II EPF was 5.6 (490 epg/87.5 females) while for Group III EPF was 8.2 (180 epg/22 females). On this basis, EPF of Group III best agrees with Gordon's (1950) estimate and values of EPF for groups I and II represented a reduction of 57% and 32% when compared to EPF for Group II. Gordon (1950) also observed that in resistant sheep, the length of the prepatent period may be extended and the rate of egg production depressed after administration of infective larvae; the larger the worm burden, the lower the rate of egg production by the individual worms. The observations in Groups I and II animals agree with this report.

That establishment or fecundity of worms in this experiment may have been affected is further shown by the fact that larvae administered by the seventh instalmental infection were expected to reach patency by the third week of infection and cause an abrupt and marked rise in egg counts in all infected animals. No such rise was observed. This suggests that these late helminths had very low fecundity or were not established at all. Evidence for reduced fecundity in resistant animals has been presented by Michel (1963). He observed that worms which develop and establish late in resistant animals do not become as fertile as those that develop early in the infection.

Results obtained in this study also show that animals with the least infection had higher maximal egg counts than those that received heavier infections. This is in line with the observation of Ross and Armour (1960), Gibson (1965) and Levine (1968) that egg counts are often a poor measure of the number of worms infecting a host. This is explained by the finding that individual worms within the parasite population of sheep with low worm burdens are not only heavier (Le Jambre _et al_ 1977, Allonby 1974) but also suck more blood than those from animals with large burdens.

The low number of _Haemonchus spp_ that established in each group could have been affected by three major factors. Firstly, the repeated dosing of the animals could have led to the loss of a large proportion of the worms. Evidence for this loss in experimental infections has been given by Fourie (1931), Brambel _et al_ (1964) and Christie (1970). According to Michel (1963) the experience of infection which is required to produce resistance is not only dependent on the total number of larvae but also on the number of occasions over which they are administered and on the dosing schedule.

Animals used for this experiment had experienced previous primary helminth infections of varying severities and so the effects of acquired resistance are to be considered. That resistance to H. contortus is stimulated by previous infection has been demonstrated by Stewart (1950), Stoll (1958), Jarret et al (1959) as well as Lopez and Urquhart (1968). Bawden (1969) stated that the number of nematodes recovered from an infected animal reflects the degree of success firstly of the initial establishment and secondly subsequent survival of the parasites. These phenomena are in turn indirect manifestations of the innate natural and acquired immune mechanisms of the host. The capacity to manifest this resistance depends on age, nutritional status and the rate and duration of the primary infection. Of these only nutritional status can be considered since animals of different ages and worm histories were used.

According to Chandler (1953) the nutritional status of the host animal can exert a profound influence on its natural and acquired immunities to nematode parasites. The reports of different investigators on the effects of a high carbohydrate diet in the form of maize supplement on the establishment and fecundity of helminth parasites could be relevant in this study since maize was used as a major constituent (63%) of the concentrate fed. Vegors _et al_ (1955) observed that calves which received corn supplement generally had fewer worms than those on the same type of pasture without corn supplement. Amos (1968) observed a marked decline in egg counts and worm populations in feedlot cattle fed on a high level of corn supplement. Two experiments reported by Bilkovich (1962, 1967) determined that calves fed corn had up to 50% fewer stomach worms than similarly exposed controls that did not receive corn. Bilkovich (1962) credited corn with causing expulsion of the worms. Goldberg (1965) determined that rations containing 30% to 42.5% corn did not affect the establishment of _Ostertagia_ and _Cooperia_ infections in calves as measured 42 days post exposure. Todd (1963) reported a marked decline in egg counts in calves infected with _H. placei_ when corn was substituted for a grain mixture in their diet. Although these investigators were unable to explain the phenomenon, some stated that corn lacked a substance necessary for egg production while others speculated that the effects of corn on the parasites may simply be mechanical. The former hypothesis may be more valid since corn is low in calcium and phosphorus which are essential in egg production and probably are essential for survival of the parasite as well.

Failure of the different dose of _Haemonchus larvae_ to significantly alter haematological values up till thirteen weeks of infection may be linked to the low patent infections established with all the doses given. This is similar to results obtained by Allonby and Urquhart (1975) in a chronic

Haemonchus infections in Merino sheep. In infected animals, apart from slight fluctuations in PCV, Hb and RBC counts, considerable alterations did not occur in these parameters until the thirteenth week of infection when these became consistently low. This observation is important since a casual examination of the blood before the thirteenth week may not reveal any alarming changes. This insidious chronic syndrome however, occurs widely under field conditions and is an important stage in the development of sudden anaemia and unthrifitiness in previously healthy-looking sheep.

Serum iron values did not show any marked changes. This finding is similar to those of Baker et al (1959), Dargie and Allonby (1975) who observed that infected sheep have some ability to reabsorb iron lost into the intestinal tract as a result of haemorrhage. This, coupled with the fact that reserve iron stored in the liver, spleen and bone marrow as ferritin is released to replace iron lost during haemorrhagic conditions, makes serum iron alterations minimal. According to Dargie and Allonby (1975) significant serum iron alterations do not take place until hepatic iron reserves have fallen to values between 60 to 80 ug%. Young animals however, have an immature haematopoietic system, and so do not have substantial iron reserves to withstand the demands of infection as well as met up normal demands of growth and metabolic processes.

The absence of marked effects of the infection on either total leucocyte or differential counts suggest that toxemia may not be associated with haemonchosis. Reports by Holman and Pattison (1960) as well as Silverman et al (1970) show a decrease in total leucocytes accompained by a relative decrease in lymphocytes and an equally consistent relative increase in polymorphonuclear leucocytes in a challenge infection of Haemonchus in sheep. In this study there were no marked differences between infected and uninfected groups but the negative correlation between total leucocytes and lymphocytes suggests that increases in leucocyte numbers in haemonchus infection is not associated with increase in lymphocyte numbers. This is supported by studies by Adams and Beh (1981) who reported that H. contortus infection produces a depletion in lymphocytes leading to a mild lymphopenia.

The slight eosinophilia in infected groups is similar to the findings of Charleston (1964) and Silverman et al (1970) but was not necessarily an eosinophilia of recovery as reported by Fourie (1931) and Andrews(1942). Slight eosinophilia in haemonchosis is probably due to the fact that the parasite is not a tissue invader although the fourth larval stage burrows into the submucosa of the abomasum and may not be secreting any soluble substances which attract large numbers of eosiniphils into the circulating blood.

Results obtained show that there were no significant changes in total protein and serum protein fractions. Most authors who investigated blood protein levels in parasitized animals (Kutler and Marble 1960, Cornelius et al 1962, Herlich 1962, Leland et al 1959, Ross 1963) reported a hypoproteinaemia produced by a reduction in circulating albumin usually accompanied by a rise in one or more of the globulin fractions. Failure of the innoculating levels of Haemonchus to significantly alter serum protein in this study may be attributed to the low levels of infection established. Anderson et al (1960) did not observe any marked changes in serum protein values in calves experimentally infected with 13,000 and 165,000 H. placei larvae. Kutler and Marble (1960) also were not able to obtain significant changes in serum proteins in lambs with a 'moderate' (4,647 epg) infection but got changes in clinically parasitized lambs. Allonby (1973) has shown that even in the face of exessive drain of body protein in Haemonchus infected animals, serum protein concentration were still maintained. This is because the parasite sucks blood and passes the constituents into the abomasum where they are broken down by proteolytic enzymes and the protein reabsorbed. Secondly, infected animals have an increased catabolic rate as well as increased albumin synthesis. In one year old sheep infected with 10,000 larvae and challenged with a further 10,000 larvae seven weeks later, Allonby (1973) observed a 0.5 gm% change in it's serum albumin concentration. The results of serum protein values in this study agrees with that of Allonby (1973) who observed that changes in serum protein are closely related to immune status of the animal. Increased gamma globulin levels in groups with heavy infections is in agreement with observations by Turner and Wilson (1960) and Kutler and Marble (1960) and may be one of the factors in the increase in resistance, but its precise role in resistance to parasitic infections has not been resolved. The negative correlation of Albumin and egg counts and its significant correlation with PCV, RBC and Hb obtained in this study lends further support to findings by Ross and Armour (1960) that serum albumin levels and packed cell volume percentages may be useful measures of pathogenicity when used in conjuction with faecal egg counts.

The differences in the concentration of sodium in infected and uninfected animals were significant but potassium concentrations did not show significant differences. Pradhan and Johnstone (1972) observed a rise in serum sodium and a fall in potassium concentrations after infection with H. contortus. They concluded that this was a reverse trend to what was happening to erythrocyte concentrations of these electrolytes. The plasma changes in serum sodium and potassium could not be sufficiently related by them to the changes in the erythrocyte. In this study serum potassium concentrations increased in infected groups between the fifth and seventh weeks of the infection but this fell to normal levels thereafter. Serum sodium concentrations on the other hand, showed a marked increase in infected groups especially during the third and eleventh weeks of infection. Since no similar changes took place in the controls, the electrolyte changes may be a response to haematological alterations during infection. Changes in serum concentrations of sodium and potassium in _Haemonchus_ infections have been attributed to influx of young erythrocytes into the circulation. Evans _et al_ (1963) drew attention to the possible association between sodium and potassium content of the erythrocytes and susceptibility to infections of _H._ contortus. Probably such changes are not sufficiently reflected in the serum.

SUMMARY AND CONCLUSION

Three groups of sheep were infected with a high (35,000) moderate (14,000) and low (2,800) Haemonchus third stage larvae respectively while one group served as uninfected controls. The infected groups showed some resistance to the infection as was reflected in their low worm burdens and low egg counts. This resistance was manifected more in the animals that received high and moderate infections and also in older animals.

The loss of a high proportion of infective larvae and death in young animals reflects conditions operational in the field. It gives an insight as to why grazing animals are not overwhelmed by large numbers of infective larvae which they ingest on pasture, and that young animals remain most susceptible to Haemonchus infection even after they have had a previous exposure. This may be due to the fact that capability to manifest resistance depends on such factors as breed and age, previous experience as well as nutritional status of the host. Diet supplements may prevent the establishment of Haemonchus or reduce them or they may cure the disease by the helminth. More experimental data are needed to evaluate properly the effects of dietary variables in haemonchosis in ruminants.

This study also shows the contribution of subclinical levels of infection to unthriftiness and susceptibility of sheep to other disease or stress factors that may normally be encountered in the intensive rearing of sheep. These insidious effects while not as dramatic as acute infections, play important roles in the attainment of maximal productivity and weight gains in sheep, especially if they are infected at an early age.

In farm practice, it is not necessary to remove all parasites from the hosts because animals unaccustomed to parasites are more liable to disease than those accustomed to a moderate worm burden. The aim of control, then in a prolific helminth like Haemonchus should be to restrain the biotic potential of the parasite to a level at which it is compatible with the biological requirements of economic husbandry. Further research into all aspects of host-parasite interactions leading to resistance is required so as to elucidate methods of stimulating protective immunity in animals and substantially limit helminth infection.

REFERENCES

Adams, D. B. and Beh, K.J. (1981). Immunity acquired by sheep from experimental infection with Haemonchus contortus. International Journal for Parasitology. 11: 381-386.

Akerejola, O. O. (1976). Comparative Protein Metabolism in Bunaji (White Fulani) and Friesian cattle in Nigeria. Ph.D Thesis. Ahmadu Bello University, Zaria, Nigeria.

Allonby, E.W. (1973). Ovine Haemonchosis: Epidemiology, clinical signs and Diagnosis In: Helminth Diseases of Cattle, Sheep and Horses in Europe. Edited by Urquhart, G.M and Armour, J. 59 - 63.

Allonby, E.W. and Urquhart, G.M. (1975). The epidemiology and pathogenic significance of haemonchosis in a Merino flock in East Africa. Veterinary Parasitology. 1: 129-143.

Allonby, E.W. and Urquhart, G. M. (1976). A possible relationship between haemonchosis and haemoglobin polymorphism in Merino Sheep in Kanya. Research in Veterinary Science. 20: 212-214.

Altif, K. I. and Dargie, J.D. (1978), Genetic resistance to helminths: The influence of breed and haemoglobin type on the response of sheep to primary infection with Haemonchus contortus. Parasitology 77: 161-175.

Ames, E.R. (1968). Namatode parasitism in feedlot cattle. The influence of Ostertagi ostertagi and Cooperia on performance. Ph.D Thesis. Colarado State University,

Anderson, F.L., Graff, D.J., Hammond, D.M., Fitsgerald, P.R. and Miner, M.L. (1960) cited by Soulsby, E.J.L. (1965). In Textbook of Veterinary Clinical Parasitology. E.A. Davis and Company. Philadelphia p. 664.

Andrews, J.S. (1942). Stomach worm (Haemonchus contortus) infection in lambs and its relation to gastric haemorrhage and general pathology. Journal of Agricultural Research. 65: 1-18.

Anon (1975). Beckman Microzone Electrophoresis Mannual Number 015-083630-A.

Anosa, V.O. (1977). Haematological observations on helminthiasis caused by Haemonchus contortus in Nigerian Dwarf Sheep Tropical Animal Health Production. 9: 11-17.

Baker, N.F., Cook, E.F., Douglas, J.R. and Cornelius, C.E. (1959). The Pathogenesis of Trichostrongylid Parasites III. Some physiological observations in lambs suffering from acute parasitic gastro-enteritis Journal of Parasitology. 45: 643-651.

Bawden, R.J. (1969). The establishment and survival of Oesophagostomum columbianum in male and female sheep given high and low protein diets. Australian Journal of Agricultural Research 20: 1151-1159.

Beaton, W.G. (1937). Goat husbandary in Northern Nigeria including a descriptive survey of the causes of mortality. In: Appendix II, Annual Report of Veterinary Department, Ministry of Animal Health and Forestry, Northern Nigeria pp. 31-48.

Bilkovich, R.R. (1962). Host Nutrition and the Bionomics of Haemonchus placei. Masters Thesis. University of Wisconsin.

Bilkovich, R.R. (1967) (1). Nematodiasis in Lousiana Dairy Cattle; (II) Impact of Host Nutrition upon experimental Ostertagiosis in the Bovine. Ph.D. Thesis. University of Wisconsin. Madison, P. 170 In: Dissertation Abstracts 12, 108.

Blood, D.C. and Henderson, J.A. (1963). In Veterinary Medicine second edition. Williams and Wilkins Company. Baltimore pp. 821 - 826.

Blunt, M.H. and Evans, J.V. (1963). Changes in the concentration of potassium in the erythrocytes and in haemoglobin type in Merino sheep under a severe anaemic stress. Nature. 200: 1215-1216.

Bothwell, T.H.; Charlton, R.W.; Cook, J,D. and Finch, C.A. (1979). In: Iron metabolism in man. Blackwell Scientific Publications. Oxford. England pp. 291-294.

Boughton, I.B. and Hardy, W.T. (1935). Stomach worms (Haemonchus contortus) of sheep and goats. In: Report of Texas Experimental Station. 48: 236-239.

Brambell, M.B.; Charleston, W.A.G. and Tothill, P. (1964). Abomasal bleeding caused by immature stages of Haemonchus contortus in sheep showing age resistance. Journal of Comparative Pathology and Therapeutics 74: 338-345.

Cameron, W.T.M. (1939). In: Immunity against Animal Parasites - Culbertson, J.T. (1941). Columbia University Press p. 23.

Chandler A.C. (1935). Studies on the nature of immunity to intestinal infections II. A study of the correlation between degree of resistance of white rats to Nippostrongylus and interval between infections. American Journal of Hygiene 22: 243-256.

Chandler, A.C. (1953). The Relation of Nutrition to Parasitism. Journal of Egyptian Medical Association. 36. 811-834.

Charleston, W.A.G. (1964). Comparison of blood and bone marrow changes in lambs with haemonchosis and in lambs repeatedly bled. Journal of Comparative Pathology and Therapeutics. 74: 224-240.

Christie, M.C. (1970). The fate of very large doses of Haemonchus contortus and their effect on conditions in the ovine abomasum. Journal of Comparative Pathology. 80: 89-100.

Christie, M.G. and Brambell, M.R. and Charleston, W.A.G. (1964). Resistance to a challenge infection with ~~Haemonchus contortus~~ confered by previous experience of immature stages only. Journal of Comparative Pathology and Therapeutics 74: 427-434.

Christie, M.G. and Brambell, M.R. (1966). Acquired Resistance to Haemonchus contortus in young lambs. Journal of Comparative Pathology 76: 207-216.

Clark, C.H.; Kiesel, G.K. and Goby, C.H. (1962). Measurements of blood loss caused by Haemonchus contortus infection in sheep. American Journal of Veterinary Research 23: 977-980.

Coadwell, W.J. and Ward, P.R. (1975). Observations on the development of Haemonchus contortus in young sheep given a single infection. Parasitology. 71: 505-515.

Cobb, N.A. (1898). Report on the Parasites of Livestock In: Key to Parasitic Nematodes s'rjabin K.I. (Ed) Israel Programme for Scientific Translations Ltd. pp. 413-414.

Coles, E.H. (1968). Veterinary Clinical Pathology. Saunders Company. London p. 127.

Cornelius, C.E.; Baker, N.F.; Kaneko, J.J. and Douglas, J.R. (1962). Distribution and turnover of iodine - 131 - tagged bovine albumin in normal and parasitized cattle. American Journal of Veterinary Research 23: 23: 837-842.

Culbertson, J.F. (1941). In: Immunity against Animal parasites. Columbia University Press. New York p. 274.

Dargie, J.D. and Allonby, E.W. (1975). Pathophysiology of single and challente infections of Haemonchus contortus in Merino sheep. Studies on red cell kinetics and the self-cure phenomenon. International Journal for Parasitology. 5: 147-157.

Delaune, E.T. and Mayhew, R.L. (1943). Studies on bovine gastro-intestinal parasites VI. The blood picture in stomach worm Haemonchus contortus infection. Transactions of the American Microscope Society. 62: 179-193.

Dinaburg, A.G. (1944). Development and survival under outdoor conditions of eggs and larvae of the common ruminant stomach worm Haemonchus contcrtus. Journal of Agricultural Research. 69: 421-433.

Dineen, J.K. and Wagland, B.M. (1966a). The dynamics of the host-parasite relationship IV. The response of sheep to graded and repeated infection with Haemonchus contortus. Parasitology 56: 639-650.

Dineen, J.K. and Wagland, B.M. (1966b). The dynamics of the host-parasite relationship V. Evidence for immunological exhaustion in sheep experimentally infected with Haemonchus contortus. Parasitology. 56: 665-677.

Dinnik, J.K. and Dinnik, N.N. (1958). Observations on the development of Haemonchus contortus larvae under field conditions in the Kenyan highlands. Bulletin of Epizootic Diseases of Africa. 6: 11-21.

Dinnik, J.A. and Dinnik, N.N. (1961). Observations on the longevity of Haemonchus contortus larvae on pasture herbage in the Kenyan highland. Bulletin of Epizootic Diseases of Africa. 9: 193-208.

Evans, J.V.; Blunt, H.M. and Southcott, W.M. (1963). The effects of infection with Haemonchus contortus on the sodium and Potassium concentrations in the erythrocytes and plasma in sheep of different haemoglobin types. Australian Journal of Agricultural Research. 14: 549-558.

Evans, J.V. and Whitlock, J.H. (1964). Genetic relationship between maximum haematocrit values and haemoglobin type in sheep. Science. 145: 1318.

Food and Agricultural Organization (1966). Agricultural Development in Nigeria. 1965-80. Rome. p. 216.

Fabiyi, J.P. (1968). An investigation into some aspects of nematodology in Nigerian goats. M.Sc Thesis. Ahmadu Bello University. Zaria.

Fabiyi, J.P. (1973). Seasonal fluctuations of nematode infestations in goats in the savannah belt of Nigeria. Bulletin of Epizootic Diseases of Africa. 21: 277-284.

Foster, A.O. (1935). The immunity of dogs to Ancylostoma caninum. American Journal of Hygiene. 22: 65-105.

Fourie, P.J. (1931). The haematology and Pathology of haemonchosis in sheep. Report (17th) Veterinary Research Union of South Africa. 495-572.

Franklin, M.C.; Gordon, H Mcl and Macgregor, C. H. (1946). A study of Nutritional and Biochemical effects in sheep of infestation with Trichostrongylus colubriformis Journal of the Council of Science and Industrial Research. 49: 46-60.

Fraser, A.H. and Robertson, D. (1933). The influence of the nutritional state of sheep on its susceptibility to infestation with the stomach worm Haemonchus contortus. Journal of Experimental Agriculture. 1: 17-21.

Gibson, T.E. (1954a). Studies on Trichostrongylus axei. I. The pathogenesis of Trichostrongylus axei in sheep maintained on a low plane of nutrition. Journal of Comparative Pathology and Therapeutics. 64: 127-140.

Gibson, T.E. (1965). Helminthiasis in sheep. Veterinary Record. 77: 1034-1041.

Goldberg, A. (1965). Relation of feeding level to gastro-intestinal helminth parasitism in cattle. Journal of Parasitology. 51: 948.

Gordon, H. Mcl. (1948). The epidemiology of Parasitic disease with special reference to studies with nematode parasites of sheep. Australian Veterinary Journal. 24: 17-45.

Gordon, H. Mcl (1950). Some aspects of parasitic gastro-enteritis of sheep. Australian Veterinary Journal. 26: 14-28.

Harness, E.; Fitzsimmons, W.. and Sellwood, S.A. (1970), Experimental Haemonchus placei infection in calves. Journal of Comparative Pathology. 80: 173-179.

Henderson, W.W. (1928). Annual Report of the Veterinary Department, Ministry of Animal Health and Forestry, Northern Nigeria. p. 8.

Herlich, H. (1962). Studies on Calves Experimentally Infected with combination of Four Nematode Species. American Journal of Veterinary Research. 23: 521-528.

Holman, H.H. and Pattison, I.H. (1941). A study of the Blood Picture of lambs suffering from Parasitic-gastritis. Veterinary Record. 53: 491-498.

Holmes, P.H. and Dargie, J.D. (1975). Pathophysiological mechanisms in ovin anaemias. In: The Blood of Sheep, Composition and Function. Edited by M.H. Blunt. Springer-verlag. Berlin. 197-206.

Jarret, W.F.; Jennings, F.W.; McIntyre, W.I.; Mulligan, W. and Sharp, N.C. (1959). Studies on immunity to Haemonchus contortus vaccination of sheep using a single dose of x-irradiated larvae. American Journal of Veterinary Research. 20: 527-531.

Jarret, W.F.; Jennings, F.W.; McIntyre, W.I.; Mulligan, W. and Sharp, N.C. (1961). Studies on immunity to Haemonchus contortus infection. Double vaccination sheep with irradiated larvae. American Journal of Veterinary Research. 22: 86-88.

Jilek, A.F. and Bradley, R.E. (1969). Haemoglobin types and Resistance to Haemonchus contortus in sheep. American Journal of Veterinary Research. 30: 1773-1778.

Kates, K.C.; Allen, R.W. and Wilson, G.I. (1962). Effects of two diets on experimental haemonchosis in lambs. Journal of Parasitology. 48: 865-870.

Kerr, K.B. (1938). The cellular response in acquired resistance in guinea pigs to an infection with Pig Ascaris. American Journal of Hygiene. 28: 51.

Kuil, H. (1972). Gastrointestinal Namatodes in the Zaria areas of Northern Nigeria. Mimeograph, Institute of Tropical Diseases, Utretch and Department of Parasitology and Entomology, Ahmadu Bello University, Zaria.

Kutler, K.L. and Marble, D.W. (1960). Serum protein changes in lambs with Naturally Acquired Nematode Infections. American Journal of Veterinary Research. 21: 445-448.

Lee, R.P. (1955). Annual Report of Veterinary Division, Ministry of Animal Health and Forestry, Northern Nigeria. 1955-56. p. 10.

Leland, S.E. (1961). Blood and Plasma Volume, Total Serum Protein and Electrophoretic Studies in Helminthic Diseases. Ney York Academy of Sciences. 94: 163-182.

Leland, S.E.; Drudge, J.H. and Dillard, R.P. (1966). Influence of Superimposed Nematode Infection and grain supplement on some blood constituents of calves on pasture. American Journal of Veterinary Research. 27: 1555-1564.

Leland, S.E.; Drudge, J.H. and Wyant, Z.N. (1958). Blood and Plasma volume, total serum protein and electrophortic serum fractionation of calves experimentally infected with Trichostrongylus axei. Journal of Parasitology. 44 (27-28).

Le Jambre, L.F.; Ractlife, L.H.; Uhazy, L.S. and Whitlock, J.H. (1971). Faecal egg output of lambs in relationship to Haemonchus contortus burden. International Journal for Parasitology. 59: 649-661.

Levine, N.D. (1968). In: Nematode Parasites of Domestic Animals of Man. Burgess Publishing Company. Minneapolis Minnesota. p. 412.

Lindquist, W.D. (1958). Unpublished, cited by Fabiyi (1968).

Lopez, V. and Urquhart, G.M. (1968). Immune Response of Merino sheep to Haemonchus contortus. (Helminthological Abstracts. 39 Abstract 4224 (1969).

Lucker, T.J. and Neumayer, E.H. (1947). An experiment on the relationship of diet to Hookworm disease in lambs. American Journal of Veterinary Research. 6: 400-412.

Marht, J.L.; Hammond, D.M. and Miner, L.M. (1964). Changes in serum proteins and other blood values associated with experimental Ostertagia ostertagi infections in calves Cornell Veterinarian. 54: 453-474.

Manton, J.A.; Peacock, R.; Poynther, D.; Silverman, P. and Terry, R.J. (1962). The influence of age on naturally acquired resistance to Haemonchus contortus in lambs. Research in Veterinary Science. 3: 308-314.

Martin, C.J. and Ross, C.I. (1934). Minimal Computation of the amount of blood removed daily by Haemonchus controtus. Journal of Helminthology. 12: 137-142.

Michel, J.F. (1952). Inhibition of development of Trichostrongylus retortaeformis. Nature. 169: 881.

Michel, J.F. (1963). The phenomenon of host resistance and the course of infection of Ostertagia ostertagi in calves.Parasitology. 53: 63-84.

Misra, S.C. and Ruprah, N.S. (1972). Haemonchus contortus infection in experimental lambs. Indian Veterinary Journal. 49: 554-560.

Monnig, H.O. (1930). Studies on the Bionomics of the free living stages of Trichostrongylus spp and other parasitic Nematodes. In: Report of Veterinary Research of South Africa. 16: 175 - 198.

Mulligan, W.; Gordon, H. Mcl, Stewart, D.F. and Wagland, B.M. (1961). The use of irradiated larvae as immunising agents in Haemonchus controtus and Trichostrongylus columbriformis infection sheep. Australina Journal of Agricultural Research. 12: 1175-1187.

Neklyudor, V.N.; Bolkhovitinov, D.V. and Sominski. Z.F. (1956). Pathogenesis of Haemonchus infection in sheep. Veterinaria. 33: 66-69 (Veterinary Bulletin 27: 173. 1951).

Oduye, O.O. (1976). Haematological values of Nigerian Goats and Sheep. Tropical Animal Health and Production. 8: 131-136.

Ogunsusi, R.A. (1978). The Epidemiology of Trichostrongylid Infections of Sheep in the Northern Guinea Savannah of Nigeria. Ph.D. Thesis. Ahmadu Bello University, Zaria.

Okon, E.D. and Enyenihi, U.K. (1977). Development and survival of Haemonchus contortus larvae on pastures in Ibadan. Tropical Animal Health and Production. 9: 7-10.

Oshio, Y. (1952). Studies on the anaemia by Haemonchus contortus (I) on the Anaemia-producing substance. Bulletin of the National Institute of Agricultural Science. Chiba 3: 171-178 (Helminthological Abstracts 21 1952).

Oshio, Y. (1953). Studies on the anaemia by Haemonchus contortus (II) On the mechanism of the anaemia. Bulletin of the National Institute of Agricultural Science. Chiba 5: 59-64 (Helminthological Abstracts 22: 1953).

Otto, G.F. and Kerr, K.B. (1939). The Immunization of Dogs Against Hookworm Ancylostoma canninun, by subcutaneous injection of graded doses of living larvae. American Journal of Hygiene. 29: 25-45.

Pradhan, S.L. and Johnstone, I.L. (1972). Haemonchus contortus. The effect on Lambs of Prolonged exposure of daily and weekly doses of infective larvae. Parasitology: 64: 143-152.

Radhakrishnan, C.V.; Bradley, R.E. and Loggins, P.E. (1972). Host responses of worm-free Florida Native and Rambouillet Lambs Experimentally infected with Haemonchus contortus. American Journal of Veterinary Research. 33: 817-823.

Ransom, B.H. (1906). The life history of the twisted wire-worm (Haemonchus contortus) of sheep and other ruminants In: Key to Parasitic Nematodes. Skrjabin K.I. (Ed) Israel Programme for Scientific Translations pp. 413-414.

Richard, R.M.; Shumard, R.F.; Pope, A.L.; Phillips, P.H.; Herrick, C.A. and Bohstedt, G. (1954). The effect of certain mineral supplements on lambs infected with the stomach worm. (Haemonchus contortus). Journal of Animal Science. 13: 694-705.

Roberts, F.H.S. (1957). Reactions of Calves to Infestation with the Stomach worm Haemonchus placei (Place 1893 Ransom 1911) Australian Journal of Agricultural Research 8: 740-767.

Roberts, F.H.S. and O'Sullivan, B,M. (1950). The Significance of Faecal egg counts in the diagnosis of Parasitic gastro-enteritis of cattle. Australian Veterinary Journal. 27: 16-18.

Ross, J.G. (1963). Experimental Infections of Calves with the nematode parasite Ostertagi Ostertagi. Veterinary Record. 75: 129-132.

Ross, J.G. and Armour, J. (1960). The Significance of Faecal Egg Counts and the use of Serum Albumin Levels and packed Cell Volume Percentages to Assess. Pathogenicity of Helminthiais. Veterinary Record. 72: 137-139.

Ross, J.G.; Armour, J. and Hart, J.A. (1962). Investigations of Haemonchosis in Nigerian Zebu Cattle. Journal of Parasitology. 48: 496.

Ross, J.G. and Gordon, J.E. (1933). Nutritional factors affecting resistance to haemonchosis. Australian Veterinary Journal. 9: 100-107.

Ross, J.G.; Lee, R.P. and Armour, J. (1959). Haemonchosis in Nigerian Zebu Cattle, Influence of Genetical Factors in Resistance. Veterinary Record. 71: 27-31.

Rudolphi, C.A. (1803). In:Key to Parasitic Nematodes. Skrjabin, K.I. (ed) Isreal Programme for Scientific Translations Ltd. pp. 43-414.

Sarles, M.P. and Taliaferro, W.H. (1936). The Local Points of Defence and the Passive Transfer of Acquired Immunity to Nippostrongylus muris in rats. Journal of Infectious Diseases. 59: 207-220.

Saror, D.I. and Schilhorn Van Veen T.W. (1973). Ruminant Health Problems in Northern Nigeria with emphasis on helminthiasis. Journal of Nigerian Veterinary Medical Association. 2: 26-31.

Schilhorn Van Veen T.W. (1978). Haemonchosis in sheep during the dry season in the Nigerian Savannah. Veterinary Record. 102 (16) 364-365.

Schilhorn Van Veen, T.W. and Folaranmi D.O.B. (1978). The haemoglobin types of Northern Nigerian Sheep. Veterinary Science. 25 (3) 397-398.

Schwartz, B.; Alicata J.E. and Lucker, J.T. (1931). Resistance of Rats to superinfection with a nematode, Nippostrongylus muris and an apparently similar resistance of Horses to superinfection with nematodes. Journal of Washinton Academy of Science. 21: 259-261.

Scott, H.L.; Silverman, P.H.; Mansfied, M.E. and Levine, H.S. (1971). Haemonchus contortus. Infection in Sheep: Active and Passive immunity in Sheep given oral iron supplement. American Journal of Veterinary Research. 32: 244-262.

Semrad, J.E. and Coors, M.J. (1951). The rise and fall of Immunity to Trichinella spiralis in the albino rat and its effect on growth and reproduction of the parasite. Transactions of Illinois Academy of Science. 44: 253-258.

Shearer, G.D. and Stewart, J. (1933). The effects of Namatode Infestations on the Metabolism of the Host (III). The Effects of Nematode Infestations on Mineral Metabolism In: Institute of Animal Pathology, Report Directory, University of Cambridge. pp. 87-97.

Sheldon, A.J. (1937). Some Experimental Studies on Strongyloides ratti. American Journal of Hygiene. 25: 39-52.

Shorb, D.A. (1943). Survival on Grass Plots of Eggs and pre-infective larvae of the common sheep stomach worm Haemonchus contortus. Journal of Parasitology. 29. 284-289.

Shorb, D.A. (1944). Factors influencing embryonation and survival of eggs of the stomach worms Haemonchus contortus. Journal of Agricultural Research. 69: 279-287.

Shumard, R.F.; Bolin, D.W. and Eveleth, D.F. (1957). Physiological and Nutritional Changes in lambs infected with the nematodes Haemonchus contortus, Trichostrongylus colubriformis and Nematodirus spathiger. American Journal of Veterinary Research. 18: 330-337.

Shumard, R.F.; Emerick, R.J.; Bemrick, W.E.; Herrick, C.A. Pope, A.L. and Phillips, P.H. (1956). Effects of Trace Minerals, Dicalcium, Phosphate, Phenothiazine and Combinations of these on the resistance of lambs to Haemonchus contortus and other Nematodes. American Journal of Veterinary Research. 17: 252-255.

Shumard, R.F. and Eveleth, D.F. (1955). Some Nutritional and Physiological changes taking place in lambs infected with Haemonchus contortus, Trichostrongylus colubriformis and Nematodirus spathiger. Journal of Parasitology. 41: 42-43.

Shumard, R.F. and Herrick, C.A. (1954). The effect of various minerals on the growth of Haemonchus contortus. Journal of Parasitology. 40 (16).

Silverman, P.H. and Campbell, J.A. (1959). Studies on Parasitic worms of sheep (I). Embryonic and larval development of Haemonchus contortus at constant conditions. Parasitology. 49: 23-28.

Silverman, P.H.; Mansfied, M.E. and Scott, H.L. (1970). Haemonchus contortus infection in sheep. Effects of various levels of primary infections in non-treated lambs. American Journal of Veterinary Research. 31: 841-854.

Silverman, P.H. and Patterson, J.E. (1960). Histotrophic (Parasitic) stages of Haemonchus contortus. Nature. 185: 54-55.

Soulsby, E.J.L. (1958). Immunity to Helminths - Recent Advances. Veterinary Record. 72: 322-327.

Soulsby, E.J.L. (1965). In: Textbook of Veterinary Clinical Parasitology I Helminths. F.A. Davis Company. Philadelphia. pp. 310-393.

Soulsby, E.J.L. and Stewart, D.F. (1960). Serological studies on the self cure reaction in sheep infected with Haemonchus contortus. Australian Journal of Agricultural Research 11: 595-603.

Steel, G.D. and Torrie, J.H. (1960). In: Principles and Procedures of Statistics. Mcgraw-Hill Book Company. New York.

Stewart, D.F. (1950). Studies on Resistance of sheep to infestation with Haemonchus contortus and Trichostrongylus spp. and on the immunological reactions of sheep exposed to infection. IV. The antibody response to natural infestation in grazing sheep and the self-cure phenomenon. Australian Journal of Agricultural Research I: 427-439.

Stewart, D.F. (1953). Studies on the resistance of sheep to infestation with Haemonchus contortus and Trichostrongylus spp. and on the immunological reactions of sheep exposed to infestation V. The nature of the self cure phenomenon. Australian Journal of Agricultural Research. 4: 100-117.

Stewart, D.F. (1955). Self-cure in Nematode infestation of sheep. Nature. 175: 1273-1274.

Stewart, D.F. and Gordon Mcl (1958). Studies on resistance of sheep to infestation with Haemonchus contortus, Trichostrongylus spp. and on the immunological reactions of sheep exposed to infestation VI. The influence of age and nutrition on resistance to Trichostrongylus colubriformis. Australian Journal of Agricultural Research. 4: 340-348.

Stoll, N.R. (1928). The occurrence of self-cure and protection with special reference to experimental vaccination against Haemonchus. Rice Institute Pamphlet 45: 184-208 (In Veterinary Bulletin 1959).

Taylor, E.L. (1934). The epidemiology of winter outbreaks of parasitic gastritis in sheep with special reference to outbreaks which occurred during the winter of 1933-1934. Journal of Comparative Pathology and Therapeutics. 47: 235-254.

Tayler, E.L. (1943). Effect of diet on resistance to Haemonchus contortus. Veterinary Record. 55: 117.

Threlkeld, W.L.; Price, N.O.; Linkous, W.N. (1956). An observation on the relationship of cobalt deficiency to internal parasites of sheep. American Journal of Veterinary Research. 15: 230

Todd, A.C. (1963). Parasitism and its relation to nutrition and performance. In: Scientific Proceedings of the 100th Annual Meeting of the American Veterinary Medical Association. pp. 50-53.

Turner, J.H. and Wilson, G.I. (1960). Serum protein studies on Sheep and Goats. Studies on Targhee lambs raised under varied exposure to parasitism. American Journal of Veterinary Association. 26: 645-650.

Veglia, F. (1915). The anatomy and life history of Haemonchus contortus (Rud.) Report (3rd and 4th) Veterinary Research, Union of South Africa. 347-500.

Vegors, H.H.; Baird, D.M.; Sell, O.E. and Stewart, T.B. (1955). Internal parasitisms of Beef yearlings of affected by type of pasture, supplemental corn feeding and age of calf. Journal of Animal Science. 14: 256-267.

Wagland, B.M. and Dinnen, J.K. (1967). The dynamics of the host-parasite relationship. Regeneration of the immune response in sheep infected with Haemonchus contortus. Parasitology. 57: 59-65.

Weir, W.C.; Bahler, T.L.; Pope, A.L.; Phillips, P.H.; Herrick, C.A. and Bohstedt G. (1948). The effect of hemopoietary dietary factors in the resistance of lambs to parasitism with the stomach worm Haemonchus contortus. Journal of Animal Science. 7: 466-474.

Whitlock, J.H. (1948). Some modifications of the McMaster Helminth egg counting technique and apparatus. Journal of the Council of Science, Industry and Research, Australia 21: 177-180

Whitlock, J.H. (1948). The relationship of nutrition to the development of Trichostrongylidiosis. Cornell Veterinarian. 39: 146-182.

Whitlock, J.H.; Callaway, H.P. and Jeppesen, Q.E. (1943). The relationship of diet to the development of haemonchosis in sheep. Journal of American Veterinary Medical Association. 102: 34-35.

Wilson, G.I. and Turner, J.H. (1965). Serum protein studies on Sheep and Goats. Studies on Targhee Lambs raised under varied exposures to Parasitism. American Journal of Veterinary Research. 26: (112) 645-650.

APPENDIX I

Means and standard deviations of helminth egg counts, PCV, Hb, RBC, WBC, Sodium and Potassium, serum iron and serum Protein values of Sheep infected with varying levels of *Haemonchus* larvae.

TABLE 3: Means for faecal egg counts (EPG) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III
1	0	8.3	80.0
2	1160.0	948.9	580.0
3	93.3	1190.4	1740.0
4	28.0	2957.8	1608.9
5	200.0	972.5	2084.4
7	1706.7	1390.0	3282.5
9	1273.3	1370.0	2468.7
11	1230.0	1147.5	917.1
13	573.3	747.5	490.0
15	790.0	772.5	373.3
17	433.3	542.5	246.7
19	566.7	450.0	345.0
21	620.0	490.0	180.0
X	626.0	982.2	1172.8

X = mean of means

TABLE 4: Means and standard deviations for Packed cell volume(%) of sheep infected with varying levels of *Haemonchus* larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	31.6 ± 2.5[a]	28.1 ± 3.3[b]	29.9 ± 3.0[ab]	28.8 ± 3.8[b]
1	32.0 ± 4.9	29.1 ± 3.4	30.2 ± 3.8	28.6 ± 4.7
2	27.8 ± 5.0	26.1 ± 4.1	28.6 ± 2.5	28.5 ± 4.3
3	31.1 ± 3.4[a]	25.4 ± 4.3[b]	27.6 ± 4.6[ab]	28.3 ± 3.8[ab]
4	28.8 ± 4.5	23.4 ± 5.9	26.2 ± 4.9	28.1 ± 3.2
5	29.9 ± 4.0	28.6 ± 3.6	27.2 ± 6.3	27.8 ± 4.6
7	24.9 ± 1.0	25.5 ± 3.1	28.6 ± 4.5	25.5 ± 3.3
9	28.0 ± 2.7	27.4 ± 3.1	28.2 ± 4.4	27.3 ± 1.9
11	31.8 ± 1.8	31.3 ± 4.8	29.6 ± 6.3	31.6 ± 2.5
13	24.3 ± 3.3	23.4 ± 4.9	22.3 ± 3.4	26.2 ± 3.4
15	21.0 ± 3.0	21.8 ± 4.6	21.3 ± 2.9	23.2 ± 3.4
17	26.8 ± 5.4[ab]	24.1 ± 4.2[b]	23.3 ± 4.6[b]	29.0 ± 3.0[a]
19	21.6 ± 3.1	20.2 ± 3.9	22.0 ± 3.8	23.1 ± 4.4
21	22.3 ± 2.7[a]	19.1 ± 3.3[b]	20.6 ± 2.4[ab]	23.6 ± 0.9[a]
$\bar{X}$	27.8 ± 5.1[a]	25.5 ± 5.1[b]	26.6 ± 5.1[ab]	27.4 ± 4.0[a]

$\bar{X}$ = Mean of means

ab Means with different superscript(s) within each row differ (P .05).

TABLE 5: Means and standard deviations for Haemoglobin values (gm/dl) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	10.7 ± 0.5^{a}	9.6 ± 1.1^{b}	10.3 ± 1.0^{ab}	10.0 ± 1.3^{ab}
1	10.2 ± 0.5	9.9 ± 1.1	10.3 ± 1.1	10.2 ± 1.5
2	8.9 ± 1.4	9.2 ± 1.0	9.9 ± 0.8	9.9 ± 1.4
3	10.0 ± 0.8	8.8 ± 1.4	9.4 ± 1.3	9.9 ± 1.3
4	9.3 ± 1.4^{ab}	8.2 ± 1.8^{b}	9.1 ± 1.6^{ab}	9.7 ± 1.0^{a}
5	9.5 ± 0.9	9.1 ± 1.2	9.4 ± 2.0	9.7 ± 1.5
7	8.1 ± 0.3	8.7 ± 1.0	8.2 ± 1.5	9.0 ± 1.1
9	9.1 ± 0.8	8.9 ± 0.9	9.7 ± 1.4	9.5 ± 0.6
11	9.7 ± 0.7	9.7 ± 1.2	9.7 ± 2.0	10.8 ± 0.9
13	8.1 ± 1.0^{ab}	7.7 ± 1.5^{b}	7.7 ± 1.2^{b}	9.2 ± 1.1^{a}
15	7.0 ± 0.9	7.1 ± 1.5	7.1 ± 0.9	8.2 ± 1.3
17	7.9 ± 0.9^{b}	8.2 ± 1.6^{b}	7.5 ± 1.5^{b}	9.8 ± 0.9^{a}
19	7.2 ± 0.9	6.7 ± 1.2	7.1 ± 1.3	8.1 ± 1.2
21	7.4 ± 0.8^{ab}	6.4 ± 1.4^{b}	6.8 ± 0.9^{b}	8.3 ± 0.3^{a}
$\bar{\bar{X}}$	8.9 ± 1.4^{bc}	8.6 ± 1.4^{c}	9.1 ± 1.7^{b}	9.5 ± 1.3^{a}

$\bar{\bar{X}}$ = Mean of means

abc Means with different superscript(s) within each row differ (P .05)

TABLE 6: Means and standard deviations for erythrocyte counts (RBC. x 10^6/ul) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTED	GROUP I	GROUP II	GROUP III	GROUP IV
0	9.5 ± 0.8^a	8.4 ± 0.9^b	8.9 ± 0.8^{ab}	8.6 ± 1.0^b
1	9.5 ± 1.3	8.5 ± 0.9	8.9 ± 0.9	8.8 ± 1.3
2	8.3 ± 1.5	7.7 ± 1.2	8.6 ± 0.8	8.7 ± 1.2
3	9.3 ± 1.0^a	7.6 ± 1.3^b	8.2 ± 1.3^{ab}	8.5 ± 1.2^{ab}
4	8.6 ± 1.3^a	6.9 ± 1.8^b	7.9 ± 1.4^{ab}	8.5 ± 0.9^a
5	8.9 ± 1.2	8.1 ± 1.1	8.1 ± 1.8	8.3 ± 1.4
7	7.5 ± 0.4	7.5 ± 1.3	7.2 ± 1.4	7.7 ± 1.0
9	8.4 ± 0.8	8.0 ± 0.9	8.4 ± 1.3	8.3 ± 0.6
11	9.4 ± 0.5	9.1 ± 1.2	8.9 ± 1.8	9.4 ± 0.6
13	7.2 ± 1.0	6.9 ± 1.4	7.2 ± 0.9	8.0 ± 0.9
15	6.3 ± 0.8	6.4 ± 1.4	6.5 ± 0.9	7.0 ± 1.0
17	7.9 ± 1.5^{ab}	7.0 ± 1.2^b	7.0 ± 1.4^b	8.7 ± 0.6^a
19	6.8 ± 0.9	5.9 ± 1.1	6.7 ± 1.2	7.0 ± 1.0
21	6.7 ± 0.8^a	5.6 ± 0.9^b	6.3 ± 0.7^{ab}	7.1 ± 0.2^a
$\bar{\bar{X}}$	8.3 ± 1.5^a	7.5 ± 1.5^b	8.0 ± 1.4^a	8.3 ± 1.2^a

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

ab Means with different superscript(s) within each row differ (P .05)

TABLE 7: Means and standard deviations for total Leucocyte counts (WBC x 10^3/ul) of sheep infected with varying levels of *Haemonchus* larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	11.3 ± 0.7	10.4 ± 1.0	10.7 ± 0.9	10.6 ± 1.3
1	11.4 ± 1.5	10.5 ± 1.0	10.8 ± 1.1	10.7 ± 1.5
2	10.4 ± 1.6	9.4 ± 1.4	10.4 ± 0.9	10.4 ± 1.5
3	11.3 ± 1.1	9.5 ± 1.7	9.9 ± 1.5	9.4 ± 3.2
4	10.5 ± 1.6	8.7 ± 2.1	9.6 ± 1.8	10.4 ± 1.2
5	10.6 ± 0.9	10.1 ± 1.3	9.7 ± 2.1	10.2 ± 1.6
7	9.3 ± 0.6	10.0 ± 0.9	8.7 ± 1.7	9.4 ± 1.2
9	10.2 ± 1.0	10.1 ± 1.0	10.4 ± 1.6	10.1 ± 0.7
11	11.5 ± 0.7	11.6 ± 1.9	10.7 ± 2.1	11.4 ± 0.7
13	9.1 ± 1.2	8.8 ± 1.7	8.8 ± 1.1	10.0 ± 1.0
15	8.1 ± 0.8	8.3 ± 1.6	8.0 ± 1.1	8.9 ± 1.3
17	9.4 ± 1.1^{ab}	9.3 ± 1.7^{ab}	8.5 ± 1.5^{b}	10.7 ± 1.0^{a}
19	7.9 ± 1.1	7.5 ± 1.3	8.2 ± 1.4	8.7 ± 1.1
21	8.3 ± 0.9	7.3 ± 1.6	7.3 ± 1.3	8.6 ± 0.4
$\bar{X}$	10.1 ± 1.6^{a}	9.5 ± 1.6^{b}	9.7 ± 1.7^{ab}	10.0 ± 1.6^{a}

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

ab Means with different superscript(s) within each row differ (P .05).

TABLE 8: Means and standard deviations for Eosinophil counts (%) of sheep infected with varying levels of *Haemonchus* larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	2.1 ± 2.5	2.7 ± 1.7	2.1 ± 1.6	2.6 ± 1.1
1	2.9 ± 3.3	4.4 ± 4.1	1.7 ± 2.2	3.7 ± 2.7
2	3.1 ± 1.9	4.6 ± 4.6	3.3 ± 3.1	4.2 ± 1.7
3	5.0 ± 3.8^{ab}	5.7 ± 3.9^{a}	2.3 ± 3.2^{b}	3.9 ± 2.5^{ab}
4	3.9 ± 3.0	6.0 ± 5.5	6.0 ± 4.6	3.9 ± 1.8
5	5.6 ± 3.8	4.9 ± 2.6	4.6 ± 4.1	5.2 ± 2.7
7	6.2 ± 4.9	7.0 ± 5.2	6.4 ± 5.4	4.7 ± 2.3
9	6.9 ± 3.7	8.5 ± 5.1	9.2 ± 4.9	4.7 ± 2.7
11	5.3 ± 3.9	7.5 ± 4.8	8.1 ± 5.9	4.9 ± 2.2
13	8.3 ± 2.6	6.9 ± 6.3	8.3 ± 7.3	5.0 ± 2.4
15	4.3 ± 2.9^{ab}	5.4 ± 4.4^{a}	1.0 ± 1.2^{b}	3.3 ± 2.9^{ab}
17	3.0 ± 2.9	6.0 ± 3.7	2.8 ± 4.2	1.7 ± 2.9
19	5.0 ± 1.6^{ab}	5.4 ± 4.5^{ab}	7.5 ± 2.1^{a}	2.2 ± 1.7^{b}
21	2.6 ± 1.9	4.9 ± 3.9	5.2 ± 3.2	2.3 ± 1.3
$\bar{X}$	4.3 ± 2.4^{b}	5.6 ± 4.4^{a}	4.4 ± 4.5^{b}	3.9 ± 2.4^{b}

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

ab Means with different superscript(s) within each row differ (P .05).

TABLE 9: Means and standard deviations for lymphocyte counts (%) of sheep infected with varying levels of <u>Haemonchus</u> larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	40.6 ± 11.2	36.9 ± 8.7	41.4 ± 5.7	37.3 ± 8.7
1	27.7 ± 12.9	31.0 ± 11.6	27.5 ± 9.3	35.7 ± 10.2
2	39.0 ± 11.1	31.6 ± 13.7	28.3 ± 11.7	34.2 ± 9.3
3	29.0 ± 5.9	25.8 ± 8.4	31.9 ± 8.6	34.2 ± 11.0
4	37.5 ± 9.5	37.3 ± 7.3	35.7 ± 7.2	37.8 ± 10.9
5	35.2 ± 13.5	32.0 ± 9.9	31.2 ± 5.1	34.2 ± 8.6
7	31.2 ± 5.4	25.8 ± 9.9	28.8 ± 9.6	26.4 ± 9.9
9	27.8 ± 5.3	25.3 ± 9.1	25.9 ± 8.2	31.2 ± 11.4
11	40.8 ± 7.3^{ab}	32.9 ± 9.7^{b}	32.0 ± 7.3^{b}	42.4 ± 8.6^{a}
13	41.0 ± 11.0	36.9 ± 8.1	42.4 ± 2.9	36.9 ± 19.1
15	42.6 ± 6.5	42.3 ± 9.2	45.2 ± 4.3	40.5 ± 11.6
17	53.0 ± 9.0	43.0 ± 2.9	38.7 ± 12.1	41.3 ± 14.4
19	51.0 ± 10.4	37.8 ± 13.8	38.8 ± 5.7	39.7 ± 6.9
21	40.8 ± 11.6	41.1 ± 11.6	36.5 ± 6.9	42.4 ± 17.6
$\bar{\bar{X}}$	37.7 ± 11.8^{a}	33.8 ± 11.0^{b}	33.6 ± 10.7^{b}	36.2 ± 11.5^{ab}

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

ab Means with different superscript(s) within each row differ (P .05).

TABLE 10: Means and standard deviations for Neutrophil counts (%) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	49.8 ± 12.7	58.3 ± 10.1	54.8 ± 5.6	55.7 ± 11.0
1	63.3 ± 15.9	59.2 ± 13.9	66.9 ± 10.4	55.0 ± 11.0
2	53.1 ± 11.0	59.2 ± 14.3	64.8 ± 14.0	59.9 ± 9.3
3	54.3 ± 8.2	64.8 ± 8.2	63.8 ± 8.3	56.8 ± 15.1
4	55.6 ± 10.7	55.4 ± 9.1	57.8 ± 6.9	50.5 ± 20.7
5	54.8 ± 15.3	58.6 ± 9.6	60.3 ± 6.1	56.3 ± 11.2
7	58.0 ± 9.9	63.9 ± 12.6	62.8 ± 7.8	64.9 ± 14.3
9	62.5 ± 6.3	62.6 ± 13.0	64.5 ± 7.2	59.4 ± 11.3
11	50.5 ± 9.2	52.7 ± 9.6	53.0 ± 7.9	49.2 ± 9.7
13	52.0 ± 11.3	55.4 ± 5.8	53.0 ± 12.0	58.2 ± 15.7
15	57.2 ± 9.9	50.1 ± 8.6	53.0 ± 3.3	51.1 ± 12.1
17	43.8 ± 8.9	48.3 ± 6.7	55.3 ± 12.9	57.4 ± 13.9
19	44.3 ± 10.7	52.3 ± 13.4	51.0 ± 3.7	57.7 ± 6.6
21	54.3 ± 11.0	49.6 ± 11.9	61.6 ± 4.0	54.3 ± 13.4
$\overline{X}$	54.9 ± 12.7^{b}	56.9 ± 11.5^{ab}	59.7 ± 9.7^{a}	56.4 ± 12.9^{b}

$\overline{X}$ = Means of means.

ab Means with different superscript(s) within each row differ (P .05).

TABLE 11: Means and standard deviations for monocyte counts (%) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	2.6 ± 1.3[a]	2.5 ± 1.1[ab]	1.5 ± 0.9[b]	2.3 ± 0.9[ab]
1	5.5 ± 2.4	5.9 ± 3.3	4.4 ± 2.3	5.5 ± 2.2
2	4.4 ± 2.2	3.8 ± 2.7	3.6 ± 4.1	1.7 ± 1.2
3	1.7 ± 1.0	2.7 ± 2.6	1.8 ± 1.7	3.4 ± 1.9
4	1.7 ± 0.8	0.9 ± 1.2	1.1 ± 1.2	2.1 ± 1.6
5	4.8 ± 1.8	4.6 ± 2.3	3.9 ± 3.7	3.3 ± 2.1
7	4.3 ± 3.6	3.4 ± 2.2	2.5 ± 2.9	2.4 ± 2.5
9	2.8 ± 1.8	2.4 ± 2.4	1.9 ± 1.6	2.9 ± 2.6
11	3.5 ± 2.1	5.6 ± 2.7	3.4 ± 2.4	3.5 ± 1.8
13	1.2 ± 2.7	0.7 ± 0.4	0.6 ± 1.1	0.5 ± 1.3
15	1.2 ± 1.8	1.9 ± 2.0	0.8 ± 1.8	1.3 ± 1.4
17	0.2 ± 0.4	1.4 ± 3.1	1.0 ± 1.4	2.3 ± 1.5
19	0.5 ± 0.5	1.3 ± 1.5	0.3 ± 0.5	0.5 ± 0.8
21	0.7 ± 0.5[a]	0.0 ± 0[b]	0.5 ± 0.6	0.4 ± 0.5[ab]
$\bar{X}$	2.7 ± 2.5	2.7 ± 2.7	2.2 ± 2.6	2.5 ± 2.2

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

ab Means with different superscript(s) within each row differ (P .05).

TABLE 12: Means and standard deviations for serum iron values (um/l) of sheep infected with varying levels of <u>Haemonchus</u> larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	24.7 ± 1.1	23.8 ± 2.9	24.8 ± 2.4	23.2 ± 2.3
1	24.2 ± 3.6	24.1 ± 2.6	24.9 ± 3.1	24.5 ± 3.7
2	22.0 ± 3.3	21.9 ± 2.8	24.1 ± 1.9	24.0 ± 3.1
3	23.2 ± 1.8	21.1 ± 3.3	22.7 ± 3.6	23.6 ± 3.1
4	22.2 ± 3.4^{ab}	19.2 ± 4.4^{b}	21.7 ± 4.0^{ab}	23.4 ± 2.6^{a}
5	22.2 ± 2.2	22.6 ± 2.8	21.7 ± 4.4	23.1 ± 3.5
7	19.4 ± 1.0	21.7 ± 2.3	19.8 ± 3.8	21.8 ± 2.8
9	20.9 ± 1.2	22.3 ± 2.0	22.6 ± 2.8	23.1 ± 1.5
11	23.2 ± 0.9^{ab}	24.2 ± 1.9^{ab}	22.9 ± 4.1^{b}	25.7 ± 1.4^{a}
13	20.3 ± 1.2	20.5 ± 3.7	20.7 ± 3.2	23.0 ± 2.5
15	18.4 ± 1.9	19.0 ± 3.6	18.7 ± 2.9	19.8 ± 3.1
17	21.1 ± 2.2^{ab}	21.4 ± 3.2^{ab}	19.0 ± 3.1^{b}	23.9 ± 1.8^{a}
19	19.1 ± 2.3	17.8 ± 2.8	18.3 ± 3.1	20.2 ± 2.2
21	18.9 ± 2.7^{ab}	16.4 ± 2.7^{b}	17.5 ± 2.0^{ab}	20.2 ± 0.8^{a}
$\bar{X}$	21.7 ± 2.9^{b}	21.3 ± 3.6^{b}	22.1 ± 3.8^{b}	23.0 ± 2.9^{a}

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

^{ab} Means with different superscript(s) within each row differ (P .05).

TABLE 13: Means and standard deviations for Total Protein values (gm/dl) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	5.6 ± 0.7	5.6 ± 0.4	5.9 ± 0.6	5.9 ± 0.4
1	5.4 ± 0.9	5.5 ± 0.6	5.0 ± 0.8	5.7 ± 0.8
2	5.8 ± 1.5	5.6 ± 0.6	5.4 ± 0.6	5.8 ± 0.6
3	5.7 ± 1.0	5.5 ± 0.6	5.3 ± 0.6	5.9 ± 0.6
4	5.5 ± 0.9^{ab}	5.0 ± 0.8^{b}	5.2 ± 0.7^{ab}	5.8 ± 0.6^{a}
5	5.9 ± 1.1	5.9 ± 0.6	5.5 ± 0.8	6.0 ± 0.5
7	5.9 ± 0.8	5.5 ± 0.8	5.5 ± 0.8	5.9 ± 0.4
9	5.4 ± 0.5	5.2 ± 0.6	5.5 ± 0.8	5.5 ± 0.2
11	6.4 ± 0.9^{a}	5.9 ± 0.7^{ab}	5.6 ± 0.5^{ab}	5.4 ± 0.6^{b}
13	6.2 ± 0.2	6.0 ± 0.5	5.9 ± 0.7	6.1 ± 0.5
15	5.3 ± 0.9	5.7 ± 0.4	5.6 ± 1.0	6.0 ± 0.4
17	6.2 ± 0.8^{ab}	5.8 ± 0.4^{ab}	5.5 ± 1.3^{b}	6.4 ± 0.4^{a}
19	5.2 ± 0.9	5.2 ± 0.5	5.6 ± 0.4	5.6 ± 0.2
21	5.9 ± 0.6^{ab}	6.0 ± 0.5^{a}	5.9 ± 0.4^{ab}	5.5 ± 0.1^{b}
$\overline{X}$	5.7 ± 0.9^{ab}	5.6 ± 0.6^{bc}	5.5 ± 0.7^{c}	5.8 ± 0.6^{a}

$\overline{X}$ = Mean of means.

abc Means with different superscript(s) within each row differ (P .05).

TABLE 14: Means and standard deviations for Albumin values (%) of sheep infected with varying levels of <u>Haemonchus</u> larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	50.6 ± 5.8	48.7 ± 3.3	52.1 ± 4.1	49.2 ± 6.8
1	47.4 ± 9.6	47.7 ± 5.3	50.0 ± 7.4	47.6 ± 6.7
2	47.8 ± 7.0	52.7 ± 3.6	49.8 ± 6.9	49.5 ± 5.4
3	45.9 ± 5.5	48.9 ± 4.1	47.4 ± 5.4	47.2 ± 5.4
4	44.6 ± 3.3	49.3 ± 3.9	50.2 ± 7.0	50.1 ± 5.2
5	43.3 ± 6.9^{b}	48.1 ± 3.9^{ab}	48.3 ± 6.4^{ab}	51.1 ± 4.3^{a}
7	46.9 ± 4.2	50.9 ± 3.7	51.9 ± 7.8	47.5 ± 4.4
9	45.9 ± 4.4	50.5 ± 3.1	48.8 ± 3.1	49.2 ± 4.6
11	50.6 ± 2.4	49.9 ± 4.5	50.8 ± 6.7	51.8 ± 3.6
13	52.2 ± 3.1	48.4 ± 5.1	50.1 ± 7.3	50.3 ± 4.3
15	43.3 ± 9.0	43.3 ± 7.4	46.9 ± 5.5	47.8 ± 7.5
17	45.0 ± 10.2	47.6 ± 5.3	49.9 ± 6.7	52.0 ± 4.0
19	46.2 ± 6.2	51.7 ± 5.6	51.4 ± 2.8	49.7 ± 3.2
21	42.9 ± 3.3^{b}	50.1 ± 5.4^{a}	52.8 ± 1.7^{a}	51.6 ± 4.2^{a}
$\bar{\bar{X}}$	46.9 ± 6.6^{b}	49.1 ± 4.9^{a}	49.9 ± 6.0^{a}	49.5 ± 5.2^{a}

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

ab Means with different superscript(s) within each row differ (P .05)

TABLE 15: Means and standard deviations for alpha-1 globulin values (%) of sheep infected with varying levels of *Haemonchus* larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	4.0 ± 0.7	4.5 ± 1.0	4.5 ± 0.9	4.4 ± 0.9
1	5.9 ± 3.2	5.9 ± 2.9	5.8 ± 3.5	4.9 ± 1.6
2	4.2 ± 2.3	3.5 ± 1.7	5.4 ± 2.9	4.1 ± 1.1
3	4.7 ± 1.4^{b}	5.6 ± 1.7^{ab}	6.5 ± 1.4^{a}	4.5 ± 1.6^{b}
4	4.3 ± 1.6	4.3 ± 1.1	5.2 ± 1.1	4.1 ± 1.2
5	4.2 ± 1.9	3.5 ± 1.9	3.9 ± 1.9	3.8 ± 0.9
7	5.7 ± 2.1	4.5 ± 2.3	4.4 ± 1.5	4.2 ± 1.1
9	6.5 ± 0.8^{a}	5.9 ± 1.4^{a}	6.2 ± 1.2^{a}	4.9 ± 0.9^{b}
11	3.5 ± 1.0	4.1 ± 1.7	4.6 ± 1.3	3.8 ± 1.5
13	3.8 ± 0.9	3.1 ± 1.2	3.3 ± 1.7	3.4 ± 1.2
15	3.5 ± 1.5	3.4 ± 1.8	3.2 ± 1.2	3.6 ± 1.3
17	3.6 ± 1.6	3.8 ± 1.3	3.7 ± 1.4	4.3 ± 1.1
19	2.8 ± 1.2	3.9 ± 2.0	3.9 ± 0.7	4.1 ± 1.0
21	3.2 ± 1.0	3.9 ± 1.9	4.2 ± 1.4	3.5 ± 0.7
$\bar{X}$	4.3 ± 1.9^{b}	4.3 ± 1.9^{b}	4.8 ± 2.0^{a}	4.1 ± 1.2^{b}

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

ab Means with different superscript(s) within each row differ. (P .05)

TABLE 16: Means and standard deviations for Alpha-2 glubulin values (%) of sheep infected with varying levels of *Haemonchus* larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	12.8 ± 2.4	12.9 ± 2.4	12.5 ± 2.2	12.2 ± 0.8
1	13.6 ± 4.8	11.9 ± 2.5	12.1 ± 2.4	10.9 ± 2.5
2	10.2 ± 2.7	9.2 ± 1.3	11.1 ± 2.7	8.9 ± 2.1
3	12.9 ± 2.1	12.3 ± 1.2	12.8 ± 2.0	12.3 ± 1.0
4	12.2 ± 3.2	10.9 ± 2.1	12.4 ± 2.5	10.9 ± 1.5
5	10.6 ± 3.2	10.7 ± 2.9	11.7 ± 1.8	12.5 ± 2.4
7	11.7 ± 3.3	12.2 ± 2.6	11.1 ± 1.7	11.9 ± 2.2
9	13.3 ± 2.0	13.5 ± 1.9	12.9 ± 2.2	12.2 ± 1.8
11	10.8 ± 1.5	11.2 ± 1.5	12.2 ± 2.1	10.5 ± 1.9
13	11.8 ± 1.8	10.8 ± 2.5	12.9 ± 2.7	10.5 ± 1.7
15	13.6 ± 4.5	12.8 ± 2.7	14.4 ± 6.0	11.8 ± 1.2
17	13.6 ± 3.8	13.3 ± 3.4	13.2 ± 1.5	11.8 ± 1.6
19	9.4 ± 2.8^{b}	10.0 ± 1.4^{ab}	12.1 ± 0.6^{a}	11.8 ± 0.7^{a}
21	10.9 ± 2.1	9.6 ± 2.8	11.1 ± 1.7	8.7 ± 1.7
$\bar{X}$	12.1 ± 3.2^{a}	11.6 ± 2.5^{ab}	12.3 ± 2.5^{a}	11.3 ± 2.0^{b}

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

ab Means with different superscript(s) within each row differ (P .05)

TABLE 17: Means and standard deviations for Beta globulin values (%) of sheep infected with varying levels of *Haemonchus* larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	8.1 ± 1.2	7.7 ± 1.9	6.9 ± 1.5	7.0 ± 1.1
1	9.8 ± 3.9^{a}	7.3 ± 3.2^{ab}	8.9 ± 3.2^{ab}	6.6 ± 2.0^{b}
2	7.1 ± 1.7	6.0 ± 1.3	7.4 ± 2.3	6.4 ± 1.6
3	8.9 ± 2.8^{a}	5.9 ± 1.3^{b}	8.1 ± 2.9^{ab}	6.5 ± 1.4^{b}
4	7.0 ± 1.2	7.5 ± 2.3	6.3 ± 2.2	6.3 ± 1.0
5	7.4 ± 1.9	6.0 ± 2.0	7.4 ± 2.1	5.9 ± 1.0
7	9.0 ± 1.8	6.8 ± 1.3	7.7 ± 1.8	8.0 ± 2.1
9	9.1 ± 1.9	7.3 ± 1.3	8.4 ± 2.9	8.6 ± 2.4
11	6.0 ± 0.9	6.4 ± 1.6	7.2 ± 4.2	6.3 ± 1.7
13	8.6 ± 1.6	8.0 ± 2.7	8.4 ± 3.2	9.0 ± 3.2
15	7.6 ± 1.2	7.9 ± 2.7	10.3 ± 2.9	8.2 ± 3.0
17	7.7 ± 2.1	7.1 ± 1.9	7.4 ± 1.7	6.7 ± 0.9
19	6.1 ± 0.7	6.9 ± 2.4	7.3 ± 0.8	5.9 ± 1.2
21	5.3 ± 1.2	4.8 ± 2.1	6.2 ± 0.6	4.9 ± 1.3
$\bar{\bar{X}}$	7.8 ± 2.3^{a}	6.9 ± 2.2^{b}	7.7 ± 2.6^{a}	7.0 ± 2.1^{b}

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

ab Means with different superscript(s) within each row differ (P ,05).

TABLE 18: Means and standard deviations for Gamma globulin values (%) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	24.4 ± 6.7^{ab}	26.2 ± 3.3^{ab}	23.0 ± 5.2^{b}	27.9 ± 4.8^{a}
1	23.7 ± 7.3	27.9 ± 3.8	24.7 ± 3.8	24.7 ± 5.7
2	28.7 ± 11.1	24.8 ± 4.3	25.7 ± 4.7	26.6 ± 5.2
3	27.6 ± 7.7	27.2 ± 3.5	25.2 ± 4.9	27.9 ± 4.0
4	30.2 ± 8.8	28.0 ± 2.7	26.1 ± 4.6	27.7 ± 4.1
5	34.5 ± 7.4^{a}	28.6 ± 4.9^{ab}	28.4 ± 5.0^{ab}	26.8 ± 4.4^{b}
7	26.7 ± 6.1	26.7 ± 4.1	23.9 ± 5.8	26.7 ± 4.3
9	26.1 ± 4.6	24.0 ± 5.0	24.2 ± 5.7	23.9 ± 3.1
11	27.5 ± 5.9	22.8 ± 2.5	24.3 ± 6.0	23.7 ± 5.1
13	27.0 ± 6.0	25.9 ± 2.8	26.8 ± 4.7	24.8 ± 2.9
15	30.6 ± 9.8^{ab}	33.3 ± 6.4^{a}	23.7 ± 2.4^{b}	23.8 ± 8.3^{b}
17	29.9 ± 5.9	27.5 ± 3.1	25.3 ± 4.1	24.8 ± 3.8
19	32.0 ± 5.5^{a}	27.0 ± 3.5^{b}	25.3 ± 2.7^{b}	24.4 ± 0.7^{b}
21	30.8 ± 5.7^{a}	30.1 ± 3.6^{a}	25.7 ± 3.1^{ab}	22.6 ± 2.2^{b}
$\bar{X}$	28.0 ± 7.2^{a}	27.1 ± 4.4^{a}	25.1 ± 4.7^{b}	25.6 ± 4.7^{b}

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

ab Means with different superscript(s) within each row differ. (P .05).

TABLE 19: Means and standard deviations for Sodium values (um/l) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	130.1 ± 17.6	133.2 ± 16.4	142.5 ± 9.4	140.3 ± 18.2
1	129.1 ± 9.5^{b}	128.9 ± 19.5^{b}	146.4 ± 11.3^{a}	138.9 ± 11.4^{ab}
2	139.4 ± 11.4^{ab}	129.6 ± 13.1^{ab}	144.3 ± 10.9^{a}	127.3 ± 24.9^{b}
3	157.1 ± 16.6^{a}	133.4 ± 17.9^{b}	148.7 ± 9.6^{a}	126.3 ± 19.5^{b}
4	146.3 ± 13.3^{a}	129.3 ± 11.9^{bc}	139.4 ± 7.6^{ab}	122.1 ± 19.2^{c}
5	131.1 ± 7.2^{ab}	119.8 ± 14.2^{b}	138.8 ± 13.1^{a}	118.9 ± 15.9^{b}
7	128.9 ± 20.4	126.8 ± 14.3	142.6 ± 15.4	129.5 ± 8.7
9	140.6 ± 16.3	136.5 ± 12.9	145.9 ± 6.1	137.9 ± 9.0
11	147.9 ± 7.9	141.9 ± 13.1	142.6 ± 6.5	126.7 ± 32.5
13	132.7 ± 8.0	137.8 ± 10.4	139.1 ± 8.9	129.2 ± 10.1
15	134.5 ± 10.7^{a}	132.9 ± 9.2^{a}	141.8 ± 11.7^{a}	119.6 ± 14.3^{b}
17	141.7 ± 11.3^{a}	130.8 ± 6.7^{b}	142.8 ± 8.4^{a}	124.0 ± 11.9^{b}
19	131.8 ± 15.3	124.9 ± 18.3	140.9 ± 6.5	124.8 ± 8.6
21	136.2 ± 14.9^{a}	126.1 ± 5.4^{ab}	136.4 ± 12.2^{a}	117.5 ± 8.2^{b}
$\bar{\bar{X}}$	136.9 ± 15.2^{b}	130.9 ± 14.4^{c}	142.8 ± 10.1^{a}	128.0 ± 17.8^{c}

$\bar{\bar{X}}$ = Mean of means

abc Means with different superscript(s) within each row differ (P .05)

TABLE 20: Means and standard deviations for Potassium values (um/l) of sheep infected with varying levels of Haemonchus larvae

WEEKS AFTER INFECTION	GROUP I	GROUP II	GROUP III	GROUP IV
0	4.7 ± 0.4	4.8 ± 0.4	5.1 ± 0.2	4.7 ± 0.9
1	4.2 ± 0.8^{ab}	4.6 ± 0.7^{ab}	4.9 ± 06^{a}	3.8 ± 1.4^{b}
2	4.4 ± 1.1	4.8 ± 0.6	4.8 ± 0.5	4.2 ± 0.6
3	4.1 ± 1.0	4.6 ± 0.3	4.5 ± 0.6	4.2 ± 0.6
4	4.5 ± 0.3	4.7 ± 0.7	4.6 ± 0.4	4.2 ± 1.0
5	5.0 ± 0.5^{a}	4.9 ± 0.5^{a}	4.7 ± 0.5^{a}	3.8 ± 1.1^{b}
7	5.2 ± 0.4^{a}	4.9 ± 0.6^{ab}	4.8 ± 0.7^{ab}	4.4 ± 0.7^{b}
9	4.8 ± 0.6	4.9 ± 0.7	4.9 ± 0.6	4.6 ± 0.8
11	4.5 ± 0.3	4.4 ± 0.3	4.8 ± 0.4	4.6 ± 0.5
13	4.7 ± 0.3	4.4 ± 0.2	4.5 ± 0.5	4.7 ± 0.6
15	4.3 ± 0.4	4.4 ± 0.2	4.4 ± 0.6	4.5 ± 0.5
17	4.3 ± 0.3	4.1 ± 0.7	4.6 ± 0.8	4.7 ± 0.6
19	4.6 ± 0.5	4.7 ± 0.6	5.3 ± 0.8	4.5 ± 0.5
21	4.5 ± 0.4	4.3 ± 0.2	4.8 ± 0.4	4.4 ± 0.8
$\bar{X}$	4.5 ± 0.6^{bc}	4.6 ± 0.5^{ab}	4.8 ± 0.6^{a}	4.4 ± 0.8^{c}

$\bar{X}$ = Mean of means

abc Means with different superscript(s) within each row differ (P .05).

APPENDIX II

Summary of computations for analysis of variance data of egg counts, PCV, RBC, WBC, serum iron, serum protein, sodium and Potassium values of sheep infected with varying levels of *Haemonchus* larvae.

TABLE 21: Summary of analysis of variance for egg counts, PCV, RBC, WBC, serum iron, serum protein sodium and potassium values of sheep infected with varying levels of Haemonchus larvae

SOURCE OF VARIATION		PCV		HB		RBC		WBC		EGG COUNT		
	DF	MS	F	MS	F	MS	F	MS	F	DF	MS	F
Between Groups	3	114.16	7.49***	18.69	12.98***	16.55	12.94***	10.62	5.39***	2	6357168.5	1.48
Between weeks	13	295.04	19.35***	33.38	23.18***	24.16	18.89***	31.31	15.92***	12	8927707.2	2.08*
Group x week	39	13.19	0.87	1.30	0.90	0.99	0.78	1.99	1.01	24	2745844.7	0.64
Error	397	15.25	-	1.44	-	1.28	-	1.97	-	243	4299094.0	-
Total	452	23.82	-	2.48	-	2.02	-	2.87	-	281	4391681.3	-

* Significant at .05 level of probability.

*** Significant at .001 level of probability.

TABLE 21 continued

SOURCE OF VARIATION	DF	TOTAL PROTEIN MS	TOTAL PROTEIN F	DF	SODIUM MS	SODIUM F	POTASSIUM MS	POTASSIUM F
Between Groups	3	2.49	5.44***	3	4633.51	27.76***	3.42	8.64***
Between weeks	13	1.55	3.38***	13	431.55	2.12	0.86	2.18**
Group x week	39	0.45	0.99	39	250.68	1.23	0.49	1.26
Error	397	0.46	-	377	203.58	-	0.39	-
Total	452	0.51	-	432	246.06	-	0.44	-

** Significant at 0.01 level of probability.

*** Significant at 0.001 level of probability.

TABLE 21 continued

SOURCE OF VARIATION	DF	EOSINOPHIL		LYMPHOCYTE		NEUTROPHIL		MONOCYTE		SERUM IRON	
		MS	F	MS	F	MS	F	MS	F	MS	F
Between Groups	3	74.79	6.03***	439.95	4.33**	371.14	2.91*	15.17	3.66*	45.27	5.58***
Between weeks	13	70.95	5.72***	762.09	7.50***	530.36	4.16***	63.57	15.32***	113.39	13.97***
Group x week	39	13.24	1.07	93.95	0.93	106.48	0.84	3.41	0.82	7.02	0.87
Error	350	12.40	-	270.88	-	127.48	-	4.15	-	8.12	-
Total	405	14.77	-	101.59	-	140.63	-	6.01	-	11.76	-

* Significant at 0.05 level of probability.

** Significant at 0.01 level of probability.

*** Significant at 0.001 level of probability.

TABLE 21 continued

SOURCE OF VARIATION	DF	ALBUMIN		ALPHA-1		ALPHA-2		BETA		GAMMA	
		MS	F	MS	F	MS	F	MS	MS	F	
Between Groups	3	271.47	7.94***	6.83	1.93	16.74	2.36	29.31	5.34**	274.44	11.15***
Between weeks	13	58.43	1.71	20.81	5.88***	26.92	3.79***	17.95	3.27***	53.76	2.18**
Group x week	39	25.64	0.75	2.37	0.67	3.61	0.51	4.50	0.82	36.18	1.47
Error	375	34.21	-	3.54	-	11.67	-	5.47	-	24.61	-
Total	431	36.26	-	4.23	-	7.0	-	6.13	-	28.33	-

* Significant at 0.05 level of probability.

** Significant at 0.01 level of probability.

*** Significant at 0.001 level of probability.

APPENDIX III

Summary of worm recovery data of Groups I, II and III sheep infected with varying levels of Haemonchus larvae.

TABLE 22: Summary of worm recovery data of Group I sheep infected with 35,000 Haemonchus larvae

ANIMAL NUMBER	NO OF PARASITES RECOVERED	% INNOCULUM RECOVERED
074	205	0.59
076	93	0.27
105	250	0.71
112	190	0.54
138	112	0.32
2801*	1205	3.4
2807*	1101	3.1
2811*	476	1.36
2814*	288	0.83
2820*	109	0.31
2821*	22	0.06
2825	234	0.67
Total	4285	-
Group mean	357	1.01

* Died before end of experiment.

TABLE 23: Summary of worm recovery data of Group II sheep infected with 14,000 Haemonchus larvae

ANIMAL NUMBER	N̲O OF PARASITES RECOVERED	% INNOCULUM RECOVERED
104	56	0.4
122	30	0.21
132	78	0.56
136*	134	0.96
141	182	1.3
145	138	0.98
146	200	1.43
150	129	0.92
2806*	750	5.4
2810*	143	1.0
2812*	137	0.98
2813*	126	0.9
Total	2103	-
Group mean	175	1.25

* Died before the end of experiment.

TABLE 24: Summary of worm recovery data of Group III sheep infected with 2,800 *Haemonchus* larvae.

ANIMAL NUMBER	NO OF PARASITES RECOVERED	% INNOCULUM RECOVERED
108	26	0.92
116	19	0.68
127	-	-
129	33	1.2
2805*	19	0.68
2808*	50	1.8
2816*	22	0.78
2817*	53	1.89
2818*	59	2.1
2819*	120	4.3
2822*	61	2.2
2824*	62	2.2
Total	535	-
Group mean	44	1.70

* Died before the end of experiment.

APPENDIX IV

Summary of computations of correlation matrix data of egg counts, haematological and serum parameters of sheep infected with varying levels of Haemonchus larvae.

TABLE 25: Summary of computations of correlation matrix data of egg counts, haematological and serum parameters of sheep infectd with varying levels of Haemonchus larvae.

	Egg count	PCV	Hb	RBC	WBC	Neut.	Eosin.	Monocyte	Lympho-cyte	Serum iron	Total protein	Alb.	Alpha-1	Alpha-2	Beta	Gamma	Sodium
Egg count																	
PCV	-.29**																
Hb	-.27**	.94**															
RBC	-.29**	.98**	.95**														
WBC	-.29**	.93**	.91**	.93**													
Neutrophil	-.14*	.03	.04	.03	.03												
Eosinophil	-.09	-.03	-.05	-.03	.00	-.22**											
Monocyte	-.09	.30**	.32**	.3**	.30**	-.07	.08										
Lymphocyte	-.08	-.10	-.11*	-.09*	-.11*	-.82**	-.14**	-.17									
Serum iron	-.32*	.92	.94**	.93**	.89**	.02	-.03	.28**	-.08								
Total Protein	-.28**	.28	.25**	.28**	.28**	-.19**	.10*	.05	.17**	.29**							
Albumin	-.08	.16**	.21**	.16**	.18**	-.10*	.09*	.08*	.05	.22**	.09*						
Alpha-1	.09	.02	.03	.01	.01	.22**	.05	.08*	-.24**	-.02	-.31**	-.26**					
Alpha-2	-.03	.08*	.04	.08*	.06	.15**	-.16**	-.01	-.09*	.04	-.26**	-.15**	.18**				
Beta	.01	.07	.04	.07	.07	.11	-.01	-.01	-.09*	.03	-.11*	.32	.23**	.26**			
Gamma	.06	.22	-.26**	-.21**	-.23**	-.03	-.01	-.06	.03	-.26	.18**	-.57	-.07	-.26**	-.09		
Sodium	.11*	.04	.02	.05	.05	.03	-.01	.00	-.03	.01	.09*	-.05	.09*	.09*	.12**	.03	
Potassium	-.21**	.08*	.06	.06	.08	.07	.10*	.01	-.11	.06	.07	.17**	-.11	-.05	.02	-.07	.05

* Significant at 5%.

** Significant at 1%.