A STUDY MEASURING METABOLIC PARAMETERS OF FALL WEANLING LAMBS DURING THE ACUTE STAGE OF HAEMONCHOSIS

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PREFACE

This study is concerned with analysis of metabolic parameters in susceptible sheep exposed to various levels of the pathogenic helminth, Haemonchus contortus, during the acute stage of induced disease. The primary objective is to determine alterations in physiologic, hematologic, and biochemical indices routinely monitored in diseased animals in order to formulate a metabolic profile for acute haemonchosis.

The author wishes to express her appreciation to her major adviser, Dr. Helen E. Jordan, for her guidance, enthusiasm, and assistance. Appreciation is also expressed to the other committee members, Dr. Ralph Buckner and Dr. Delbert Whitenack, for their cooperation and assistance. Gratitude is also extended to Dr. L. Claypool for his assistance with the statistical analysis of the data, and Dr. L. Stratton for his assistance in providing financial resources.

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CHAPTER I

INTRODUCTION

Explanation of the Problem

The U.S.D.A. (1976) estimated the direct losses from internal parasites for 1973 to be $39,700,000.00, approximately six percent of the total value, $661,200,000.00, of sheep production for the year. An additional $24,400,000.00 was estimated to include the cost of medication and animal care. Losses of feed efficiency were estimated at $33,600,000.00. These losses emphasize the necessity for developing diagnostic methods for early detection of parasitic infections and research to clarify the pathogenesis of parasitic gastroenteritis in sheep.

Parasitism in a flock of sheep is determined primarily by recognition of clinical signs in conjunction with data from selected laboratory tests. Clinical symptoms usually include anorexia, reduced weight gains or weight loss with resulting poor growth, intermittant diarrhea and constipation, pallor, rough hair coat, depression, and death. Routine laboratory test data includes results from fecal flotations, packed cell volumes, hemoglobin determinations, total serum proteins, albumin levels, white blood cell counts, and albumin:globulin ratios. Parasites commonly incriminated are Haemonchus, Trichostrongylus, Ostertagia, Bunostomum, Chabertia and Oesphagostomum. The pathogenic

mechanisms responsible are not clearly understood. With the advancement of modern technology, some aspects of parasitic gastroenteritis are becoming clarified. Reliable methods for early detection are needed because of the insidious nature of the parasites. Although metabolic profiling of infectious diseases is not always definative, it has become an accepted diagnostic approach. Application of this method should facilitate the understanding and recognition of parasitic gastroenteritis. Variations in experimental methods and individual variation in the host makes extrapolation of previous experimental and field data into a metabolic profile exceedingly difficult. Therefore, an experiment designed to monitor alterations in metabolic parameters through the acute stage of clinical parasitism in sheep due to the most common pathogenic nematode, Haemonchus contortus, was conducted. Determination of a metabolic profile for acute haemonchosis should facilitate early detection of the infection and should contribute valuable information on the pathogenesis of this common ovine helminth disease.

Purpose of the Study

The primary purpose of the research was to monitor alterations in serum electrolyte values of sheep during the acute stage of clinical haemonchosis in conjunction with changes in the hemogram and serum protein levels. Different larval inocula were used to determine the effects exposure dosage and established adult parasite population levels have on parameters measured.

CHAPTER II

LITERATURE REVIEW

The blood sucking activities of larval and adult Haemonchus contortus have been recognized as the major pathogenic mechanism of the severe anemia produced in sheep. Veglia (1915) observed that fourth stage larvae have a piercing mouth apparatus by which they attach to the abomasal mucosa and produce hemorrhage forty-eight hours after entering the host. The fourth ecdysis occurred between the ninth and eleventh days post-inoculation and was preceded by a twenty-four hour quiescent period. Fresh hemorrhage was detected fifteen days after infection, coinciding with the presence of numerous adult worms.

Fourie (1931) studied the hematology and pathology of haemonchosis in sheep. The effects of experimental infection consisted of progressive and fatal disease, anemia with recovery, and cases in which no effects were observed. Icterus, hemaglobinuria, and hemaglobinemia were not present, thereby eliminating hemolysis as an element in the pathogenesis. Disturbances in erythropoiesis, and erythrophagocytosis were not observed. The parasite's ingestion of blood and the hemorrhages produced were considered responsible for the anemia. The anemia was characterized by an initial polycythemia, primarily an erythrocytosis. Active hematopoiesis was evident by the presence of anisocytosis, polychromasia, punctate basophilia, and Howell Jolly bodies in erythrocytes and myloid hyperplasia of the bone marrow. Terminally,

exhaustion of blood-forming organs was evident by 'pessaforamen' (pale cells), poikilocytosis, and basophilic stippling. The decrease in volume:color index suggested a relative deficiency in hemoglobin. Fourie's findings suggested that the anemia of haemonchosis was due primarily to gastric hemorrhage with compensatory failure and final exhaustion of the hematopoietic system.

Evidence of iron deficiency with haemonchosis and other trichostrongylids was suggested by Whitlock (1950). Depending on the nutritional plane of the host diet, a microcytic, hypochromic, leptomorphic anemia or a macrocytic, hyperchromic, normocytic anemia occurred from infections of either H. contortus or Trichostrongylus spp. and Cooperia spp.. The microcytic, hypochromic, leptomorphic anemia appeared with an iron deficiency.

Baker et al. (1959) established that iron deficiency was responsible for the terminal phase of mixed trichostrongyle infections due primarily to H. contortus. There was no interference with mobilization of liver iron stores. Plasma iron levels did not decrease until liver iron stores fell below 0.7 to 2.5 mg per 100 g of fresh tissue. Plasma iron decreased slightly prior to or simultaneously with decreases in hemoglobin. Total iron binding capacity did not increase significantly in severely anemic animals, suggesting that other complicating factors such as plasma protein loss, poor nutrient absorption, or inadequate transferrin synthesis were present. However, these effects were attributed to the other parasitic genera present.

Kuttler and Marble (1960) studied changes in serum proteins with naturally acquired nematode infections of sheep in which H. contortus predominated. Significant changes were recorded in clinically and

moderately parasitized animals. The total serum protein and albumin levels decreased while globulins increased. Similar changes were produced by phlebotomy induced anemia, but the alpha-2 globulin level was higher and the albumin level was lower in parasitized animals.

Clark et al. (1962) estimated the blood loss caused by H. contortus as 0.049 cc per parasite per day. Blood first appeared in the feces six to twelve days after infection. Two peaks in blood loss were observed, coinciding with eruption of fourth stage larvae and patency.

Brambell and Charleston (1964) detected a decrease in blood loss by measuring fecal radioactivity after the ninth day post-infection in sheep showing age resistance. Blood loss from the sixth to eighth day was caused by fourth stage larvae. Blood loss ten to fourteen days after infection was more severe and caused by fifth stage larvae and adults.

Dyshaematopoiesis was investigated by Charleston (1964). He detected changes in the bone marrow and peripheral blood of parasitized sheep which differed from changes observed in sheep with phlebotomy induced anemia. More pronounced basophilic stippling and polychromasia and megaloblasts with abnormal morphology in the bone marrow were found in parasitized sheep. Mean cell volume and anisocytosis were greater in bled sheep. These differences suggested that interference in maturation and hemoglobination of the erythrocytes occurred due to the parasites.

Charleston (1965) described the gross and microscopic changes in the abomasums of experimentally infected sheep. Four days after infection mucus production increased due to mucosal hyperplasia. Developing larvae were within or below the mucus layer. By day ten, larvae had piercing mouth parts. From the time of the fourth molt, the integrity

of the abomasal mucosa was increasingly disrupted. Hemorrhage was evident six days post-infection. Four to six days after infection an influx of eosinophils and lymphocytes was detected around vessels. Sixteen days after infection large lymphocytes and plasma cells were present in the lamina propria. Lymphoid hyperplasia and eosinophilia were noted in regional lymph nodes by the fourth to eighth day. Tissue eosinophilia peaked between the tenth and twelfth day. Mast cells also increased in the wall of the abomasum and lymph nodes during these periods. The cellular changes were considered to represent repair of the abomasum, expulsion of the parasites, and prevention of establishment of new parasite populations. The peak of lymph node eosinophilia, and the abomasal eosinophilia during lymphoid and plasmacytic infiltration suggested that antigens released during larval molts stimulated mobilization of antibody-producing cells. The hypertrophy of the abomasal mucosa and disruption of the mucosal integrity were considered factors contributing to the hypoproteinemia of haemonchosis.

Silverman et al. (1970) attempted to determine pathological and hematological base lines for different experimental infection levels of H. contortus in lambs. Lambs receiving up to 10,000 larvae (1,500 - 7,650 adults) survived. Two of five lambs died after exposure to 25,000 larvae (10,000 - 14,500 adults). Four of five lambs exposed to 50,000 larvae (5,800 - 10,000 adults) died. Body weight and age were considered important host tolerance factors. Reduction of egg counts and prolongation of patency were observed in animals re-exposed to as many as 10,000 larvae. The greatest resistance to re-exposure was demonstrated in animals receiving 25,000 larvae initially. Resistance was inhibited by initial exposure to 50,000 larvae. These findings

suggested that immunity retarded maturation and adversely affected the fecundity of the parasites. Overwhelming primary infections paralyzed or inactivated immune mechanisms. Anemia based on hemoglobin determinations was evident seven days post-exposure. Evidence of erythropoiesis was detectable during the first seven days of prepatency, regardless of the larval dose. With larval doses of 10,000, 25,000, and 50,000, exhaustion of the hematopoietic system occurred. Death resulted from inadequate synthesis of hemoglobin rather than inadequate erythropoiesis, possibly due to depletion or inadequate stores of iron in growing lambs. Variations in worm burdens of lambs which died indicated the complexity of the relationship between the parasite population and condition of the host and the outcome of an infection. Larval exposure, age, and condition of the host appeared to determine the severity of the disease.

Dargie and Allonby (1975), using radioisotopic, hematologic, and parasitologic techniques, evaluated patterns of erythrocyte loss and hematopoietic activity of sheep and correlated these with parasite egg production, hematologic findings, and iron reutilization. They also monitored hematophagic activity and egg production after challenge, and correlated these with worm burden. In single infections, packed cell volume, hemoglobin, and red blood cell counts fell progressively from the twelfth to twenty-fifth day post-infection. Hemorrhage began seven to ten days after infection and increased for the following ten to fourteen days. There was considerable variability in red cell loss between individual animals. After patency, erythrocyte loss declined for four to ten days then increased or fluctuated in a cyclic manner. Increased red cell production was evident since the

packed cell volumes of most animals were maintained over the post-patent period. Upon challenge, four responses were observed: 'self-cure' and protection, classical 'self-cure', temporary suppression of egg production followed by hyperinfection, and no change. Correlation of erythrocyte loss, egg production, and worm burden showed that the metabolic activity and reproductive efficiency of individual worms was highest in sheep harboring smaller numbers of parasites. During the first eight weeks, there was increased synthesis of hemoglobin, evident by a rapid and progressive increase in iron turnover. Following challenge, 'self-cure' and protection resulted in reduction of plasma ^{59}Fe clearance and improvement of serum iron levels, but plasma iron turnover rate was reduced. With re-infection, plasma iron turnover rate declined because of exhaustion of iron stores. Only a small portion of iron was reabsorbed through the intestine. Reabsorption was greatest in animals suffering the greatest loss. Dargie and Allonby concluded that there were three stages in the development of anemia due to H. contortus. The first stage, occurring from the seventh to the twenty-fifth day post-infection, was characterized by a decreased packed cell volume, low fecal egg counts, and normal serum iron concentrations. This stage was initiated by fourth stage larvae but reflected the voracious blood letting activities of young adult parasites. During this stage, the blood loss was not sufficient nor was there enough time for adequate red cell production. The second stage was characterized by stabilization of hematologic indices for six to fourteen weeks. During this period, hemorrhage was continuous, but erythrocyte production was increased. The third stage represented progressive depletion of the host iron reserves. Iron deficiency resulted from limited reabsorption of

iron lost through hemorrhage.

Sinclair and Pritchard (1975) studied the pathogenicity of arrested fourth stage larvae. Elevations of plasma pepsinogen and abomasal pH, negative dry matter balance, and increased fluid loss including plasma into the gastrointestinal tract were observed. They concluded that arrested larvae damaged the mucosa. There were no alterations in total plasma protein levels, albumin levels, packed cell volumes, hemoglobin concentrations, or total and differential leucocyte counts.

Dargie (1975) explained alterations in serum protein composition with helminth infections. In haemonchosis, the fractional catabolic rate of albumin increases progressively between the first and third week after infection and remains elevated until termination of the infection by the 'self-cure' phenomenon. Diet and inappetance complicate the hypoalbuminemia. The high rate of albumin degradation appears to be due to loss of plasma into the gut. Some of the loss can be explained by the blood sucking activity of the parasite. However, additional losses may represent increased permeability of the hyperplastic mucosa to protein macromolecules. Changes in immunoglobulins also reflect changes in catabolic rates of these proteins which may be related to a homeostatic mechanism controlling colloid osmotic pressure. Therefore, it appears that the distribution of synthesis and extent of catabolism within the various protein pools are altered; and a large portion of the available amino acids are channeled to organs producing proteins needed for survival during the parasitic crisis. The mechanisms involved are not understood, but may be under hormonal control. Protein reabsorption is determined by nitrogen balance studies.

Disturbances in nitrogen balance result from failure of the host to conserve amino acids derived from excessive degradation of muscle and blood proteins. Loss occurs through urinary excretion of nitrogenous materials rather than through impaired digestion and absorption.

Studies measuring changes in serum electrolytes and enzymes due to H. contortus are few. Coop (1971) studied alterations in abomasal pH and electrolyte composition, and plasma pepsinogen levels. Abomasal pH increased, sodium ion concentration increased, and potassium ion and chloride ion concentrations decreased at the time of emergence of fourth stage larvae from the gastric glands. A four-fold increase in plasma pepsinogen levels occurred five days after infection. Serum levels of the electrolytes were normal two weeks after infection. These findings suggested that there was increased permeability of the abomasal mucosa to plasma elements. Increased mucinous secretions from the abomasum may have contributed to the increase in abomasal pH.

Zajicek et al. (1976) measured serum calcium, inorganic phosphorus, magnesium, and buffer capacity in sheep infected with H. contortus and Trichostrongylus colubriformis. Calcium levels decreased and phosphorus levels increased during the period of most intensive development of the parasite. Magnesium levels were unaltered. Buffer capacity was also unaltered. Zajicek et al. (1972) also reported decreased activity of SGOT, SGPT, cholinesterase, and alkaline phosphate in sheep with H. contortus and T. colubriformis.

Yashchenko (1967) measured serum calcium, inorganic phosphorus, and catalase activity in sheep experimentally infected with H. contortus. Inorganic phosphorus and catalase decreased while larval forms of Haemonchus were developing. After parasites reached sexual maturity,

a further decrease in catalase activity and a decrease in serum calcium levels were detected.

Shumard et al. (1957) found rapid weight loss, hyperglycemia, hypophosphatemia, and hypoproteinemia in lambs experimentally infected with Haemonchus contortus, Nematodirus spathiger, and Trichostrongylus colubriformis. Lambs demonstrated variations in susceptibility to acquisition and to affects of the nematodes. Hyperglycemia and hypophosphatemia developed ten to fifteen days after exposure. Hemoglobin and hematocrit values indicated severe haemonchosis in all but one infected animal from which H. contortus was not recovered.

It appears that the anemia of haemonchosis results from frank hemorrhage complicated by depletion of iron reserves and protein loss through a damaged mucosa. Larval stages contribute to the pathogenesis, but adults produce the most severe disturbances. The ultimate outcome depends upon nutritional status, age, and innate ability of the host to resist infection. Immunologic mechanisms function in the host to reduce the parasite burden. Larval exposure, exposure rate, and adult populations of the parasite influence the severity of the disease.

CHAPTER III

MATERIALS AND METHODS

Experimental Animals

Lambs reared worm free were not available for this experiment. Twenty cross-bred fall weanling lambs were purchased from the Fort Reno, Oklahoma State University, Research Facility, and transported to Stillwater, Oklahoma. Fecal flotations and physical examinations were conducted, and the lambs were treated with levamisole hydrochloride orally.

Nutrition

The lambs were on a starter ration consisting of forty percent alfalfa, forty-five percent milo, five percent molasses, and ten percent soybean meal at the time of purchase. They were gradually changed to a ration consisting of eighty percent special thirty-two percent meal, forty-five percent milo, forty-two percent dehydrated alfalfa, and five percent molasses. This ration contained twenty-five grams of aureomycin per ton of feed. The special meal consisted of vitamin A and thirty-two percent protein. The lambs had free access to feed and water.

Housing

The lambs were transported to the Oklahoma State University,

Pawhuska Research Station where they were separated into four groups consisting of five lambs each. They were housed in sheltered pens with dirt floors bedded with straw. The pens were cleaned daily. In Pawhuska, the lambs were treated for coccidia with amprolium in their water at a rate of ten milligram per kilogram of body weight for the first five days. The lambs were observed for a three-week period before the start of the experiment.

Experimental Design

Body weight, heart rate, respiratory rate, and body temperature were monitored weekly during the three-week observation period and the six-week post-infection period. Fecal samples were collected rectally from each animal weekly for fecal flotations. Three successive daily blood samples were drawn from each lamb immediately prior to administration of the infective larvae to determine pre-exposure patterns of the following parameters: erythrocyte count, hemoglobin, hematocrit, white blood cell count, total serum protein level, globulin levels, albumin level, albumin:globulin ratio, and serum magnesium, potassium, phosphorus, chloride, calcium and sodium levels. Blood samples were collected by jugular venipuncture, using vacutainer multiple sample twenty gauge one inch needles, and plain and EDTA vacuum blood collection tubes. Larvae were administered orally at the end of week three. Lambs 1 through 5 served as the uninfected control group. Lambs 6 through 10, group I, received 20,000 larvae. Lambs 11 through 15, group II, received 100,000 larvae. Lambs 16 through 20, group III, received 200,000 larvae. Blood samples were then collected weekly from the lambs to determine post-infection patterns of the above mentioned

parameters until termination of the experiment at week nine, six weeks after exposure. Upon death or euthanasia at the designated time, animals were necropsied. Worm burden estimates and parasite identifications were conducted. An eggs per gram of feces was done with the last fecal specimen collected from each lamb.

Parasitology

Fecal flotations were conducted using saturated sodium nitrate levitation solution. Eggs per gram of feces were determined by the Stoll dilution method (Stoll, 1923). The infective larvae were provided by North Carolina State University, Grinells Animal Health Laboratory, Raleigh, North Carolina. Dosages were determined by suspending the larvae in fifty milliliters of water and counting the number of larvae in 0.005 milliliters. The number of larvae per milliliter of suspension was then calculated.

Worm counts were conducted after necropsy. The digestive tracts were removed and separated into segments consisting of abomasum, small intestine, and large intestine. Each segment was placed in a separate bucket, opened, and washed in cool water by gently massaging the mucosal surface. The contents of each segment were then flushed through a successive series of U. S. standard sieves ranging from a ten mesh to a sixty mesh. The adhering material was then flushed back into a bucket and transferred to one gallon jars. Forty percent formaldehyde was added to the jars in sufficient quantity to approximate a final concentration of ten percent formalin solution. Worm burden estimates were determined by adjusting the volume of the jars containing abomasal and small intestinal contents to 3675 ml. Three thirty-five milliliter

aliquots were withdrawn after suspending the contents by vigorous stirring. The worms in each aliquot were counted using a stereoscopic microscope, and the average of the three counts was multiplied by a dilution factor of 105. Total counts, not aliquots, of the large intestinal contents were made. The parasites in one aliquot of each segment of gastrointestinal tract from each animal were identified to species.

Hematology

Hemoglobin, hematocrit, red blood cell counts, and white blood cell counts were determined from the non-clotted blood samples. Red and white blood cell counts were determined on a Coulter Counter. Hemoglobin was determined by the cyanomethhemoglobin method. Hematocrits were determined by the microhematocrit method.

Serum Electrolytes and Electrophoresis

Clotted blood samples were centrifuged. The serum was pipetted off and transferred to clean tubes for freezing. Serum electrolytes determined were sodium, potassium, chloride, phosphorus, calcium, and magnesium. Magnesium was determined by atomic absorption spectroscopy using a Varion model 1200 atomic absorption spectrometer. The remaining electrolytes were determined on a Coulter-Chem 22 instrument with Coulter reagents. Electrophoresis was done on cellulose acetate using a Helena Electrophoresis System.

Necropsy

Standard necropsy procedure was performed. Abdominal and thoracic

viscera were inspected for gross abnormalities. Tissues were collected and stored in ten percent formalin-saline for reference.

Analysis of Data

The data was subjected to statistical analysis consisting of an analysis of variance procedure and Duncan's multiple range tests. Significant differences were determined at the alpha .05 level ($P < .05$).

CHAPTER IV

RESULTS

General Observations

The lambs were healthy at the time of purchase. Fecal flotation results demonstrated coccidial infections in all of the lambs, a strongyle infection in lamb 12, and Strongyloides infections in lambs 2, 8, 10, 12, 13, 16, 18, and 19. All of the lambs continued to shed oocysts after treatment with amprolium. After treatment with levamisole hydrochloride, Strongyloides eggs were recovered from the feces of lamb 8 during weeks 1, 2, 3, and 9, and lamb 13 during weeks 4, 7, 8, and 9. Trichuris sp. eggs were recovered from the feces of lamb 1 at week 9, lamb 3 at weeks 3 and 6, lamb 9 at weeks 4, 6, 8, and 9, lamb 11 at week 5, lamb 16 at weeks 2, 3, and 7, and lamb 17 at weeks 8 and 9, and lamb 20 at week 6.

All the lambs exposed to infective larvae began shedding strongyle eggs in their feces by week 6, twenty-one days after exposure. Strongyle eggs were recovered from one control lamb's feces, lamb 1, on weeks 7, 8, and 9.

Four animals died during the course of the experiment. Lamb 6 of group I died by the end of week 7, four weeks after exposure. Lamb 15 and lamb 11 of group II died by the end of weeks 6 and 8, respectively. Lamb 20 of group III died by the end of week 6, three weeks after exposure. Clinical signs were observed near death and consisted of pallor,

reluctance to move, weakness, and collapse upon exertion.

Results are presented for significant differences ($P < .05$) in weekly mean levels of the parameters monitored for the four experimental groups. The results of the three successive daily pre-infection bleedings have been considered as weekly values for convenience.

Clinical Examination

The mean weight of the control group at week 1 was significantly higher than the mean weights of the other groups. At weeks 2 and 3, there were no differences in weight between the groups. The mean weights of group I and III were significantly lower than the mean weight of the control group at week 4. There were no significant differences in mean weights at week 5. At week 6, the mean weight of group III was significantly lower than that of the control group, group I, and group II. During weeks 7 and 8, the mean weights of groups I, II, and III were significantly lower than the mean weight of the control group. The mean weight of group III was significantly lower than the mean weights of the control group and group I by week 9. Figure 1, page 19, illustrates the weekly mean weights in pounds of the four groups. Table VI, page 76, lists weekly weights of the lambs and group weekly mean weights and standard deviations.

Significant differences in mean respiratory rate between the four groups were not detected until the fifth week. The mean respiratory rates for groups II and III were higher than the control group mean respiratory rate. At week 6, the mean respiratory rate of group III was significantly lower than that of the control group, group I, and group II. Differences were not detected for the remainder of the

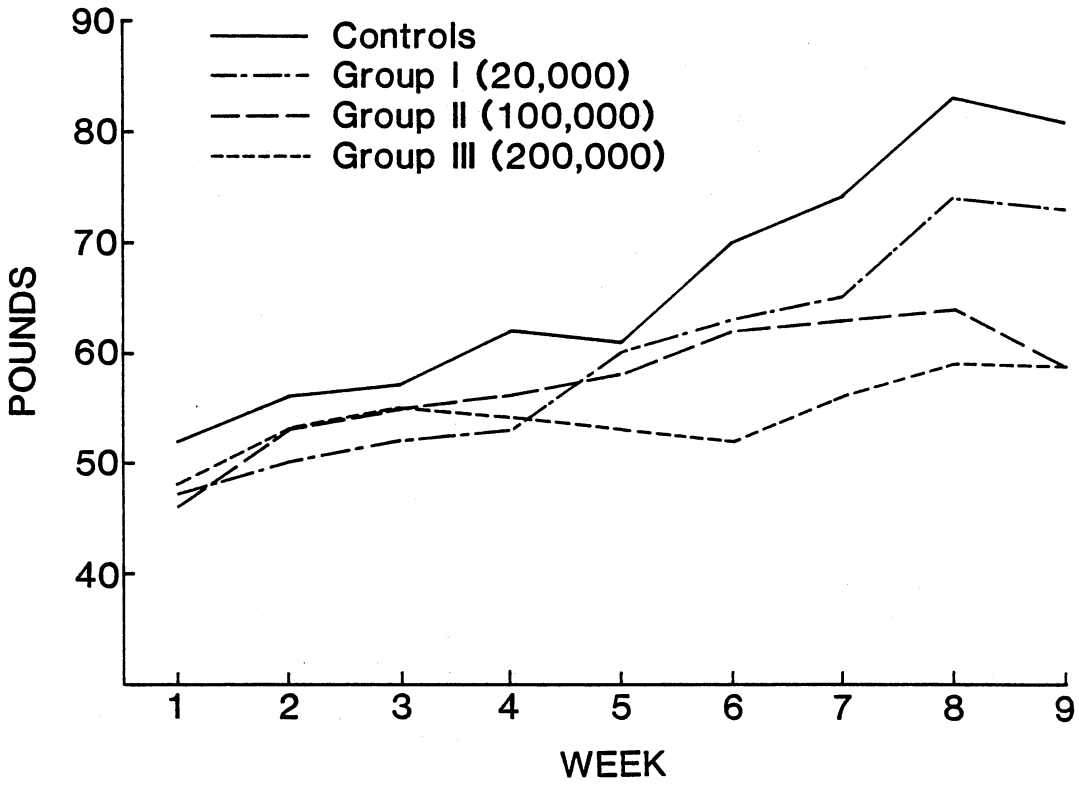

Figure 1. Weekly Mean Weights of the Four Experimental Groups.

experiment. Figure 2, page 21, depicts the weekly mean respiratory rates of the four groups. Table VII, page 77, lists weekly respiratory rates of the lambs, and group weekly mean respiratory rates and standard deviations.

Significant differences in mean heart rates were observed at week 6. The control group mean heart rate was significantly lower than the mean heart rates of group III and I. Significant differences were not detected after week 6. Figure 3, page 22, illustrates the weekly mean heart rates of the four groups. Table VIII, page 78, lists weekly heart rates of the lambs, and group weekly mean heart rates and standard deviations.

Analysis of body temperature means was not performed for week 6 because of inadequate data. The mean body temperature of group I was significantly higher than that of the three other groups at week 2. Differences were not detected at any other time. Figure 4, page 23, illustrates the weekly mean body temperatures of the four experimental groups. Table IX, page 79, list the weekly body temperatures of the lambs, and group weekly means and standard deviations.

Hematology

Hematologic parameters for lamb 17 week 1, lamb 16 week 2, lambs 4, 5, 6, 11, and 12 week 6, lamb 4 week 7, and lamb 9 week 8, were not conducted because the blood samples were clotted. Figure 5, page 24, depicts the weekly mean total white blood cell counts of the four groups. The only significant difference was detected at week 3. The mean white blood cell count for group I was higher than that of the control group and group II. Table X, page 80, lists weekly white blood

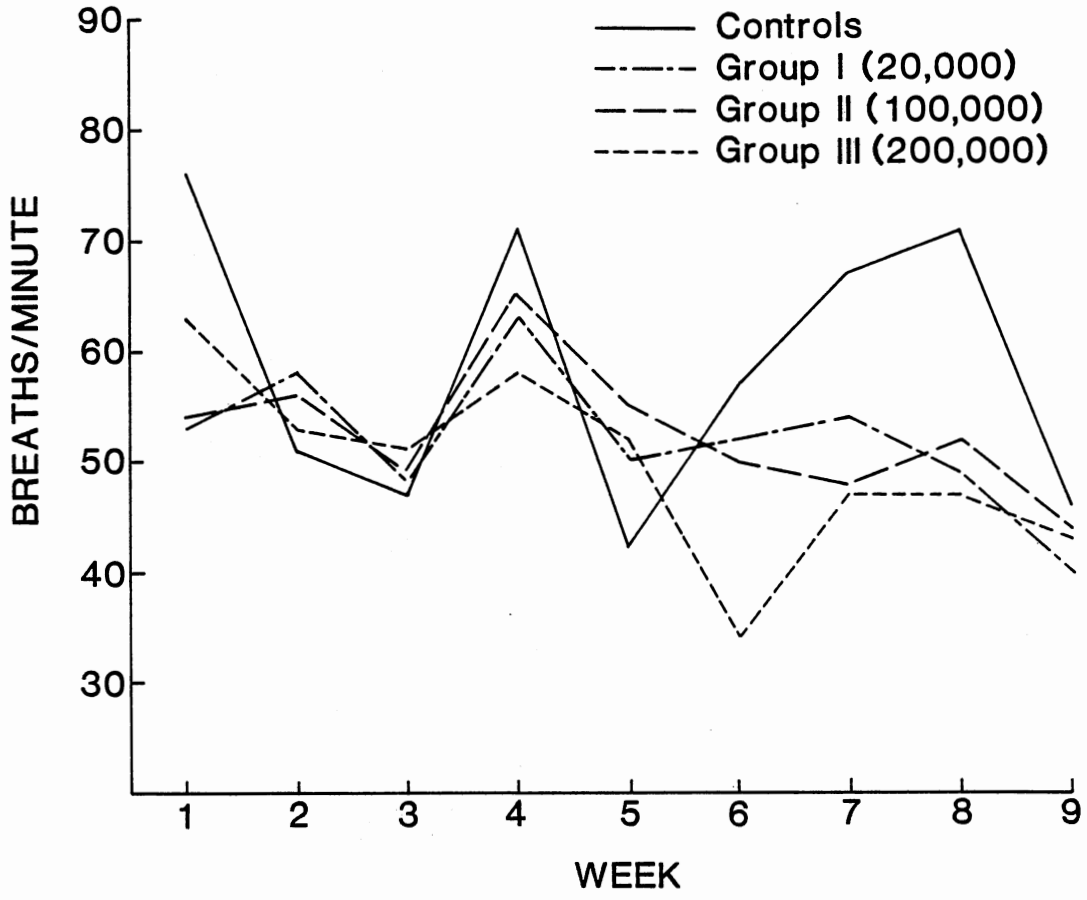

Figure 2. Weekly Mean Respiratory Rates of the Four Experimental Groups.

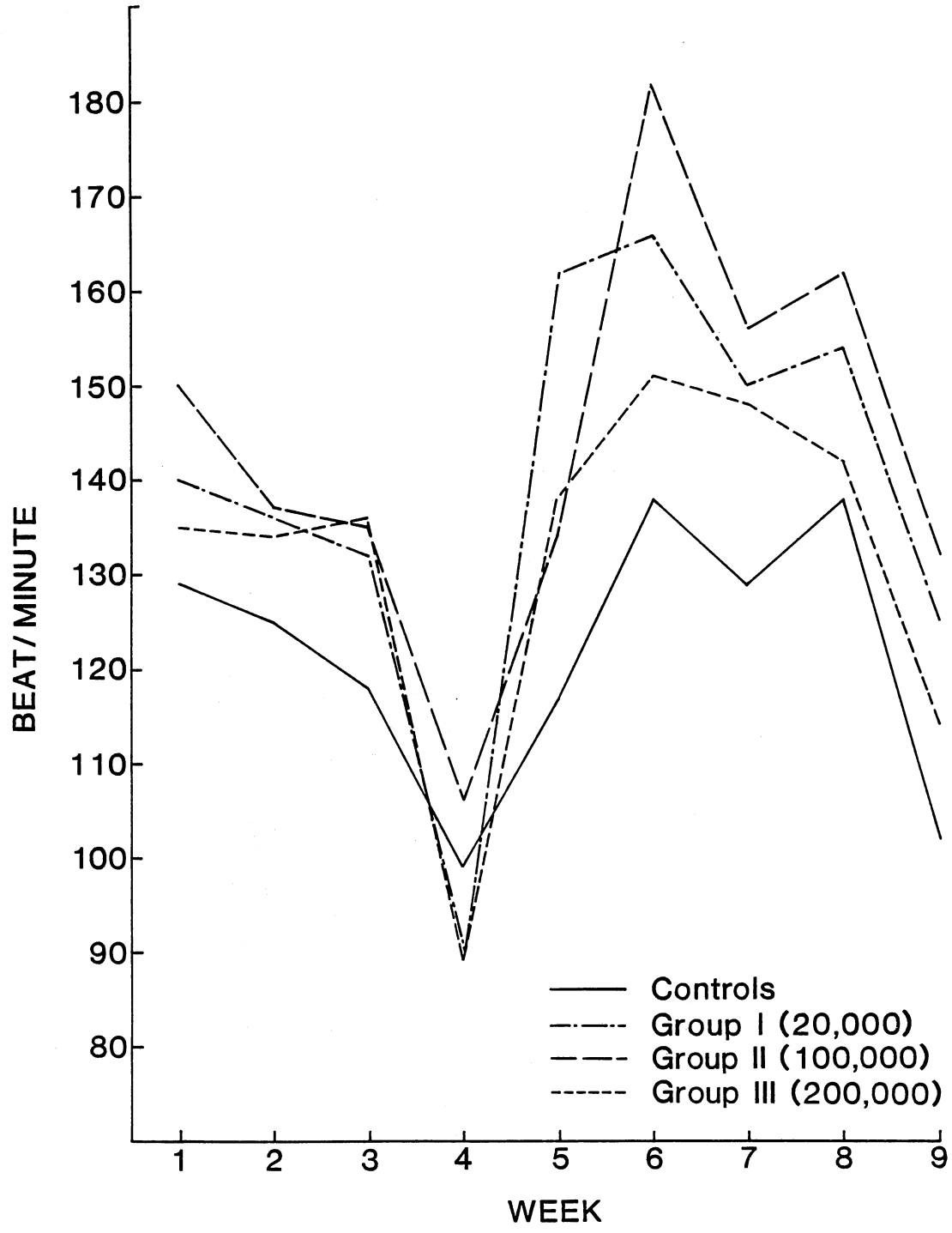

Figure 3. Weekly Mean Heart Rates of the Four Experimental Groups.

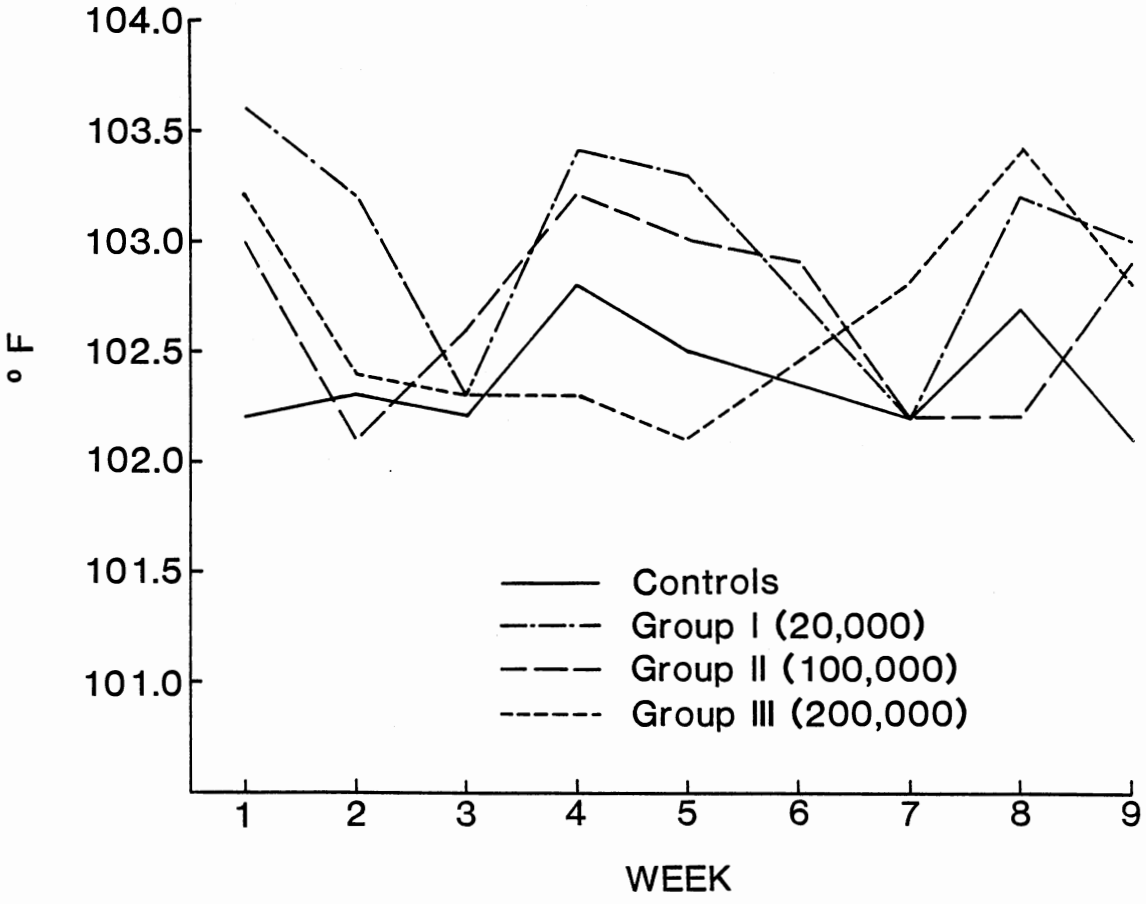

Figure 4. Weekly Mean Body Temperatures of the Four Experimental Groups.

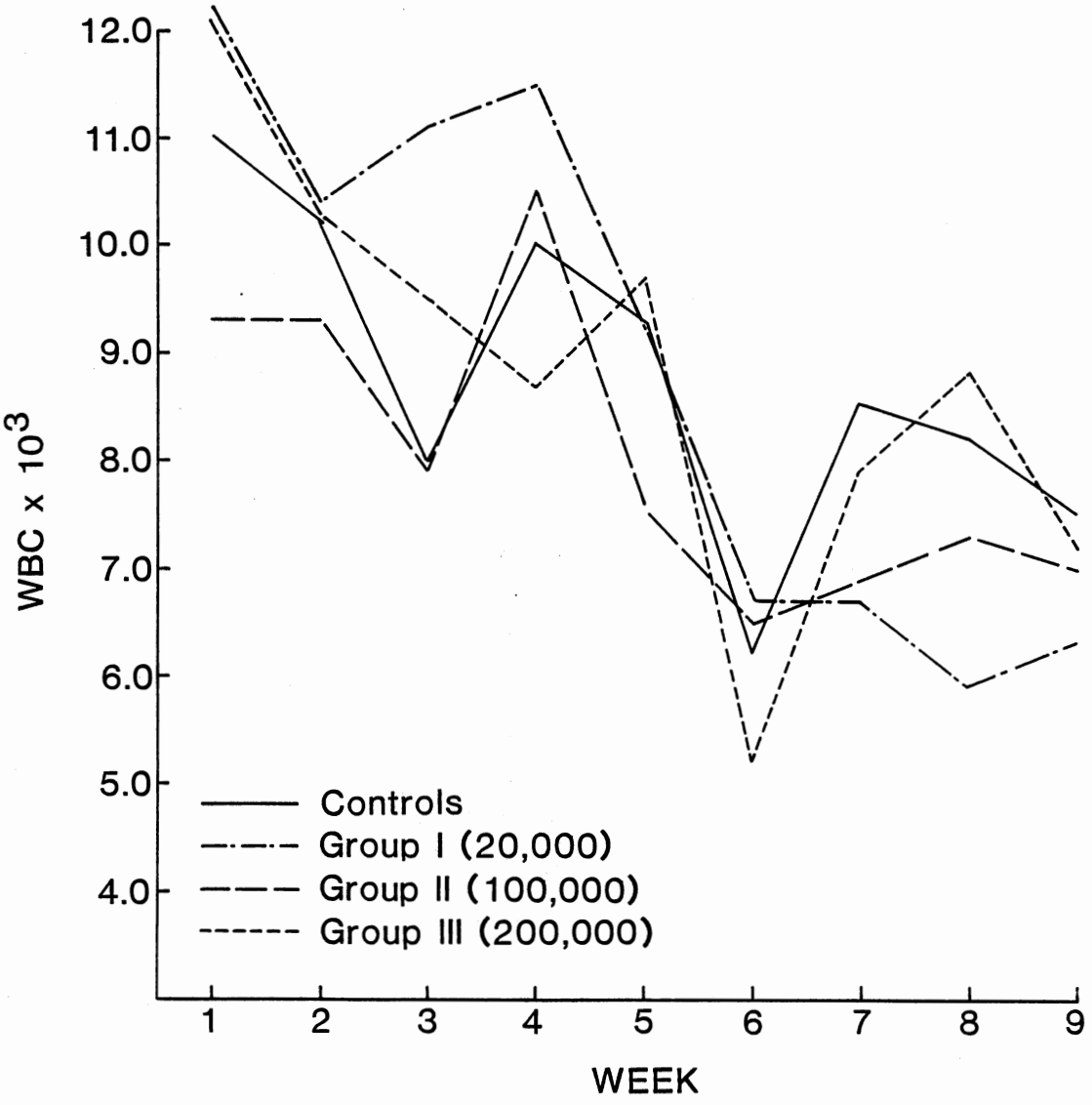

Figure 5. Weekly Mean White Blood Cell Counts of the Four Experimental Groups.

cell counts of the lambs and group means and standard deviations.

Weekly mean erythrocyte counts of the four groups are depicted in figure 6, page 26. The control group mean erythrocyte count was significantly higher than the mean erythrocyte counts of groups II, III, and I during weeks 7 and 8. At week 9, the control group mean erythrocyte count was significantly higher than the mean erythrocyte count of group I. Table XI, page 81, lists weekly erythrocyte counts of the lambs and group weekly means and standard deviations.

Weekly mean hematocrits of the four groups are illustrated in figure 7, page 27. During weeks 7, 8, and 9, the control group mean hematocrits were significantly higher than the mean hematocrits of groups III, II, and I. Table XII, page 82, lists weekly hematocrits of the lambs and group weekly means and standard deviations.

Figure 8, page 28, depicts the weekly mean hemoglobin levels of the four groups. Significant differences were detected at week 5. The mean hemoglobin level of the control group was higher than the mean levels of groups I and II. Differences were not significant at week 6. During weeks 7, 8, and 9, the mean hemoglobin levels of the control group were significantly higher than the mean levels of groups III, II, and I. Table XIII, page 83, lists the weekly hemoglobin levels of the lambs and group weekly mean hemoglobin levels and standard deviations.

Serum Proteins

Serum protein determinations were not performed for lambs 1 and 19 week 1, lamb 4 week 2, lamb 11 week 3, lamb 9 week 4, lamb 18 week 5, and lambs 3, 6, and 11 week 7, because of insufficient serum sample

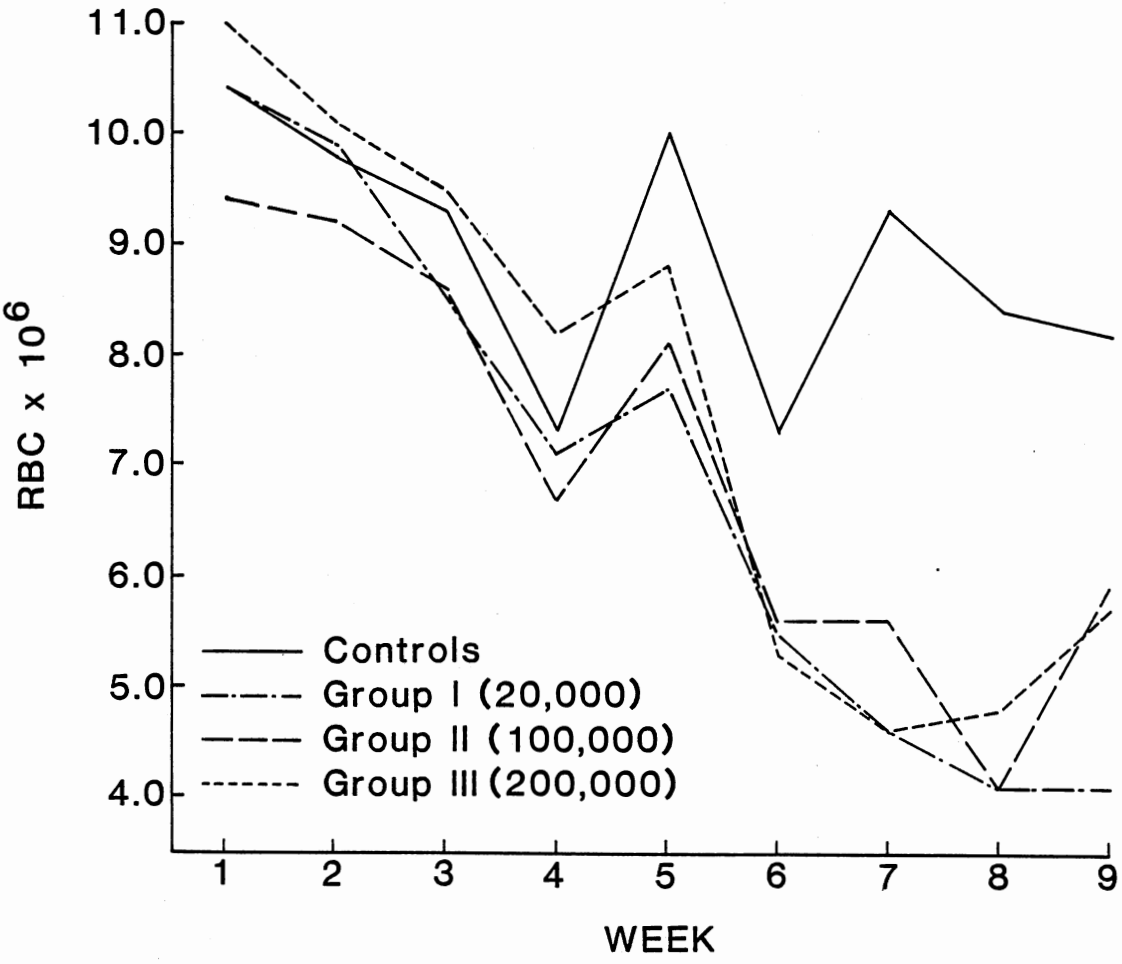

Figure 6. Weekly Mean Erythrocyte Counts of the Four Experimental Groups.

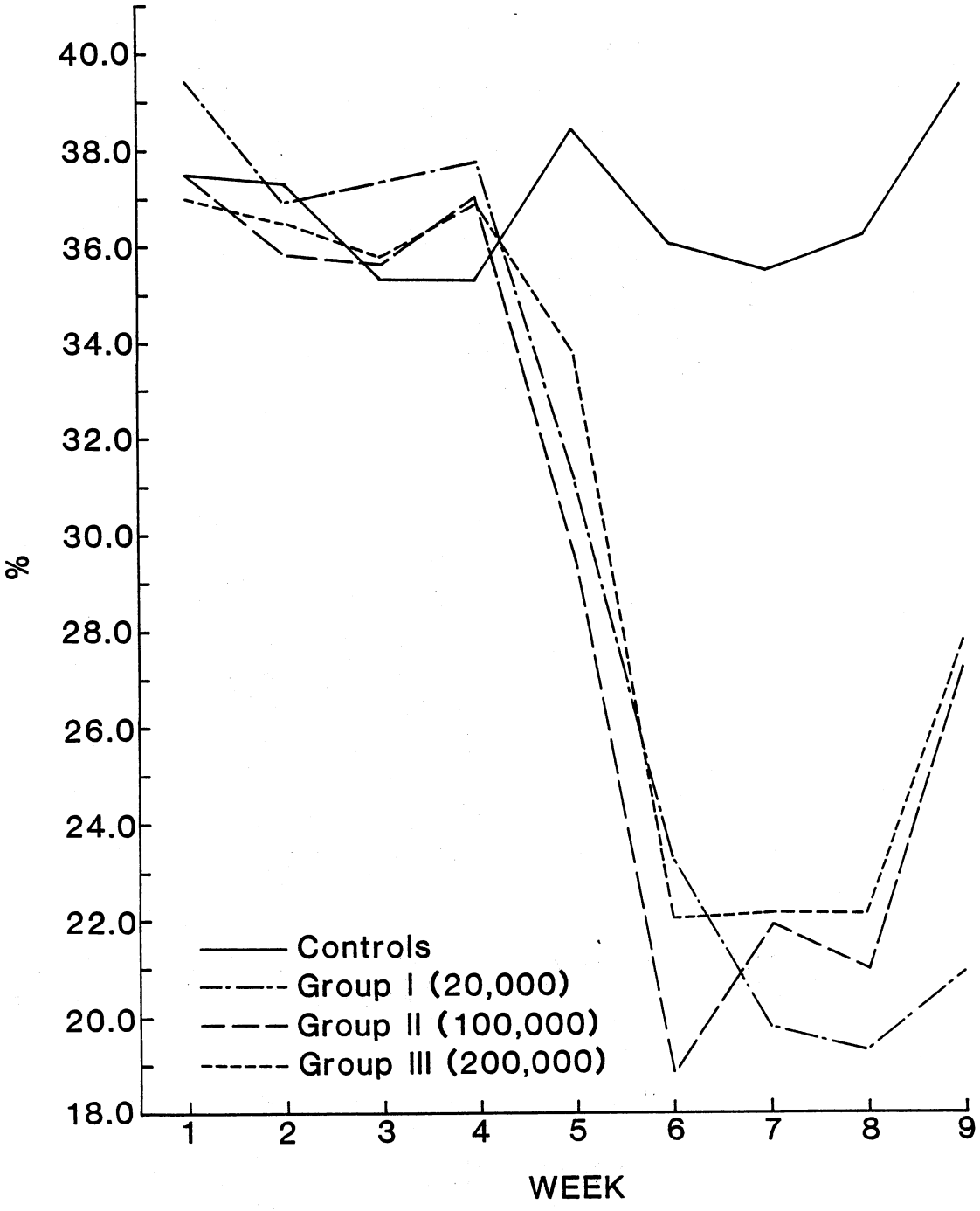

Figure 7. Weekly Mean Hematocrits of the Four Experimental Groups.

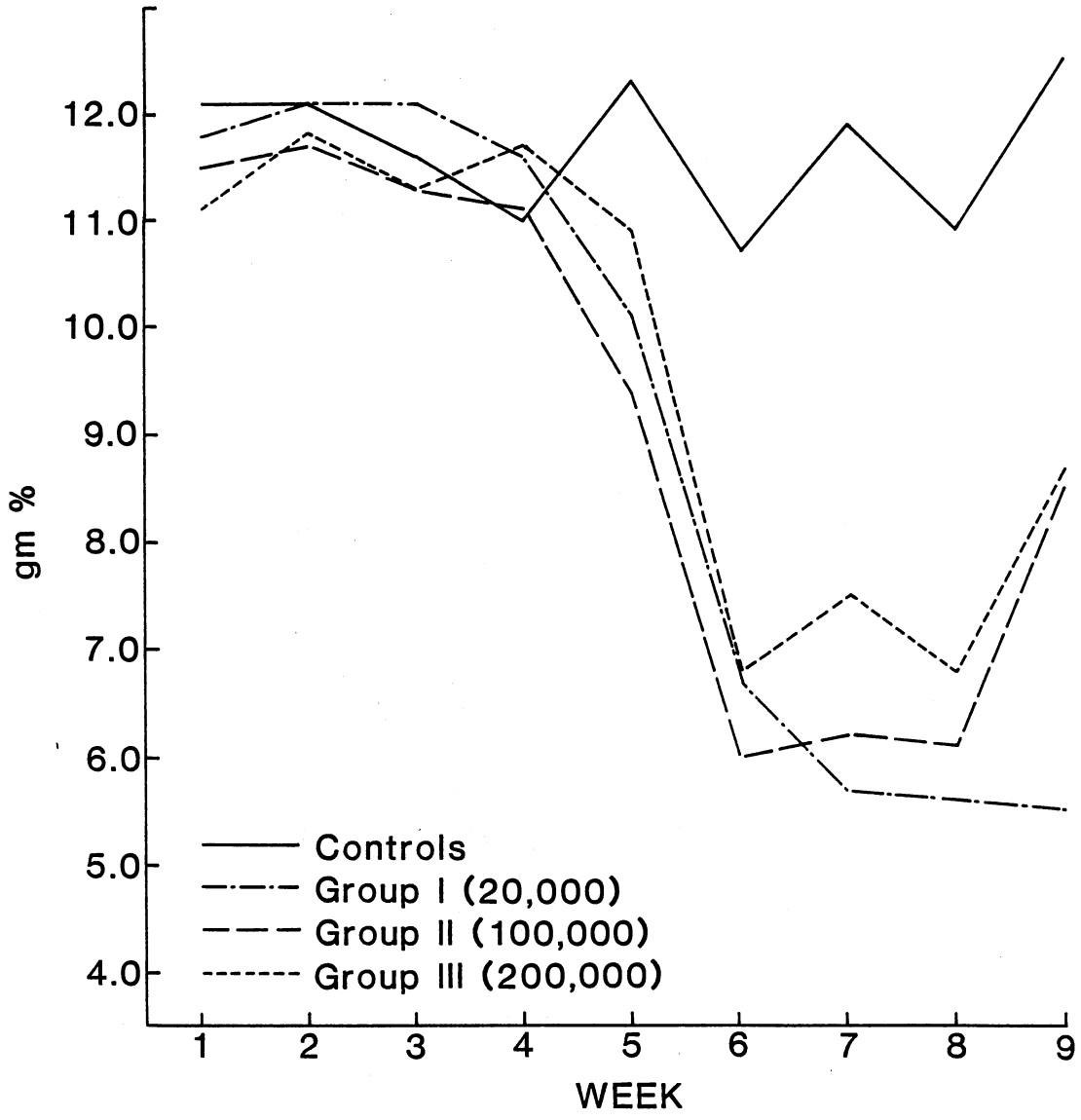

Figure 8. Weekly Mean Hemoglobin Levels of the Four Experimental Groups.

or loss of the samples.

Weekly mean total protein levels of the four groups are presented in figure 9, page 30. The control group mean total protein level was significantly higher than the mean levels of groups I and II during week 5. By week 6, the control group and group II mean total protein levels were significantly higher than the mean total protein level of group I. Differences were not detected during week 7. By week 8, the control group mean total protein level was significantly higher than the levels of groups III, II, and I. Differences were not significant during week 9. Table XIV, page 84, lists weekly total protein levels of the lambs, and group weekly mean total protein levels and standard deviations.

The weekly mean albumin level of the control group was significantly higher than the levels of groups III and I by week 6. Differences were not significant at week 7. During weeks 8 and 9, the control group mean albumin level was higher than the mean levels of the other three groups. Weekly mean albumin levels are depicted in figure 10, page 31. Table XV, page 85, lists weekly albumin levels of the lambs, and group means and standard deviations.

Weekly mean alpha-1 globulin levels of the four groups were not significantly different throughout the course of the experiment. Figure 11, page 32, illustrates the weekly mean alpha-1 globulin levels of the four groups. Table XVI, page 86, lists weekly alpha-1 globulin levels of the lambs, and group means and standard deviations.

Weekly mean alpha-2 globulin levels are depicted in figure 12, page 33. By week 2, the mean alpha-2 globulin level of group III was significantly higher than the alpha-2 globulin levels of group I, the

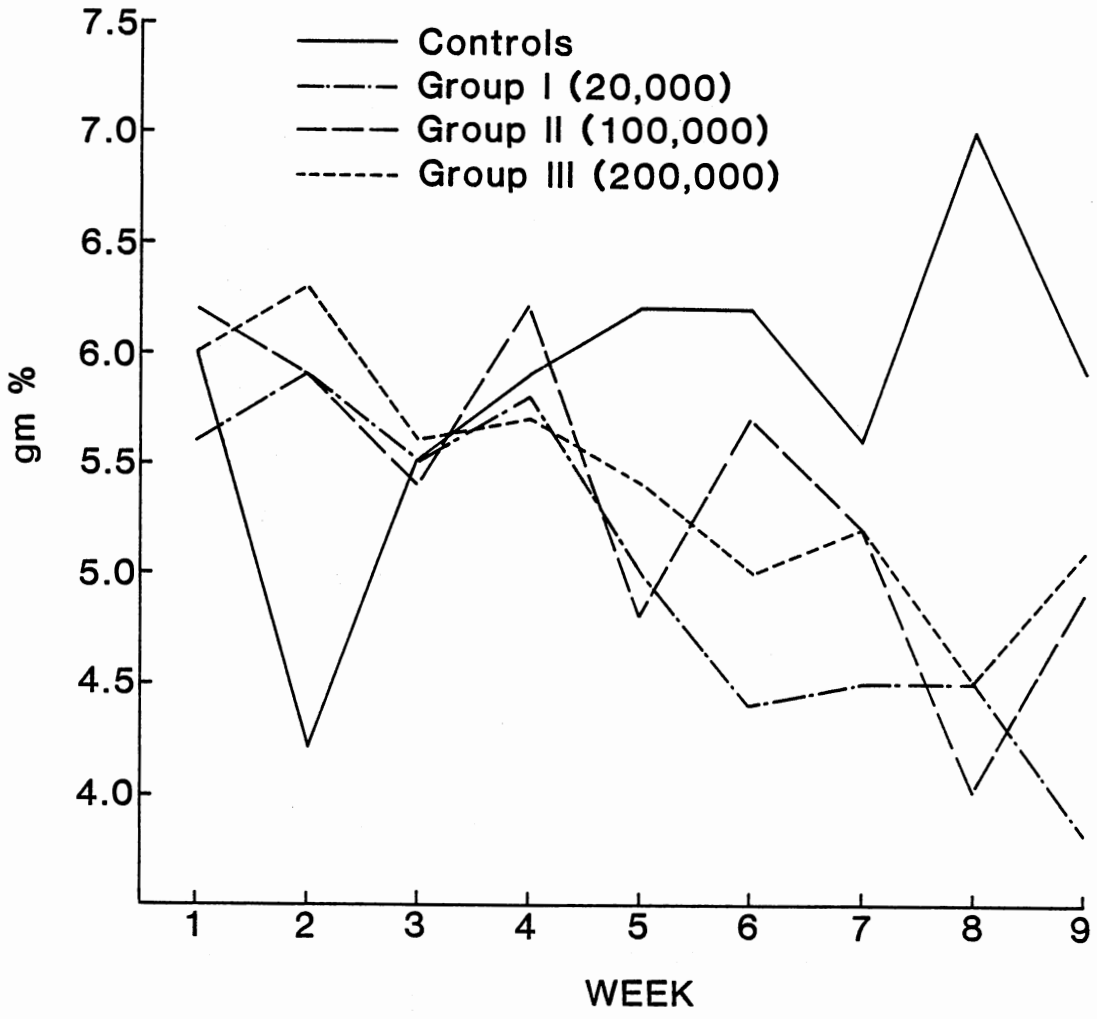

Figure 9. Weekly Mean Total Protein Levels of the Four Experimental Groups.

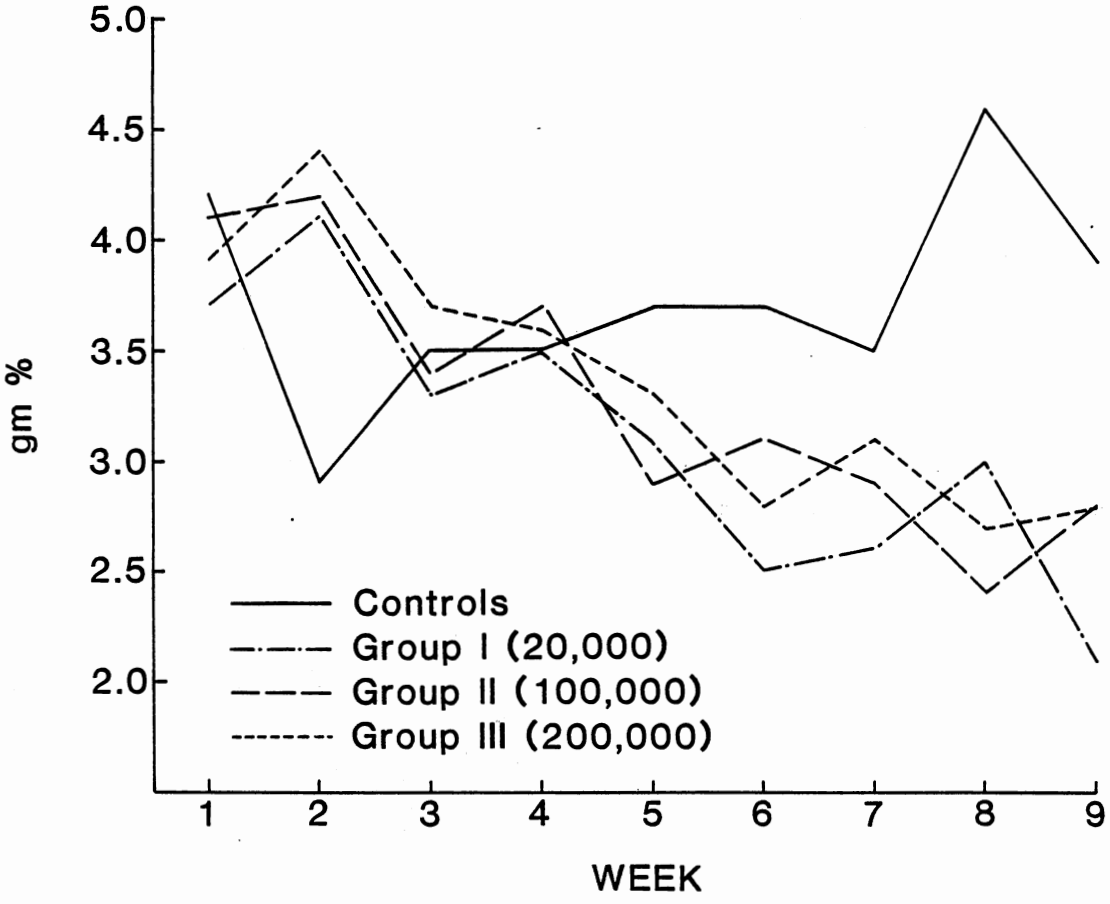

Figure 10. Weekly Mean Albumin Levels of the Four Experimental Groups.

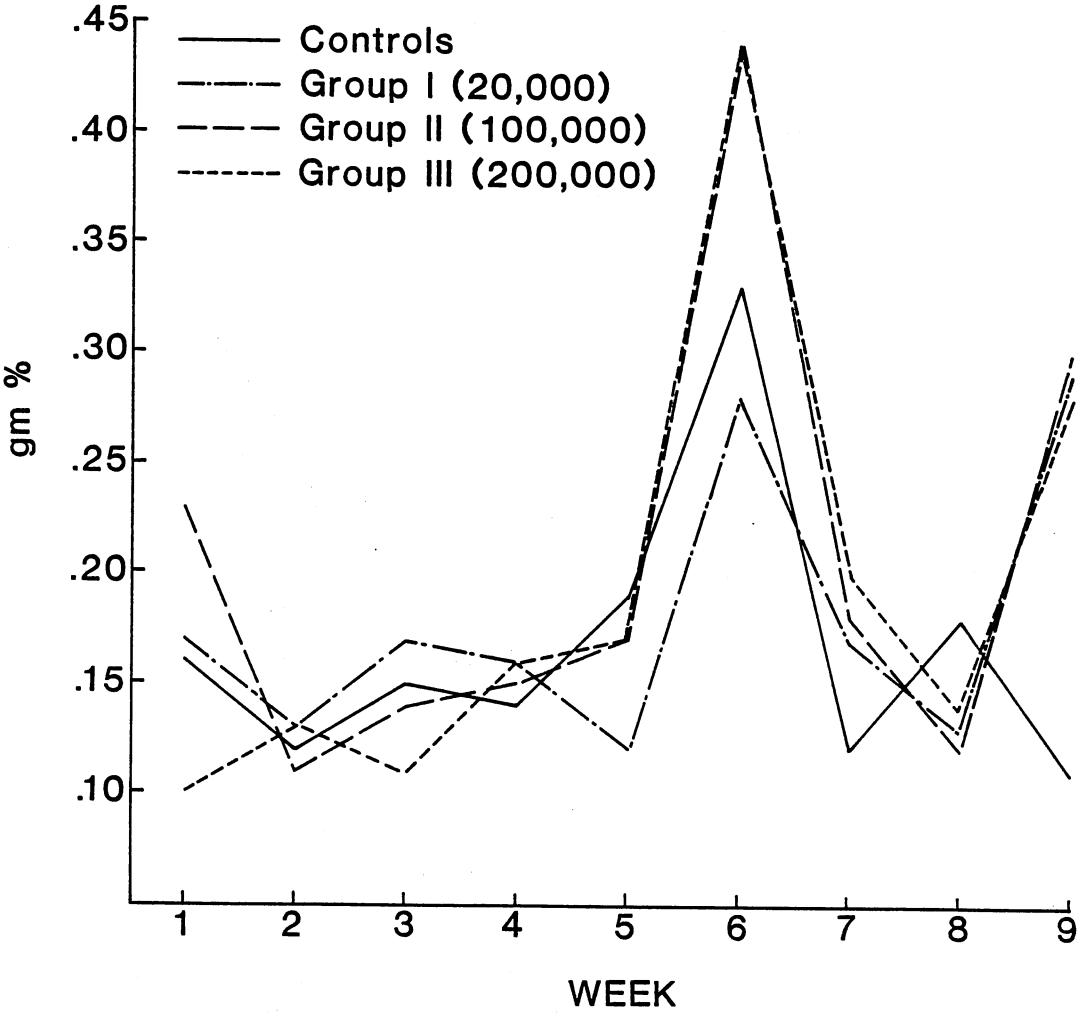

Figure 11. Weekly Mean Alpha-1 Globulin Levels of the Four Experimental Groups.

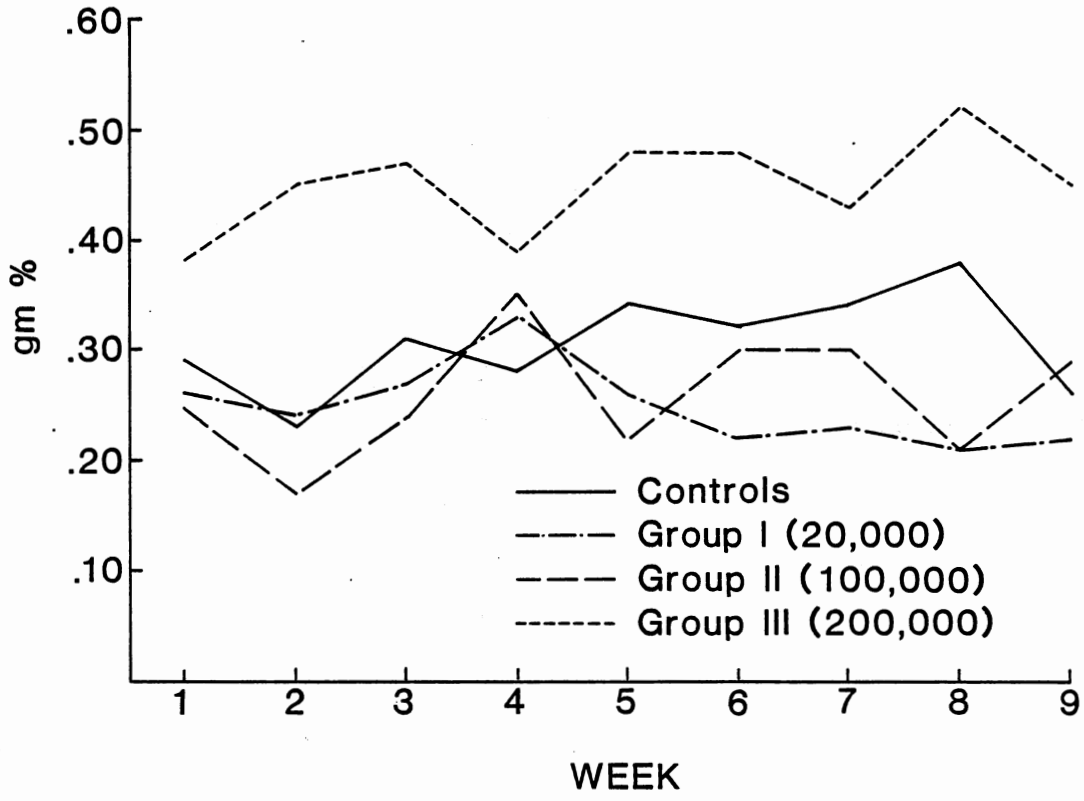

Figure 12. Weekly Mean Alpha-2 Globulin Levels of the Four Experimental Groups.

control group, and group II. During week 3, the mean alpha-2 globulin level of group III was significantly higher than the mean alpha-2 globulin levels of the control group, group I, and group II. Differences were not significant during week 4. The mean alpha-2 globulin level of group III was significantly higher than the mean alpha-2 globulin levels of the control group, group I, and group II during week 5. During week 6, the mean alpha-2 globulin level of group III was significantly higher than that of group I. Differences were not significant during week 7. By week 8, the alpha-2 globulin level of group III was higher than that of groups I and II. Differences were not significant during week 9. Table XVII, page 87, lists weekly alpha-2 globulin levels of the lambs, and group means and standard deviations.

Weekly mean beta-1 globulin levels of the four groups are presented in figure 13, page 35. By week 2, the mean beta-1 globulin level of group I was significantly higher than the mean beta-2 globulin levels of group III and the control group. By week 3, the levels of beta-1 globulin in group I and the control group were significantly higher than the mean beta-1 globulin level of group III. Differences were not significant during week 4. The mean beta-1 globulin level of group III was significantly lower than the mean beta-1 globulin levels of group I, the control group, and group II during week 5. At week 6, the beta-1 globulin level of group III was significantly lower than the level of group II. Significant differences were not discovered in the mean beta-1 globulin levels of the four groups during weeks 7, 8, and 9. Table XVIII, page 88, lists weekly beta-1 globulin levels of the lambs, and group means and standard deviations.

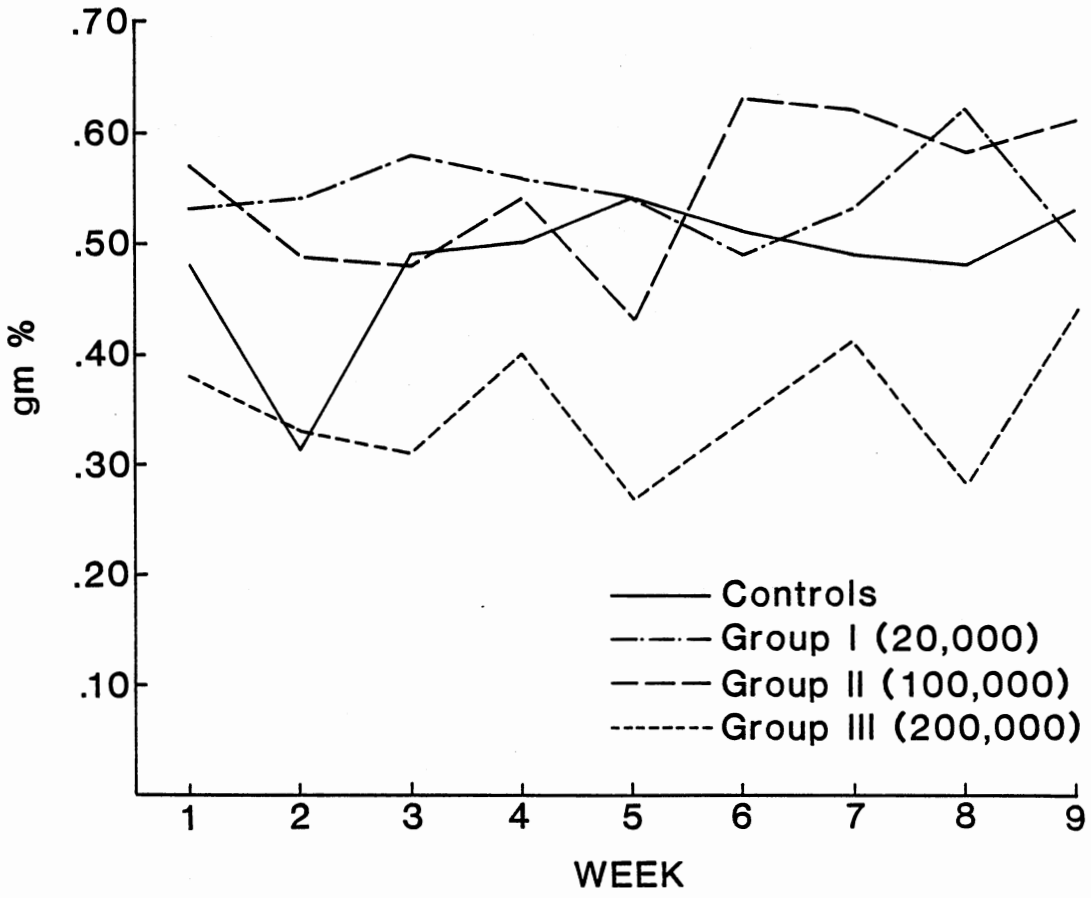

Figure 13. Weekly Mean Beta-1 Globulin Levels of the Four Experimental Groups.

Weekly mean beta-2 globulin levels of the four groups are demonstrated in figure 14, on page 37. The only significant difference was detected during week 5 when the mean beta-2 globulin level of the control group was significantly higher than the mean beta-2 globulin levels of groups I, II, and III. Table XIX, page 89, lists the weekly beta-2 globulin levels of the lambs, and group weekly mean beta-2 globulin levels and standard deviations.

Weekly mean gamma-1 globulin levels are presented in figure 15, on page 38. During week 6, the gamma-1 globulin levels of group II and the control group were significantly higher than those of group I. Differences were not significant during week 7. The control group gamma-1 globulin level was significantly higher than the levels of groups II and I during week 8. At week 9, differences were not significant. Table XX, page 90, lists weekly gamma-1 globulin levels of the lambs, and group mean gamma-1 globulin levels and standard deviations.

Figure 16, on page 39, presents weekly mean gamma-2 globulin levels of the four groups. The gamma-2 globulin fractions were significantly higher in group II and the control group than the levels in group III and group I at week 4. By week 5, the mean gamma-2 globulin level of the control group was significantly higher than the level of group I. Significant differences were not present during weeks 6, 7, 8, and 9. Table XXI, page 91, lists weekly gamma-2 globulin levels of the lambs, and group means and standard deviations.

Weekly mean albumin:globulin ratios are depicted in figure 17 on page 40. At week 6, the control group ratio was significantly higher than that of groups III and II. Significant differences were not

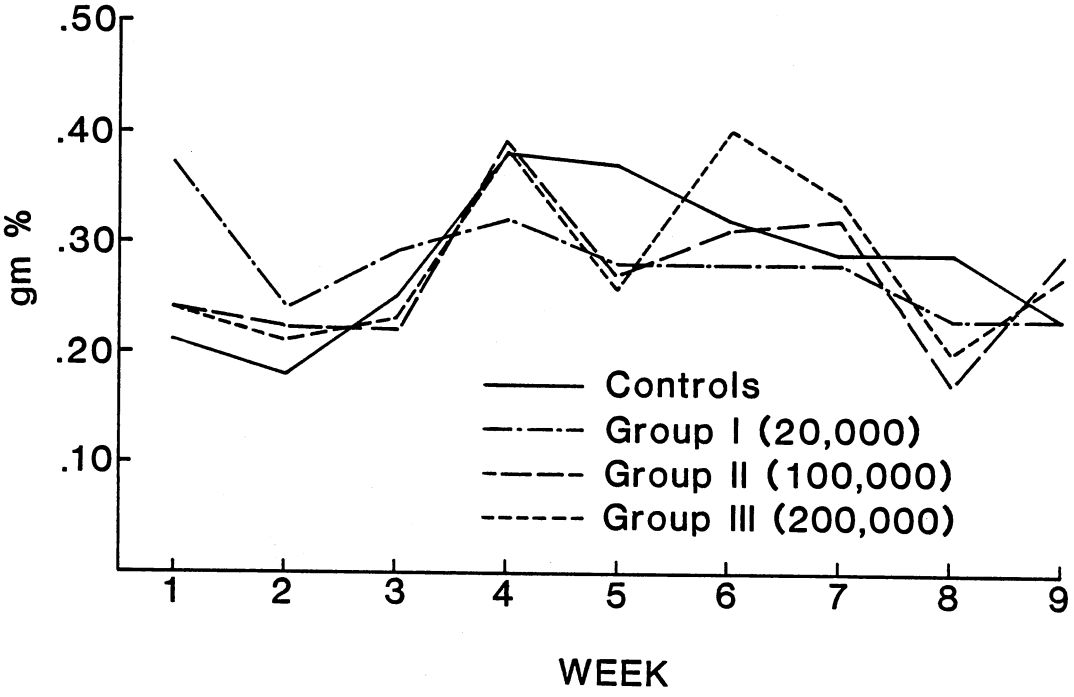

Figure 14. Weekly Mean Beta-2 Globulin Levels of the Four Experimental Groups.

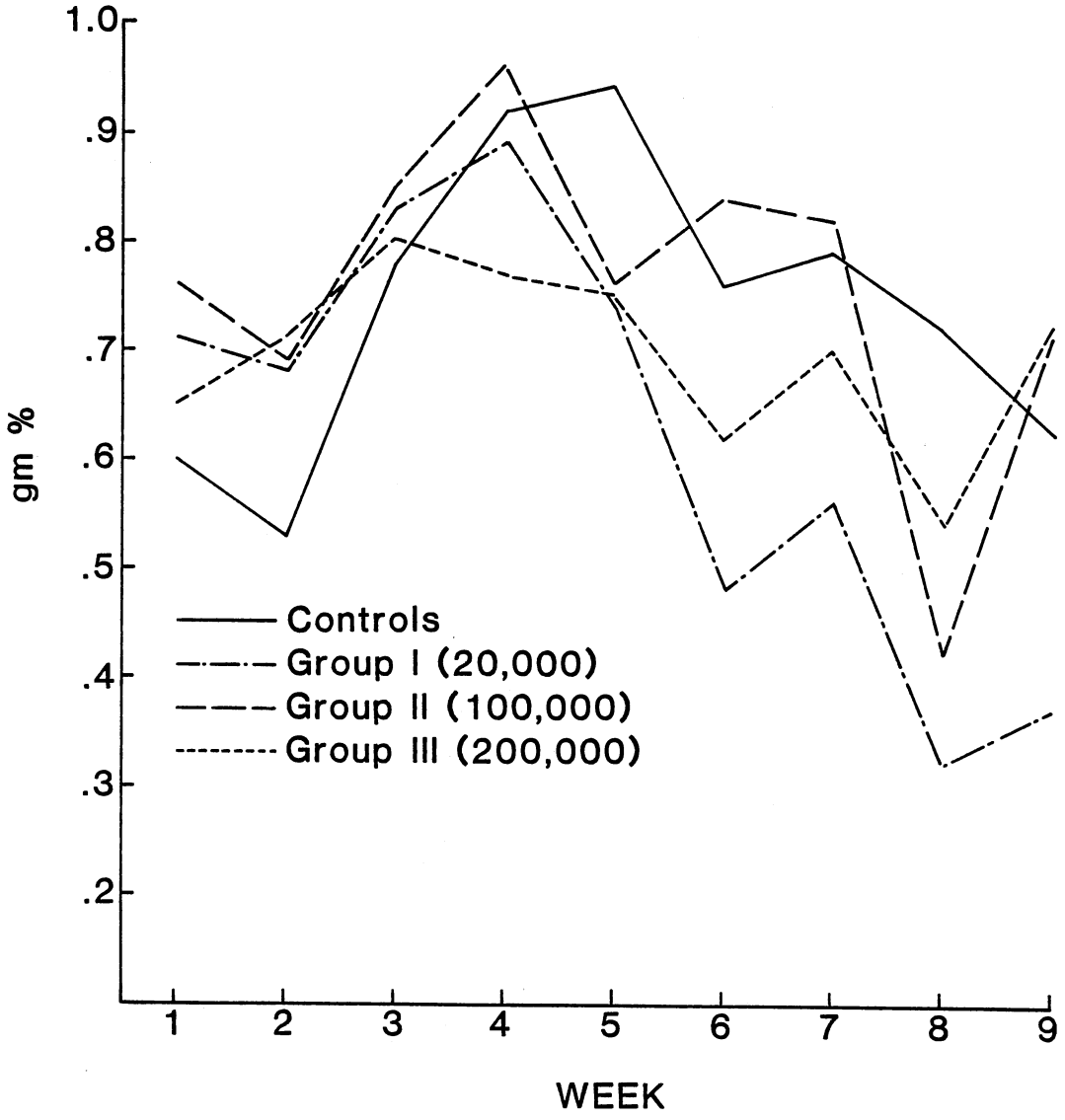

Figure 15. Weekly Mean Gamma-1 Globulin Levels of the Four Experimental Groups.

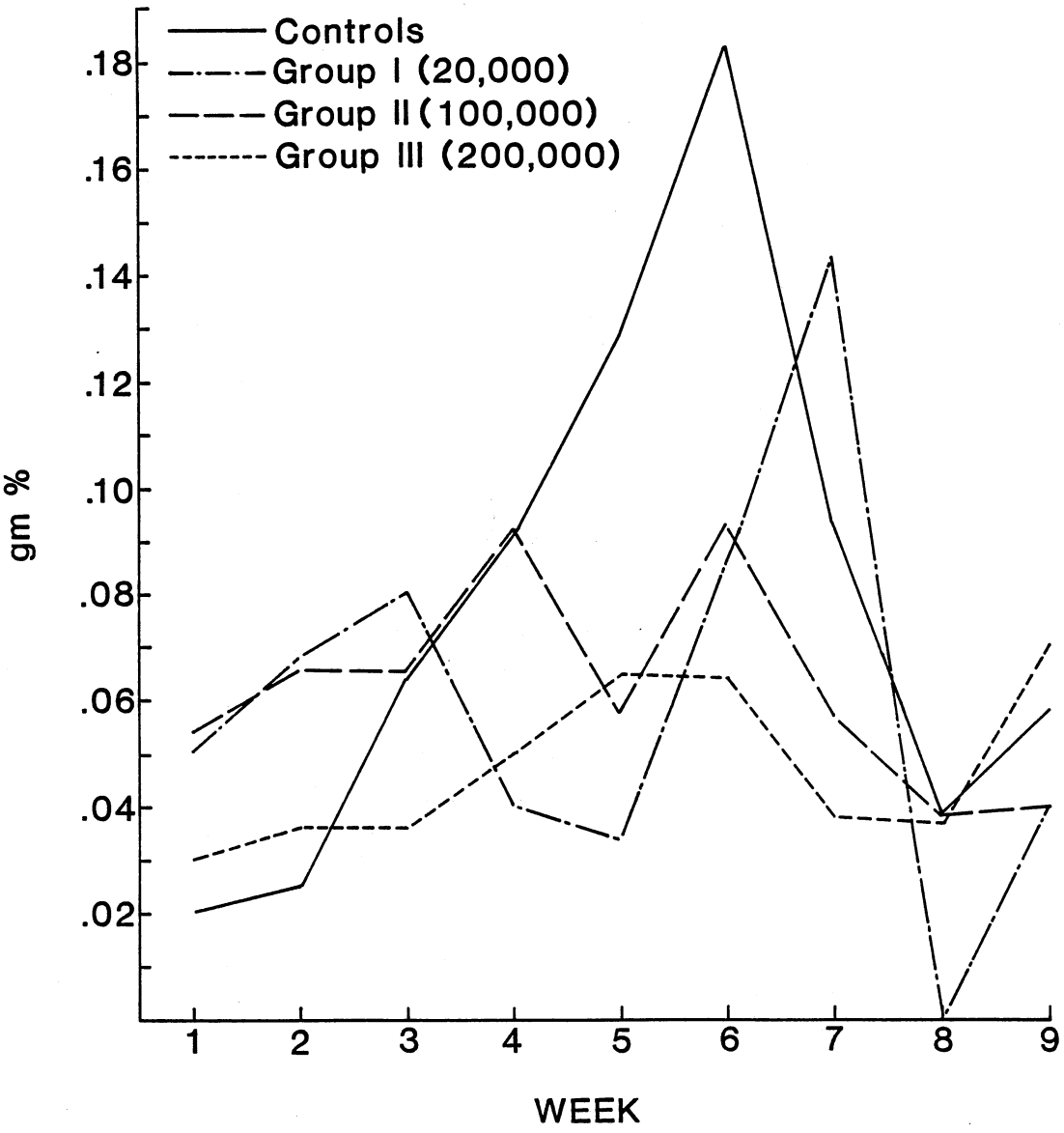

Figure 16. Weekly Mean Gamma-2 Globulin Levels of the Four Experimental Groups.

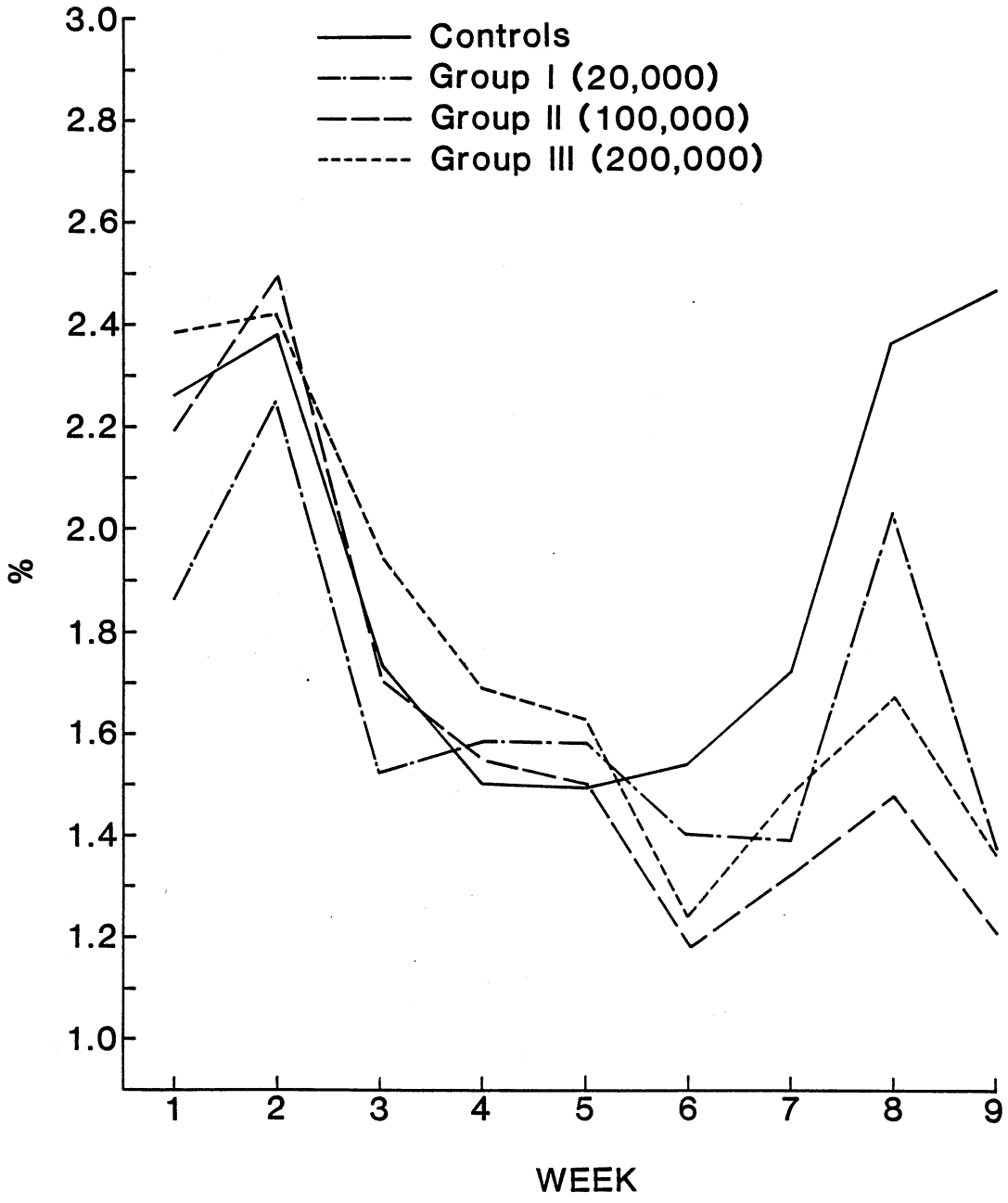

Figure 17. Weekly Mean Albumin:Globulin Ratios of the Four Experimental Groups.

observed again until week 9, at which time the control group ratio was significantly higher than the ratios of groups III, I, and II. Table XXII, page 92, lists weekly albumin:globulin ratios of the lambs, and group mean ratios and standard deviations.

Serum Electrolytes

Serum magnesium levels were not recorded for any of the lambs during week 4. Sodium, calcium, and potassium levels were not recorded for lamb 7 week 4. Chloride levels were not recorded for lamb 10 week 2, lambs 3, 12, and 20 week 3, and lamb 7 week 4.

Weekly mean serum magnesium levels are depicted in figure 18, on page 42. During week 6, the mean serum magnesium level of group II was significantly higher than the levels of groups III and I. Significant differences were not detected during week 7. By week 8, the mean serum magnesium level of the control group was significantly higher than the level of group I. Differences were not significant at week 9. Table XXIII, page 93, lists the weekly serum magnesium levels of the lambs, and group means and standard deviations.

Figure 19, page 43, demonstrates the weekly mean serum potassium levels of the four groups. During week 8, the mean serum potassium level of the control group was significantly higher than the mean levels of group II and group I. By week 9, the mean serum potassium level of group I was significantly lower than the levels of group II, the control group, and group III. Table XXIV, page 94, lists the weekly serum potassium levels of the lambs, and group mean levels and standard deviations.

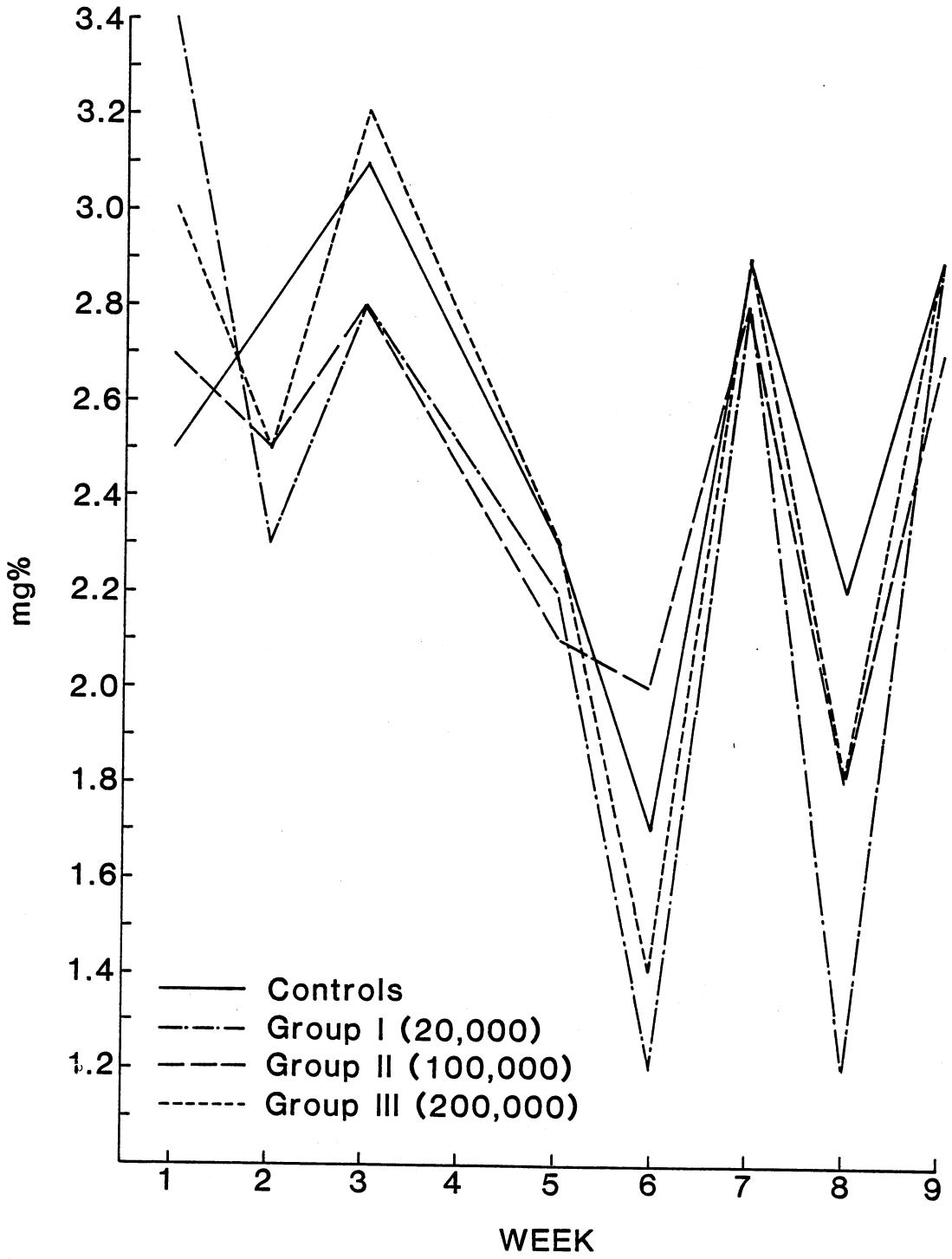

Figure 18. Weekly Mean Serum Magnesium Levels of the Four Experimental Groups.

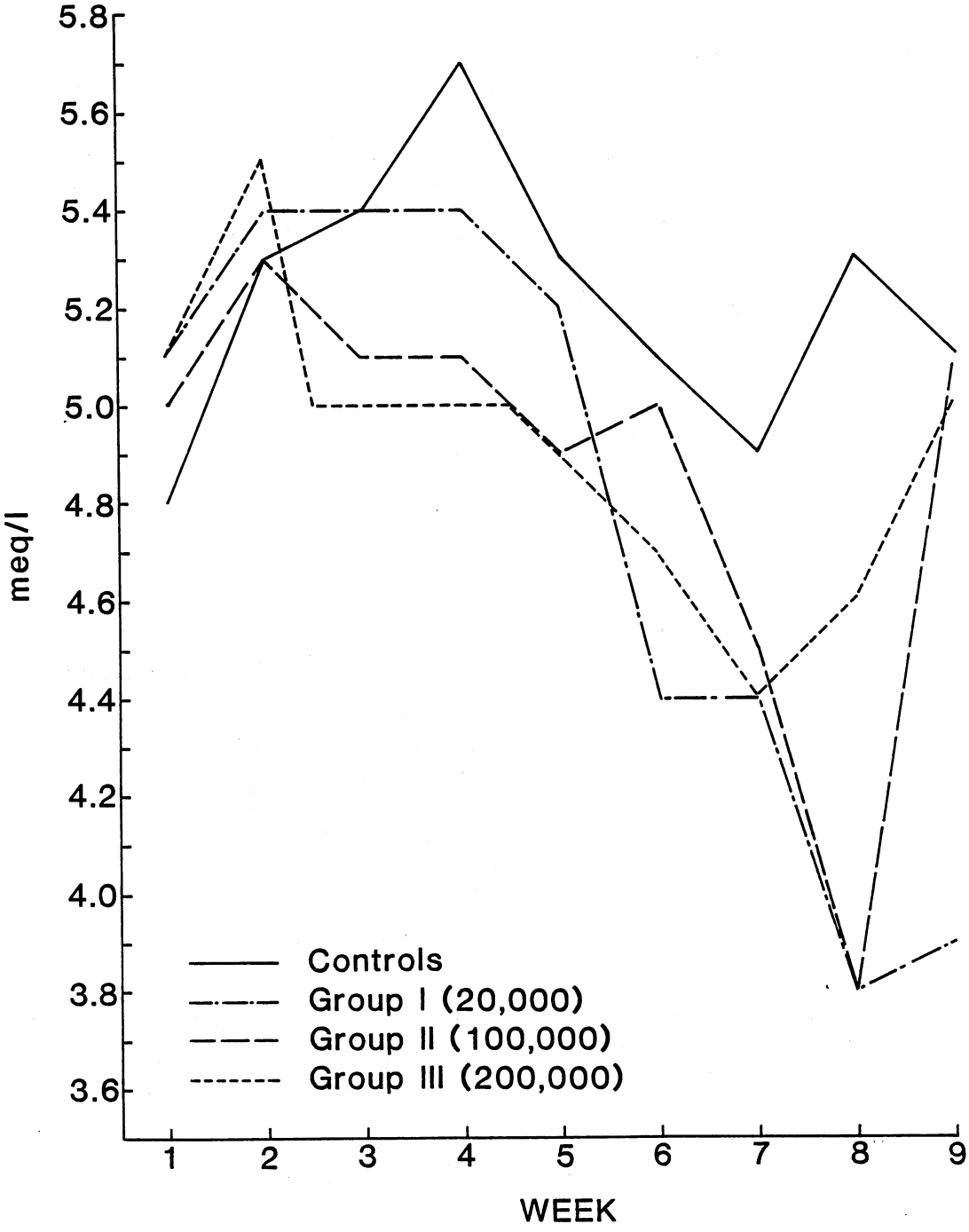

Figure 19. Weekly Mean Serum Potassium Levels of the Four Experimental Groups.

The mean serum chloride level of the control group was significantly higher than the mean serum chloride levels of groups III, II, and I during week 8. During week 9, the serum chloride level of group I was significantly lower than the levels of group II, the control group, and group III. Weekly mean serum chloride levels of the four groups are illustrated in figure 20, page 45. Table XXV, page 95, lists weekly serum chloride levels, and group means and standard deviations.

Figure 21, page 46, illustrates weekly mean serum calcium levels of the four groups. The mean serum calcium level of the control group was significantly higher than the levels of serum calcium in groups III, II, and I by week 8. During week 9, the mean serum calcium level of group I was significantly lower than the control group, group II, and group III levels. Table XXVI, page 96, lists weekly serum calcium levels of the lambs, and group means and standard deviations.

Weekly mean serum phosphorus levels of the four groups are demonstrated in figure 22, page 47. Table XXVII, page 97, lists weekly serum phosphorus levels of the lambs, and group means and standard deviations. During week 4, the mean serum phosphorus level of group III was significantly lower than the mean serum phosphorus levels of the control group, group I, and group II. At week 5, the mean serum phosphorus level of group III was significantly lower than the level of the control group. Significant differences were not present during weeks 6 and 7. By week 8, the serum phosphorus level of the control group was significantly higher than the serum phosphorus levels of groups II, I, and III. By week 9, significant differences were not present.

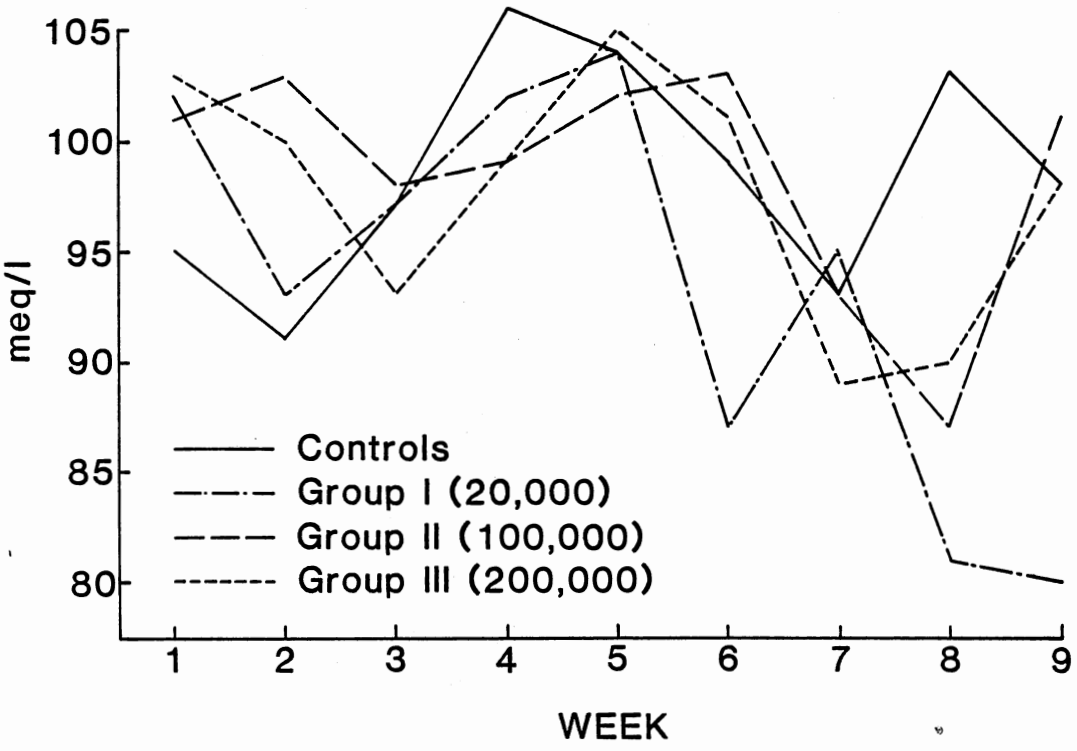

Figure 20. Weekly Mean Serum Chloride Levels of the Four Experimental Groups.

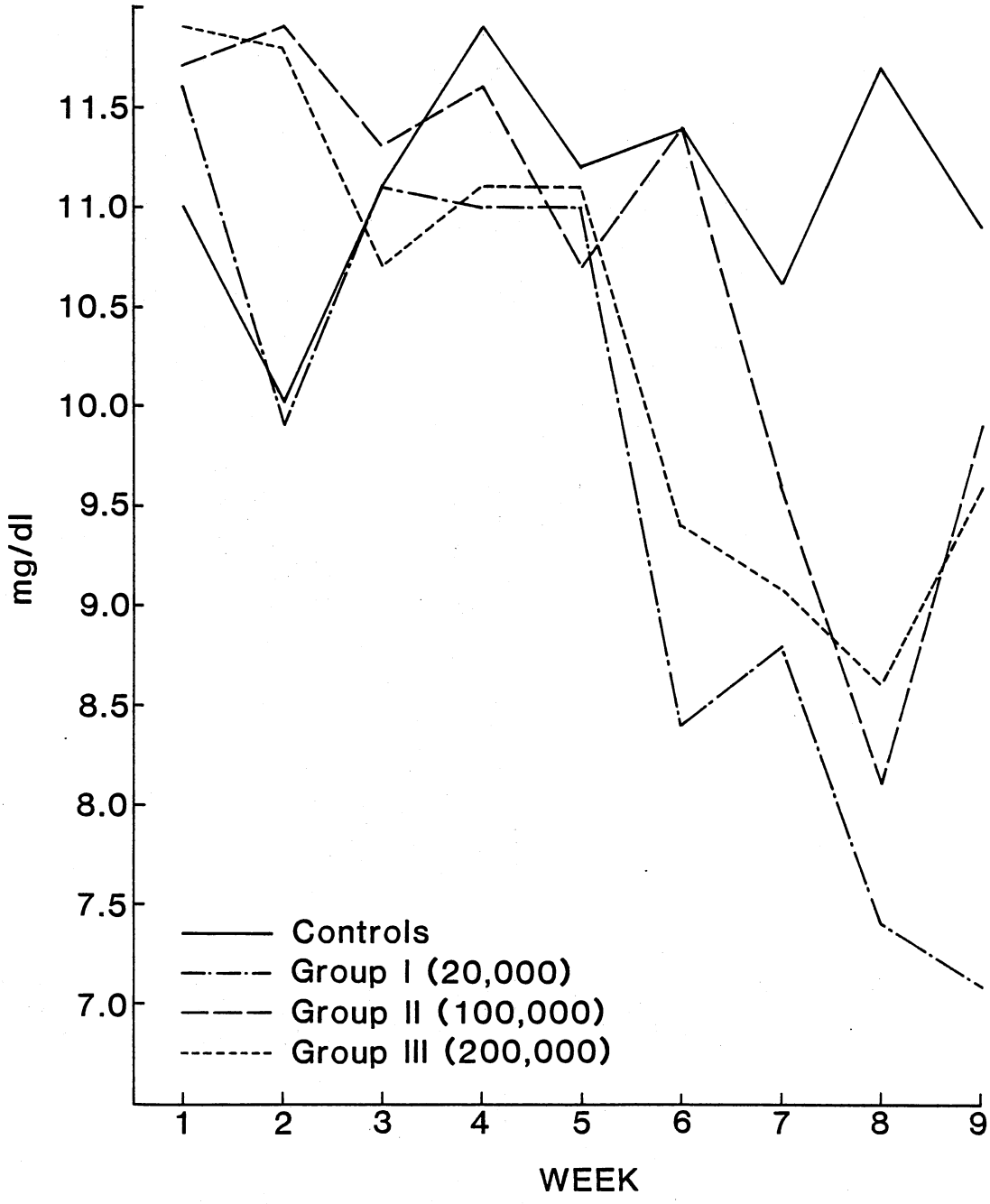

Figure 21. Weekly Mean Serum Calcium Levels of the Four Experimental Groups.

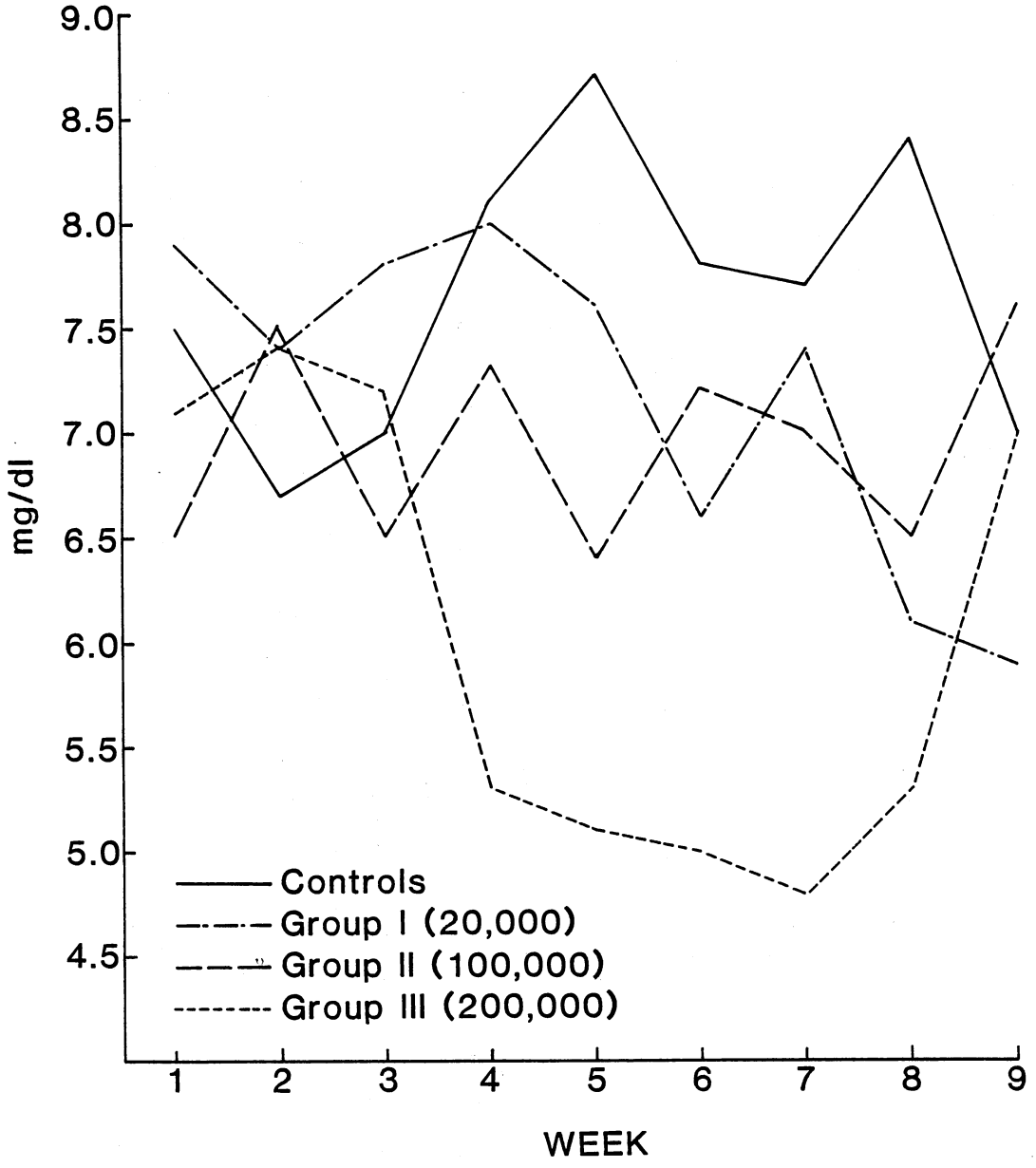

Figure 22. Weekly Mean Serum Phosphorus Levels of the Four Experimental Groups.

Weekly mean serum sodium levels are depicted in figure 23, page 49. Table XXVIII, page 98, lists weekly serum sodium levels of the lambs, and group means and standard deviations. During week 8, the control group mean serum sodium level was significantly higher than the serum sodium levels of groups III, II, and I. By week 9, the serum sodium level of group I was significantly lower than the levels of group II, the control group, and group III.

Postmortem Parasitologic Findings

Estimates of the parasites recovered from the abomasums, small intestines and large intestines of the lambs are presented in Tables I, II, and III, pages 50, 51, 52, respectively. Table IV, page 53, lists the total worm burden estimates, percent recovery of the larval inocula, and percentages of the worm burden estimates consisting of H. contortus. Table V, page 54, lists the eggs per gram of feces of the lambs and a range of H. contortus worm burden estimates based on egg counts.

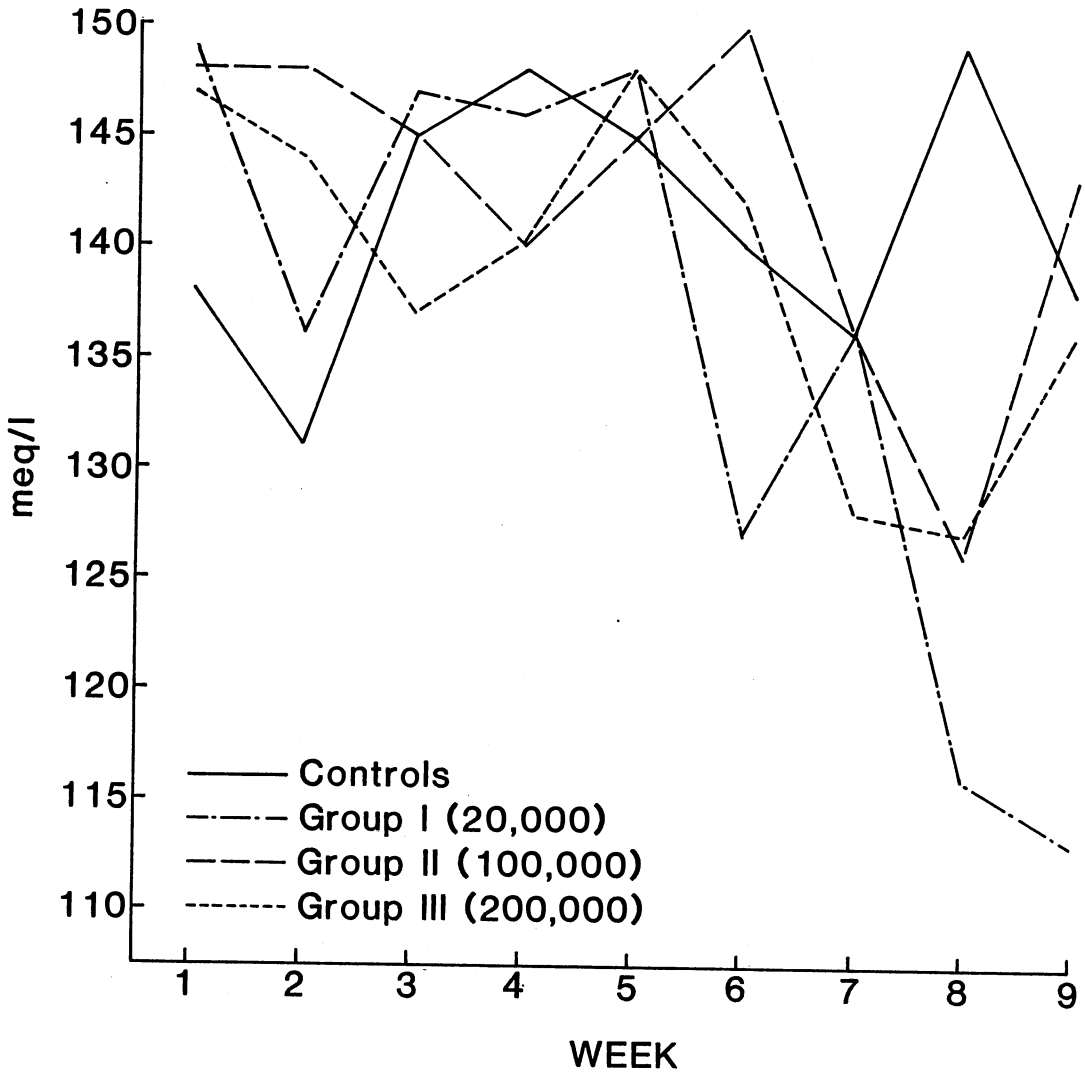

Figure 23. Weekly Mean Serum Sodium Levels of the Four Experimental Groups.

TABLE I

GENERA AND NUMBERS OF PARASITES RECOVERED FROM THE ABOMASUMS OF THE EXPERIMENTAL LAMBS

Animal	Group	Haemonchus contortus	Ostertagia circumcinta	Trichostrongylus axei
1		0	0	0
2		0	0	0
3	Control	0	0	0
4		0	0	0
5		0	0	0
6**		10,598	0	0
7		8,699	0	0
8	I	8,408	0	0
9		6,968	63	0
10		5,520	0	137
11***		9,870	0	452
12		0	32	766
13	II	5,564	63	420
14		3,795	0	378
15*		18,834	0	0
16		9,697	32	32
17		7,724	0	63
18	III	210	63	63
19		542	49	420
20*		24,197	0	241

*died three weeks after exposure.
**died four weeks after exposure.
***died five weeks after exposure.

TABLE II

GENERA AND NUMBERS OF PARASITES RECOVERED FROM THE SMALL INTESTINES OF THE EXPERIMENTAL LAMBS

Animal	Group	Trichostrongylus Colubriformis	Nematodirus filicollis
1		0	17
2		0	0
3	Control	0	0
4		0	0
5		0	0
6**		557	0
7		872	0
8	I	285	0
9		644	0
10		285	0
11***		0	0
12		490	0
13	II	1187	0
14		1157	0
15*		206	0
16		1653	0
17		715	0
18	III	644	0
19		228	0
20*		2255	0

*died three weeks after exposure.
**died four weeks after exposure.
***died five weeks after exposure.

TABLE III

GENERA AND NUMBERS OF PARASITES RECOVERED FROM THE LARGE INTESTINES OF THE EXPERIMENTAL LAMBS

Animal	Group	Oesophagostomum venulosum	Trichuris sp.
1		0	0
2		0	0
3	Control	0	0
4		0	0
5		0	0
6**		11	0
7		50	1
8	I	32	0
9		38	0
10		40	1
11***		303	0
12		7	0
13	II	433	0
14		52	1
15*		1	0
16		398	2
17		293	2
18	III	0	0
19		105	0
20*		1	0

*died three weeks after exposure.
**died four weeks after exposure.
***died five weeks after exposure.

TABLE IV

TOTAL WORM BURDEN ESTIMATES, PERCENT RECOVERY OF LARVAL DOSE, AND PERCENTAGES OF WORM BURDENS CONSISTING OF *HAEMONCHUS CONTORTUS*

Animal	Group	Total Worm Burden	Percent Recovery L_3	Percent *H. contortus*
1		17	0	0
2		0	0	0
3	Control	0	0	0
4		0	0	0
5		0	0	0
6**		11,166	55.8	94.9
7		9,622	48.1	90.4
8	I	8,725	43.6	96.4
9		7,713	38.6	90.3
10		5,983	29.9	92.2
11***		10,625	10.6	92.9
12		1,295	1.3	0
13	II	7,667	7.7	72.6
14		5,383	5.4	70.5
15*		19,041	19.0	98.9
16		11,814	5.9	82.1
17		8,797	4.4	87.8
18	III	980	.5	21.4
19		1,344	.7	40.3
20*		26,694	13.3	90.6

*died three weeks after exposure.
**died four weeks after exposure.
***died five weeks after exposure.

TABLE V

EGGS PER GRAM OF FECES AND HAEMONCHUS WORM BURDEN ESTIMATES BASED ON EGG COUNTS OF THE EXPERIMENTAL LAMBS

Animal	Group	EPG	Haemonchus Worm Burden Estimates (Range)
1		100	20-40
2		0	0
3	Control	0	0
4		0	0
5		0	0
6**		68,300	13,660-27,320
7		118,600	23,720-47,440
8	I	68,800	13,760-27,520
9		51,400	10,280-20,560
10		12,800	2,560-5,120
11***		78,500	15,700-31,400
12		100	20-40
13	II	69,400	13,880-27,760
14		2,700	540-1,080
15*		500	100-200
16		15,800	3,160-6,320
17		47,600	9,520-19,040
18	III	1,700	340-680
19		3,300	660-1,320
20*		700	140-280

*died three weeks after exposure.
**died four weeks after exposure.
***died five weeks after exposure.

CHAPTER V

DISCUSSION

General Observations

Treatment with levamisole hydrochloride was relatively effective for removal of the parasites present in the lambs at the time of purchase. Strongyloides eggs and Trichuris eggs were recovered by fecal flotation in several animals periodically throughout the course of the experiment. Re-infection with Strongyloides sp. may have occurred. Since the prepatent period for Trichuris sp. is several months, infection with this parasite from the larval inoculum is unlikely because eggs were detected as early as two weeks after anthelmintic treatment. Recovery of Strongyloides eggs and Trichuris eggs probably represented development of immature stages of these parasites which were present at the time of purchase but were not susceptible to the anthelmintic, or alternatively, ineffective elimination of adult parasites and temporary suppression of egg production. The inconsistent weekly fecal flotation findings demonstrates the various host, parasite, and technical factors which produce false negative flotation results, and emphasizes the necessity for repeated examinations.

Treatment with amprolium was not effective in eliminating the coccidial infections present in the lambs. Again, this probably represented development of stages which are not altered by the coccidiostat. However, treatment and sanitation procedures should have

minimized exposure. Since alterations in the parameters monitored were not detected until after exposure to the infective larvae of H. contortus, it was concluded that the coccidial infection was not the major contributor to the changes observed although an exaggerated pathogenic effect may have occurred.

Four of the five control animals consistently had negative fecal flotation findings for strongyle eggs. Strongyle eggs were detected in the feces of lamb 1 of the control group during weeks 7, 8, and 9. The only parasite recovered from this animal at necropsy was Nematodirus fillicolis. Cross infection or contamination of the premises seems unlikely because this was the only animal that harbored this parasite, and it was present in extremely low numbers. Stongyle eggs were detected in the feces of all the lambs within twenty-one days after exposure to the larval inocula.

Clinical Findings

The mean weights of the four groups were similar by week 3, the time of exposure to Haemonchus. Reduced weight gains were obvious in the three infected groups. Group II and III were the most severely affected. This suggested that larval exposure was an important factor. Larval pathogenicity due to blood loss by fourth and fifth stage larvae has been described by Veglia (1915), Clark et al. (1962), Brambell et al. (1964), and Charleston (1965). Charleston (1965) observed hypértrophy of the abomasal mucosa as early as four days after infection and suggested that increased permeability to protein macromolecules may contribute to the hypoproteinemia associated with H. contortus infections. Coop's (1971) findings of decreased abomasal acidity, increased

abomasal sodium ion concentration, and increased plasma pepsinogen levels also suggested that increased abomasal permeability and interchange between serum and abomasal constituents occurred at the time of emergence of fourth stage Haemonchus larvae and persisted for at least twenty-seven days after exposure. Sinclair and Pritchard (1975) found similar alterations in experimentally infected sheep treated with disophenol to arrest the development of H. contortus larvae at the fourth larval stage. Alterations persisted for fifty-five days after exposure. Greater fluid and plasma loss occurred in infected sheep than in non-infected sheep. Dargie (1975) stated that Haemonchus causes an increased catabolic rate of albumin associated with development of larval and adult worms due to loss of plasma from hemorrhage and possibly from loss of plasma due to increased permeability of the hyperplastic gastric mucosa. Protein production for growth is inhibited because of compensatory production of proteins needed for survival. Negative nitrogen balance develops due to poor conservation of amino acids derived from catabolism of body proteins through urinary excretion of nitrogenous materials. In the present experiment, initial weights of the lambs at the time of exposure did not consistently affect the reduction in weight gains or response to the infection, as observed by Silverman et al. (1970) who found that lighter lambs gained less and died more acutely. In the present experiment some of the heavier lambs succumbed to the infection. Reduced weight gains were most obvious in the lambs receiving the massive larval doses.

Differences in respiratory rate, heart rate, and body temperature were inconsistent and probably reflected excitability upon handling the animals, and difficulties or errors in monitoring these parameters.

With anemia and hypoproteinemia, tachycardia and hyperventilation might be expected due to hypoxia and reduced plasma colloidal osmotic pressure. Severely affected animals were reluctant to move or exert themselves, thus reducing their oxygen requirements.

Elevated body temperature is not associated with H. contortus infections. Body temperature was monitored to help assess the health status of the lambs. Body temperatures were not greatly elevated, nor was there gross evidence at necropsy of other ongoing disease processes in any of the lambs.

Hematology

Reports in the literature conflict concerning changes in white blood cell counts due to H. contortus infections. Fourie (1931) reported an initial leucopenia when red cell counts began to decrease fourteen days after infection followed by a neutrophilic leucocytosis during the period of active bone marrow response in fatal cases of haemonchosis. Charleston (1964) found that total leucocyte counts did not follow any trend and neutrophils declined in all groups. Silverman et al. (1970) did not detect marked effects on total or differential counts. The higher mean white blood cell count of group I at week 3 may represent a stress response or mild infectious process. There were no obvious alterations in white blood cell counts due to the parasites.

Declining mean erythrocyte counts, hematocrits, and hemoglobin levels before patency represented blood loss initiated by fourth and fifth stage larvae. The precipitous fall in these parameters after patency illustrated the severity of the hemorrhage produced by the adult parasites. The greatest declines were observed in group I since

mean red blood cell count, hematocrit, and hemoglobin level were still significantly lower than the control group value at week 9. The differences between the groups are best explained by differences in adult populations in the surviving lambs of the 3 infected groups. Group I had four surviving lambs in which worm burdens ranged from 5,520 to 8,699 adult H. contortus. Group II had three surviving lambs and their worm burdens ranged from 0 to 5,564 adult H. contortus. Group III had four surviving lambs in which worm burdens ranged from 210 to 9,697 adult H. contortus. Changes in hematologic parameters were consistent with those reported by Silverman et al. (1970), and Dargie and Allonby (1975).

Serum Proteins

The most consistent changes in mean serum total protein levels were observed in group I. The mean total protein level of this group continued to decline through week 9. Initial decreases in total protein levels of the groups during week 5 suggested protein loss through the abomasum due to activity of immature stages of the parasite. Decreases in total protein levels by week 6 reflected the hematophagic activity of adult parasites. Differences between the groups again implied differences in worm burden populations in surviving lambs at the time of sampling. Changes in mean albumin levels of the three infected groups followed a similar trend.

Weekly mean alpha-1 globulin levels were not significantly different throughout the course of the experiment. Significantly elevated alpha-2 globulin levels of group III during weeks 2, 3, 5, 6, and 8 were not considered to be due to the parasitic infection but rather

were ascribed to an ongoing inflammatory response. Evidence of an inflammatory condition was not detected in this group of lambs during clinical examination or at necropsy. Kuttler and Marble (1970) detected significant increases in alpha-2 globulin levels of clinically parasitized sheep as compared to animals with phlebotomy-induced anemia. The increased alpha-2 levels in the group III lambs could have represented previous exposure to parasites since two of the four surviving animals in this group had low worm burdens at necropsy. However, fecal flotation findings were negative during the pre-infection observation period.

Group III also had consistently lower mean beta-1 globulin levels. According to Kaneko (1980), beta-globulin decreases are associated with chronic liver disease, iron storage disease, hemolytic anemia, acute liver disease, autoimmune disease, and disseminated intravascular coagulation. Changes in the alpha-2 and beta-1 globulin levels of group III could have reflected alterations in serum protein constituents of these electrophoretic areas. However, the reciprocal nature of the relationship between the alpha-2 and beta-1 values suggested that subjectivity in alpha-2 and beta-1 marking may have contributed to the changes in alpha-2 and beta-1 levels (Tyler, 1983).

The mean beta-2 globulin levels of the 3 infected groups were significantly lower than the control group level at week 5 only. Disease processes associated with beta-globulin decreases were not evident in these lambs. With an iron deficiency, or anemia, an increase in the transferrin portion of the beta-2 globulin fraction would be expected. At this point in the course of the experiment, the anemia was just becoming evident by hematologic parameters. Erythropoietic stimulus was minimal. Therefore, transferrin synthesis for iron transport was

probably not markedly elevated.

Gamma globulin decreases probably represented plasma loss due to hemorrhage and movement of interstitial fluids into the vascular compartment. Return of values of infected groups to control values could have represented increased production due to the antigen stimulus of the parasites and/or differences in worm burden populations in surviving lambs.

Changes in serum proteins detected during this experiment were not consistent with those reported by Kuttler and Marble (1960) in lambs with naturally acquired parasite infections due primarily to H. contortus. Hypoproteinemia and hypoalbuminemia were present, but globulin levels either declined or were similar to levels of the control group. The discrepancies may be related to differences in the duration of the infections reported by Kuttler and Marble and those in the present study.

Decreases in the albumin:globulin ratios were obvious after patency, and weekly differences between the groups reflected variations in the various protein fractions. Globulins were not elevated in the infected lambs. Therefore, the results suggested that the decreases were primarily due to albumin loss and movement of interstitial fluids to restore blood volume.

Serum Electrolytes

The most consistent changes in serum electrolytes were observed in group I during weeks 8 and 9. Differences between the groups suggested variations due to adult populations in surviving lambs.

The only significant decrease from the control group mean magnesium

level occurred at week 8 in group I. Examination of the data in Table XVIII, page 93, shows one extremely low magnesium value of 0.9 mg% in lamb 10. This may represent a technical error. Zajicek et al. (1976) did not detect changes in serum magnesium levels of sheep infected with H. contortus and T. colubriformis.

Declining mean serum potassium levels were present in groups II and I during week 8, and group I during week 9. According to Tasker (1980), hypokalemia may occur in anorectic animals which continue to drink water because renal excretion of potassium continues despite decreased consumption and intestinal absorption. Shumard et al. (1957) did not detect changes in water consumption until twenty-five days after infection with Haemonchus, Trichostrongylus, and Nematodirus. Alkalosis causes increased exchange of potassium for hydrogen ions in the renal tubular fluid and causes hypokalemia. Renal excretion of potassium also occurs when aldosterone is released to maintain serum sodium levels. The exact mechanism responsible for the reduced serum potassium levels in the present experiment was not determined, but probably involve renal conservation of sodium and water due to the hypovolemia resulting from the hemorrhage and hypoproteinemia associated with haemonchosis.

Mean serum sodium levels decreased during week 8 in all the infected groups, and remained depressed by week 9 in group I. Owen (1971) demonstrated increased plasma volumes in H. contortus infections. Blood volume remained constant. He concluded that compensation for reduced red cell volume occurred by increasing the plasma volume, despite a marked hypoproteinemia. Sodium is the major cation regulating osmolarity of the extracellular fluid compartments. Coop (1971)

measured changes in abomasal electrolyte concentration in sheep receiving one million H. contortus larvae and in sheep with low or nil levels of infection, and recorded a significant decrease in abomasal acidity and increase in sodium ion concentration occurring three to six days after dosing with one million larvae. Potassium and chloride levels also decreased slightly. Significant differences were still present in hydrogen ion and sodium ion concentrations nineteen and twenty-seven days post-infection. Plasma electrolyte concentrations were not altered for two weeks after infection. The changes were believed to be due to increased permeability of the abomasal mucosa. Serum sodium level decreases, five weeks after exposure, in the present experiment may represent excessive loss through a damaged mucosa. Net intestinal absorption occurs when flow from the intestinal lumen to the plasma exceeds that in the opposite direction (Cardielhac, 1971). Scott (1982) listed several diseases that cause hyponatremia. Acute hemorrhage and hypoalbuminemia due to protein-losing enteropathy were included in the lists. The pathogenesis involves impaired excretion of free water by the kidneys. The renin-angiotensin system is stimulated by decreased extracellular fluid volume, primarily of the vascular compartment, to produce thirst and non-osmotic release of arginine vasopressin (anti-diuretic hormone) via baroreceptors in the carotid sinus, aortic arch, greater pulmonary veins, and the left atrium. As a result of decreased glomerular filtration rate and maximum sodium and water retention by the proximal tubules, delivery of salt and water to the loops of Henle are reduced. Excretion of free water depends upon sufficient delivery of isotonic fluid to the loops of Henle.

Mean serum chloride levels of the three infected groups followed

a similar trend to that of sodium. Decreases in chloride levels may have resulted from a decline in sodium level because electrical neutrality of the extracellular fluid compartment must be maintained and enhances renal excretion of chloride.

Mean serum calcium levels of the three infected groups also followed a similar pattern. This resulted from loss of serum proteins since half of serum calcium is protein bound, primarily to albumin. Simesen (1980) stated that moderate hypocalcemia is a common finding in diseases with protein-losing gastroenteropathies. He also stated that the abomasal digesta of sheep contains materials which bind calcium above a pH of 5.0. Formation of insoluble calcium compounds may reduce intestinal absorption. Coop (1971) and Christie (1970) demonstrated increases in abomasal pH above 5.0 with experimental infections of H. contortus.

Mean serum phosphorus levels of group III were decreased during weeks 4, 5, and 8. The levels of group II and I were also decreased by week 8. Yashchenko (1967) detected declining serum inorganic phosphorus levels while the larval forms of H. contortus were developing. After sexual maturity of the parasites, serum calcium levels declined and phosphorus levels remained depressed. Shumard et al. (1957) detected hypophosphatemia in mixed trichostrongyle infections. Zajicek et al. (1976) found decreased serum calcium levels and increased inorganic phosphorus levels during the period of most intensive development of H. contortus and T. colubriformis. The present results were consistent with those of Yashchenko. Decreased inorganic phosphorus levels during the period of larval development in group III suggested that changes in abomasal pH may interfer with phosphorus absorption. Hays and Swenson (1977) stated that normal gastric secretion of

hydrochloric acid is necessary for calcium and phosphorus absorption because a low intestinal pH is needed for their solubility. Decreased serum phosphorus levels after patency of the H. contortus infection could also have represented increased urinary excretion of phosphorus that results from parathyroid hormone released to maintain serum calcium levels.

Parasitology

The percent recovery of the larval dosage decreased as the larval dosage increased. Kates and Turner (1960) observed a similar effect with Trichostrongylus axei. The most consistent levels of infection with H. contortus were produced in group I lambs which received larval inocula of 20,000 and had worm burdens ranging from 5,520 to 10,598.

Lamb 12 of group II did not harbor H. contortus adults. Age, weight, and previous exposure to the parasite are factors which could account for this resistance to experimental infection. Procedures were not performed to recover inhibited larvae. The exact ages of the lambs at the time of purchase were not known. The mean weights of the lambs were similar at the start of the experiment, and heavier lambs succumbed to the infection. A strongyle infection was detected by fecal flotation at the time of purchase. Therefore, it appeared that the resistance was an acquired one. T. axei and T. colubriformis were recovered from this animal. Steward (1953) demonstrated that recent H. contortus infections could cause 'self-cure' of established T. axei and T. colubriformis infections. Therefore, these parasites probably were acquired from the larvae employed in the experimental procedure. This is supported by the finding that the controls did not harbor these parasites.

Lambs 18 and 19 in group III developed small populations of H. contortus which consisted of 210 and 542 adults, respectively. Again weight did not appear to play a role in the resistance observed. Previous exposure prior to the experiment was not evident from fecal flotations. However, acquired immunity can not be eliminated as a possible explanation. Christie (1970) demonstrated that conditions for development of H. contortus were less favorable when a large challenge was given even in the absence of previous exposure. The antigenic stimulus in this group was the greatest due to the large larval inoculum administered. Hematologic parameters of lamb 19 decreased slightly. Hematologic parameters of lamb 18 decreased by week 6 then rose back toward normal values by week 9. This suggested that expulsion of parasites in lamb 19 occurred during the third ecdysis, and expulsion of parasites in lamb 18 occurred during the fourth ecdysis. Charleston (1965) interpreted mononuclear cellular infiltration of the abomasum and hypertrophy of regional lymph nodes in terms of immune phenomena related to larval stages, including the fourth molt.

Five animals, lambs 6, 7, 8, 9, and 15, did not develop T. axei infections. One animal, lamb 11, did not develop T. colubriformis infection. Low level infections could have been missed by the worm burden procedure employed.

Estimations of adult worm burdens based on eggs per gram of feces did not accurately reflect the populations of adult H. contortus present. Estimates tended to be either high or low. Estimates were extremely low in animals which succumbed at patency. Gordon (1950) stated that egg production was higher in animals with lower levels of adult parasites. Dargie and Allonby (1975) stated that egg production

was greater in light infections.

Summary

Reduced weight gains were evident in the groups of lambs exposed to infective larvae of H. contortus. The groups exposed to massive larval inocula demonstrated the most severe reductions in weight gain. Declining hematologic values two weeks after exposure were useful in detecting prepatent infections. Hemoglobin level was the most sensitive indicator. Total protein levels declined two to five weeks after exposure. Albumin levels declined three weeks after exposure and remained depressed six weeks after exposure. Globulin levels were unaltered or declined. The albumin:globulin ratios declined after patency. Serum phosphorus level declined one week after exposure in the group receiving 200,000 larvae and remained depressed five weeks after exposure in this group. Five weeks after exposure serum phosphorus levels of the other infected groups declined. Serum potassium, chloride, sodium, and calcium levels declined in the infected groups five weeks after exposure also. Serum magnesium levels remained close to control group levels. The most consistent changes were observed in the group receiving 20,000 larvae, probably due to the more uniform adult populations within this group of lambs. Egg counts did not accurately predict worm burden levels, particularly in lambs which died at the time of patency.

Based on significant changes detected during the period of larval development (prepatency) and changes detected after sexual maturity of the parasite (patency), the following metabolic profiles for acute haemonchosis were suggested from the results of this experiment: 1) prepatency; normal weight gain, normal red blood cell count, normal

hematocrit, reduced hemoglobin level, reduced total protein level, normal albumin level, reduced beta-2 globulin level, and reduced serum phosphorus level, 2) patency; reduced weight gain, reduced red blood cell count, reduced hematocrit, reduced hemoglobin level, reduced total protein level, reduced albumin level, reduced gamma-1 globulin level, reduced albumin:globulin ratio, reduced serum phosphorus level, reduced serum potassium level, reduced serum chloride level, reduced serum calcium level, reduced serum sodium level. Variations in parameter values are dependent upon larval exposure, adult populations, duration of the infection, and immune and nutritional status of the host.

CHAPTER VI

CONCLUSIONS

Twenty fall weanling lambs were divided into four groups of five lambs each, and were observed for three weeks prior to experimental exposure to H. contortus and for six weeks after exposure. One group served as unexposed controls. Groups I, II, and III were inoculated orally with 20,000, 100,000, and 200,000 infective larvae, respectively. Patency was detected twenty-one days post-exposure. Mortality reached forty percent in group II. A twenty percent mortality was observed in each of the remaining infected groups. Reduced weight gains were observed in all three infected groups. The most dramatic reductions in weight gains occurred in groups II and III, which received massive larval doses. Respiratory rate, heart rate, body temperature, and white blood cell count changes were inconsistent and reflected excitability of the animals upon handling.

Hematologic parameters followed a consistent pattern of declining values two to three weeks after exposure in the infected groups. Hemoglobin was the most sensitive monitor of the developing anemia. The most seriously affected group was group I and this seemed to result from the uniformly heavy adult parasite populations established in the lambs of this group.

Serum protein changes consisted of decreased total protein levels and albumin levels. Globulins either remained normal or decreased.

Albumin:globulin ratios also declined. The most consistent changes were observed in group I.

Serum electrolyte changes were detected one week after exposure in group III and were signaled by declining inorganic phosphorus levels. Serum phosphorus levels were depressed for five weeks after exposure in this group and declined in groups I and II at this time. Serum magnesium levels of the three infected groups remained unaltered. Serum sodium, potassium, chloride, and calcium levels declined five weeks after exposure in all three infected groups. The most consistent changes were observed in group I.

Parasitologic findings at necropsy demonstrated that H. contortus was the primary parasite responsible for the alterations in metabolic parameters observed. Some animals apparently were able to resist infection with Haemonchus. Other parasites recovered were Trichostrongylus axei, Trichostrongylus colubriformis, Ostertagia circumcinta, Nematodirus fillicolis, Trichuris sp., and Oesophagastomum venulosum. Egg counts did not accurately reflect the levels of infection achieved, particularly in animals which died at the time of patency. Changes in most of the metabolic parameters related to the adult populations that established and are thought to be a consequence of their hematophagic activity.

It is obvious that physiologic studies need to be conducted to determine pathophysiologic mechanisms responsible for the alterations detected. In view of the technical difficulties encountered during the experimental procedure, the experiment should be repeated to confirm the validity of the present results. Difficulties included a mixed larval inoculum, lambs that were not parasite free, and deficiencies

in the data due to clotting of samples, loss of specimens or insufficient samples. Addition of other test procedures such as urine electrolyte concentrations and specific gravity, blood urea nitrogen levels, serum enzyme levels, and serum osmolarity should facilitate determination of the pathogenic mechanisms involved. Statistical analysis of the data with groupings based on adult populations of H. contortus would also be valuable since the most consistent results were observed in group I which had the most uniform adult populations.

BIBLIOGRAPHY

1. Baker, N. F., E. C. Cook, J. R. Douglas, and C. E. Cornelius. 1959. The Pathogenesis of Trichostrongyloid Parasites. III. Some Physiological Observations in Lambs Suffering from Acute Parasitic Gastroenteritis. J. Parasitol. 45:643-651.

2. Brambell, M. R., and W. A. G. Charleston. 1964. Abomasal Bleeding Caused by Immature Stages of Haemonchus contortus in Sheep Showing 'Age-Resistance'. J. Comp. Path. 74:338-345.

3. Cardielhac, P. J. 1971. Mineral Metabolism. In Textbook of Veterinary Physiology. James E. Brazile (ed.), pp. 448-456. Philadelphia: Lea and Febiger.

4. Charleston, W. A. G. 1964. Comparison of Blood and Bone Marrow Changes in Lambs with Haemonchosis and in Lambs Repeatedly Bled. J. Comp. Path. 74:223-240.

5. Charleston, W. A. G. 1965. Pathogenesis of Experimental Haemonchosis in Sheep, with Special Reference to the Development of Resistance. J. Comp. Path. 75:55-67.

6. Christie, M. G. 1970. The Fate of Very Large Doses of Haemonchus Contortus and Their Effect on Conditions in the Ovine Abomasum. J. Comp. Path. 80:89-100.

7. Clark, C. H., G. K. Kiesel, and C. H. Goby. 1962. Measurements of Blood Loss Caused by Haemonchus contortus Infection in Sheep. Am. J. Vet. Res. 23:977-980.

8. Coop, R. L. 1971. The Effect of Large Doses of Haemonchus contortus on the Level of Plasma Pepsinogen on the Concentration of Electrolytes in the Abomasal Fluid of Sheep. J. Comp. Path. 81:213-219.

9. Dargie, J. D. 1975. Application of Radioisotopic Techniques to the Study of Red Cell and Plasma Protein Metabolism in Helminth Diseases of Sheep. Symposia of the British Society of Parasitology. 13:1-26.

10. Dargie, J. D. and E. W. Allonby. 1975. Pathophysiology of Single and Challenge Infections of Haemonchus contortus in Merino Sheep: Studies on Red Cell Kinetics and The 'Self-cure' Phenomenon. Int. J. Parasit. 5:147-157.

11. Fourie, P. J. J. 1931. The Hematology and Pathology of Haemonchosis in Sheep. (Rep. 17th). Vet. Res. Un. S. Afr.:495-572.

12. Gordon, H. McL. 1950. Some Aspects of Parasitic Gastroenteritis of Sheep. Aust. Vet. J. 26(1):14-28.

13. Hays, V. W., and M. L. Swenson. 1977. Minerals, Bones, and Joints. In Dukes' Physiology of Domestic Animals, 9th ed., M. J. Swenson (ed.), pp. 395-397. Ithaca: Cornell University Press.

14. Kaneko, J. J. 1980. Serum Proteins and the Dysproteinemias. In Clinical Biochemistry of Domestic Animals, 3rd. ed., J. J. Kaneko (ed.), pp. 104-105. New York: Academic Press.

15. Kates, K. C., and J. H. Turner. 1960. Experimental Trichostrongylosis (axei) in Lambs, with a Discussion of Recent Research on this Disease in Ruminants. Am. J. Vet. Res. 21:254-261.

16. Kuttler, K. L., and D. W. Marble. 1960. Serum Protein Changes in Lambs with Naturally Acquired Nematode Infections. Am. J. Vet. Res. 21:445-448.

17. Owen, N. C. 1971. Plasma and Blood Volume Changes in Sheep Experimentally Infected with Haemonchus contortus. J. S. Afr. Vet. Med. Ass. 42(1):9-12.

18. Scott, R. C. 1982. Disorders of Sodium Metabolism. Vet. Cl. N. Am. 12(3):375-409.

19. Shumard, R. F., D. W. Bolin, and D. F. Evelith. 1957. Physiological and Nutritional Changes in Lambs Infected with the Nematodes, Haemonchus contortus, Trichostrongylus colubriformis, and Nematodirus spathiger. Am. J. Vet. Res. 18:330-336.

20. Silverman, P. H., M. E. Mansfield, and H. L. Scott. 1970. Haemonchus contortus Infection in Sheep: Effects of Various Levels of Primary Infections on Nontreated Lambs. Am. J. Vet. Res. 31(5): 841-857.

21. Simesen, M. G. 1980. Calcium, Phosphorus, and Magnesium Metabolism. In Clinical Biochemistry of Domestic Animals, 3rd. ed., J. J. Kaneko (ed.), pp. 577-589. New York: Academic Press.

22. Sinclair, K. B., and R. K. Pritchard. 1975. The Use of Disophenol in Studies of the Pathogenicity of the Arrested Fourth Stage Larvae of Haemonchus contortus in Sheep. Res. Vet. Sci. 19:232-234.

23. Steward, D. F. 1953. Studies on 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. Aust. J. Agric. Res. 4(1):100-117.

24. Stoll, N. R. 1923. Investigations on the Control of Hookworm Disease. XV. An Effective Method of Counting Hookworm Eggs in Feces. Am. J. Hyg. 3:59-70.

25. Tasker, J. B. 1980. Fluids, Electrolytes, and Acid-base Balance. In Clinical Biochemistry of Domestic Animals, 3rd. ed., J. J. Kaneko (ed.), pp. 414-426. New York: Academic Press.

26. Tyler, R. 1983. Personal communication. Stillwater, Oklahoma, March 3, 1983.

27. U. S. D. A. 1976. Control of Sheep and Other Animal Diseases - Infectious, Non-infectious, and Parasitic. Washington: Agriculture Research Service, NPR No. 20440.

28. Veglia, F. 1915. The Anatomy and Life History of the Haemonchus contortus (Rud.). (Rep. 3rd and 4th). Vet. Res. Un. S. Afr.: 347-500.

29. Whitlock, J. H. 1950. The Anemias in the Trichostrongylidoses. Cornell Vet. 40:288-299.

30. Yashchenko, N. F. 1967. Biochemical Changes in the Blood Composition of Sheep Infected with Haemonchus. Veterinariia, Kiev. 15:64-65.

31. Zajicek, D., Z. Makoc, and F. Vorel. 1972. The Activities of SGOT, SGPT, Cholinesterase and Alkalic Phosphatase in Lambs Experimentally Infected with Haemonchus contortus and Trichostrongylus colubriformis, and Changes After Nilverm (ICI) Treatment. Acta Vet. BRNO. 41:405-412.

32. Zajicek, D., M. Marova, and F. Kalous. 1976. Values of Ca, Inorganic P, Mg, and Buffer Capacity in the Blood Serum of Lambs Experimentally Infected with Haemonchus contortus and Trichostrongylus colubriformis, and Changes After Treatment with Nilverm. SBORNIK Vedeckych Praci Ustredniko Statniko Vsdaternarinarniko Ustayu. 6:24-27.

APPENDIXES

TABLE VI

WEEKLY WEIGHTS IN POUNDS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN WEIGHTS

	Animal	Group	Weight (lbs.) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		50.0	52.5	53.0	61.0	58.0	68.0	70.0	81.5	77.0
	2		53.0	58.0	58.5	61.0	59.0	70.5	64.0	84.0	81.0
	3	Control	48.0	49.0	53.5	57.5	55.0	62.0	74.0	63.5	69.0
	4		54.0	56.5	60.0	68.0	67.0	78.0	82.0	94.0	88.0
	5		53.5	57.5	61.0	60.5	65.0	72.0	80.0	91.0	88.0
Group $\bar{X} \pm SD$			51.70 ± 2.59	56.13 ± 2.50	57.20 ± 3.72	62.60 ± 3.86	60.80 ± 5.12	70.10 ± 5.83	74.00 ± 7.35	82.80 ± 11.92	80.60 ± 8.02
	6		51.5	54.5	56.0	52.0	63.0	62.0	57.5		
	7		49.5	53.8	56.5	58.0	63.0	65.0	73.0	72.0	73.0
	8	I	44.0	47.5	46.5	48.0	52.0	54.0	60.5	63.0	60.0
	9		43.0	45.0	47.0	48.0	57.0	61.0	62.0	66.0	67.0
	10		47.5	51.5	55.5	58.5	66.0	73.0	71.0	74.0	73.0
Group $\bar{X} \pm SD$			47.10 ± 3.50	50.46 ± 4.10	52.30 ± 5.08	52.90 ± 5.15	60.20 ± 5.93	63.00 ± 6.89	64.80 ± 6.81	68.75 ± 5.12	68.25 ± 6.18
	11		50.0	56.0	61.0	62.0	64.0	70.5	65.5	64.5	
	12		44.5	55.0	52.5	54.5	53.0	53.0	55.5	57.5	55.0
	13	II	48.5	55.0	58.0	62.0	60.0	62.0	66.5	64.5	60.0
	14		44.0	50.0	52.5	54.5	58.0	63.0	67.0	67.5	63.0
	15		43.5	49.5	52.5	45.0	54.0				
Group $\bar{X} \pm SD$			46.10 ± 2.95	53.10 ± 3.09	55.30 ± 3.98	55.60 ± 7.01	57.80 ± 4.49	62.13 ± 7.17	63.23 ± 5.45	63.50 ± 4.24	59.33 ± 4.04
	16		48.5	54.0	54.5	53.5	57.0	53.5	56.0	51.0	55.0
	17		45.0	49.5	54.5	50.5	53.0	50.0	57.0	64.0	62.0
	18	III	47.5	53.5	56.0	53.0	52.0	55.0	56.5	61.0	60.0
	19		50.5	55.0	55.5	53.5	51.0	51.5	55.0	59.0	57.0
	20		48.0	52.0	55.0	57.0	53.0	51.5			
Group $\bar{X} \pm SD$			47.90 ± 0.80	52.80 ± 2.14	55.10 ± 0.65	53.50 ± 2.32	53.20 ± 2.28	52.30 ± 1.96	56.13 ± 0.85	58.75 ± 5.56	58.50 ± 3.11

TABLE VII

WEEKLY RESPIRATORY RATES OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN RESPIRATORY RATES

	Animal	Group	Respiratory Rate								
			Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		68	40	52	80	44	48	68	64	44
	2		136	68	40	100	44	72	76	120	56
	3	Control	48	44	48	52	40	60	68	56	40
	4		68	52	48	56	40	60	64	68	44
	5		60	44	48	68	44	44	60	48	44
Group $\bar{X}$ ± SD			76.0 ±34.5	51.0 ±12.4	47.2 ± 4.4	71.2 ±19.5	42.4 ± 2.2	56.8 ±11.1	67.2 ± 5.9	71.2 ±28.3	45.6 ± 6.1
	6		52	68	44	56	48	48	32		
	7		40	52	44	48	44	48	36	32	32
	8	I	40	52	44	52	48	48	76	60	48
	9		60	48	56	84	68	60	60	52	40
	10		72	68	48	74	44	56	68	52	40
Group $\bar{X}$ ± SD			52.8 ±13.7	57.6 ± 9.6	47.2 ± 5.2	62.8 ±15.4	50.4 ±10.0	52.0 ± 5.7	54.4 ±19.5	49.0 ±11.9	40.0 ± 6.5
	11		52	48	60	64	48	60	48	36	
	12		60	52	52	52	52	36	40	64	48
	13	II	56	72	48	56	60	44	44	48	36
	14		52	52	44	80	52	60	60	60	48
	15		48	56	40	74	64				
Group $\bar{X}$ ± SD			53.6 ± 4.6	56.0 ± 9.4	48.8 ± 7.7	65.2 ±11.8	55.2 ± 6.6	50.0 ±12.0	48.0 ± 8.6	52.0 ±12.7	44.0 ± 6.9
	16		52	60	48	68	52	40	44	44	40
	17		72	56	60	54	48	28	52	60	44
	18	III	60	40	44	44	48	28	44	52	44
	19		72	52	52	48	60	28	48	32	44
	20		60	56	52	76	52	44			
Group $\bar{X}$ ± SD			63.2 ± 8.7	52.8 ± 7.7	51.2 ± 5.9	58.0 ±13.6	52.0 ± 4.9	33.6 ± 7.8	47.0 ± 3.8	47.0 ±11.9	43.0 ± 2.0

TABLE VIII

WEEKLY HEART RATES OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN HEART RATES

	Animal	Group	Heart Rate								
			Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		128	116	112	100	188	136	120	148	104
	2		160	152	132	130	88	128	128	144	108
	3	Control	96	108	120	84	100	140	148	120	80
	4		140	128	120	84	108	164	132	148	108
	5		120	104	108	96	100	124	116	132	108
Group $\bar{X}$ ± SD			128.8 ± 23.7	125.00 ± 20.5	118.4 ± 9.2	98.8 ± 18.9	116. ± 40.4	138.4 ± 15.7	128.8 ± 12.5	138.4 ± 28.3	101.6 ± 12.2
	6		144	128	132	100	144	180	128		
	7		100	144	112	92	140	180	160	156	108
	8	I	180	156	180	93	220	180	176	160	156
	9		120	132	112	100	148	128	140	148	120
	10		156	120	124	64	160	160	144	152	116
Group $\bar{X}$ ± SD			140.0 ± 31.1	136.0 ± 14.1	132.0 ± 28.1	89.8 ± 14.9	162.4 ± 33.1	165.6 ± 22.7	149.6 ± 18.7	154.0 ± 5.2	125.0 ± 21.3
	11		144	132	152	106	176	200	172	184	
	12		148	124	120	100	80	168	136	132	132
	13	II	160	140	116	100	148	180	160	172	144
	14		140	132	108	118	172	180	156	160	120
	15		160	156	180	104	92				
Group $\bar{X}$ ± SD			150.4 ± 9.2	136.8 ± 12.1	135.2 ± 30.1	105.6 ± 7.4	133.6 ± 45.0	182.0 ± 13.3	156.0 ± 15.0	162.0 ± 22.3	132.0 ± 12.0
	16		104	148	112	88	120	128	164	168	140
	17		160	132	160	88	156	160	148	160	120
	18	III	132	120	148	88	136	168	160	120	96
	19		132	112	116	68	96	160	120	120	100
	20		148	156	144	112	180	140			
Group $\bar{X}$ ± SD			135.2 ± 21.1	133.6 ± 18.5	136.0 ± 21.0	88.8 ± 15.6	137.6 ± 32.3	151.2 ± 16.6	148.0 ± 19.9	142.0 ± 25.6	114.0 ± 20.3

TABLE IX

WEEKLY BODY TEMPERATURES OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN BODY TEMPERATURES

	Animal	Group	Body Temperature (^{0}F) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		102.0	101.5	101.5	102.1	101.2		102.5	101.2	101.0
	2		102.0	102.5	102.5	102.8	103.3		102.5	103.2	102.4
	3	Control	102.2	102.7	102.0	102.2	102.9		101.0	102.2	100.6
	4		103.6	102.9	102.5	104.0	103.3		102.6	104.0	103.8
	5		101.0	102.4	102.7	102.9	101.8		102.2	102.8	102.6
Group $\bar{X}$ ± SD			102.16 ± 0.93	102.33 ± 0.59	102.24 ± 0.49	102.80 ± 0.76	102.50 ± 0.95		102.16 ± 0.67	102.68 ± 1.05	102.08 ± 1.29
	6		103.6	103.5	102.3	103.7	103.5		99.5		
	7		103.0	102.0	101.6	103.4	102.1		102.1	104.0	102.8
	8	I	103.0	104.0	102.5	103.4	103.5		103.4	103.0	103.4
	9		102.8	103.4	101.8	102.9	103.3		102.5	102.4	103.0
	10		105.4	102.9	103.3	103.5	103.9		103.3	103.4	102.6
Group $\bar{X}$ ± SD			103.56 ± 1.07	103.16 ± 0.76	102.30 ± 0.67	103.38 ± 0.29	103.26 ± 0.68		102.16 ± 1.58	103.20 ± 0.67	102.95 ± 0.34
	11		102.7	102.4	103.0	103.1	103.2	104.4	101.2	101.0	
	12		104.1	101.9	103.0	103.2	103.1	101.7	102.5	102.4	102.8
	13	II	102.6	102.0	101.7	102.9	101.0	102.6	102.2	103.0	103.4
	14		102.6	102.0	103.0	102.9	102.6	102.7	102.7	102.2	102.4
	15		102.9	102.0	102.5	103.9	104.9				
Group $\bar{X}$ ± SD			102.98 ± 0.64	102.06 ± 0.19	102.64 ± 0.57	103.20 ± 0.41	102.96 ± 1.40	102.85 ± 1.13	102.15 ± 0.67	102.15 ± 0.84	102.87 ± 0.50
	16		103.2	102.0	101.4	101.1	102.2		104.3	104.6	102.4
	17		102.4	102.3	102.0	102.7	101.9		103.4	103.0	102.4
	18	III	102.4	102.7	103.7	101.8	102.0		101.6	102.8	103.2
	19		104.0	103.0	102.6	102.5	102.1		101.9	103.2	103.0
	20		104.0	102.0	102.0	103.5	102.5				
Group $\bar{X}$ ± SD			103.20 ± 0.80	102.40 ± 0.44	102.34 ± 0.87	102.32 ± 0.91	102.14 ± 0.23		102.80 ± 1.27	103.40 ± 0.82	102.75 ± 0.41

TABLE X

WEEKLY TOTAL WHITE BLOOD CELL COUNTS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN WHITE BLOOD CELL COUNTS

	Animal	Group	Total White Blood Cell Count								
			Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		9379	8304	7389	8447	8558	6850	6750	7502	6272
	2		10497	8557	9252	8308	7696	5200	8300	7704	7277
	3	Control	9022	7751	7334	8687	7848	6550	4100	6513	6825
	4		10281	9762	7616	9871	8700			7883	8029
	5		15629	14220	8182	14907	13807		14900	11233	8961
Group $\bar{X}$ ± SD			10951.60 ±2680.04	10210.75 ±2747.49	7954.60 ±799.16	10044.00 ±2787.66	9321.80 ±2544.72	6200.00 ±878.92	8512.50 ±4597.89	8167.00 ±1793.97	7472.80 ±1051.67
	6		8830	7769	7700	10852	7119		5550		
	7		10912	9595	9166	6749	8080	5650	5800	5251	5905
	8	I	13681	12366	13277	14194	12988	4900	6800	5654	7296
	9		13687	14645	13665	12974	8273	11850	7500		5554
	10		14149	7727	11541	12926	9538	4350	8050	6903	6544
Group $\bar{X}$ ± SD			12241.80 ±2294.78	10420.40 ±3024.82	11069.80 ±2588.86	11539.00 ±2934.62	9199.60 ±2286.23	6687.50 ±3482.67	6740.00 ±1072.03	5936.00 ±861.35	6324.75 ±766.30
	11		7004	6988	8949	10586	9213		7400	5152	
	12		12897	14351	8888	12406	10903		11700	10453	8645
	13	II	10447	9202	7815	14076	6691	5750	7350	6766	6502
	14		8085	7879	8519	6728	7405	7200	7650	6954	5907
	15		7881	8009	5123	8583	3557				
Group $\bar{X}$ ± SD			9262.80 ±2398.36	9285.80 ±2939.07	7858.80 ±1594.54	10475.80 ±2929.50	7553.80 ±2770.88	6475.00 ±1025.30	8525.00 ±2120.73	7331.25 ±2232.81	7018.00 ±1440.09
	16		11310		8165	8162	11574	4400	7300	7554	6581
	17			8842	9824	8566	7196	7100	6850	9782	6452
	18	III	13095	11707	10535	9987	11267	5600	9900	8578	8652
	19		11943	10214	9132	7764	10058	5500	7450	9439	7065
	20		11865	10387	9928	9027	8620	3400			
Group $\bar{X}$ ± SD			12053.25 ± 749.50	10287.50 ±1171.86	9516.80 ±904.93	8701.20 ±858.31	9743.00 ±1838.66	5200.00 ±1391.04	7875.00 ±1373.86	8838.25 ±994.75	7187.50 ±1011.36

TABLE XI

WEEKLY ERYTHROCYTE COUNTS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN ERYTHROCYTE COUNTS

	Animal	Group	Erythrocyte Count x 10^6								
			Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		10.36	9.39	8.78	6.99	9.40	6.58	8.24	7.40	7.53
	2		11.80	10.10	10.06	10.39	10.14	7.70	11.14	8.65	10.58
	3	Control	8.36	9.32	10.37	6.61	10.39	7.46	8.46	9.07	7.75
	4		10.72	9.97	9.25	6.84	10.37			9.12	8.38
	5		10.90	9.84	8.08	5.63	9.77		9.18	7.65	6.88
Group $\bar{X}$ ± SD			10.42 ± 1.27	9.83 ± 0.31	9.31 ± 0.93	7.29 ± 1.81	10.01 ± 0.42	7.25 ± 0.59	9.26 ± 1.32	8.38 ± 0.80	8.22 ± 1.42
	6		10.36	10.06	9.69	7.08	7.68		1.65		
	7		9.26	9.90	7.60	6.18	7.71	6.77	4.26	3.66	2.83
	8	I	12.10	11.0	10.14	8.56	9.41	5.91	5.34	4.33	3.82
	9		11.00	10.06	7.30	7.33	7.70	4.67	5.55		4.80
	10		9.27	8.51	7.90	6.23	6.09	4.57	6.07	4.26	4.92
Group $\bar{X}$ ± SD			10.40 ± 1.21	9.91 ± 0.89	8.53 ± 1.30	7.06 ± 0.97	7.72 ± 1.17	5.48 ± 1.05	4.57 ± 1.76	4.08 ± 0.37	4.09 ± 0.98
	11		10.02	8.78	8.89	6.26	7.73		3.27	1.36	
	12		6.74	9.55	9.07	6.36	9.55		10.53	7.16	8.10
	13	II	10.37	8.44	7.14	5.70	7.88	5.03	3.27	3.67	4.55
	14		9.17	9.61	8.79	7.30	10.98	6.18	5.37	4.92	4.91
	15		10.49	9.82	9.00	7.83	4.35				
Group $\bar{X}$ ± SD			9.36 ± 1.55	9.24 ± 0.60	8.58 ± 0.81	6.69 ± 0.85	8.10 ± 2.48	5.61 ± 0.81	5.61 ± 3.43	4.12 ± 2.39	5.85 ± 1.95
	16		11.14	10.06	10.04	8.48	9.02	4.28	3.34	2.92	3.65
	17			9.76	9.93	7.48	8.62	6.03	3.21	3.59	3.60
	18	III	10.72	9.86	8.27	7.12	8.39	4.88	4.12	5.61	9.04
	19		10.84	10.07	9.08	7.52	10.61	8.45	7.67	6.91	6.34
	20		11.11	10.56	10.19	10.37	7.56	2.67			
Group $\bar{X}$ ± SD			10.95 ± 0.21	10.06 ± 0.31	9.50 ± 0.81	8.19 ± 1.31	8.84 ± 1.12	5.26 ± 2.16	4.61 ± 2.09	4.76 ± 1.83	5.66 ± 2.59

TABLE XII

WEEKLY HEMATOCRITS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN HEMATOCRITS

	Animal	Group	Hematocrit								
			Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		36.5	35.0	34.0	35.0	34.0	32.0	34.0	39.0	39.0
	2		38.0	38.5	37.0	37.0	40.0	37.5	38.0	41.0	41.0
	3	Control	37.5	35.5	34.5	37.0	42.5	38.5	37.0	39.0	39.0
	4		38.5	40.0	37.0	35.0	38.0			42.0	42.0
	5		37.0	35.5	34.0	32.5	37.5		33.0	35.0	35.0
Group $\bar{X} \pm SD$			37.50 ± 0.79	37.25 ± 2.40	35.30 ± 1.57	35.30 ± 1.86	38.40 ± 3.15	36.00 ± 3.50	35.50 ± 2.38	36.20 ± 3.11	39.30 ± 2.68
	6		40.0	37.0	37.5	35.0	28.0		10.0		
	7		36.5	34.0	34.0	34.5	33.0	22.0	20.5	16.5	16.0
	8	I	42.5	41.0	42.0	39.0	36.0	26.5	23.5	20.5	18.5
	9		40.0	37.5	38.0	37.0	31.5	23.0	23.5		24.0
	10		38.0	35.0	35.0	33.0	27.0	21.5	21.5	21.0	25.0
Group $\bar{X} \pm SD$			39.40 ± 2.27	36.90 ± 2.70	37.30 ± 3.11	37.70 ± 2.33	31.10 ± 3.68	23.25 ± 2.25	19.80 ± 5.63	19.33 ± 2.47	20.88 ± 4.33
	11		36.0	35.5	38.0	38.0	30.0		16.0	8.5	
	12		38.5	37.0	37.0	38.5	36.5		35.5	35.0	35.0
	13	II	38.0	34.0	34.5	36.5	29.5	18.0	18.0	20.0	22.5
	14		39.0	37.0	34.5	38.0	34.0	19.5	18.0	20.5	24.0
	15		35.5	35.5	34.0	34.0	17.0				
Group $\bar{X} \pm SD$			37.40 ± 1.56	35.80 ± 1.25	35.60 ± 1.78	37.00 ± 1.84	29.40 ± 7.51	18.75 ± 1.06	21.88 ± 9.13	21.00 ± 10.86	27.17 ± 6.83
	16		40.0	37.5	40.0	40.0	34.5	18.5	15.5	14.5	24.0
	17		33.5	35.0	32.5	36.0	33.0	25.0	18.0	18.0	18.5
	18	III	36.5	37.0	35.0	35.0	33.5	16.5	26.5	26.5	36.0
	19		38.0	37.0	35.0	37.0	39.5	42.0	29.5	29.5	32.5
	20		37.0	36.0	36.0	36.5	28.0	8.0			
Group $\bar{X} \pm SD$			37.00 ± 2.37	36.50 ± 1.00	35.70 ± 2.73	36.90 ± 1.88	33.70 ± 4.10	22.00 ± 12.72	22.13 ± 9.78	22.13 ± 7.04	27.75 ± 7.96

TABLE XIII

WEEKLY HEMOGLOBINS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN HEMOGLOBINS

	Animal	Group	Hemoglobin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		11.0	11.3	11.0	10.9	11.6	9.4	13.2	10.3	12.5
	2		12.4	12.2	12.0	12.0	12.8	11.6	11.2	9.7	13.0
	3	Control	13.0	11.3	11.0	10.8	12.7	11.1	11.8	11.5	12.5
	4		11.6	12.2	12.8	11.3	12.0			12.8	13.3
	5		12.7	12.5	11.3	9.8	12.2		11.2	10.0	11.0
Group $\bar{X}$ ± SD			12.14 ± 0.82	12.05 ± 0.52	11.62 ± 0.78	10.96 ± 0.80	12.26 ± 0.50	10.70 ±1.15	11.85 ± 0.94	10.86 ± 1.28	12.46 ± 0.88
	6		12.3	12.2	12.0	11.6	9.8		3.8		
	7		12.7	11.3	10.9	10.5	10.8	6.8	5.6	4.5	3.5
	8	I	11.0	13.3	13.3	14.3	11.6	7.3	6.2	6.0	4.2
	9		10.6	12.0	12.3	11.6	9.8	6.6	6.5		6.5
	10		12.5	11.6	11.8	10.1	8.6	6.0	6.5	6.2	7.7
Group $\bar{X}$ ± SD			11.82 ± 0.95	12.08 ± 0.77	12.06 ± 0.87	11.62 ± 1.64	10.12 ± 1.14	6.68 ±0.54	5.72 ± 1.13	5.57 ± 0.93	5.48 ± 1.96
	11		12.7	11.6	11.6	10.8	9.8		3.3	2.5	
	12		12.7	12.0	12.0	11.0	11.3		12.1	11.0	12.5
	13	II	12.3	11.6	10.9	10.6	9.5	5.9	4.8	5.3	6.8
	14		10.6	12.1	11.3	12.6	11.0	6.0	4.5	5.6	6.3
	15		9.0	11.3	10.5	10.6	5.4				
Group $\bar{X}$ ± SD			11.46 ± 1.63	11.72 ± 0.33	11.26 ± 0.59	11.12 ± 0.84	9.40 ± 2.36	5.95 ±0.07	6.18 ± 4.00	6.10 ± 3.55	8.53 ± 3.44
	16		10.6		11.3	12.6	11.0	5.5	3.5	4.2	5.6
	17			11.3	10.8	11.3	10.6	7.3	6.5	4.5	8.0
	18	III	11.3	12.2	11.3	11.5	11.0	5.1	7.3	8.0	11.0
	19		10.6	12.5	12.0	12.0	12.7	12.7	12.7	10.3	10.3
	20		12.0	11.3	11.3	11.3	9.3	3.3			
Group $\bar{X}$ ± SD			11.13 ± 0.67	11.83 ± 0.62	11.34 ± 0.43	11.74 ± 0.56	10.92 ± 1.22	6.78 ±3.60	7.50 ± 3.83	6.75 ± 2.92	8.73 ± 2.45

TABLE XIV

WEEKLY TOTAL PROTEIN VALUES OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN TOTAL PROTEIN VALUES

	Animal	Group	Total Protein (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			6.10	5.60	6.00	6.20	6.10	6.30	6.80	6.00
	2		6.20	1.12	6.20	4.70	5.60	5.80	6.00	6.00	6.20
	3	Control	6.00	5.60	5.10	6.40	7.50	6.50		6.80	6.40
	4		6.20		5.30	5.90	6.00	6.50	4.40	6.80	6.90
	5		5.60	5.40	5.40	6.60	5.70	5.90	5.70	8.40	4.10
Group $\bar{X}$ ± SD			6.000 ± 0.283	4.200 ± 2.707	5.520 ± 0.421	5.920 ± 0.740	6.200 ± 0.765	6.160 ± 0.329	5.600 ± 0.837	6.960 ± 0.876	5.920 ± 1.071
	6		6.60	6.00	5.80	6.50	5.40	4.30			
	7		5.00	5.00	5.30	5.70	5.30	4.70	4.80	5.50	1.81
	8	I	5.60	6.00	5.20	5.80	5.20	4.20	4.70	4.60	3.90
	9		5.50	7.00	6.40		4.70	4.20	4.20	3.10	4.80
	10		5.10	5.60	5.00	5.30	4.50	4.50	4.20	4.80	4.60
Group $\bar{X}$ ± SD			5.560 ± 0.635	5.920 ± 0.729	5.540 ± 0.563	5.825 ± 0.499	5.020 ± 0.396	4.380 ± 0.216	4.475 ± 0.320	4.500 ± 1.010	3.775 ± 1.372
	11		6.30	5.90		6.10	5.40	4.70		2.60	
	12		5.60	5.40	5.50	6.60	5.20	5.50	5.80	5.50	5.60
	13	II	6.40	5.80	5.50	5.70	4.50	5.10	5.10	3.20	4.20
	14		6.70	5.70	5.40	6.20	5.40	7.40	4.60	4.80	5.00
	15		6.10	6.90	5.10	6.40	3.40				
Group $\bar{X}$ ± SD			6.220 ± 0.409	5.940 ± 0.568	5.375 ± 0.189	6.200 ± 0.339	4.780 ± 0.856	5.675 ± 1.195	5.167 ± 0.603	4.025 ± 1.352	4.933 ± 0.702
	16		6.40	8.70	6.50	5.50	6.30	5.80	4.10	4.20	4.60
	17		6.20	5.30	4.50	5.20	5.00	5.50	4.80	4.10	4.00
	18	III	5.60	6.10	6.20	5.90		4.70	6.00		5.90
	19			6.10	4.80	6.10	5.50	6.10	6.00	5.10	5.70
	20		5.60	5.40	6.00	6.00	4.70	3.10			
Group $\bar{X}$ ± SD			5.950 ± 0.412	6.320 ± 1.383	5.600 ± 0.892	5.740 ± 0.378	5.375 ± 0.699	5.040 ± 1.203	5.225 ± 0.939	4.467 ± 0.551	5.050 ± 0.904

TABLE XV

WEEKLY ALBUMIN LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN ALBUMIN LEVELS

	Animal	Group	Albumin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			3.92	3.79	3.40	3.90	3.57	3.41	4.34	3.99
	2		4.40	0.79	3.81	3.06	3.01	3.61	3.81	4.43	3.66
	3	Control	3.90	3.97	3.31	3.77	4.31	4.00		4.07	4.77
	4		4.15		2.92	3.45	3.75	3.85	2.92	4.09	4.06
	5		4.16	4.01	3.54	3.95	3.47	3.63	3.79	6.29	3.17
Group $\bar{X} \pm SD$			4.153 ± 0.204	2.907 ± 1.834	3.474 ± 0.371	3.526 ± 0.346	3.688 ± 0.486	3.732 ± 0.185	3.465 ± 0.402	4.644 ± 0.933	3.930 ± 0.587
	6		4.42	3.98	3.50	4.06	3.38	2.54			
	7		4.10	4.42	3.30	3.65	3.17	2.86	2.85	3.87	1.16
	8	I	3.60	3.97	2.79	2.99	2.86	2.05	2.34	2.98	1.94
	9		3.28	4.75	3.99		2.94	2.47	2.61	2.15	2.83
	10		3.16	3.78	3.09	3.42	2.92	2.76	2.50	2.97	2.62
Group $\bar{X} \pm SD$			3.724 ± 0.535	4.180 ± 0.396	3.334 ± 0.451	3.530 ± 0.445	3.054 ± 0.217	2.536 ± 0.315	2.575 ± 0.214	2.993 ± 0.702	2.138 ± 0.754
	11		4.55	4.22		3.74	3.41	2.64		1.27	
	12		3.18	4.13	3.39	3.74	3.14	3.04	3.55	3.80	3.14
	13	II	3.57	3.62	3.21	3.37	2.74	2.46	2.58	1.95	2.78
	14		4.70	4.09	3.34	3.86	3.00	4.13	2.64	2.59	2.59
	15		4.71	4.91	3.51	3.99	2.02				
Group $\bar{X} \pm SD$			4.142 ± 0.716	4.194 ± 0.463	3.363 ± 0.124	3.740 ± 0.231	2.862 ± 0.529	3.068 ± 0.749	2.923 ± 0.544	2.403 ± 1.076	2.837 ± 0.279
	16		3.85	6.21	4.42	3.36	4.11	3.29	2.32	2.21	2.21
	17		3.69	3.84	2.85	3.48	3.04	3.10	2.89	2.33	2.42
	18	III	3.97	3.93	3.94	3.45		2.60	3.58		3.21
	19			4.66	3.36	3.84	3.35	3.57	3.71	3.67	3.35
	20		4.17	3.56	3.87	3.80	2.84	1.48			
Group $\bar{X} \pm SD$			3.920 ± 0.202	4.440 ± 1.070	3.688 ± 0.600	3.586 ± 0.219	3.335 ± 0.558	2.808 ± 0.822	3.125 ± 0.646	2.737 ± 0.811	2.798 ± 0.567

TABLE XVI

WEEKLY ALPHA-1 GLOBULIN LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN ALPHA-1 GLOBULIN LEVELS

Animal	Group	Alpha-1 Globulin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
1			0.24	0.08	0.13	0.11	0.31	0.15	0.13	0.13
2		0.11	0.02	0.20	0.16	0.15	0.36	0.10	0.14	0.17
3	Control	0.22	0.12	0.17	0.13	0.22	0.31		0.33	0.09
4		0.09		0.17	0.21	0.17	0.52	0.13	0.13	0.12
5		0.23	0.09	0.11	0.07	0.30	0.15	0.10	0.17	0.05
Group $\bar{X}$ ± SD		0.163 ± 0.073	0.118 ± 0.092	0.146 ± 0.049	0.140 ± 0.051	0.190 ± 0.073	0.330 ± 0.132	0.120 ± 0.024	0.180 ± 0.085	0.112 ± 0.045
6		0.10	0.12	0.10	0.20	0.08	0.15			
7		0.20	0.06	0.14	0.05	0.15	0.39	0.14	0.10	0.10
8	I	0.14	0.14	0.29	0.22	0.10	0.40	0.21	0.15	0.44
9		0.23	0.16	0.13		0.15	0.28	0.20	0.11	0.25
10		0.18	0.15	0.18	0.18	0.13	0.19	0.11	0.16	0.38
Group $\bar{X}$ ± SD		0.170 ± 0.051	0.126 ± 0.040	0.168 ± 0.074	0.163 ± 0.077	0.122 ± 0.031	0.282 ± 0.113	0.165 ± 0.048	0.130 ± 0.029	0.293 ± 0.150
11		0.24	0.11		0.17	0.14	0.39		0.08	
12		0.35	0.03	0.16	0.15	0.17	0.24	0.14	0.06	0.37
13	II	0.24	0.19	0.17	0.16	0.13	0.58	0.20	0.16	0.24
14		0.16	0.08	0.11	0.10	0.27	0.53	0.21	0.16	0.28
15		0.14	0.16	0.10	0.17	0.14				
Group $\bar{X}$ ± SD		0.226 ± 0.083	0.114 ± 0.063	0.135 ± 0.035	0.150 ± 0.029	0.170 ± 0.058	0.435 ± 0.153	0.183 ± 0.038	0.115 ± 0.053	0.297 ± 0.066
16		0.15	0.04	0.07	0.17	0.10	0.32	0.26	0.16	0.39
17		0.10	0.11	0.07	0.10	0.16	0.30	0.14	0.20	0.06
18	III	0.10	0.23	0.13	0.20		0.27	0.18		0.44
19			0.12	0.11	0.20	0.21	0.40	0.20	0.06	0.24
20		0.06	0.14	0.08	0.12	0.21	0.33			
Group $\bar{X}$ ± SD		0.103 ± 0.037	0.128 ± 0.068	0.112 ± 0.046	0.158 ± 0.046	0.170 ± 0.052	0.324 ± 0.048	0.195 ± 0.050	0.140 ± 0.072	0.283 ± 0.171

TABLE XVII

WEEKLY ALPHA-2 GLOBULIN LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN ALPHA-2 GLOBULIN LEVELS

	Animal	Group	Alpha-2 Globulin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			0.24	0.26	0.26	0.31	0.21	0.28	0.41	0.29
	2		0.26	0.04	0.28	0.17	0.31	0.26	0.34	0.21	0.32
	3	Control	0.27	0.12	0.17	0.26	0.29	0.28		0.27	0.13
	4		0.20		0.32	0.33	0.25	0.33	0.13	0.30	0.21
	5		0.43	0.50	0.54	0.37	0.55	0.54	0.62	0.70	0.33
Group $\bar{X}$ ± SD			0.290 ± 0.098	0.225 ± 0.201	0.314 ± 0.138	0.278 ± 0.077	0.342 ± 0.189	0.324 ± 0.128	0.343 ± 0.205	0.378 ± 0.194	0.256 ± 0.085
	6		0.32	0.27	0.37	0.41	0.29	0.26			
	7		0.23	0.19	0.21	0.30	0.31	0.24	0.22	0.21	0.10
	8	I	0.22	0.23	0.31	0.32	0.24	0.24	0.25	0.22	0.24
	9		0.27	0.26	0.18		0.19	0.18	0.18	0.11	0.25
	10		0.26	0.26	0.28	0.28	0.25	0.19	0.25	0.29	0.28
Group $\bar{X}$ ± SD			0.260 ± 0.039	0.242 ± 0.037	0.268 ± 0.075	0.328 ± 0.057	0.256 ± 0.047	0.222 ± 0.035	0.225 ± 0.033	0.208 ± 0.074	0.218 ± 0.080
	11		0.24	0.13		0.25	0.18	0.31		0.14	
	12		0.25	0.14	0.25	0.29	0.24	0.29	0.30	0.28	0.29
	13	II	0.28	0.17	0.20	0.30	0.22	0.23	0.31	0.11	0.18
	14		0.25	0.18	0.29	0.29	0.29	0.37	0.28	0.29	0.40
	15		0.21	0.22	0.23	0.60	0.19				
Group $\bar{X}$ ± SD			0.246 ± 0.025	0.168 ± 0.036	0.243 ± 0.038	0.346 ± 0.143	0.224 ± 0.044	0.300 ± 0.058	0.297 ± 0.015	0.205 ± 0.093	0.290 ± 0.110
	16		0.68	0.79	0.59	0.36	0.67	0.72	0.57	0.71	0.78
	17		0.23	0.37	0.44	0.50	0.48	0.66	0.49	0.41	0.38
	18	III	0.23	0.26	0.30	0.30		0.29	0.38		0.30
	19			0.43	0.40	0.26	0.32	0.27	0.27	0.44	0.33
	20		0.37	0.48	0.60	0.55	0.46	0.44			
Group $\bar{X}$ ± SD			0.378 ± 0.212	0.446 ± 0.199	0.466 ± 0.128	0.394 ± 0.126	0.483 ± 0.052	0.476 ± 0.207	0.428 ± 0.131	0.520 ± 0.165	0.448 ± 0.224

TABLE XVIII

WEEKLY BETA-1 GLOBULIN LEVELS OF THE EXPERIMENTAL LAMBS AND . GROUP WEEKLY MEAN BETA-1 GLOBULIN LEVELS

Animal	Group	Beta-1 Globulin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
1			0.43	0.49	0.53	0.48	0.60	0.57	0.50	0.48
2		0.57	0.11	0.65	0.42	0.52	0.59	0.64	0.52	0.81
3	Control	0.49	0.50	0.53	0.59	0.78	0.56		0.66	0.53
4		0.61		0.45	0.60	0.58	0.57	0.46	0.36	0.62
5		0.23	0.19	0.34	0.35	0.33	0.25	0.29	0.35	0.19
Group		0.475	0.308	0.492	0.498	0.538	0.514	0.490	0.478	0.526
$\bar{X}$ ± SD	±	0.171 ±	0.187 ±	0.113 ±	0.109 ±	0.164 ±	0.148 ±	0.153 ±	0.128 ±	0.226
6		0.67	0.55	0.53	0.60	0.61	0.56			
7		0.49	0.46	0.61	0.50	0.55	0.36	0.50	0.76	0.21
8	I	0.46	0.51	0.48	0.63	0.57	0.46	0.54	0.63	0.52
9		0.52	0.73	0.71		0.52	0.46	0.50	0.40	0.74
10		0.49	0.47	0.55	0.51	0.45	0.62	0.59	0.69	0.54
Group		0.526	0.544	0.576	0.560	0.540	0.492	0.533	0.620	0.503
$\bar{X}$ ± SD	±	0.083 ±	0.110 ±	0.088 ±	0.065 ±	0.060 ±	0.101 ±	0.043 ±	0.156 ±	0.219
11		0.55	0.53		0.70	0.50	0.46		0.47	
12		0.46	0.39	0.48	0.73	0.46	0.60	0.55	0.46	0.54
13	II	0.60	0.48	0.53	0.45	0.36	0.62	0.75	0.51	0.63
14		0.60	0.39	0.51	0.55	0.56	0.85	0.56	0.88	0.67
15		0.65	0.65	0.38	0.29	0.29				
Group		0.572	0.488	0.475	0.544	0.434	0.633	0.620	0.580	0.613
$\bar{X}$ ± SD	±	0.072 ±	0.109 ±	0.067 ±	0.182 ±	0.109 ±	0.162 ±	0.113 ±	0.201 ±	0.067
16		0.29	0.39	0.28	0.51	0.33	0.32	0.24	0.41	0.35
17		0.40	0.23	0.20	0.30	0.22	0.25	0.30	0.27	0.23
18	III	0.48	0.52	0.60	0.52		0.59	0.74		0.62
19			0.19	0.19	0.34	0.25	0.32	0.34	0.16	0.54
20		0.26	0.31	0.29	0.33	0.26	0.23			
Group		0.358	0.328	0.312	0.400	0.265	0.342	0.405	0.280	0.435
$\bar{X}$ ± SD	±	0.101 ±	0.132 ±	0.167 ±	0.106 ±	0.047 ±	0.144 ±	0.227 ±	0.125 ±	0.177

TABLE XIX

WEEKLY BETA-2 GLOBULIN LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN BETA-2 GLOBULIN LEVELS

	Animal	Group	Beta-2 Globulin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			0.32	0.26	0.38	0.34	0.31	0.36	0.38	0.22
	2		0.22	0.03	0.26	0.28	0.46	0.24	0.34	0.17	0.37
	3	Control	0.32	0.21	0.24	0.59	0.48	0.42		0.50	0.22
	4		0.17		0.32	0.31	0.43	0.44	0.24	0.16	0.29
	5		0.12	0.14	0.17	0.35	0.28	0.21	0.20	0.26	0.05
Group			0.208	0.175	0.250	0.382	0.374	0.322	0.285	0.294	0.230
$\bar{X}$ ± SD		±	0.085 ±	0.122 ±	0.054 ±	0.122 ±	0.090 ±	0.101 ±	0.077 ±	0.145 ±	0.118
	6		0.22	0.25	0.37	0.30	0.26	0.24			
	7		0.23	0.19	0.21	0.34	0.31	0.29	0.27	0.24	0.10
	8	I	0.83	0.31	0.32	0.37	0.33	0.40	0.38	0.24	0.28
	9		0.31	0.20	0.32		0.28	0.25	0.21	0.11	0.24
	10		0.26	0.23	0.21	0.26	0.20	0.21	0.25	0.31	0.30
Group			0.370	0.236	0.286	0.318	0.276	0.278	0.278	0.225	0.230
$\bar{X}$ ± SD		±	0.260 ±	0.048 ±	0.072 ±	0.048 ±	0.050 ±	0.074 ±	0.073 ±	0.083 ±	0.090
	11		0.14	0.24		0.37	0.32	0.22		0.13	
	12		0.39	0.08	0.16	0.41	0.24	0.26	0.27	0.21	0.32
	13	II	0.38	0.34	0.28	0.40	0.24	0.39	0.42	0.11	0.24
	14		0.16	0.16	0.17	0.36	0.29	0.37	0.26	0.24	0.30
	15		0.14	0.28	0.27	0.43	0.27				
Group			0.242	0.220	0.220	0.394	0.272	0.310	0.317	0.173	0.287
$\bar{X}$ ± SD		±	0.131 ±	0.102 ±	0.064 ±	0.029 ±	0.034 ±	0.083 ±	0.090 ±	0.062 ±	0.042
	16		0.31	0.17	0.28	0.30	0.19	0.39	0.24	0.17	0.28
	17		0.23	0.20	0.24	0.20	0.32	0.36	0.28	0.27	0.25
	18	III	0.20	0.29	0.27	0.55		0.38	0.35		0.32
	19			0.15	0.06	0.52	0.27	0.55	0.47	0.16	0.22
	20		0.20	0.22	0.29	0.33	0.26	0.30			
Group			0.235	0.206	0.228	0.380	0.260	0.396	0.335	0.200	0.268
$\bar{X}$ ± SD		±	0.052 ±	0.054 ±	0.096 ±	0.150 ±	0.054 ±	0.093 ±	0.101 ±	0.061 ±	0.043

TABLE XX

WEEKLY GAMMA-1 GLOBULIN LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN GAMMA-1 GLOBULIN LEVELS

	Animal	Group	Gamma-1 Globulin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			0.93	0.72	1.19	1.01	0.99	1.28	0.97	0.83
	2		0.63	0.12	0.93	0.54	0.96	0.67	0.72	0.49	0.81
	3	Control	0.75	0.62	0.64	0.90	1.18	0.89		0.94	0.57
	4		0.57		0.96	0.88	0.84	0.71	0.50	0.62	0.62
	5		0.43	0.45	0.65	1.07	0.73	0.54	0.65	0.57	0.27
Group $\bar{X}$ ± SD			0.595 ±0.133	0.530 ±0.338	0.780 ±0.154	0.916 ±0.246	0.944 ±0.171	0.760 ±0.179	0.780 ±0.341	0.718 ±0.222	0.620 ±0.226
	6		0.74	0.67	0.85	0.88	0.78	0.52			
	7		0.58	0.53	0.79	0.84	0.72	0.48	0.59	0.31	0.20
	8	I	0.76	0.84	0.90	1.21	1.10	0.49	0.72	0.37	0.41
	9		0.82	0.83	0.99		0.57	0.46	0.47	0.20	0.43
	10		0.67	0.55	0.61	0.62	0.52	0.44	0.45	0.38	0.44
Group $\bar{X}$ ± SD			0.714 ±0.092	0.684 ±0.148	0.828 ±0.142	0.888 ±0.243	0.738 ±0.229	0.478 ±0.030	0.558 ±0 125	0.315 ±0.083	0.370 ±0.114
	11		0.55	0.61		0.86	0.80	0.59		0.44	
	12		0.81	0.56	0.84	1.17	0.89	0.93	0.92	0.07	0.90
	13	II	1.25	0.91	1.06	0.93	0.76	0.73	0.77	0.35	0.51
	14		0.83	0.78	0.95	1.01	0.92	1.09	0.62	0.62	0.71
	15		0.35	0.59	0.56	0.82	0.42				
Group $\bar{X}$ ± SD			0.758 ± 0.339	0.690 ± 0.150	0.853 ± 0.215	0.958 ± 0.139	0.758 ± 0.200	0.835 ± 0.220	0.770 ± 0.150	0.370 ± 0.229	0.707 ± 0.195
	16		1.04	1.09	0.83	0.73	0.86	0.70	0.45	0.50	0.59
	17		0.53	0.52	0.66	0.60	0.67	.71	0.64	0.52	0.50
	18	III	0.60	0.84	0.91	0.83		0.54	0.77		0.90
	19			0.51	0.89	0.89	0.85	0.89	0.94	0.60	0.88
	20		0.43	0.61	0.71	0.81	0.62	0.28			
Group $\bar{X}$ ± SD			0.650 ± 0.269	0.714 ± 0.249	0.800 ± 0.110	0.772 ± 0.112	0.750 ± 0.123	0.624 ± 0.229	0.700 ± 0.207	0.540 ± 0.053	0.718 ± 0.203

TABLE XXI

WEEKLY GAMMA-2 GLOBULIN LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN GAMMA-2 GLOBULIN LEVELS

	Animal	Group	Gamma-2 Globulin (gm%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			0.00	0.00	0.11	0.06	0.10	0.25	0.06	0.07
	2		0.03	0.02	0.07	0.07	0.19	0.07	0.05	0.03	0.02
	3	Control	0.05	0.06	0.04	0.05	0.24	0.06		0.03	0.09
	4		0.00		0.17	0.12	0.10	0.09	0.02	0.03	0.08
	5		0.00	0.02	0.04	0.10	0.05	0.59	0.05	0.04	0.03
Group $\bar{X}$ ± SD			0.020 ±0.024	0.025 ±0.025	0.064 ±0.064	0.090 ±0.029	0.128 ±0.083	0.182 ±0.229	0.093 ±0.106	0.038 ±0.013	0.058 ±0.031
	6		0.03	0.15	0.08	0.05	0.00	0.02			
	7		0.06	0.06	0.04	0.02	0.09	0.07	0.23	0.00	0.00
	8	I	0.03	0.00	0.12	0.06	0.00	0.16	0.26	0.00	0.07
	9		0.07	0.07	0.08		0.05	0.10	0.03	0.00	0.05
	10		0.06	0.06	0.08	0.03	0.03	0.08	0.05	0.00	0.04
Group $\bar{X}$ ± SD			0.050 ±0.019	0.068 ±0.054	0.080 ±0.028	0.040 ±0.018	0.034 ±0.038	0.086 ±0.030	0.143 ±0.119	0.000 ±0.000	0.040 ±0.029
	11		0.03	0.05		0.12	0.04	0.09		0.07	
	12		0.16	0.06	0.12	0.12	0.06	0.14	0.07	0.03	0.05
	13	II	0.08	0.10	0.05	0.09	0.05	0.09	0.07	0.03	0.02
	14		0.00	0.03	0.04	0.03	0.07	0.05	0.03	0.02	0.05
	15		0.00	0.09	0.05	0.10	0.07				
Group $\bar{X}$ ± SD			0.054 ±0.068	0.066 ±0.029	0.065 ±0.037	0.092 ±0.037	0.058 ±0.013	0.093 ±0.037	0.057 ±0.023	0.038 ±0.022	0.040 ±0.017
	16		0.08	0.00	0.03	0.07	0.04	0.05	0.02	0.02	0.00
	17		0.02	0.03	0.04	0.02	0.11	0.11	0.06	0.09	0.05
	18	III	0.02	0.03	0.05	0.05		0.04	0.00		0.11
	19			0.04	0.00	0.05	0.06	0.10	0.07	0.00	0.12
	20		0.00	0.08	0.06	0.06	0.05	0.02			
Group $\bar{X}$ ± SD			0.030 ±0.035	0.036 ±0.029	0.036 ±0.023	0.050 ±0.019	0.065 ±0.031	0.064 ±0.039	0.038 ±0.033	0.037 ±0.047	0.070 ±0.056

TABLE XXII

WEEKLY ALBUMIN:GLOBULIN RATIOS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN ALBUMIN:GLOBULIN RATIOS

	Animal	Group	Albumin:Globulin Ratio								
			Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1			1.80	2.09	1.31	1.70	1.41	1.18	1.76	1.98
	2		2.44	2.38	1.59	1.87	1.16	1.65	1.74	2.82	1.44
	3	Control	1.86	2.44	1.85	1.43	1.35	1.60		1.49	2.92
	4		2.65		1.23	1.41	1.67	1.46	1.97	2.76	2.55
	5		2.08	2.88	1.91	1.49	1.55	1.60	1.98	2.98	3.41
Group $\bar{X}$ ± SD			2.258 ±0.354	2.375 ±0.443	1.734 ±0.334	1.502 ±0.216	1.486 ±0.228	1.544 ±0.103	1.718 ±0.375	2.362 ±0.684	2.460 ±0.773
	6		2.22	1.98	1.52	1.66	1.67	1.44			
	7		2.27	2.98	1.65	1.78	1.49	1.56	1.46	2.36	1.70
	8	I	1.69	1.96	1.16	1.06	1.22	0.96	0.99	1.85	.99
	9		1.48	2.12	1.66		1.67	1.43	1.64	2.27	1.44
	10		1.63	2.20	1.62	1.82	1.85	1.59	1.47	1.62	1.32
Group $\bar{X}$ ± SD			1.858 ±0.362	2.248 ±0.421	1.522 ±0.210	1.580 ±0.353	1.580 ±0.238	1.396 ±0.254	1.390 ±0.279	2.025 ±0.350	1.363 ±0.295
	11		2.61	2.51		1.58	1.71	1.29		0.95	
	12		1.31	3.27	1.61	1.30	1.52	1.23	1.58	2.23	1.28
	13	II	1.26	1.66	1.40	1.45	1.56	0.93	1.02	1.55	1.29
	14		2.35	2.53	1.62	1.65	1.25	1.26	1.35	1.17	1.07
	15		3.40	2.47	2.20	1.66	1.46				
Group $\bar{X}$ ± SD			2.186 ±0.909	2.488 ±0.570	1.708 ±0.344	1.528 ±0.153	1.500 ±0.167	1.178 ±0.167	1.317 ±0.281	1.475 ±0.561	1.213 ±0.124
	16		1.51	2.49	2.12	1.57	1.88	1.31	1.30	1.11	0.93
	17		2.45	2.63	1.73	2.02	1.55	1.30	1.51	1.32	1.93
	18	III	2.43	1.81	1.74	1.41		1.24	1.48		1.20
	19			3.24	2.33	1.70	1.55	1.41	1.62	2.58	1.43
	20		3.13	1.93	1.82	1.73	1.53	0.92			
Group $\bar{X}$ ± SD			2.380 ±0.665	2.420 ±0.577	1.948 ±0.266	1.686 ±0.225	1.628 ±0.169	1.236 ±0.187	1.478 ±0.133	1.670 ±0.795	1.373 ±0.424

TABLE XXIII

WEEKLY SERUM MAGNESIUM LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN SERUM MAGNESIUM LEVELS

	Animal	Group	Serum Magnesium (mg%) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		1.1	4.2	2.6		2.2	2.2	3.0	2.3	3.0
	2		3.1	2.1	2.2		2.5	1.2	3.0	2.2	3.0
	3	Control	2.1	3.0	3.9		2.2	2.0	2.8	2.0	2.8
	4		2.5	1.5	4.4		2.4	1.8	2.7	2.8	2.7
	5		3.5	3.2	2.2		2.1	1.5	2.8	1.9	2.8
Group $\bar{X}$ ± SD			2.46 ±0.93	2.80 ±1.04	3.06 ±1.02		2.28 ±0.16	1.74 ±1.40	2.86 ± 0.13	2.24 ±0.35	2.86 ±0.13
	6		4.0	3.1	2.8		2.3	1.2	2.6		
	7		3.3	2.5	3.3		2.2	1.2	3.0	1.2	3.0
	8	I	3.2	2.4	3.0		2.2	1.3	2.3	1.3	2.3
	9		3.9	1.2	2.5		2.2	1.3	3.5	1.3	3.5
	10		2.4	2.2	2.6		1.9	1.2	2.7	.9	2.7
Group $\bar{X}$ ± SD			3.36 ±0.64	2.28 ±0.69	2.84 ±0.32		2.16 ±0.15	1.24 ±0.05	2.82 ±0.45	1.18 ±0.19	2.88 ±1.47
	11		1.8	2.7	3.1		2.0	1.4	2.5	1.4	
	12		2.2	2.3	2.3		2.3	2.3	2.4	2.4	2.4
	13	II	2.9	2.9	3.1		2.0	1.6	2.9	1.7	2.5
	14		3.1	2.7	2.4		2.3	2.8	3.2		3.2
	15		3.5	2.0	3.1		1.9				
Group $\bar{X}$ ± SD			2.70 ±0.69	2.52 ±0.36	2.80 ±0.41		2.10 ±0.19	2.03 ±0.64	2.75 ±0.37	1.83 ±0.51	2.70 ±0.44
	16		2.4	3.0	2.0		2.4	1.4	3.0	1.4	3.0
	17		2.6	2.2	2.4		2.3	1.0	2.5	1.5	2.5
	18	III	4.2	2.3	3.9		2.3	1.3	3.2	1.5	3.2
	19		2.1	1.9	4.5		2.3	1.4	2.9	2.6	2.9
	20		3.8	3.2	3.0		2.1	1.8			
Group $\bar{X}$ ± SD			3.02 ±0.92	2.52 ±0.55	3.16 ± 1.04		2.28 ± 0.11	1.38 ± 0.29	2.90 ± 0.29	1.75 ± 0.57	2.90 ±0.29

TABLE XXIV

WEEKLY SERUM POTASSIUM LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN SERUM POTASSIUM LEVELS

	Animal	Group	Serum Potassium (meq/l) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		4.0	6.2	5.4	6.0	5.1	5.0	5.3	4.7	4.6
	2		5.2	6.1	6.2	6.8	6.2	5.3	5.9	5.7	6.1
	3	Control	5.1	5.5	5.0	5.2	5.2	5.4	5.0	5.6	5.1
	4		5.3	4.2	5.2	5.4	4.9	5.0	4.2	5.5	5.4
	5		4.6	4.5	5.1	4.9	4.9	5.0	4.0	5.0	4.1
Group $\bar{X}$ ± SD			4.84 ±0.54	5.30 ±0.91	5.38 ±0.48	5.60 ±0.75	5.26 ±0.54	5.14 ±0.19	4.88 ±0.79	5.30 ±0.40	5.06 ±0.76
	6		5.4	6.8	6.2	6.1	5.0	3.8	3.4		
	7		5.1	6.1	5.4		5.3	5.8	4.3	3.3	3.1
	8	I	4.9	5.6	5.3	5.5	5.5	4.2	5.6	4.2	4.2
	9		5.0	3.1	5.0	4.8	4.8	4.6	4.2	3.6	4.2
	10		5.1	5.5	5.3	5.1	5.3	3.7	4.5	4.0	4.1
Group $\bar{X}$ ± SD			5.10 ±0.19	5.42 ±1.40	5.44 ±0.45	5.38 ±0.56	5.18 ±0.28	4.42 ±0.85	4.40 ±0.79	3.78 ±0.40	3.90 ±0.54
	11		4.9	5.2	5.2	5.3	5.1	4.3	4.3	2.8	
	12		5.1	6.1	5.4	5.7	5.4	5.0	5.3	4.7	5.4
	13	II	5.1	5.3	4.8	4.7	4.6	4.3	4.9	4.0	5.1
	14		5.3	4.8	5.1	5.0	5.0	6.4	3.5	3.8	4.9
	15		4.7	4.9	5.2	4.9	4.6				
Group $\bar{X}$ ± SD			5.02 ±0.23	5.26 ±0.51	5.14 ±0.22	5.12 ±0.39	4.94 ±0.34	5.00 ± 0.99	4.50 ± 0.78	3.83 ± 0.78	5.13 ± 0.25
	16		5.0	5.6	4.6	4.9	5.0	5.2	3.9	4.2	4.4
	17		5.1	5.3	5.8	5.6	5.0	4.7	4.3	5.1	5.2
	18	III	5.1	5.6	4.7	5.3	5.1	3.6	4.3	4.1	4.8
	19		5.5	4.9	5.2	4.9	5.2	5.4	5.1	4.8	5.7
	20		5.0	6.1	4.7	4.3	4.7	4.4			
Group $\bar{X}$ ± SD			5.14 ±0.21	5.50 ±0.44	5.00 ±0.50	5.00 ±0.49	5.00 ±0.19	4.66 ±0.71	4.40 ± 0.50	4.55 ±0.48	5.02 ±0.56

TABLE XXV

WEEKLY SERUM CHLORIDE LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN SERUM CHLORIDE LEVELS

	Animal	Group	Serum Chloride (meq/l) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		80	99	95	105	102	103	96	98	103
	2		89	81	97	109	105	89	106	106	101
	3	Control	100	103		100	103	97	98	101	99
	4		104	82	100	110	104	100	80	109	103
	5		102	88	97	105	104	105	84	100	82
Group $\bar{X}$ ± SD			95.0 ±10.2	90.6 ±10.0	97.3 ±2.1	105.8 ±4.0	103.6 ±1.1	98.8 ±6.3	92.8 ±10.6	102.8 ±4.6	97.6 ±8.9
	6		103	105	97	105	108	78	101		
	7		102	106	98		102	107	89	74	63
	8	I	99	101	93	100	106	87	107	85	80
	9		104	61	103	101	104	91	87	80	89
	10		100		95	103	102	73	89	84	88
Group $\bar{X}$ ± SD			101.6 ± 2.1	93.3 ±21.6	97.2 ±3.8	102.3 ±2.2	104.4 ±2.6	87.2 ±13.2	94.6 ± 8.9	80.8 ±8.9	80.0 ±12.0
	11		101	104	97	102	104	86	99	84	
	12		102	102		102	101	102	102	96	99
	13	II	102	104	97	96	94	90	93	85	101
	14		101	103	100	106	104	132	78	83	104
	15		99	100	98	88	106				
Group $\bar{X}$ ± SD			101.0 ± 1.2	102.6 ± 1.7	98.0 ±1.4	98.8 ±7.0	101.8 ±4.7	102.5 ±20.8	93.0 ±10.7	87.0 ±6.1	101.3 ±2.5
	16		99	99	78	98	102	116	84	81	89
	17		104	105	105	99	109	93	89	104	96
	18	III	102	103	88	108	104	91	88	80	101
	19		101	87	99	101	102	94	96	93	104
	20		107	107		89	106	111			
Group $\bar{X}$ ± SD			102.8 ± 3.0	100.2 ±8.0	92.5 ±12.0	99.0 ±6.8	104.6 ±3.0	101.0 ±11.6	89.3 ±5.0	89.5 ±11.3	97.5 ±6.6

TABLE XXVI

WEEKLY SERUM CALCIUM LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN SERUM CALCIUM LEVELS

	Animal	Group	Serum Calcium (mg/dl) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		8.2	11.2	10.7	12.0	11.2	11.5	11.0	10.7	11.8
	2		10.5	8.9	11.2	12.1	11.6	9.6	11.6	12.5	11.5
	3	Control	11.7	11.6	11.9	11.7	10.8	11.6	11.5	11.8	11.2
	4		12.3	9.0	11.0	11.8	11.5	12.2	9.5	12.1	11.8
	5		12.2	9.5	10.7	11.8	10.8	12.1	9.6	11.6	8.4
Group $\bar{X}$ ± SD			11.00 ±1.73	10.04 ±1.27	11.10 ±0.49	11.88 ±0.16	11.18 ±0.38	11.40 ±1.05	10.64 ±1.02	11.74 ±0.67	10.94 ±1.44
	6		11.8	11.2	11.3	11.6	11.8	6.5	8.8		
	7		12.7	11.5	10.9		11.3	11.6	8.2	7.0	5.7
	8	I	10.2	10.0	9.7	10.4	10.8	8.5	10.9	7.6	6.3
	9		11.4	6.1	12.3	10.7	10.5	8.8	7.9	7.4	8.0
	10		11.8	10.9	11.2	11.2	10.7	6.7	8.6	7.6	8.2
Group $\bar{X}$ ± SD			11.58 ±0.91	9.94 ±2.22	11.08 ±0.93	10.98 ±0.53	11.02 ±0.53	8.42 ±2.06	8.88 ±1.18	7.40 ±0.28	7.05 ±1.24
	11		11.5	12.1	11.6	11.6	11.6	8.3	9.5	6.5	
	12		11.1	11.5	11.1	11.1	10.7	11.5	10.6	10.3	10.4
	13	II	11.9	12.1	11.4	11.6	9.9	9.3	10.6	8.1	9.5
	14		12.2	12.0	11.1	13.1	11.6	16.3	7.6	7.5	9.8
	15		12.0	11.7	11.3	10.6	9.5				
Group $\bar{X}$ ± SD			11.74 ±0.44	11.88 ±0.27	11.30 ±0.21	11.60 ±0.94	10.66 ±0.96	11.35 ±3.56	9.56 ±1.42	8.10 ±1.61	9.90 ±0.46
	16		13.7	14.2	10.1	12.7	12.2	11.2	7.7	7.0	7.8
	17		10.8	11.3	11.7	10.6	11.0	8.6	8.6	9.4	8.8
	18	III	11.7	11.4	9.5	11.5	10.8	7.6	9.2	8.4	10.6
	19		11.4	9.1	10.9	11.1	11.2	11.1	10.9	9.4	11.3
	20		12.0	12.9	11.5	9.8	10.4	8.3			
Group $\bar{X}$ ± SD			11.92 ±1.09	11.78 ±1.91	10.74 ±0.93	11.14 ±1.07	11.12 ±0.67	9.36 ±1.67	9.10 ±1.35	8.55 ±1.36	9.63 ±1.61

TABLE XXVII

WEEKLY SERUM PHOSPHORUS LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN SERUM PHOSPHORUS LEVELS

	Animal	Group	Serum Phosphorus (mg/dl) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		6.1	8.1	8.3	9.2	7.2	8.2	7.5	7.8	6.7
	2		6.9	5.5	6.3	8.2	10.1	7.7	9.7	9.4	8.5
	3	Control	6.6	6.4	9.9	6.4	8.0	5.7	6.8	7.8	6.0
	4		7.4	5.9	1.4	9.5	9.6	7.8	6.5	8.3	8.3
	5		10.4	7.5	9.0	7.4	8.5	9.6	8.1	8.2	5.5
Group $\bar{X}$ ± SD			7.48 ±1.70	6.68 ±1.09	6.98 ±3.39	8.14 ±1.28	8.68 ±1.18	7.80 ±1.40	7.72 ±1.27	8.36 ±0.67	7.00 ±1.35
	6		7.3	8.7	7.2	9.0	7.8	3.8	7.6		
	7		8.2	8.5	7.3	6.9	6.2	8.2	7.0	5.0	3.9
	8	I	7.3	7.1	6.9	7.5	6.7	6.7	8.5	5.0	5.7
	9		7.8	4.7	9.4	7.5	8.6	8.7	6.5	7.3	7.3
	10		8.9	8.2	8.0	9.1	8.9	5.8	7.5	6.9	6.6
Group $\bar{X}$ ± SD			7.90 ±0.67	7.44 ±1.65	7.76 ±1.00	8.00 ±0.99	7.64 ±1.17	6.64 ±1.97	7.42 ±0.75	6.05 ±1.22	5.88 ±1.47
	11		7.1	8.4	5.5	7.9	7.5	5.7	5.1	5.9	
	12		8.3	9.0	7.5	7.5	7.8	8.6	10.1	7.5	7.8
	13	II	6.7	9.8	8.7	6.9	5.8	5.5	7.5	7.0	7.4
	14		5.6	5.4	6.1	6.0	5.7	8.9	5.1	5.5	7.5
	15		4.6	5.1	4.7	8.2	5.3				
Group $\bar{X}$ ± SD			6.46 ±1.42	7.54 ±2.15	6.50 ±1.60	7.30 ±0.87	6.42 ±1.14	7.18 ±1.82	6.95 ±2.39	6.48 ±0.93	7.57 ±0.21
	16		4.1	4.5	4.2	3.9	3.4	3.7	2.7	3.5	4.5
	17		8.6	9.5	11.0	7.0	6.5	5.8	6.5	5.0	7.3
	18	III	7.2	7.6	5.6	4.2	4.0	2.8	4.9	6.3	8.5
	19		9.2	7.8	8.5	6.6	5.5	7.0	5.2	6.2	7.7
	20		6.6	7.7	6.8	4.8	5.9	5.5			
Group $\bar{X}$ ± SD			7.14 ±1.99	7.42 ±1.81	7.22 ±2.64	5.30 ±1.41	5.06 ±1.31	4.95 ±1.69	4.83 ±1.58	5.25 ±1.31	7.00 ±1.74

TABLE XXVIII

WEEKLY SERUM SODIUM LEVELS OF THE EXPERIMENTAL LAMBS AND GROUP WEEKLY MEAN SERUM SODIUM LEVELS

	Animal	Group	Serum Sodium (meq/l) Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
	1		116	149	146	151	145	146	145	140	143
	2		129	114	146	148	146	121	151	149	140
	3	Control	145	148	148	144	143	143	145	153	143
	4		151	116	142	150	145	140	119	153	145
	5		151	127	144	145	145	149	121	148	119
Group $\bar{X} \pm SD$			138.4 ±15.4	130.8 ±16.9	145.2 ± 2.3	147.6 ± 3.1	144.8 ± 1.1	139.8 ±11.0	136.2 ±15.0	148.6 ± 5.3	138.0 ±10.7
	6		148	150	145	147	148	111	150		
	7		150	150	147		150	158	123	106	91
	8	I	148	147	144	147	148	127	155	125	111
	9		149	87	150	142	147	133	119	111	122
	10		152	147	149	149	148	107	132	120	127
Group $\bar{X} \pm SD$			149.4 ± 1.7	136.2 ±27.5	147.0 ± 2.5	146.3 ± 3.0	148.2 ± 1.1	127.2 ±20.3	135.8 ±16.1	115.5 ± 8.6	112.8 ±16.0
	11		145	150	142	145	147	126	138	123	
	12		150	148	148	145	148	146	150	142	143
	13	II	150	150	145	137	133	136	145	120	142
	14		148	150	145	146	148	191	111	118	143
	15		147	142	144	125	147				
Group $\bar{X} \pm SD$			148.0 ± 2.1	148.0 ± 3.5	144.8 ± 2.2	139.6 ± 8.9	144.6 ± 6.5	149.8 ±28.7	136.0 ±17.4	125.8 ±11.0	142.7 ± 0.6
	16		149	145	117	142	147	163	117	114	123
	17		146	150	152	141	151	131	128	143	138
	18	III	145	146	129	146	147	124	122	115	141
	19		150	125	146	146	149	145	145	135	143
	20		148	155	143	124	147	148			
Group $\bar{X} \pm SD$			147.6 ± 2.1	144.2 ±11.4	137.4 ±14.2	139.8 ± 9.1	148.2 ± 1.8	142.2 ±15.3	128.0 ±12.2	126. ±14.5	136.3 ± 9.1

VITA

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