"IMMUNOSUPPRESSION IN EXPERIMENTAL *Trypanosoma (Nannomonas) congolense* INFECTION IN GOATS: STUDIES USING ANTHRAX VACCINE"

by

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A THESIS

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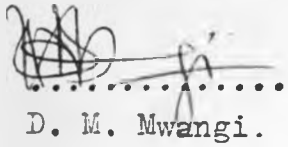
D E C L A R A T I O N

This Thesis is my original work and has not been presented for a degree in any other University.



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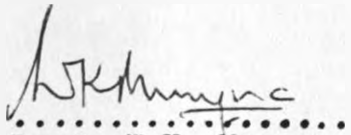

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LIST OF ABBREVIATIONS.

EDTA - Ethylene diamine tetraacetic acid (disodium salt).

"g" - Relative centrifugal force ($g = 118 \times 10^{-7} \times r \times n^2$).
(n = revolutions per minute).
(r - rotating radius).

g - Grams.

Group I - Control goats (non-infected and non-vaccinated).

Group II - Control anthrax vaccinated goats.

Group III - T. congolense infected and anthrax vaccinated goats.

Group IV - T. congolense infected, anthrax vaccinated and Veriben (R) treated goats.

Hb - Haemoglobin content (Mg/100 ml).

H and E - Haematoxylin and Eosin Stain.

MB - Methylene blue stain.

MCHC - Mean Corpuscular haemoglobin concentration (per cent).

MCV - Mean Corpuscular Volume (Microns).

PBS - Phosphate Buffered Saline (pH 7.2.)

PCV - Packed Cell Volume (per cent).

PID - Post Infection Date.

PSG - Phosphate Saline Glucose.

RBC - Red Blood Cell Counts ($x10^6$ per Microlitre).

SD	-	Standard deviation.
SE	-	Standard error.
SRBCs	-	Sheep red blood cells.
TPP	-	Total plasma protein.
WBC	-	White blood cell counts ($\times 10^3$ per microlitre).
TWBC	-	Total white blood cell counts.
L	-	Lymphocytes.
E	-	Eosinophils.
N	-	Neutrophils.
M	-	Monocytes.
B	-	Basophils.
VATs	-	Variable antigenic types.
VSGs	-	Variable surface glycoproteins.

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SUMMARY.

The humoral immune response to anthrax vaccine in Trypanosoma congolense (T. congolense) ILRAD 1180 infected goats was studied. The study revealed that anthrax antibody response of T. congolense infected goats was markedly depressed compared to the control, non-infected vaccinated goats.

Trypanocidal therapy using Veriben (diminazene aceturate) of one group of goats 14 days post infection (4 days after vaccination) resulted in a normal humoral response indicating that therapy before or after vaccination may restore immune competence.

The degree of immunosuppression seemed to be positively related or associated with high parasitaemia levels, low packed cell volumes and low red blood cell counts, indicating that the presence of trypanosomes in the blood is essential in the initiation and persistence of immunosuppression.

Leucopaenia during the incubation period and the early phase of the disease was observed in infected goats. There was no marked leucocytosis after anthrax vaccination in infected non-treated goats.

Infected non-treated goats which showed evidence of immunosuppression showed lymphoid depletion of the spleen with indistinct follicular areas and few germinal centres. There was marked hyperplasia of

the mononuclear phagocytic system especially macrophages in the red and white pulps. Trypanosomes were observed in the spleen. The lymph nodes showed diffuse cortex with lymphoid thinning, indistinct follicles and poor germinal centre formation. The mononuclear phagocytic system was enlarged especially in paracortex and medulla. The liver showed enlarged sinusoidal spaces, hepatic congestion with cellular infiltration. Three infected goats had purulent bronchopneumonia.

Infected treated goats showed marked splenic hyperplasia with adequate lymphoid population, distinct follicular areas and active germinal centres. Medulla of lymph nodes showed high numbers of plasma cells and mononuclear phagocytic cells.

The pathology of the lymphoid organs could among other mechanisms, contribute to immunosuppression in African trypanosomiasis.

1. INTRODUCTION.

Trypanosomiasis is one of the greatest disease limiting factors in livestock production in Kenya and the East African region as a whole. Other infectious diseases do also occur in this region such as anthrax. These diseases and anthrax in particular are controlled through vaccinations. Several workers among them Murray et al. (1974); Moulton and Coleman (1977); Whitelaw et al. (1979) Griffin et al. (1980) and Rurangirwa et al. (1983), have shown clearly that animals infected with trypanosomiasis do not respond optimally to heterologous antigens. However, Rurangirwa et al. (1980a) did not observe any significant immune depression in trypanosomiasis infected cattle simultaneously vaccinated with rinderpest vaccine.

Murray P.K. et al (1974) and Griffin et al. (1980) demonstrated an apparent restoration of immune competence in trypanosomiasis infected animals after trypanocidal therapy. However, Sharpe et al. (1982) did not observe any significant effect of trypanocidal therapy on same day of vaccination in cattle infected with trypanosomiasis.

To my knowledge there is no published reports on immunosuppressive effects of Trypanosoma congolense infection in goats following vaccination against anthrax. The results obtained could also be of practical application in other domestic ruminants including bovine and the ovine.

The main objectives of this study was to

i). Establish whether there exists a state of humoral immune depression in Trypanosoma congolense infection in goats following immunization with anthrax spore vaccine.

ii). Study the effect of trypanocidal therapy on the immune status of the Trypanosoma congolense infected goats.

iii) Find if there is any correlation between the degree of immune depression on one hand and the levels of parasitaemia, haematological values and pathology of the lymphoid tissues: the lymph nodes, spleen and liver in particular in Trypanosoma congolense infected goats.

2. LITERATURE REVIEW

2.1. THE DISEASE

2.1.1. Aetiology

Trypanosomiasis is a group of diseases of man, domestic and wild animals caused by various species of protozoan parasites of the genus Trypanosoma. Hornby (1949) defined trypanosomiasis as several allied diseases each of which is caused by infection with specific trypanosomes.

One of the major causes of Trypanosomiasis in goats is Trypanosoma (Nannomonas) congolense (Whiteside, 1958, Omuse, 1973). Trypanosoma congolense (T. congolense) is one of the smallest of the trypanosomes and measures about 9 - 18 u in length (Soulsby, 1982). It has no free flagellum and has a medium sized kinetoplast which is marginally located. In a wet preparation it does not move rapidly across the field, rather it is a focus of turbulence among red blood cells (RBCs). The blood forms are pleomorphic with an inconspicous, undulating membrane and a rounded or obtuse posterior end. The trypanosomes are easily stained with Giemsa stain (Mulligan, 1970) although Leishman's and Wright's stains can also be used.

2.1.2. Vectors

The main vectors of Trypanosoma congolense are the tsetseflies, the major ones being Glossina morsitans and Glossina palliditas although thirty (30) other

species of Glossina can also transmit the disease cyclically (Richardson, 1928). Tabanus and other biting flies can transmit the disease mechanically.

2.1.3. Pathogenicity and clinical signs.

T. congolense causes an acute; subacute or chronic forms of the disease in goats, cattle and sheep (Fiennes, 1954; Losos et al., 1973; Omuse, 1973; Anosa and Isoun 1974; and Griffin and Allonby, 1979). The incubation period varies between 6 - 10 days (Kaaya, 1975). The disease is characterised by high daily fluctuating temperatures, progressive anaemia and emaciation (Stephen, 1970; Wellde et al., 1974). Kaaya, (1975); and Griffin and Allonby, (1979) observed lymphadenopathy of superficial lymph nodes. Marked oedema of the face and submandibular regions, sternal recumbency coma and death were observed by Kaaya. (1975). Animals became weak and often fed when kneeling down on their front legs but appetite was invariant (Kaaya, 1975). Initial parasitaemia occurred one day before or after initial temperature rise (Kaaya, 1975). Temperatures became subnormal (35^{o}C) and there was weight loss just prior to death (Wellde et al., 1974). Wellde et al. (1974) further observed dyspnoea, while pneumonia occured concurrently with trypanosomiasis (Kramer, 1966; Kanyari, 1981).

T. congolense infection in goats is severe and usually terminates in death (Kaaya, 1975). T. congolense was thought to be a strict plasma parasite (Hornby, 1929;

Hornby and Bailey 1930; Losos and Ikede, 1972; Tizard et al., 1978 b) but work by Luckins and Gray (1978), Luckins and Gray (1979) and Gray and Luckins (1980) demonstrated T. congolense in siruses of lymph nodes. Trypanosome enzymes have been identified which catalyse the breakdown of phospholipids and perhaps proteins in the membranes of host red blood cells and lymphocytes (ILRAD, 1985) thus causing their destruction.

2.1.4. Haematology.

Packed cell volumes (PCV), red blood cell counts (RBC) and haemoglobin levels (Hb) were severely decreased in both the acute and chronic forms of the disease (Fiennes, 1954; Naylor, 1971; Losos et al., 1973; Kaaya, 1975; Dargie et al., 1979). Mean corpuscular volume (MCV) showed a slight increase on the fourth (4th) week and a drop on 14 - 16th weeks post-infection, while there were no marked changes on the mean corpuscular haemoglobin content (MCHC) (Kaaya, 1975). The marked changes on the erythrocytic indices was due to either erythrophagocytosis (Connal, 1912; Fiennes, 1954; Mackenzie et al., 1978), hemodilution (Kaaya, 1975 and Kaaya et al., 1977) or bone marrow depression (Kaaya et al 1977; Mackenzie et al., 1978).

Initial leucopaenia has been observed by Losos et al. (1973), and Wellde et al. (1974), but Kaaya (1975) reported a slight leucocytosis as well as an increase in total leucocyte counts 4 - 12 weeks post infection with a neutrophilia and a lymphocytosis.

The total serum proteins in infected cattle decreased (Fiennes, 1970; and Wellde et al., 1974) due to an increase in gamma globulins in serum and proteinuria. However, Kaaya (1975) did not observe any change in the levels of total serum proteins in T. congolense infected goats.

2.1.5. Immune response.

The host responds to the infecting trypanosomes by production of highly specific antibodies (Musoke et al., 1981 b). In an infected host, there are recurrent peaks of immunoglobulins, mainly immunoglobulin M (IgM) against the variable surface glycoprotein (VSG) of infecting trypanosomes as well as those trypanosome variants arising during the course of infection (Nantulya et al., 1979; Masake et al., 1983). This recurrent rise in antibodies could be due to a random emergence of variable antigenic types (VATs) in infected animals which are either identical or of close resemblance to infecting VATs (Nantulya et al., 1985). The mononuclear phagocytic system plays a role in the elimination of trypanosomes from blood circulation (Nantulya et al., 1985). Histological evidence of the expansion of the mononuclear phagocytic system with macrophages of the liver, lymph nodes and bone marrow displaying morphological features of increased activity was observed by Murray P. et al.,(1974.) Trypanosomes sensitised with immune whole serum or VSG specific

IgM or IgG were rapidly engulfed and ingested by cultured monocytes (Nantulya et al., 1985). The mononuclear phagocytic system acting in concert with VSG-specific antibodies could be a major mechanism of parasite clearance in cattle (Ngaira et al., 1983).

Cell mediated immune responses in African trypanosomiasis is not well documented. Development of delayed type hypersensitivity (DTH) responses in cattle immunized with T. congolense has been reported by Emery et al. (1980) and Wells et al. (1982). Marked in vitro proliferative responses of peripheral blood lymphocytes from infected cattle was observed when the lymphocytes were cultivated with ultrasonicated trypanosome antigens. Indication of DTH has been reported in laboratory animal models in trypanosomiasis (Tizard and Soltys, 1971; Finerty et al., 1978).

2.1.6. Diagnosis.

Thick blood smears made from the superficial ear vein and stained with Giemsa has been used in both the acute and chronic forms of the disease. In chronic cases and infections with very low parasitaemias, mice innoculation (MI) and haematocrit centrifugation technique (Woo, 1970) has been used (Godfrey and Killick-Kendrick, 1961; Killick-Kendrick, 1968). The use of a method combining both the thick and thin blood smears has been used for diagnosis and identification of infecting species of trypanosomes (Kaaya, 1975). Several serological tests among them the Indirect

red and white pulp (Kaaya, 1975 and Losos et al., 1973). These workers also reported increased population of plasma cells, macrophages and lymphocytes in the red pulp.

The liver was enlarged and congested (Kaaya, 1975). Microscopically, there was swelling of the Kupffer cell nuclei and an increase in white blood cells in the sinusoids, including congestion and aggregation of lymphocytes and macrophages in periportal areas.

The bone marrow was pinkish (Kaaya, 1975) and the major part inactive. Microscopically, there was accumulation of immature cells and many erythrocytic foci (Kaaya, 1975). Generalized hemosiderosis especially in macrophages of the spleen, liver and bone marrow has been reported (Kaliner, 1974; Wellde et al., 1974; Kaaya, 1975; Van den Ingh, 1976; and Mackenzie et al., 1978). Kaaya (1975) and Kanyari (1981) reported genelized infiltration of body tissue, especially the kidneys, lungs and brain with plasma cells and lymphocytes.

Rurangirwa et al. (1980a) observed atrophy and reduction in weight of the thymus in T. congolense infected cattle.

In laboratory animals Brown and Losos (1977) and Morrison et al. (1982) noted enlargement of lymph nodes and also an increase in the proliferative activity in the follicular area. Large lymphocytes and high mitotic activity leading to expansion of medullary cords with plasma cells and large lymphocytes were also

observed. Later in the course of the infection there was an increase in population of lymphocytes and macrophages and a decrease in size and activity of follicular areas. Brown and Losos (1977) observed high numbers of plasma cells in the lymph nodes and a general lymphocytic depletion of the cortex. They also observed in the spleen, an increase in plasma cells, macrophages and lymphocytes in the red pulp. Murray, M. et al. (1974), Murray P.K. et al. (1974) and Roelants et al. (1979) observed depletion of splenic white pulp and a generally loose cellular structure in infected mice.

Grossly there was a gradual atrophy of the thymus (Murray, M. et al., 1974; Murray, P.K. et al., 1974; Morrison et al., 1982). Microscopically, there was a reduction of thymic cortex during the later stages of infection and marked mitotic activity in cortical thymocytes while blood vessels showed numerous macrophages (Morrison et al., 1982).

2.2. Anthrax.

This is a peracute infectious disease of herbivores especially cattle and sheep caused by, the bacteria Bacillus anthracis (Brunner and Gillespie 1973). Animals are immunized to prevent heavy losses. The vaccine mainly used in the immunisation of cattle, sheep, and goats is living avirulent anthrax spore vaccine (Sterne, 1946).

2.3. Immunosuppresion in African trypanosomiais.

A state of immune depression caused by various human and animal diseases has been extensively reported. The depression may be of either humoral or cell mediated immunity or both. Streptococcal infections were found to be major causes of death in cases of human trypanosomiasis (Moss, 1906). Parkin and Hornby (1930), and Greenwood et al., (1973) first reported existence of a state of immunosuppression in bovine trypanosomiasis. Murine and human patients of malaria had also a depressed immune system as reported by Greenwood et al. (1971, 1972). Philips et al. (1974) and Wedderburn (1974) demonstrated that Plasmodium infection in mice caused a state of immune depression. Reports on immunedepression in bovine theileriosis (Wagner et al., 1975) also indicates that this is a phenomenon in most protozoan diseases. Kramer (1966) and Hull et al. (1971) observed that sheep infected with Trypanosoma vivax were very predisposed to bacterial infections which may be due to the depressed immune system.

2.3.1. Immunosuppression in laboratory animals.

Laboratory animals especially mice have been extensively used in attempts to demonstrate, study and elucidate on the phenomenon of immunosuppression in trypanosomiasis. Various workers including Goodwin et al. (1972); Murray M. et al. (1973); Urquhart et al. (1973); Corsini et al. (1977); Murray M et al. (1974)

and Longstaffe (1974) demonstrated unequivocally varying degrees of immunosuppression in murine trypanosomiasis. This work was extended by other workers including Mansfield and Wallace (1974); Mansfield (1975); Hudson et al. (1976); Hudson and Terry (1979); Jayawardena and Waksman (1977); Reid et al. (1979); Whitelaw et al. (1979); Albright and Albright, (1981); and more recently by Yamamoto et al. (1985); and Barrance and Hudson, (1986) who confirmed the earlier findings.

Various techniques have been used to demonstrate the state of immune depression in murine trypanosomiasis. Studies have also been done to identify the type and level of immune response affected. Humoral responses to various heterologous antigens have been shown to be depressed in murine trypanosomiasis (Goodwin et al. 1972; Urquhart et al., 1973; Reid et al., 1979; and Whitelaw et al., 1979). Mice infected with T. congolense showed reduced antibody response after immunization with sheep red blood cells (SRBCs) (Goodwin, 1970), Louping-ill vaccine (Whitelaw et al., 1979) and a reduction in development of protective antibodies to the worm Nippostrongylus brasiliensis (Urquhart et al., 1973). Mice infected with T. brucei and T. vivax were also shown to have reduced antibody response to louping-ill vaccine (Whitelaw et al., 1979) and other antigens (Barrance and Hudson, 1986). Reid

et al. (1979) observed that in T. brucei infection in mice the protective effects of louping-ill vaccine and lymphocytic choriomeningitis were immensely reduced. T. congolense infection in mice causes a more severe state of immunodepression than in infections with T. brucei and T. vivax (Whitelaw et al., 1979).

The cell mediated immunity is also depressed in murine trypanosomiasis. Yamamoto et al. (1985) observed a reduction in delayed type hypersensitivity (DTH) to Listeria monocytogenes antigens in murine trypanosomiasis. Pearson et al. (1978) demonstrated a reduction in mixed leucocyte reactions (MLR) and a compromise of allogeneic skin graft rejections in murine trypanosomiasis. Urquhart et al. (1973) showed that there was a marked reduction of immune expulsion of adult Nippostrongylus brasiliensis, a decrease in reaginic antibodies, decrease in mast cell populations of intestinal mucosa, and a reduction in response to oxazolone sensitization.

In murine trypanosomiasis it has been shown by Goodwin et al. (1972); Albright et al. (1977); Corsini et al. (1977); Jayawardena and Waksman (1977); Pearson et al. (1978); and Roelants et al (1979) that there is a profound reduction in the DNA synthetic responses of leucocytes to mitogens such as pokeweed mitogen (PWM), concanavalin (ConA) and Escherichia coli lipopolysaccharide (LPS).

2.3.2. Immunosuppression in domestic animals.

Reduced humoral responses to heterologous antigens in trypanosomiasis of domestic animals have been observed. In goats infected with T. congolense there is reduced antibody response after vaccination with killed Brucella melitensis antigens (Griffin et al., 1980) and to clostridial vaccines (Van Dam et al., 1981). Griffin et al. (1981 a) observed reduced immune expulsion of Haemonchus contortus in goats infected with T. congolense. Reduced antibody response to Brucella abortus vaccine in sheep infected with T. congolense was demonstrated by Malu and Tabel (1986). T. brucei infected goats showed a marked immunosuppression as indicated by a suppression of mitogenic reactivity of peripheral blood lymphocytes (Van der zee et al., 1985). Mackenzie et al. (1975), has shown a reduced antibody response to Vibrio fetus strain 1980 in sheep infected with T. congolense.

Cattle infected with T. congolense showed a marked depression of antibody response to clostridial vaccine (Holmes et al., 1974), Brucella abortus (Rurangirwa et al., 1979; 1983) and to Mycoplasma mycoides var. mycoides Gladysdale strain (Rurangirwa et al., (1978). Rurangirwa et al. (1979) further demonstrated a reduced antibody response to Leptospira biflexa.

Scott et al. (1977) and Sharpe et al. (1982) observed a depression of immune response to foot and mouth disease vaccine, while Whitelaw et al. (1979) showed a depressed antibody response to louping-ill vaccine in bovine trypanosomiasis. Depressed immune responses to different clones of T. congolense and T. brucei occurs as shown by Nantulya et al. (1982). Rurangirwa et al. (1980 a) showed that immune responses to live rinderpest vaccine was not affected in bovine trypanosomiasis. East Coast fever immunization was likewise not affected by chronic T. congolense infection in cattle (Taracha et al., 1986).

In vitro proliferative responses of bovine peripheral blood leucocytes (PBL) to phytohaemagglutinin (PHA), lipopolysaccharide (LPS), pokeweed mitogen (PWM) and ConA. has been studied (Sollod and Frank 1979; Masake et al., 1981. Sollod and Frank (1979) however failed to demonstrate a decrease in DNA synthesis of leucocyte cultures from infected cattle in response to mitogen stimulation, but Masake et al. (1981) did show reduced stimulation of LPS and PWM on PBL from T. congolense infected cattle.

2.3.3. Mechanisms of immunosuppression.

Several possible mechanisms mediating immunosuppression in African trypanosomiasis have been suggested.

2.3.3.1 Antigenic competition.

This is the most favourable of the mechanism of immunosuppression in large domestic animals. Gray (1970) and Goodwin et al. (1972); first suggested that antigenic competition of variants of infecting trypanosomes for the stem cells and humoral factors (Abramoff and La Va 1970), may be the main cause of immunosuppression. However, Musoke et al (1981b) and Nantulya et al (1982) suggested that since there is a dramatic rise in immunoglobulin levels which are specific against numerous variant antigenic types arising from the infection, there is a resultant antigenic competition due to infection and the test antigens. Indeed, Nantulya et al (1982) suggested that it is possible suppressor cells may be the main effectors of antigenic competition. Antigenic competition may either act at the macrophage level, or the macrophages might elaborate a factor which inhibits proper lymphocyte responses of infected host to other antigens (Liacopoulos and Ben Efraim, 1975).

2.3.3.2 Suppressor factors.

In mice immunosuppression in tryponosomiasis could be mediated by generation of suppressor cell populations, mainly T-cells and Macrophages, which could act directly or through elaboration of suppressive factors (Jayawardena and Waksman, 1977; Mansfield, 1978; Pearson et al, 1978; Roelants et al, 1979; Terry et al,

1980, and Grosskinsky and Askonas 1981). Macrophages from T. brucei and T. rhodesiense infected mice profoundly depressed the ability of normal spleen cell cultures to proliferate and secrete immunoglobulins to E. coli LPS and SRBCs as shown by Corsini et al. (1977); Eardley and Jayawardena (1977) and Wellhausen and Mansfield (1979). Trypanosome induced suppressor T-cells secrete a factor which initiates the macrophages to carry the suppressive messages to other T-cells and also B-cells (Eardley and Jayawardena, 1977; Bagasra et al 1981 and Grosskinsky and Askonas, 1981). Recent findings have shown that macrophages could exert their immunosuppressive effects by inhibiting T-cell production and secretion of Interleukin-2, a T-cell growth factor (Alcina et al. 1985; Sileghem et al 1985). In large animals, however, the involvement of suppressor cells in tryponosome mediated immunosuppression has not been demonstrated, but Nantulya et al (1982) suggested that they could be the main effectors of antigenic competition. Rurangirwa et al (1979) demonstrated that trypanosome infected cattle, treated with Berenil on the day of vaccination with Leptospira biflexa antigens showed a normal immune response and thus suggested that if at all supressor cells are involved in immunosupression in bovine trypanosomiasis, they do not have long lived effects.

2.3.3.3 Polyclonal B-cell activation.

This observation has only been demonstrated in mice. Trypanosomes and their products are thought to cause a non-specific stimulation of B-cells leading to production of excess immunoglobulins. This leads to clonal exhaustion and eventually to immunosuppression and immune dysfunction (Greenwood 1974, Hudson et al 1976, Assoku, Tizard and Nielsen, 1977, Assoku et al, 1979). There is a functional depletion of T- and B- memory cells and other lymphoid cell subpopulations (Askonas et al, 1979). However, in large animals this may not be the case since according to Rurangirwa et al (1979) and Tabel et al (1981), trypanosome infected cattle did not show any antibodies to Leptospira biflexa (Rurangirwa et al, 1979) or to chicken or sheep red blood cells (Tabel et al 1981) prior to immunisation. Furthermore, Musoke et al (1981) and Masake et al (1983) observed that antibodies produced by B-cells during bovine trypanosomiasis are specific for the infecting trypanosome variants.

2.3.3.4 Role of complement

Nielsen et al (1978), suggested that an increased immunoglobulin catabolism and hypocomplementaemia could play a role in the observed immunosuppression in bovine trypanosomiasis. A complement activating factor in trypanosomes (Musoke and Barbet, 1977, Nielsen et al 1978) react with complement to influence and inhibit leucocyte migration (Rurangirwa et al. 1980b).

Indeed, Rurangirwa et al (1980b) suggested that these factors were the immune complexes which are formed between antigens of dying trypanosomes, immunoglobulin M and complement.

2.3.3.5 Trypanosome mitogenic factors.

It has been shown that trypanosomes possess mitogenic factors which alone could induce a state of immunosuppression (Assoku and Tizard, 1978; Esuruoso, 1976; Greenwood and Oduloju 1978; Hazlet and Tizard 1978; and Masake et al. 1981). Several trypanosome fractions have been demonstrated to have immunosuppressive effects. For example, Clayton et al (1979), Sacks et al (1980a and 1982) and Yamamoto et al (1985) demonstrated immunosuppressive effects of plasma membrane fractions of trypanosomes in mice. Furthermore, complex lipids and trypanosome derived saturated fatty acids and proteins showed immunosuppressive and polyclonal B-cell activation effects in mice (Assoku et al 1982). Mitogenic effects of trypanosomes were also demonstrated in vitro by Mansfield et al. (1976). Albright and Albright on the other hand observed inhibition of murine humoral responses by substances derived from trypanosomes.

2.3.3.6 Block of antibody secretion at plasma cell level.

In the spleens of trypanosome infected mice, numerous plasma cells with distended endoplasmic reticulum, probably with cytoplasmic immunoglobulin was observed. This was in contrast to infected treated mice. This evidence would

suggest a blockage of secretion but not production of antibodies at the plasma cell level, which would also be a likely cause of immunosuppression, at least in mice (ILRAD, 1985).

2.4. Correlation of immunosuppression with other parameters.

2.4.1 Parasitaemia.

Levels of parasitaemia and degree of immune depression are positively correlated (Freeman, 1974; Jennings et al., 1974; Scott et al., 1977; Whitelae et al., 1979; Griffin et al., 1980; Rurangirwa et al., 1980; Tizard et al., 1980; Baltz et al., 1981; Van den zee et al., 1985). However, Barrance and Hudson (1986) observed an inverse relationship between level of parasitaemia of individual infected mice and degree of immune depression.

2.4.2 Haematology.

Levels of parasitaemia are inversely correlated to the PCV, the RBC and Hb (Fiennes, 1954; Naylor 1971, Kaaya et al., 1977; Mackenzie et al., 1978).

There is an imminent leucopaenia in active trypanosomiasis (Naylor, 1971; Losos et al., 1973; Wellde et al., 1974; Kaaya, 1975; Maxie and Losos 1977; Kaaya et al., 1980; Valli and Forsberg, 1979).

2.4.3 Immunology.

In animal trypanosomiasis there is dramatic rise in serum immunoglobulins especially immunoglobulin M. fraction (Clarkson. 1968; Houba et al., 1969; Naylor, 1971; Terry et al., 1973: Hudson et al., 1976 and Mackenzie et al., 1978). However, Rurangirwa et al. (1983) showed that there was profound depression of

specific Brucella abortus antibody responses of IgG_1, IgG_2 subclasses and IgM class in T. congolense infection in cattle. Malu and Tabel (1986), suggested that there is a positive correlation between high serum levels of factor B at the early stage of trypanosomiasis infection and enhanced immune responsiveness.

Mice infected with T. congolense showed a depression of B and T lymphocyte responses and appearance of suppressor cell activity in spleens. This paralleled the appearance of parasites in peripheral blood and immune depression (Roelants et al., 1979).

2.5. Effect of trypanocidal therapy on the degree of immunosuppression.

Immune competence in trypanosomiasis is restored after trypanocidal treatment of the infected animals (Murray P.K. et al., 1974; Roelants et al., 1979; Rurangirwa et al., 1979; Griffin et al., 1980; Masake et al., 1981; Malu and Tabel, 1986). However, Sharpe et al. (1982) reported that treatment at the time of vaccination did not restore immune competence significatly to primary vaccination. Malu and Tabel (1986) demonstrated an immunoenhancement when the infected animals were vaccinated four days after trypanocidal treatment. Thus, the restoration of immune competence is dependent on the time of antigen presentation after trypanocidal therapy.

3. MATERIALS AND METHODS.

The experimental study was carried out in the Department of Veterinary Pathology and Microbiology of the Faculty of Veterinary Medicine, Kabete.

3.1. Animals: Conditioning housing and feeding.

Twenty three (23) healthy goats of mixed breed, age, and sex were used. They were purchased from a trypanosomiasis free area of Central Province of Kenya, where they were bred and reared before use, the goats were screened for blood parasites and were shown to be free from trypanosome infections. Some goats were found to be having anti-anthrax antibody titres not exceeding 1:8. The animals were housed in tsetse fly-proof houses during the duration of the experiment. Two weeks before the start of the experiment, all goats were treated with Levamisole and Zanil (NILZAN^{R}; Wellcome Kenya, Limited) at the recommended dosage of 15mg/kg body weight to control internal helminths. The animals were fed on grass hay, wheat bran and supplemented with mineral licks. Water was provided ad libitum.

3.2. Parasite: Trypanosomes and establishment of infections.

The parasite used in this study was Trypanosoma congolenses (T. congolense) clone ILRAD 1180. T. congolense ILRAD 1180 had previously been derived from STIB.212 originally isolated from a lion in Serengeti

National Park, Tanzania in 1971 (Geigy and Kauffman, 1973). STIB 212 was passaged three times (3) in non-irradiated BALB/c mice and cloned in an irradiated (500R) BALB/c mouse. The clone so obtained was again passaged once into a similarly irradiated BALB/c mouse and then stabilated as IL.933. Clone IL. 933 was then innoculated into an irradiated (500R) BALB/c mouse and recloned 3 days post infection. The subsequent recloned population was stabilated after 11 days as IL.968 (Nantulya et al., 1984). Clone IL.968 was further passaged in irradiated mice (500R) and recloned seven days (7) later as ILRAD 1180.

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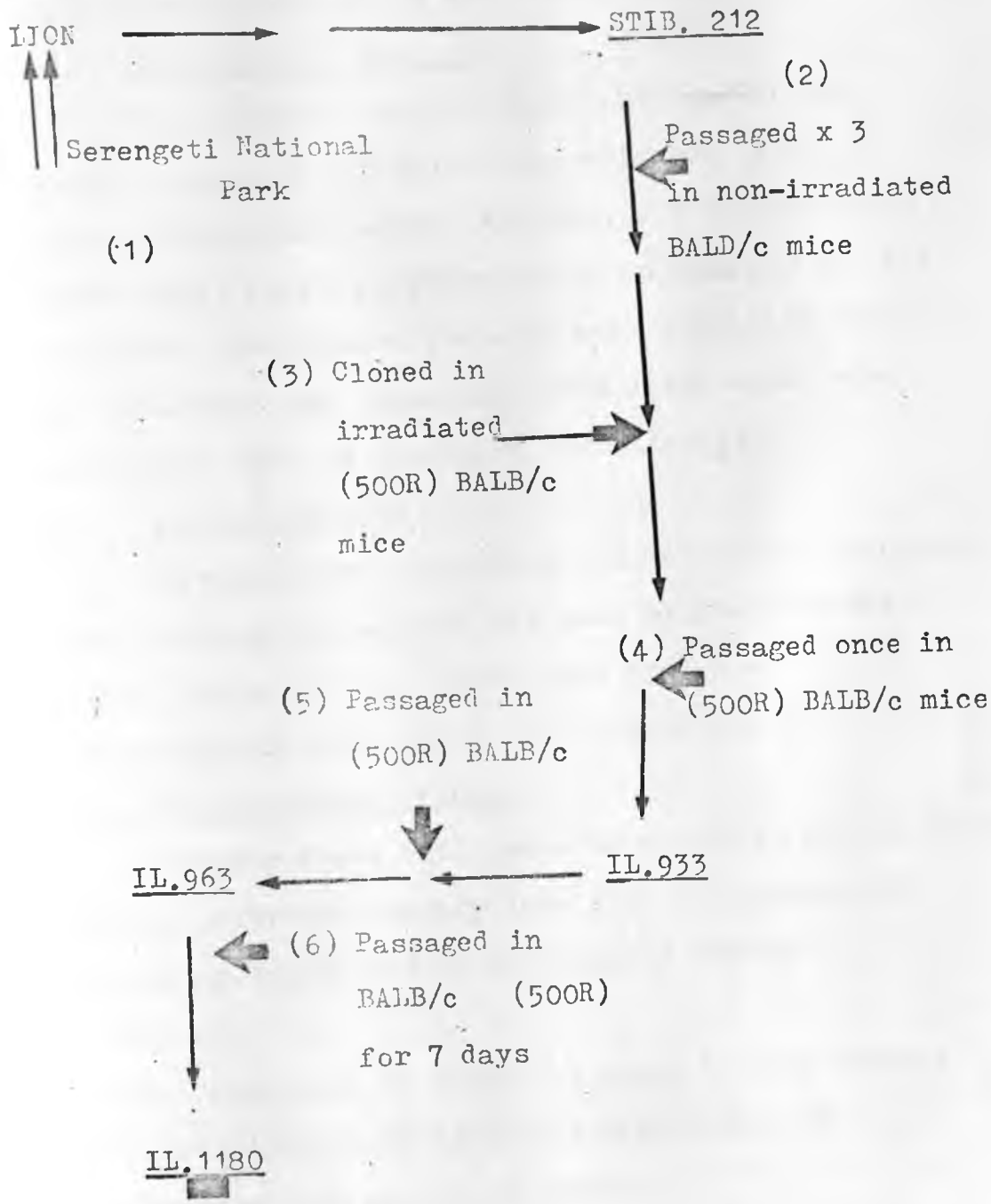
Figure 1: Derivation of T. congolense ILRAD 1180.



All experimentally infected goats received 1 x 10^6 viable blood stream forms of T. congolense ILRAD 1180 subcutaneously around the shoulder region.

3.3. Trypanocidal therapy.

One group of infected goats (Group IV; see Table 1) received 3.5 mg/kg body weight of 4,4'-dibenzamidine diaceturate (Veriben, PVU International Canada Inc.) by deep intramascular injection four (4) days after vaccination (14 days post infection day). This treatment was repeated a week later since some goats still had low levels of parasitaemia.

3.4. Anthrax vaccine.

Living avirulent anthrax spore vaccine (Wellcome, Kenya Limited) in saponin was used to immunize the goats (Sterne, 1946). Goats were immunized subcutaneously with 1.0 ml of the vaccine.

3.5. Experimental design.

Twenty three (23) goats were used in the experiment and were divided randomly into four (4) groups with each group being treated differently (Table 1) as follows:

GROUP I comprised of three (3) goats (Eartag numbers 403, 406, 408) which were left uninfected and unvaccinated and served as controls.

GROUP II comprised of seven (7) goats (Eartag numbers 429, 430, 431, 433, 443 and 444). This group was innoculated with 1.0 ml of the anthrax vaccine

3.6. Haematology.

Blood was collected from the jugular vein into bijou bottles containing 2% ethylenediaminetetracetic acid (EDTA) as anticoagulant twice a week for the determination of the haematological parameters. Red blood cell counts (RBC) in 10^6 per microlitre of blood (10^6/ul), white blood cell counts (WBC x 10^3/ul) were determined using an automatic Electronic Coulter Counter (Coulter Electronics Inc. Coulter Model Zb. Inc). The haemoglobin content (Hb) in mg/100 ml of the red blood cells was estimated as cyanmethemoglobin through a haemoglobinometer attached to the main coulter instrument.

The packed red blood cell volume (PCV%) was determined using the microhaematocrit method. The mean corpuscular volume (MCV) and the mean corpuscular haemoglobin concentration (MCHC) were calculated according to Schalm et al. (1975) as follows:

$$MCV = \frac{PCV \times 10}{RBC}$$ expressed in cubic Microns (u)

$$MCHC = \frac{Hb \times 100}{PCV}$$ expressed in percent (%).

Total plasma protein (T.P.P) was determined using a refractometer (T.S. Meter, A.H. Optical Company Keene; NH, U.S.A.) and expressed as grams per 100 ml (g/100 ml).

Thin blood smears were made from the jugular blood on clean glass slides, fixed for 3 minutes in methanol and stained for 30 minutes in a 1:10 concentration of Giemsa for differential leucocyte counts.

3.7. Serum collection.

Serum collections from each animal were made twice weekly before and during the course of the experiment. Blood was collected from the jugular vein into sterile universal bottles. After clot formation and retraction, the serum was decanted and centrifuged to remove the few suspended red blood cells. Serum was then stored at -20°C until use.

3.8. Parasitaemia.

Thick blood smears were made twice a week from the ear vein. The smears were air dried, methanol fixed for 3 minutes and stained with 1:10 Giemsa stain (MSD, E. Merck, A.G. Darmstadt Germany) for 30 minutes. Excess stain was washed off and the smears air dried. Twenty five high dry microscopic fields were observed and the total number of trypanosomes (T) and white blood cells (L) determined in these fields. The parasitaemia, trypanosomes per millilitre of blood (Tryps/ml) was then calculated (Rurangirwa et al., 1980a).

$$\text{Tryps/ml} = \frac{\text{Tryps in 25 fields} \times \text{Total WBC/ul of blood} \times 1000}{\text{WBC in 25 fields}}$$

3.9. Anthrax antibody assay.

The indirect microhaemagglutination test was used following a modification of the method used by Buchanan et al. (1971).

3.9.1. Antigen preparation.

Living avirulent non-encapsulated strain of Bacillus anthracis Sterne 134 was grown in blood agar plates for 12 hours at 37°C aerobically. The resultant growth of colonies were washed off and suspended in 0.85% physiological saline to make a thick suspension. The suspension was sonicated in a ultrasound disintegrator (Branson Sonic Power Model M52, Branson Instruments Incorporated Danburg, Connecticut) for 30 minutes at 5 amperes. The sonic extracts was then centrifuged (IEC Model HN-SII centrifuge) at 2750 r.p.m. (1150"g") to obtain a yellowish supernatant. The supernatant was then passed through 0.2 u millipore filters (Millipore Filter Coorp. Bedford, Massachusets U.S.A.) to obtain a clear yellowish filtrate. To the filtrate was added 1% sodium azide as preservative. The filtrate was then preserved at 4°C and used as the sonic extracts anthrax antigen after a dilution to 2 mg/ml.

3.9.2. Positive anthrax antisera.

Anthrax antiserum was prepared from three (3) New Zealand White rabbits. Each rabbit was immunized subcutaneously with 1.0 ml of anthrax spore vaccine and two weeks later boosted with 0.5 ml of the vaccine.

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The rabbits were test bled on day 14 post vaccination (P.V.) and a week later after boosting when they had developed a maximum haemagglutination titre of 1:256. Negative antisera was obtained from two non-vaccinated rabbits.

3.9.3. Preparation of glutaraldehyde-fixed sheep red blood cells (SRBCs).

Glutaraldehyde-fixed sheep red blood cells were prepared using modifications of the methods of Stavitsky (1954), Sever (1962), and Bing et al. (1967). Sheep blood was collected in 2% EDTA from sheep in a slaughterhouse. The blood was washed five times in phosphate buffered saline (PBS) p.H. 7.2. A solution of 2.5% commercial glutaraldehyde (SERVA, Feinbiochemica, Heidelberg Germany),(24% glutaraldehyde in water), was added to packed red blood cells at a ratio of 1:4 respectively. The suspension was incubated in room temperature for 30 minutes with frequent mixing. The cells were then washed three times in PBS. The concentration of SRBCs suspension was determined by microhaemotocrit method and made up to 20 per cent. One per cent (1%) of sodium azide was then added to the suspension after which it was stored at $4^{o}C$.

3.9.4. Tanning and sensitisation of SRBCs.

Glutaraldehyde-fixed sheep red blood cells stored at $4^{o}C$ prior to sensitisation were washed twice with phosphate buffered saline, pH 7.2 and prepared

as a 20% suspension of SRBC in PBS. The cells were mixed with an equal volume of a 1:5000 solution of tannic acid and incubated at 37°C for 10 minutes. The tanned cells were then washed in PBS and centrifuged.

Equal volumes of packed tanned cells and Bacillus anthracis sonic extract antigen (2 mg/ml) were mixed and suspension incubated in room temperature for 3 hours. The suspension was then washed in PBS to remove excess antigen. The sensitised cells were adjusted to a 2% final cell suspension in pH 7.2 PBS containing 1:200 rabbit serum. A 2% and 20% suspension of tanned, non-sensitised, glutaraldehyde fixed cells were also prepared for control red cells and serum adsorption respectively.

3.9.5. Preparation of serum samples.

Before the haemagglutination test was carried out, serum complement was first inactivated at 56°C for 30 minutes. All serum samples were adsorbed with an equal volume of 20% tanned SRBCs for 2 hours at room temperature. Cells were repacked by centrifugation and supernatant test serum obtained at a dilution of 1:2.

3.9.6. Preparation of microtiter plates.

All the wells of U-type microtiter plates (Linbro Chemical Co., New Haven, Conn.) were filled with 0.025 ml of 1:100 normal rabbit serum in p.H. 7.2 phosphate-buffered saline. Test serum samples (0.025 ml each) were then placed in predesignated wells by direct

tulip-loop transfer. Subsequently, all the serum samples were serially diluted twofold. A 0.025 ml of the 2% suspension of antigen sensitised glutaraldehyde fixed SRBCs was dispensed into all wells by microtiter technique. Control tests, for red blood cells and serum were included in the test. The serum-cell suspensions were gently mixed and incubated for 2 hours at room temperature before reading. All the tests were set in duplicate.

3.9.1. Method of reading haemagglutination test.

The haemagglutination test was read as the reciprocal of serum dilution giving complete haemagglutination (Sever, 1962).

3.10. Necropsy.

A complete necropsy was done on all animals that died during the experiment. Some control goats were sacrificed for comparison purposes. Impression smears were made from the liver, lymph nodes, spleen, lungs and kidneys. The smears were fixed in methanol for 3 minutes and stained with 10% Giemsa for 30 minutes, and examined for trypanosomes and cell types. Tissues for histopathology were obtained from various organs including lymph nodes, spleen, liver, lungs and kidneys. They were sectioned and then fixed in 10% formalin. The formalin-fixed tissues were embedded in paraplast and 6 microns thick sections cut and stained with Haematoxylin and Eosin (H and E; BDH Ltd. Poole, England) Methylene

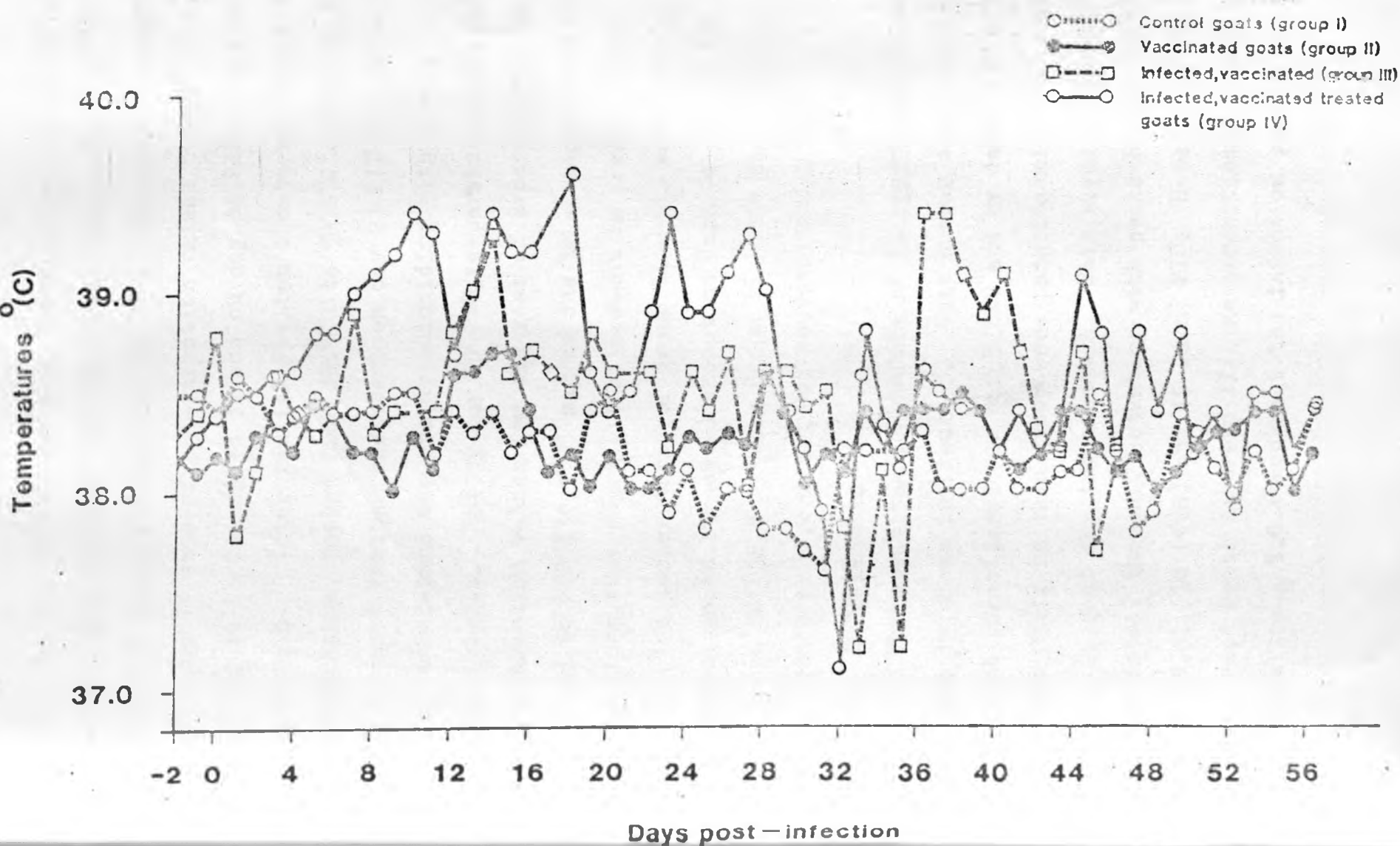
4. RESULTS.

4.1 Rectal temperatures.

All the T. congolense challenged goats became infected. Infected groups (Groups III and IV) had higher temperatures than non-infected groups from days 7 to 44 post infection date PID (Figure 2). Group III goats developed a mean temperature of 38.9°C on day 7 PID which was followed by daily fluctuations with several peaks on day 14 (39.3°C), days 36 and 37 (39.4°C), days 38 and 40 (39.1°C). Between days 15 and 28 PID Group III had lower temperatures than Group IV but higher than the control groups. This state was due to subnormal body temperatures of goats 436, 426, 428, and 437 which died of trypanosomiasis on days 16, 17, 21, and 28 PID respectively. On day 33 PID and day 35 PID the temperatures were low, 37.2°C and this was due to subnormal temperatures of Goats 447 (35°C), 445 (36°C) before their deaths due to trypanosomiasis. The mean temperatures were of normal values between 8 - 12 days, PID and from day 41 PID throughout the rest of experiment.

Group IV goats developed higher mean temperatures than control goats from day 7 PID (39.0°C) to day 11 PID (39.3°C) with a peak on day 10 PID (39.4°C). This was followed by fluctuating mean daily temperatures which were generally higher than control goats up to day 29 PID with several peaks on days 14 (39.4°C), 18 (39.6°C), 23 (39.4°C), 27 (39.3°C) and on day 44 (39.1°C)

Figure 2 Daily mean rectal temperatures (°C) of T. congolense infected and vaccinated goats, and control goats



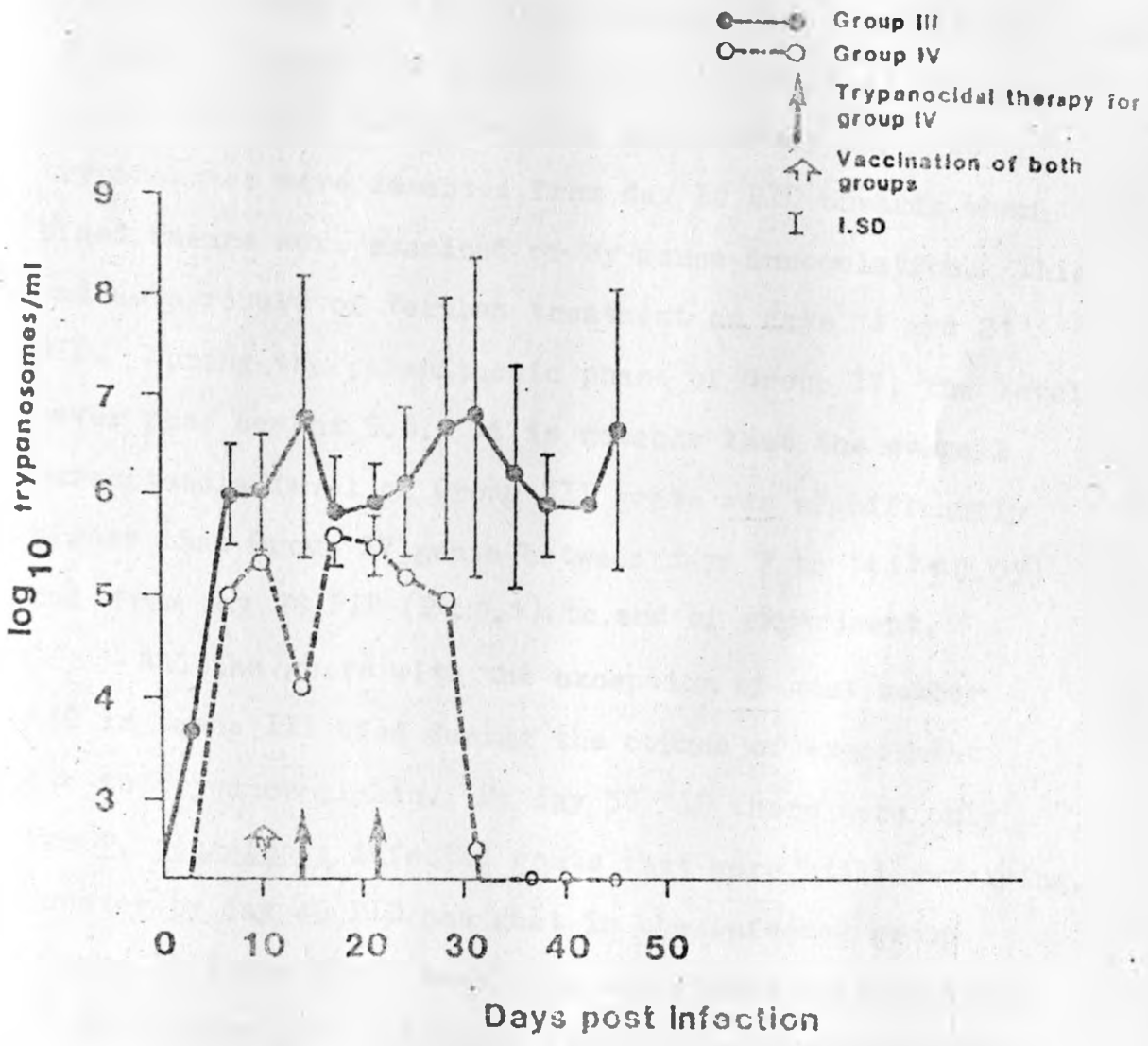
PID. On day 32 PID the mean temperature was low (37.1°C) which coincided with the subnormal temperature (35°C) of goat 401 which died on day 33 PID. The mean temperatures of Group IV were of pre-infection values between days 0 to 8, 12, 19 to 21, 29 to 31 and on day 33 PID to the end of experiment. After initial Veriben treatment on day 14 PID temperature dropped from 39.4°C to 38.6°C on day 19 PID. The second Veriben treatment on day 21 PID was given when the temperature was rising coinciding with a wave of parasitaemia. But by day 29 PID, temperatures dropped to pre-infection values for the rest of experiment. The peak on day 44 PID (39.1°C) may be due to a response to trypanosome fractions that may still be present in treated goats.

The temperatures of Group II goats were generally as control goats (Group I) except on days 14 and 15 (38.6°C) and day 23 (38.6°C) when Group II goats had higher temperatures, possibly due to vaccination effect.

4.2. Parasitaemia.

The parasitaemic profile of T. congolense infected goats is shown in figure 3. Goat number 426 in Group III became parasitaemic on day 3 PID and by day 7 PID all the eight goats in this group were parasitaemic with a mean $\log_{10}$ 6.0 of the trypanosomes. This level of parasitaemia persisted, though fluctuating with peaks on day 14 (6.8), 28 (6.6), 30 (6.8) and 45 (6.6) PID. However in the infected goats in Group III the parasitaemic levels were above $\log_{10}$ 5.8 throughout the infection period.

Figure 3 Mean parasitaemia profile (log_{10}) of T.congolense infected goats



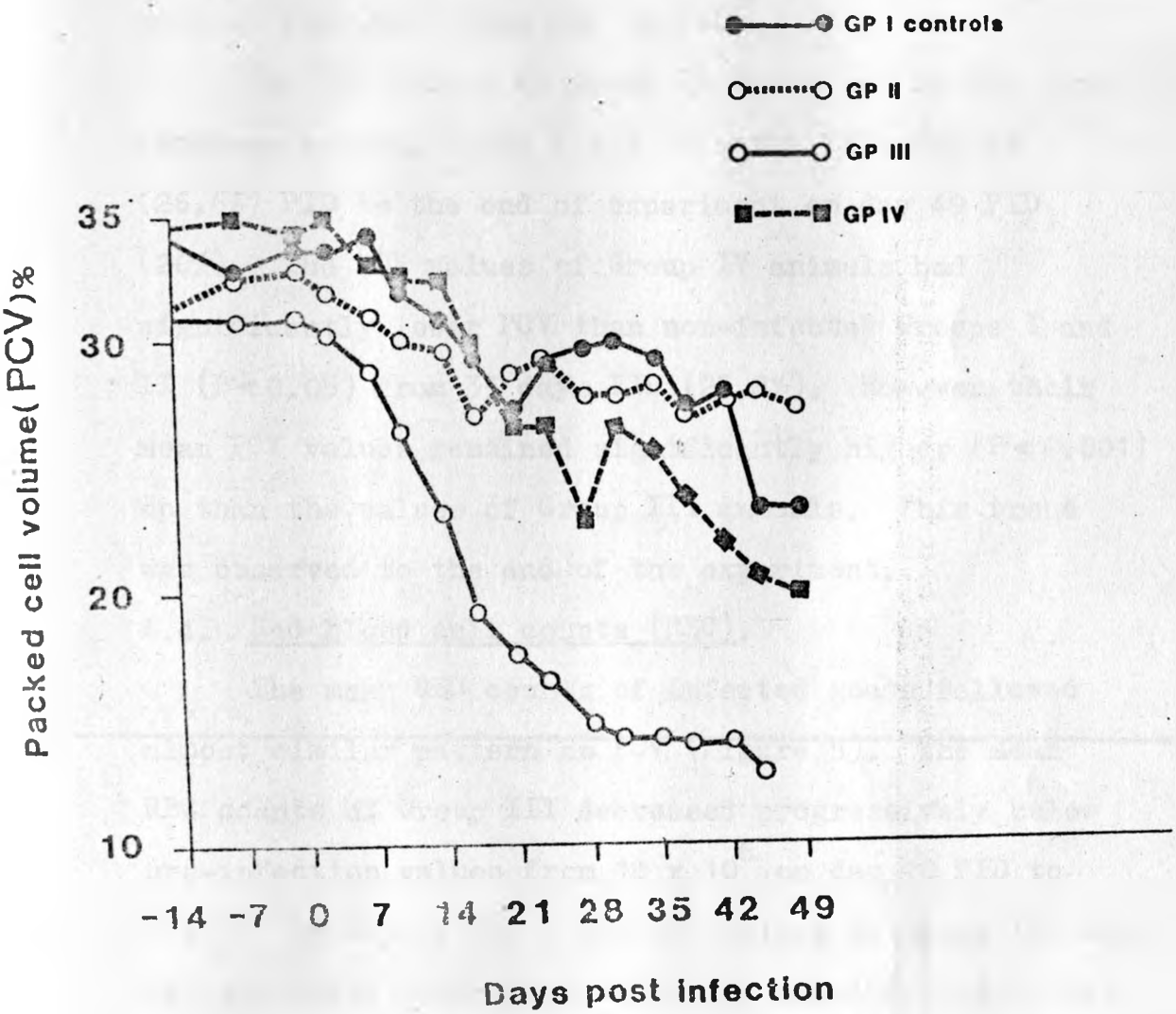
Two goats (numbers 401 and 407) of Group IV, first became parasitaemic on day 7 PID. However by day 10 PID all the five goats were parasitaemic. The parasitaemic level of Group IV goats had peaks on day 10 PID (5.4) and on day 17 PID (5.6) later dropping to 5 on day 28. No trypanosomes were detected from day 30 PID onwards when blood smears were examined or by mouse innoculation. This was as a result of Veriben treatment on days 14 and 21 PID. During the parasitaemic phase of Group IV, the level never rose beyond 5.6. It is notable that the overall parasitaemia level of Group III goats was significantly higher than Group IV goats between days 7 to 14 ($P<0.05$) and from day 28 PID ($P<0.1$) to end of experiment.

All the goats with the exception of goat number 448 in Group III died during the course of experiment due to trypanosomiasis. By day 36 PID there were only two T. congolense infected goats that were still surviving. However by day 46 PID one goat in the infected group (Group III) was still surviving and indeed this goat was at this stage post challenge found to be parasitologically negative when blood smears were examined or by mouse inoculation.

4.3. Packed cell volume.

The mean packed cell volumes (PCV%) of infected and control groups are shown in Figure 4. The mean PCV of Group III animals dropped below pre-infection values progressively from day 7 PID (29%) to 14% on days

Figure 4 Changes in means packed cell volume (PCV%) of T.congolense infected goats



38 to 42 and to 13% in the two remaining (446 and 448) infected goats on day 45 PID. The PCV values of Group III goats was significantly lower than that of the other groups ($P < 0.001$) from day 10 PID.

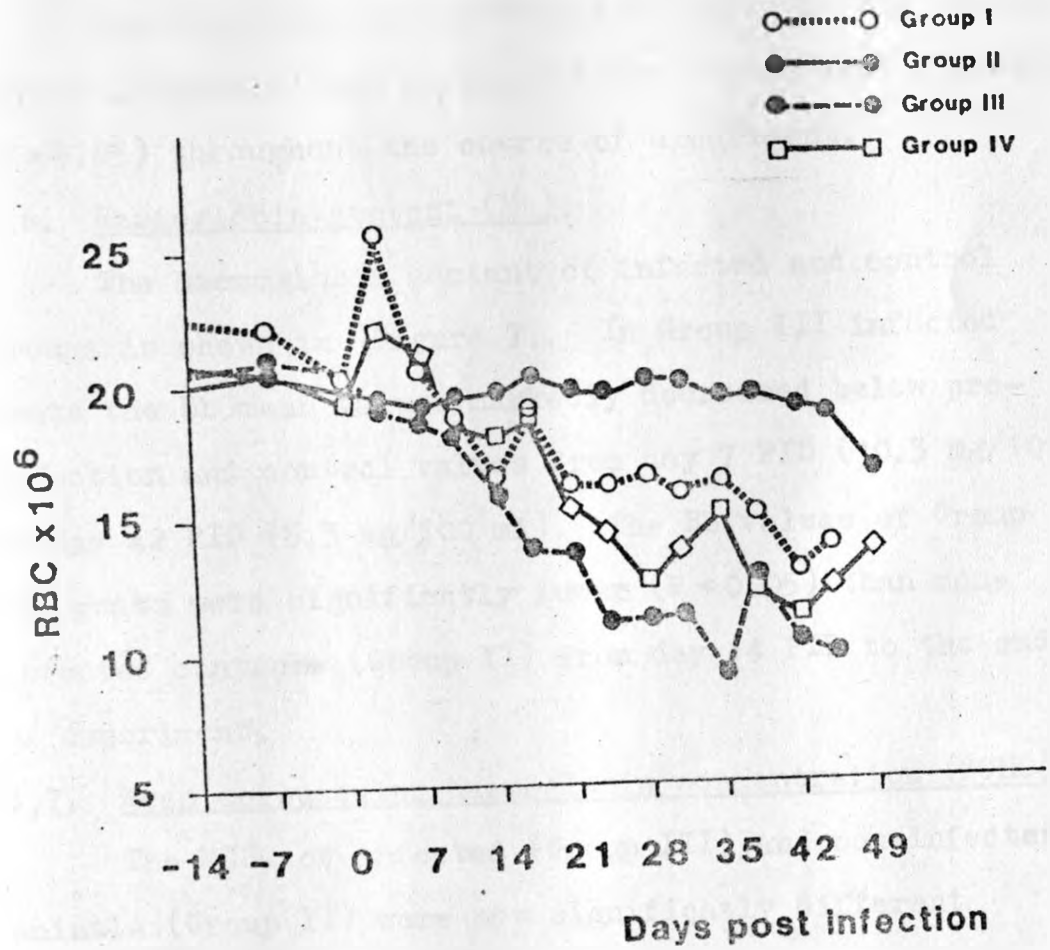
The PCV values of Group IV goats was in the same range as control Group I and II until from day 24 (26.6%) PID to the end of experiment on day 49 PID (20%). The PCV values of Group IV animals had significantly lower PCV than non-infected Groups I and II ($P < 0.05$) from 35 days PID (25.8%). However their mean PCV values remained significantly higher ($P < 0.001$) up than the values of Group III animals. This trend was observed to the end of the experiment.

4.4. Red blood cell counts (RBC).

The mean RBC counts of infected goats followed almost similar pattern as PCV (Figure 5). The mean RBC counts of Group III decreased progressively below pre-infection values from 18×10^6 on day 10 PID to 9×10^6 on day 45 PID. The RBC values of Group III were significantly lower than values of non-infected Groups I and II ($P < 0.01$) but not of Group IV ($P > 0.1$).

The mean RBC values for Group IV dropped from 15.4×10^6 on day 21 PID to 12.2×10^6 on day 45 PID. Group IV goats had significaltly lower ($P < 0.01$) RBC counts than control goats from 21 days PID. There was no significant statistical differences between the RBC

Figure 5 Changes in mean red blood cell counts (RBC in $x10^6/\mu l$ of blood) of T. congolense infected goats



counts of Group III and Group IV goats ($P > 0.05$) although Group III goats had lower RBC counts than Group IV goats from 14 days PID.

4.5. Mean cell volume (MCV).

The mean corpuscular volume of infected and control groups (Figure 6) had no significant statistical difference ($P > 0.05$) throughout the course of experiment.

4.6. Haemoglobin content (Hb).

The haemoglobin content of infected and control groups is shown in Figure 7.. In Group III infected goats the Hb mean values markedly decreased below pre-infection and control values from day 7 PID (10.3 mg/100ml) to day 42 PID (5.3 mg/100 ml). The Hb values of Group III goats were significatly lower ($P < 0.05$) than non-infected controls (Group II) from day 14 PID to the end of experiment.

4.7. Mean corpuscular haemoglobin concentration (MCHC).

The MCHC of infected (Group III) and non-infected animals (Group II) were not significatly different ($P > 0.05$) throughout the course of the experiment (Figure 8) and remained all within normal values.

4.8. Mean white blood cell counts (WBC).

The mean WBC for all the groups are shown on Figure 9. Goats in Group I showed no marked changes in WBC throughout the experiment. Group II goats showed a rise in WBC from 21 days PID (15.4×10^3) which

Figure 6 Changes in mean corpuscular volume (MCV, μ) of T. congolense infected goats

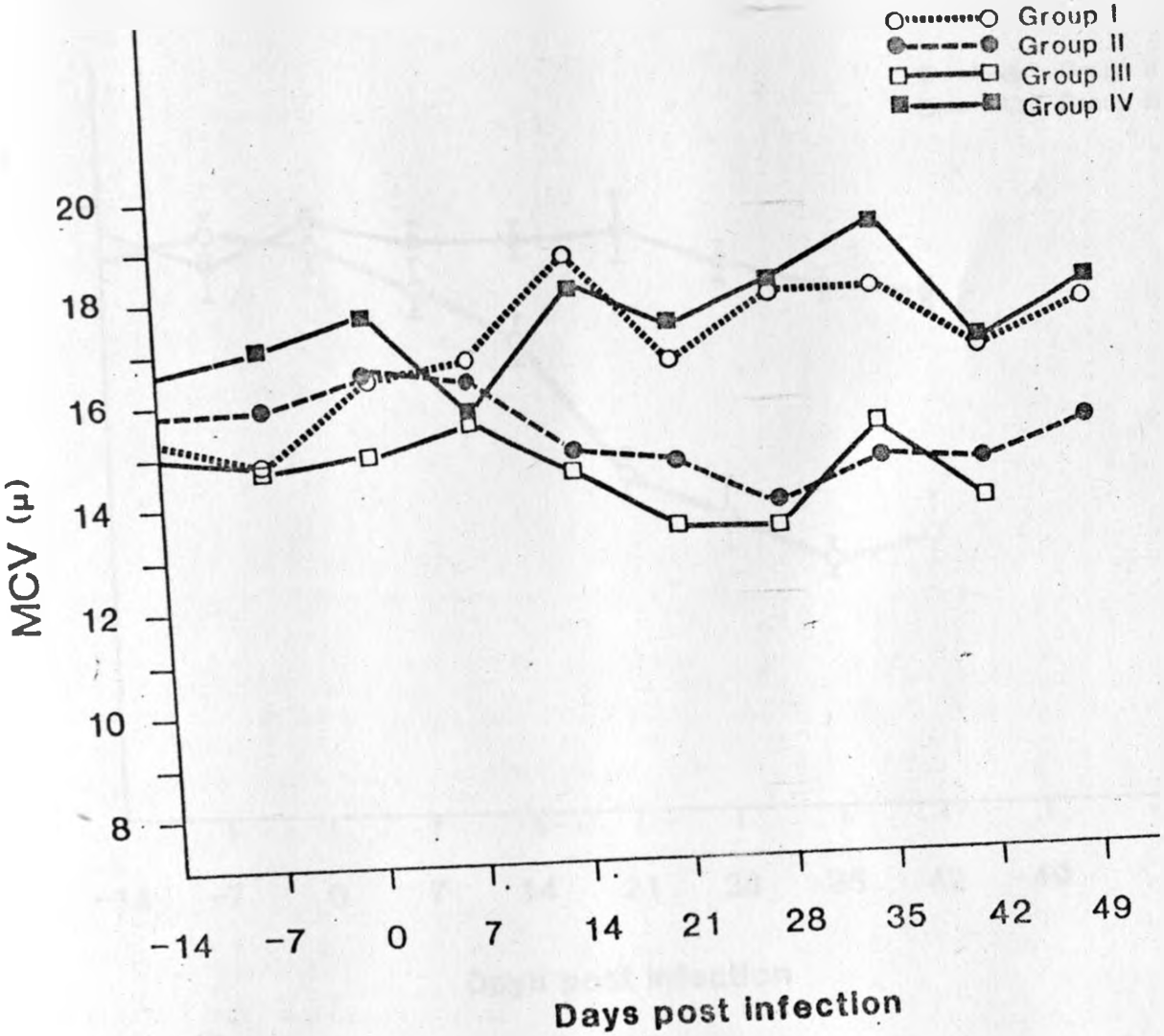


Figure 7 Changes in mean haemoglobin content (Hb mg/100ml) of T. congolense infected goats

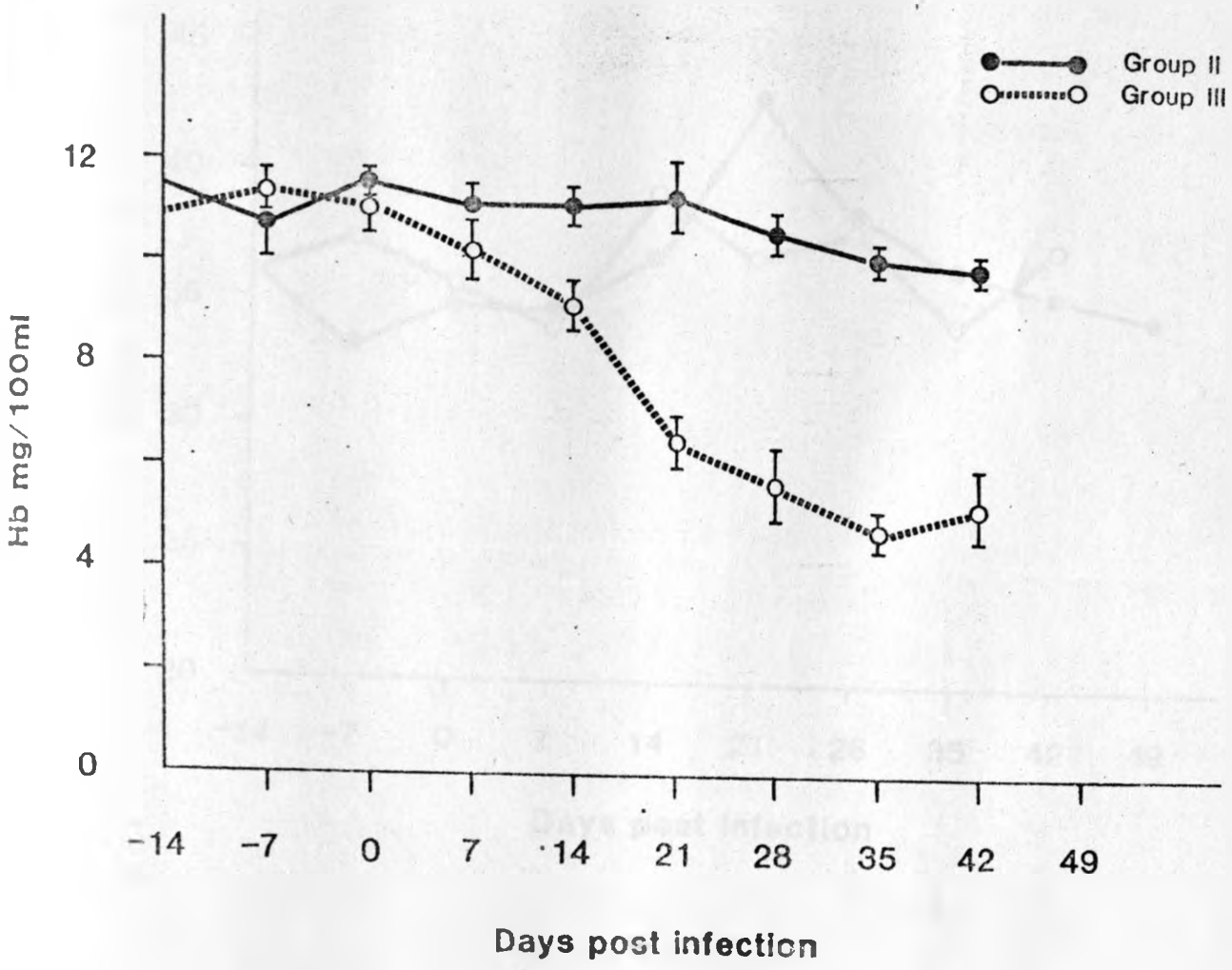
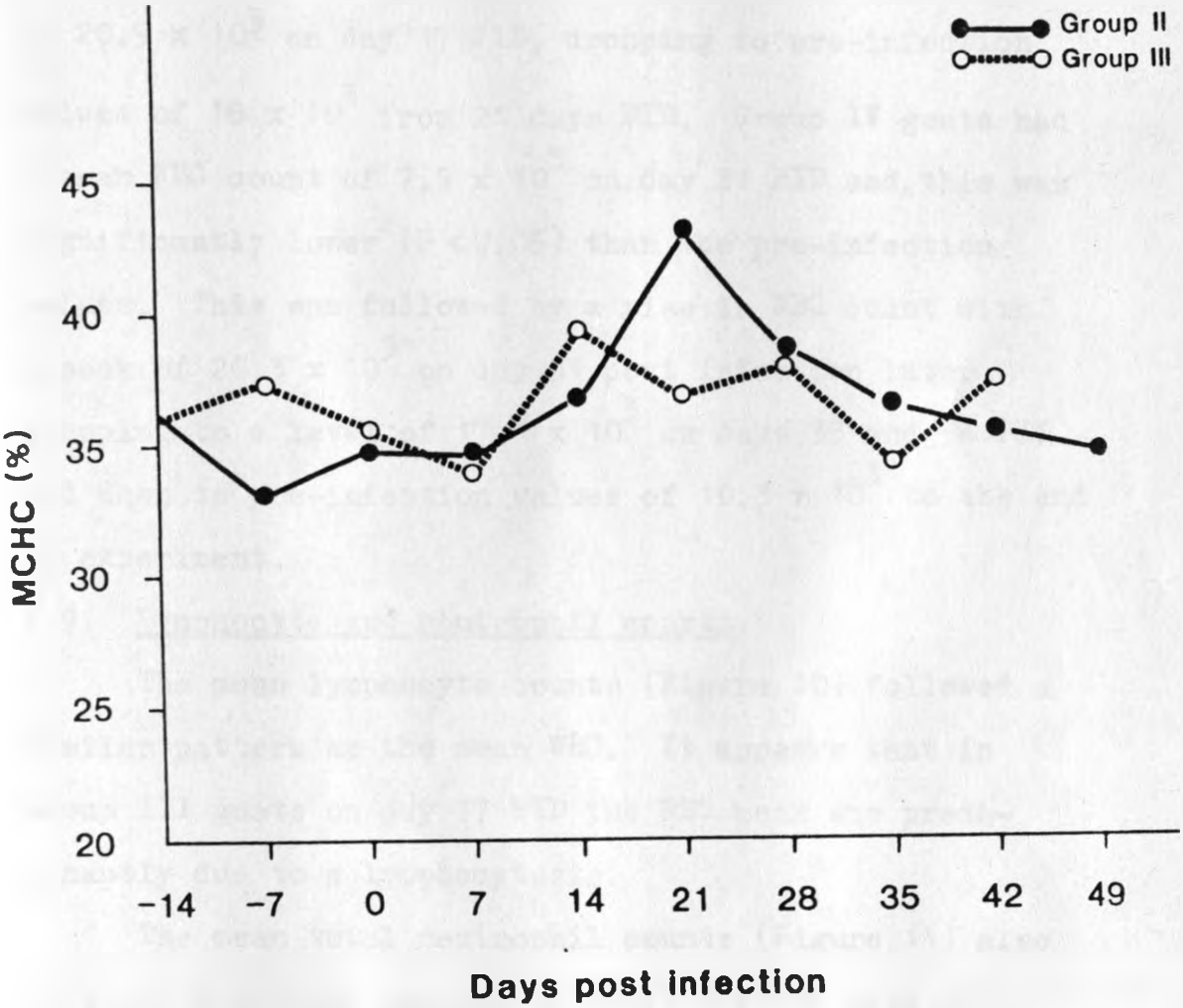


Figure 8 Changes in mean corpuscular heamoglobin concentration (MCHC %) of T. congolense infected goats



later dropped to pre-infection values (15×10^3) on day 31 and to 11.4×10^3 by day 49 PID.

Group III goats showed a rise in WBC with the peak of 20.9×10^3 on day 17 PID, dropping to pre-infection values of 18×10^3 from 21 days PID. Group IV goats had a mean WBC count of 7.9×10^3 on day 21 PID and this was significantly lower ($P < 0.05$) than the pre-infection values. This was followed by a rise in WBC count with a peak of 20.3×10^3 on day 31 post infection later dropping to a level of 17.1×10^3 on days 35 and 38 PID and then to pre-infection values of 10.3×10^3 to the end of experiment.

4.9. Lymphocyte and neutrophil counts.

The mean lymphocyte counts (Figure 10) followed a similar pattern as the mean WBC. It appears that in Group III goats on day 17 PID the WBC peak was predominantly due to a lymphocytosis.

The mean total neutrophil counts (Figure 11) also followed a similar pattern as WBC. The WBC peak of Group II on day 28 PID appeared to be mainly due to an increase in total neutrophils. The total neutrophil counts in Group III though fluctuating, decreased with the course of the disease. The WBC peak of Group IV appeared to be due to both a lymphocytosis and a neutrophilia.

Figure 9 Changes in mean white blood cell counts (WBC $\times 10^3/\mu l$ of blood) of T. congolense infected goats

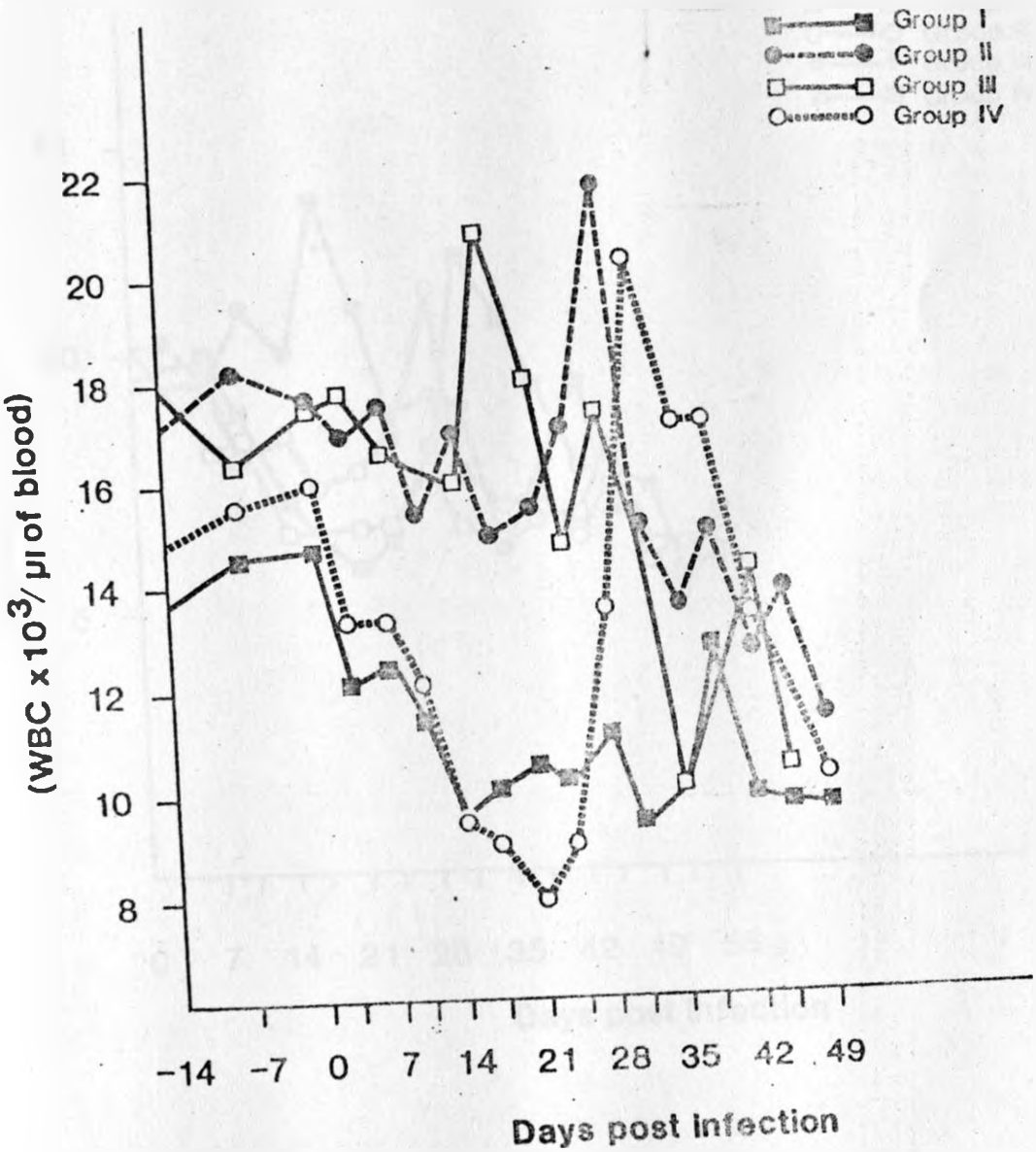


Figure 10 Changes in mean lymphocyte counts ($x10^3/\mu l$ blood) of T. congolense infected goats

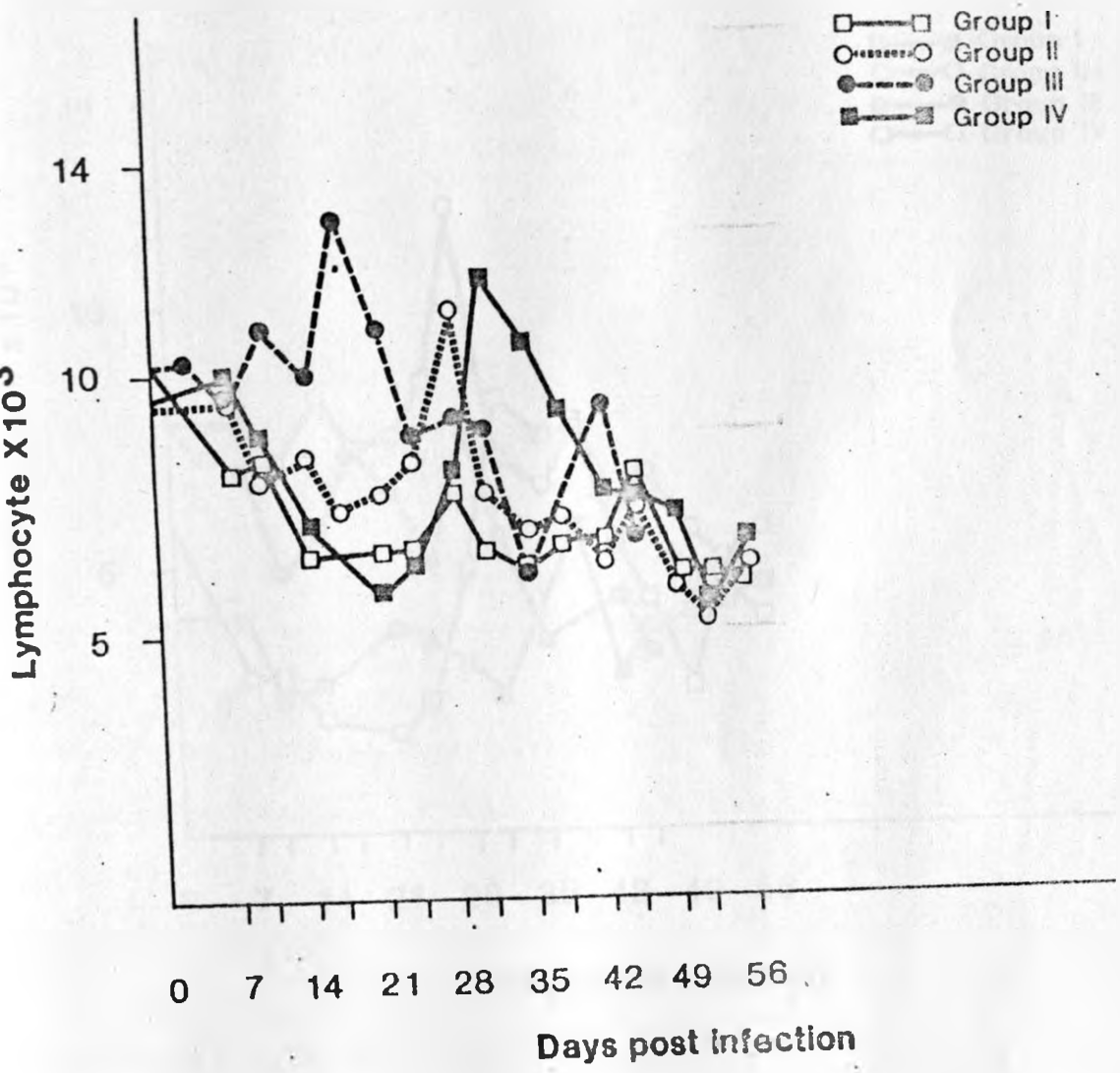
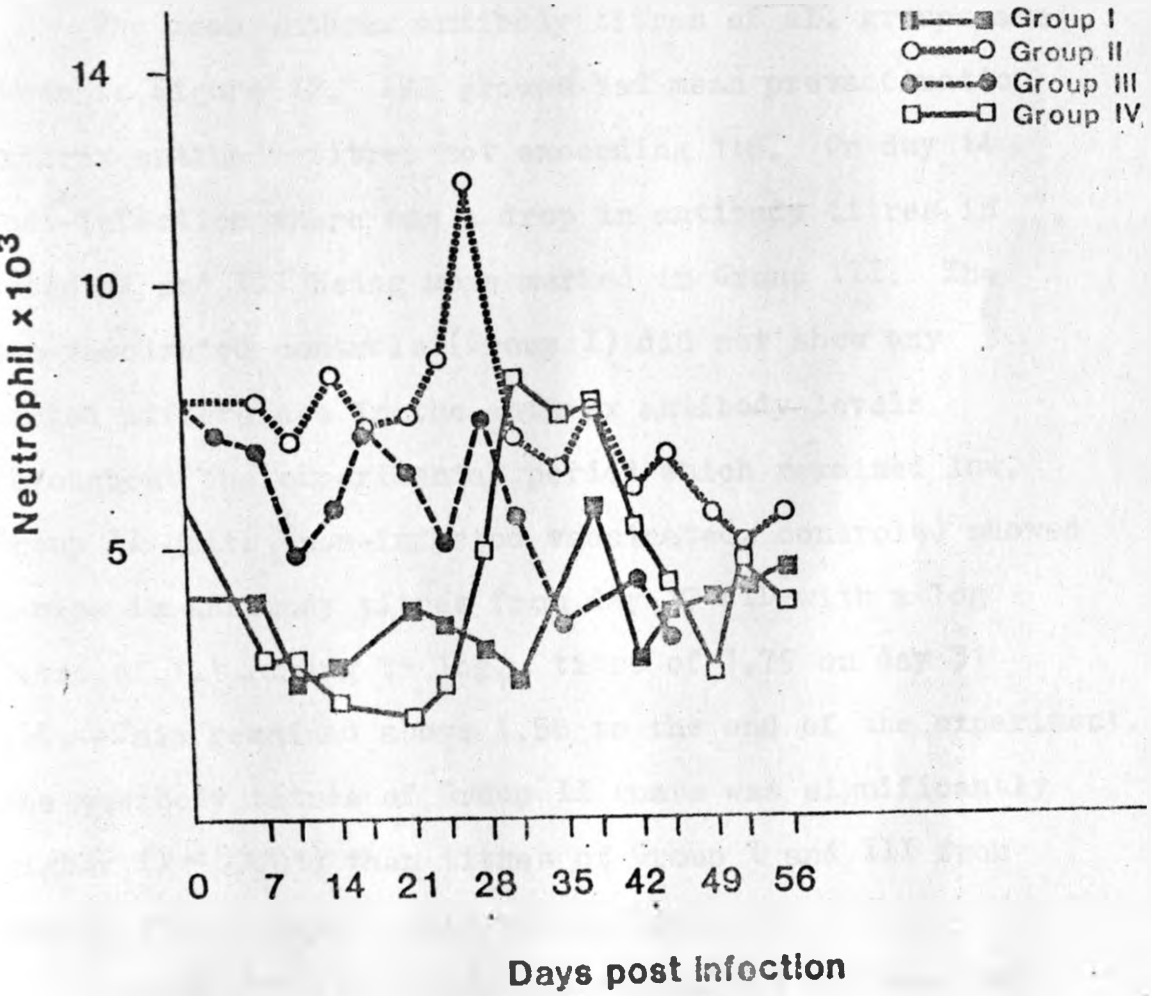


Figure 11 Changes in mean neutrophil counts ($x10^3/\mu l$ of blood) of T. congolense infected goats



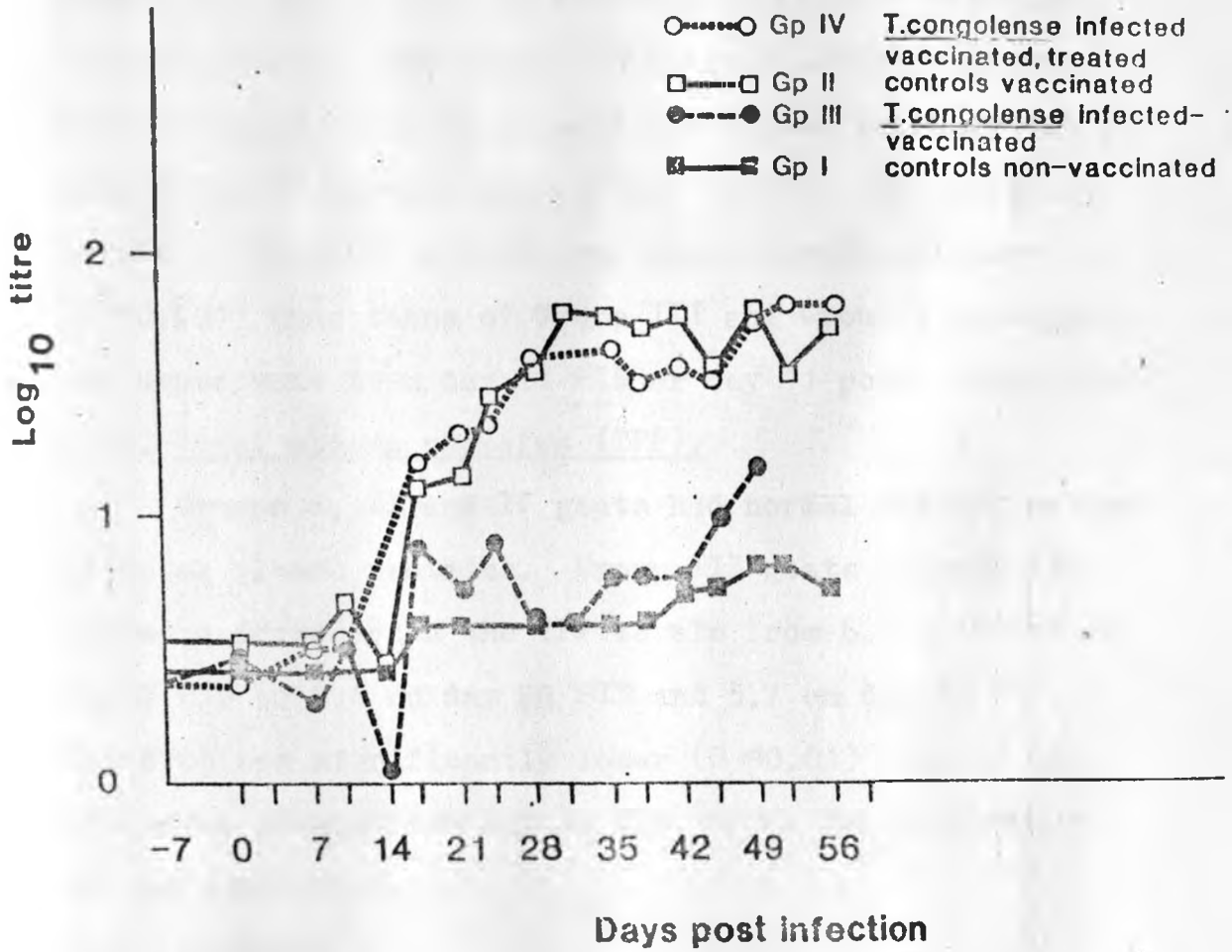
The low WBC count of Group IV goats on 21 days PID appeared to be due to a lymphopaenia and neutropaenia..

4.10. Anthrax indirect haemagglutination antibody titres (Log_{10} of IHA titre).

The mean anthrax antibody titres of all groups are shown in Figure 12. All groups had mean prevaccination anthrax antibody titres not exceeding 1:8. On day 14 post-infection there was a drop in antibody titres in Group II and III being more marked in Group III. The non-vaccinated controls (Group I) did not show any marked differences in the anthrax antibody levels throughout the experimental period which remained low. Group II goats (non-infected vaccinated, controls) showed a rise in antibody titres from day 17 PID with a log titre of 1.1 rising to log_{10} titre of 1.79 on day 31 PID. This remained above 1.58 to the end of the experiment. The antibody titres of Group II goats was significantly higher ($P < 0.001$) than titres of Group I and III from day 21 PID or day 11 post vaccination.

Group III (infected, vaccinated but not treated) goats showed only an unsteady insignificant rise of antibody titres throughout the experiment with peaks on day 17 PID (0.9), day 24 (0.9), day 47 (Only one goat survived, 1.2). There was no significant statistical difference between Group III and Group I ($P > 0.05$), although Group III had relatively higher antibody levels than Group 1 from day 21 PID or day 11 post-vaccination.

Figure 12 Mean anthrax antibody titres (log_{10} titre) following vaccination of goats infected with T. congolense



Group IV animals (infected vaccinated and treated) showed a similar rise in antibody titres as Group II. From day 14 PID the antibody titre of 1.2 rose to 1.61 on day 35 PID. This remained steady around 1.6 until day 49 when the titre was 1.75 and then rose to a maximum of 1.8 from day 52 to day 56 PID. There was however no significant statistical difference ($P > 0.05$) between Group II and Group IV anthrax antibody titres. Nevertheless, Group II animals had higher antibody titres than Group IV between days 31 and 42 PID. The antibody levels of Group IV animals was significantly higher ($P < 0.001$) than those of Group III and Group I throughout the experiment from day 21 PID or day 11 post vaccination.

4.11. Total plasma proteins (TPP).

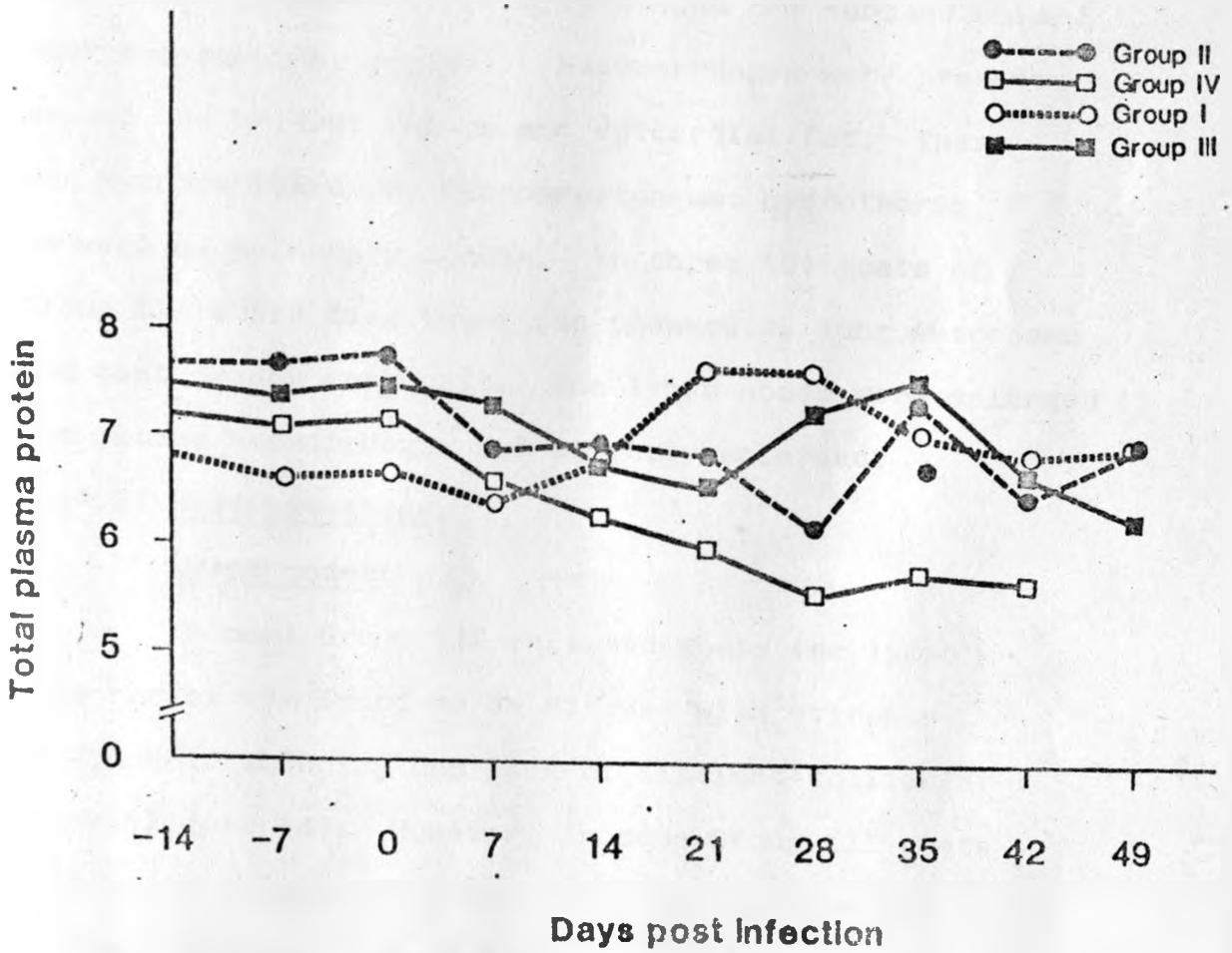
Groups I, II and IV goats had normal control values of total plasma proteins. Group III goats (Figure 13) showed a decrease in the TPP levels from 6.6 g/100 ml on day 7 PID to 5.6 on day 28 PID and 5.7 on day 42 PID. The drop was significantly lower ($P < 0.01$) than in all the other groups from day 28 PID until the termination of the experiment.

4.12. Necropsy.

4.12.1 Gross lesions.

Seven (7) out of the eight (8) T. congolense infected vaccinated goats in Group III died due to trypanosomiasis on various days during the course of

Figure 13 Changes in mean total plasma protein (TPP g/100ml) of T. congolense infected goats



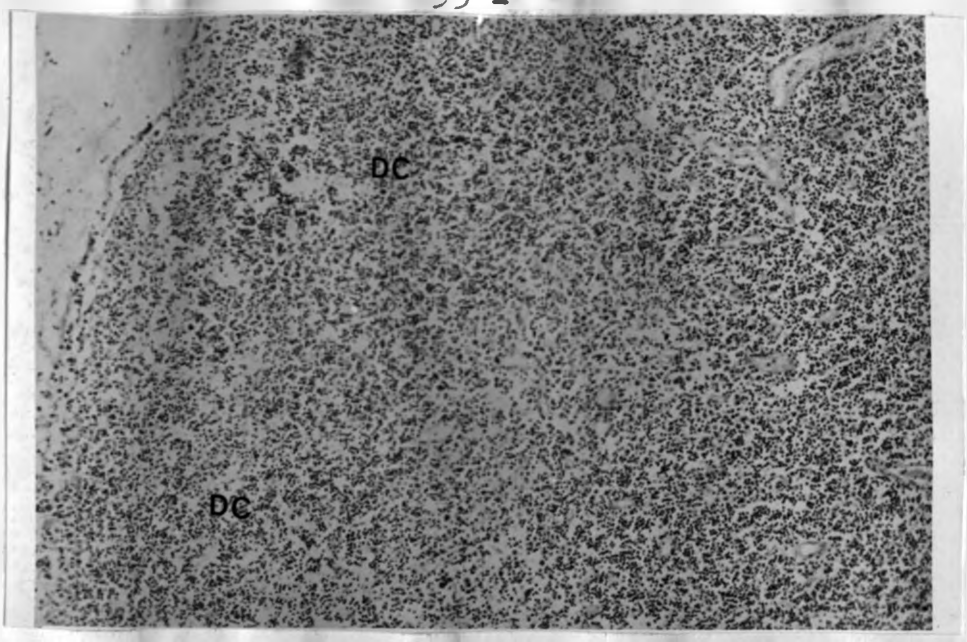


Fig. 14. Lymph node of a Group III infected goat (437) showing diffuse cortex (DC) and lack of distinct follicles.

H & E x 100

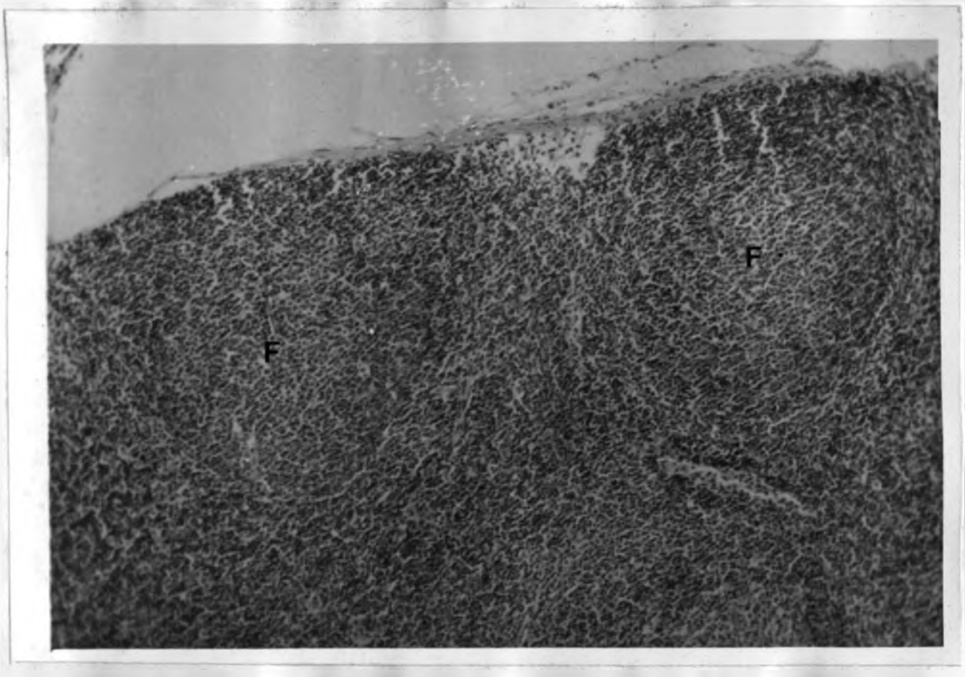


Fig. 15. Lymph node of a Group III infected goat (446) showing distinct follicles (F) but no germinal centres.

MB x 100

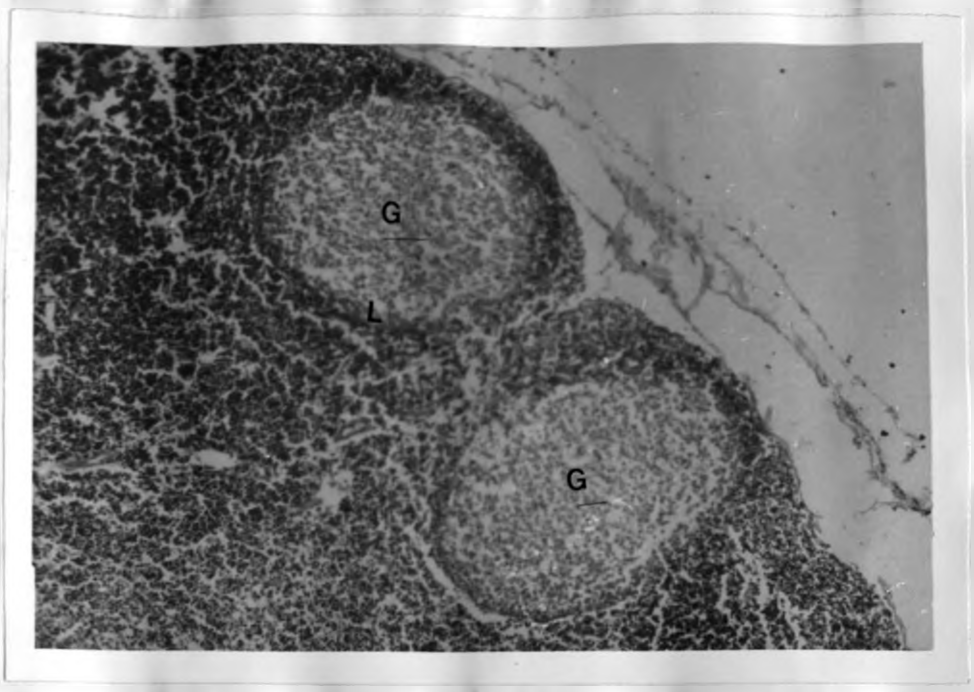


Fig. 16. Lymph node of Group IV infected goat(401) showing germinal centres (G) with a lymphocytic mantle (L).

MB x 100

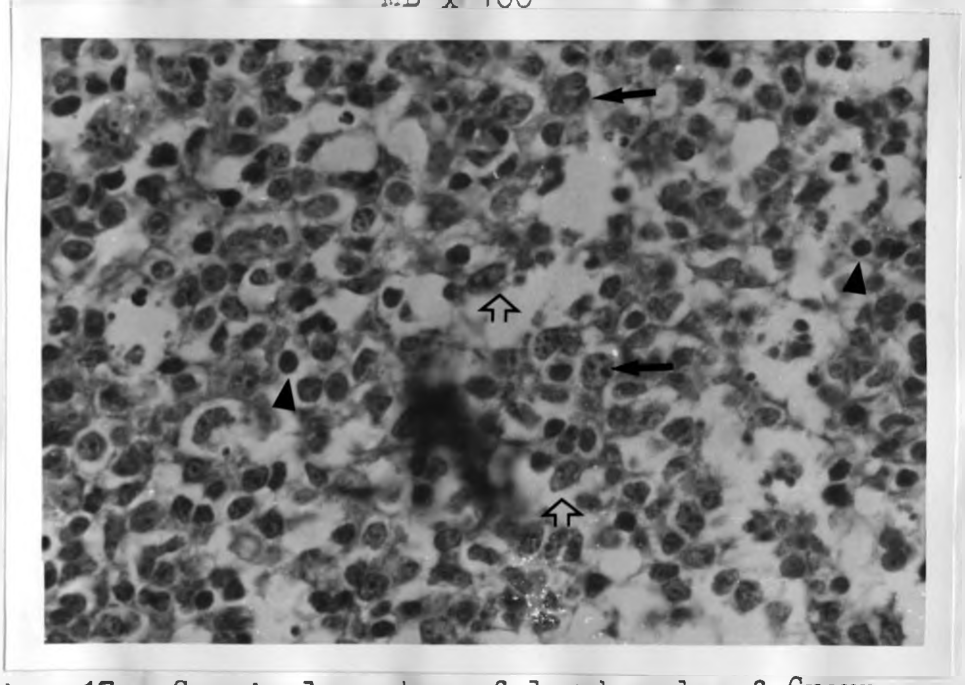


Fig. 17. Germinal centre of lymph node of Group IV goat showing active macrophages (open arrow), large lymphocytic cells (arrow head) and evidence of erythrophagocytosis (thick arrow), (Goat 402).

MB x 630

Germinal centres seen in lymph nodes of Group III goats appeared less active due to predominance of non-lymphocytic cells (Figure 18). In both Group III and Group IV goats, there was evidence of enlarged mononuclear phagocytic system. In the follicular and paracortical areas (Figure 19). Plasma cells, plasma cell precursors, lymphocytes and many mononuclear phagocytic cells especially macrophages were predominant also in the medulla of lymph nodes (Figure 20). Evidence of phagocytosis of erythrocytes and trypanosomes by macrophages and hemosiderosis were evident (Figures 19 and 20).

Spleen.

Extensive changes were observed in the spleen of both infected groups. In Group III infected goats, the red pulp was hypertrophic containing mainly mononuclear cells especially macrophages lymphoblasts, plasma cells, plasma cell precursors, erythropoietic and granulopoietic cells. The white pulp and the periarterial lymphocytic sheaths (PALS) were severely depleted, disorganised and had indistinct follicles (Figure 21 and 22). Very few germinal centres were evident. The white pulp mainly comprised of macrophages, lymphoblasts and plasma cells replacing the lymphocytic sheaths (Figure 23). Macrophages observed in the red and white pulp had high phagocytic activity. This was indicated by

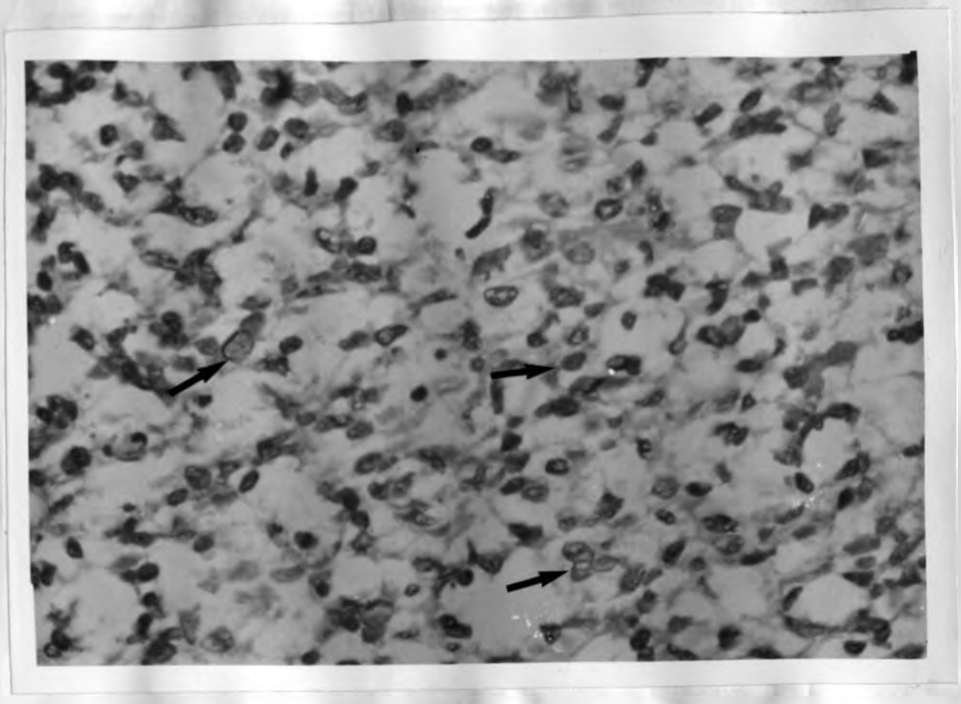


Fig. 18. Germinal centre of lymph node of Group III infected goat (445) showing predominance of non-lymphocytic cells (arrowed).

H & E x 630

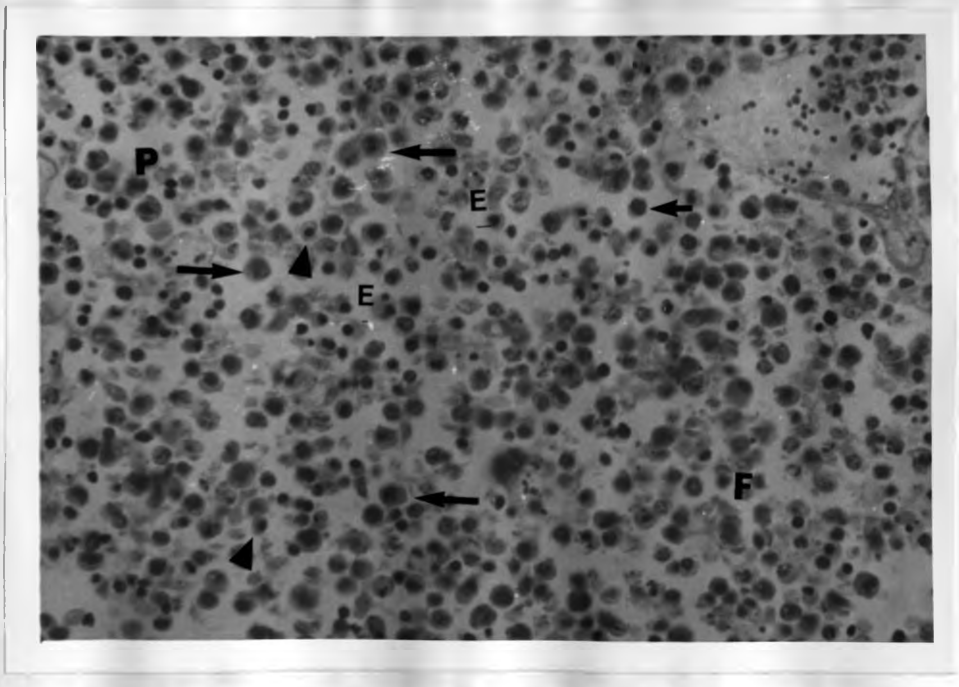


Fig. 19. Follicular (F) and paracortical (P) areas of lymph node of Group III infected goat (437) showing many macrophages (thick arrow) lymphoblasts (short arrow) plasma cells (arrow head) and erythrophagocytosis (E).

MB x 400

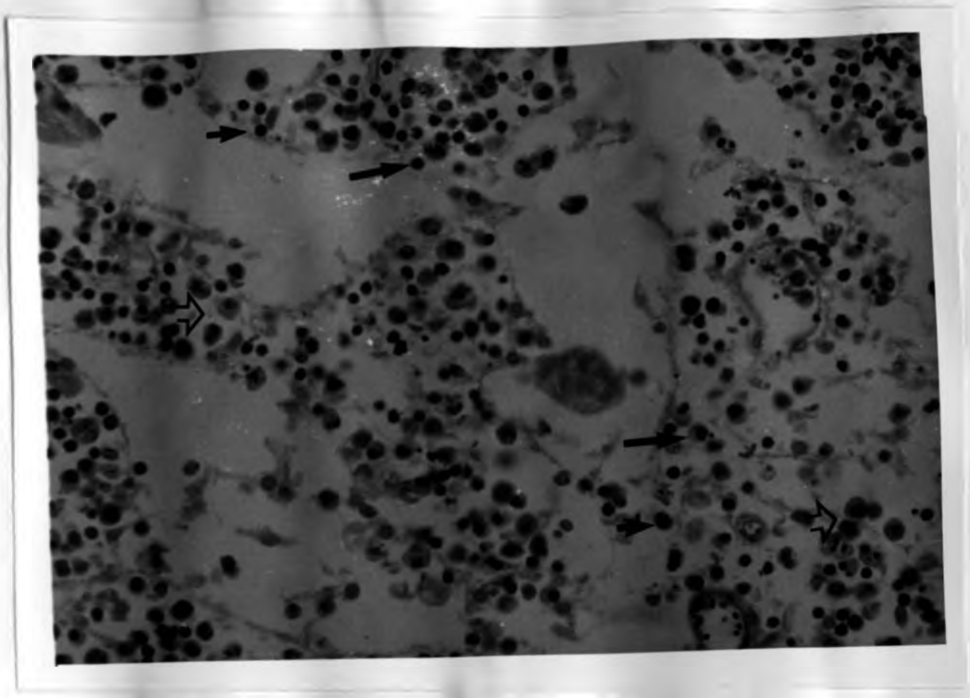


Fig. 20. Medulla of lymph node of a Group III (Goat 447) infected goat showing many macrophages (open arrow), plasma cells (thick arrow) and lymphocytes (short arrow).

MB x 400

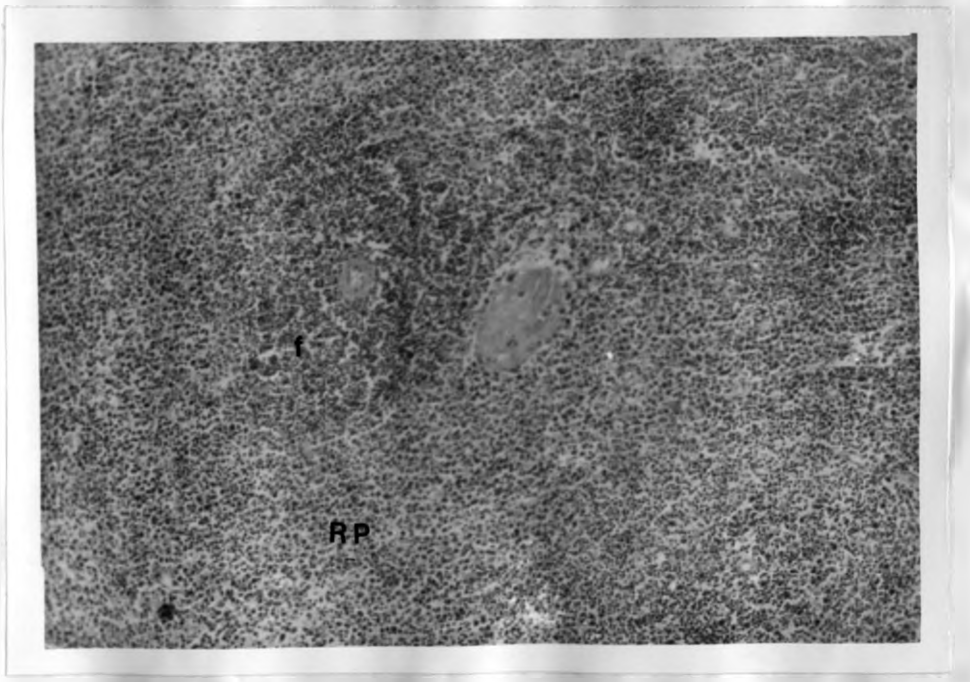


Fig. 21. Spleen of a Group III goat (447) showing disorganisation of the follicular areas (f) hypetrophic red pulp (RP) with many mononuclear cells.

H & E x 40

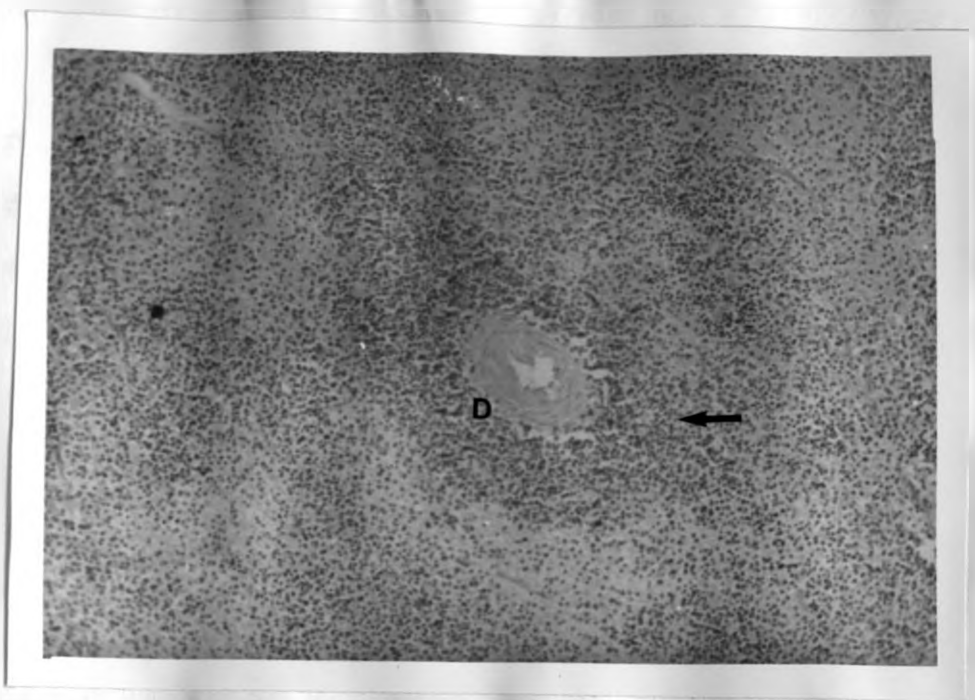


Fig. 22. Spleen of Group III infected goat (446) showing slight depletion (D) of white pulp and lack of lymphocytic mantle (arrow).

H & E x 100

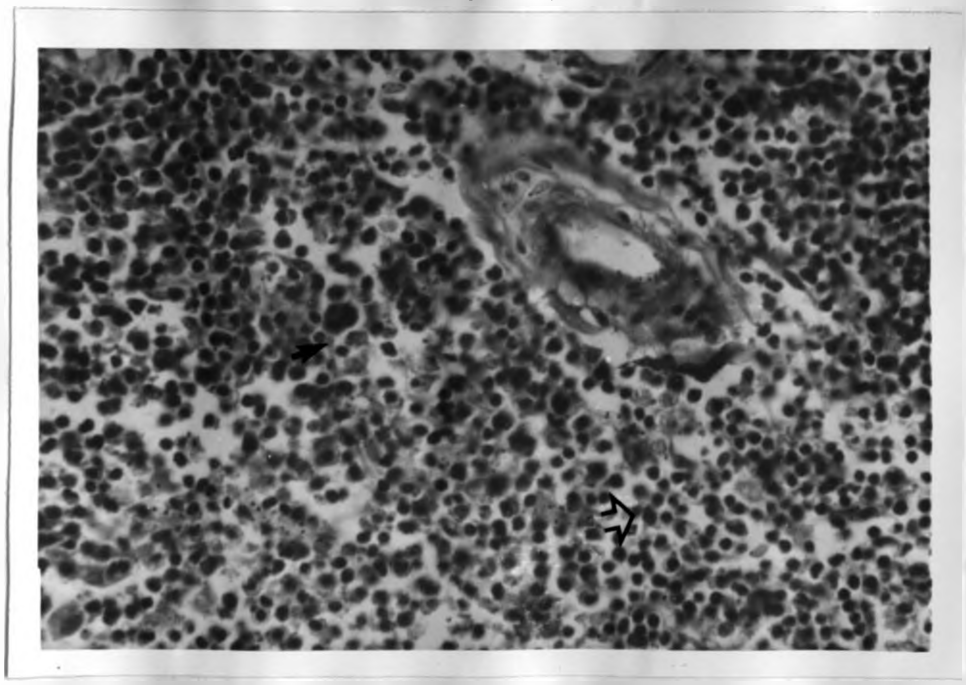


Fig. 23. Spleen of Group III infected goat (436) showing replacement of lymphocytic sheath by macrophages (arrow) and other mononuclear cell (arrow head).

MB x 400

presence of vacuolation of cytoplasm, hemosiderin, erythrophagocytosis and phagocytosis of trypanosome remnants (Figure 24). Whole trypanosomes were observed either free in the red pulp of spleen or in macrophages of Group III goats (Figures 25 and 26).

In Group IV goats the white pulp was adequately populated and was hyperplastic (Figure 27a). Follicles were evident and most had active germinal centres which had predominatly large lymphocytes and lymphoblasts with prominent nucleoli indicating mitotic activity (Figure 27b).

Liver.

Infected goats of Group III showed marked expansion of sinusoidal spaces, hepatic congestion, and evidence of increased infiltration with mononuclear cells (Figure 28). There was also evidence of necrosis of hepatocytes (Figure 29).

Lungs.

Group III goats showed high incidence of pneumonia evidenced by marked pulmonary consolidation, congestion, and oedema (Figures 30 and 31). The consolidated areas showed marked infiltration by lymphocytes, neutrophils, plasma cells and many macrophages. Some of these macrophages contained erythrocytes and hemosiderin (Figure 32).

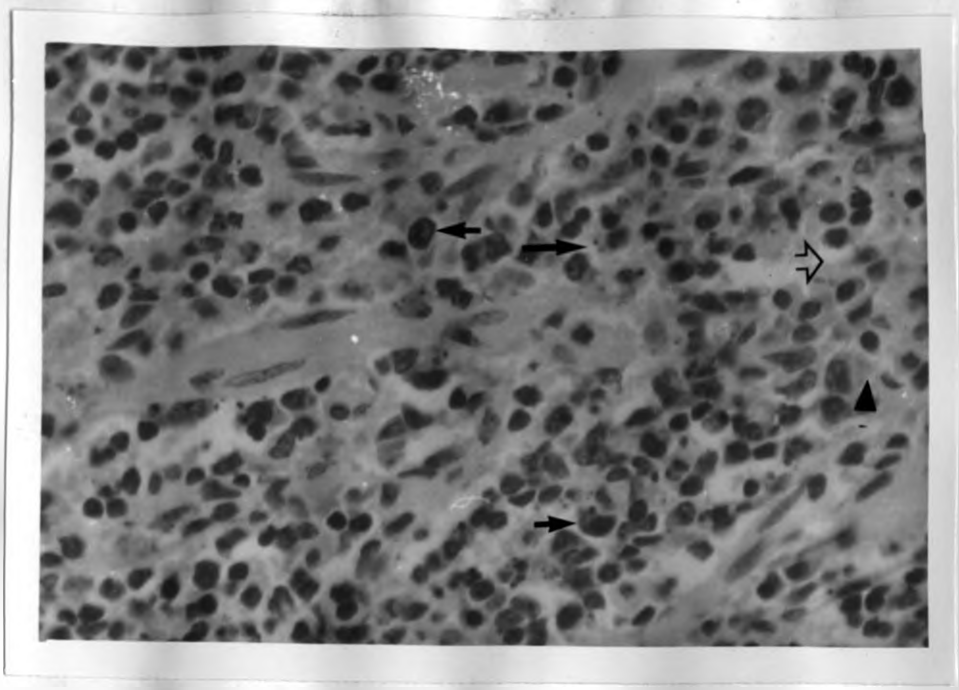


Fig. 24. Spleen of Group III goat (428) showing many macrophages containing erythrocytes (short arrow), trypanosome remnants (long arrow), vacuolation (open arrow) and hemosiderosis (arrow head).

H & E x 630

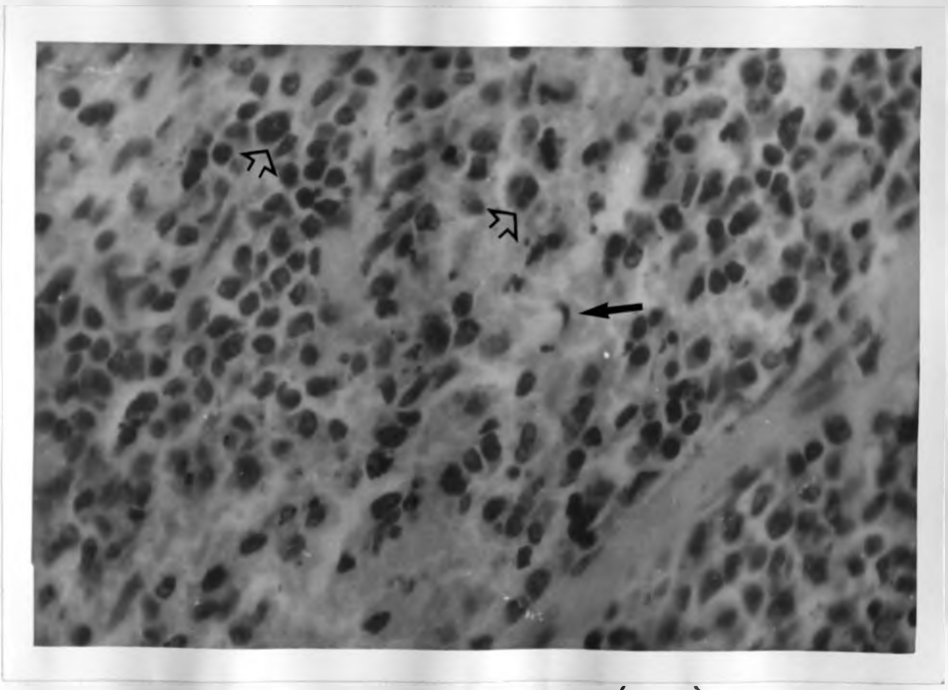


Fig. 25. Spleen of Group III goat (446) showing trypanosome free in the red pulp (arrow) and numerous macrophages (open arrow).

H & E x 630

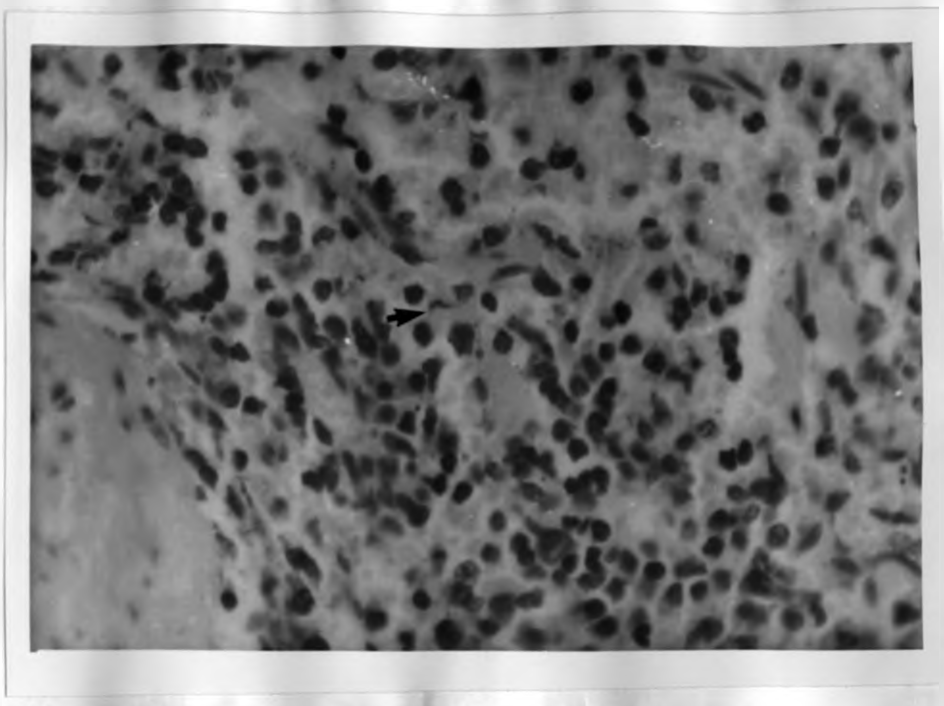


Fig. 26. Spleen of Group III goat (447) showing Trypanosoma congolense inside a macrophage (arrow).
H & E x 630

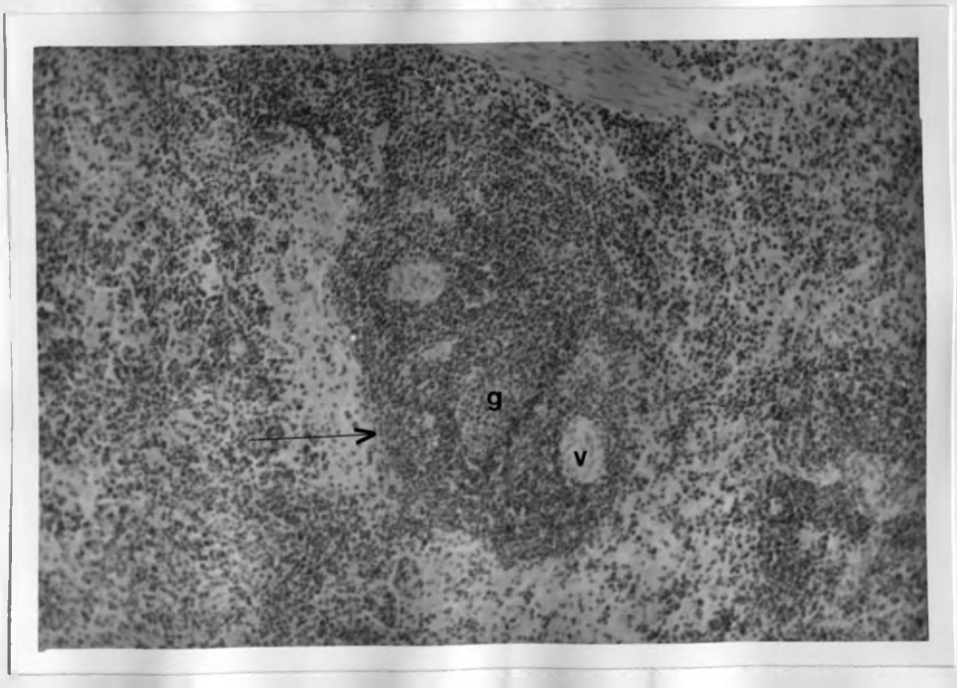


Fig. 27(a). Spleen of Group IV goat (402) showing hyperplasia and adequate population of white pulp; Follicular areas (arrows) around blood vessels (V) with germinal centre (g).
H & E x 100

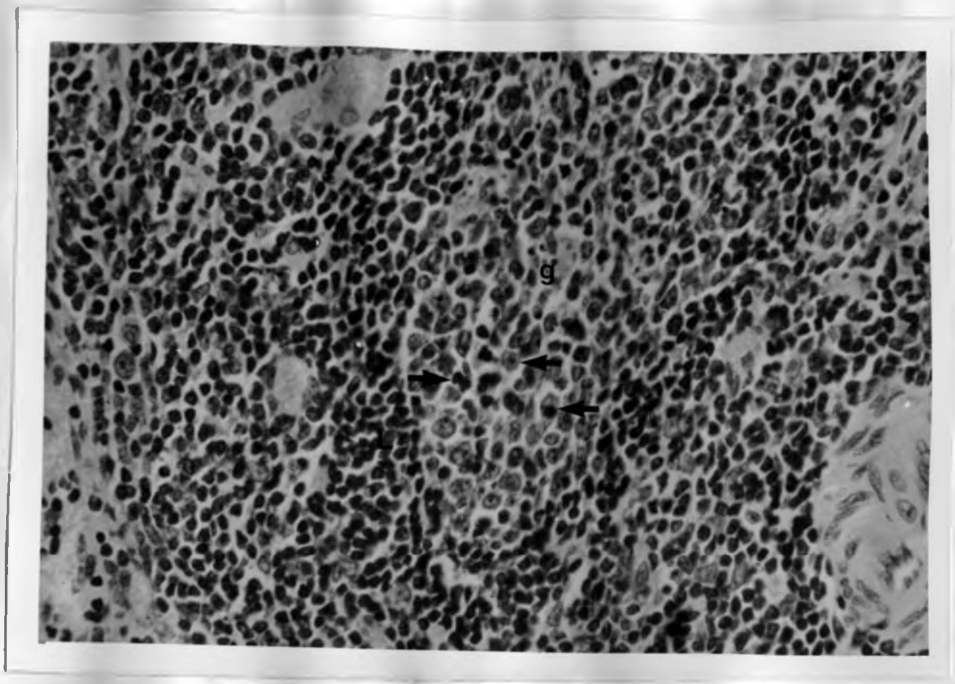


Fig. 27(b). Spleen of Group IV goat (402) showing active germinal centre (g) with large lymphocytes and prominent nucleolus, mitotic activity and lymphocytic sheath (L).

H & E x 400

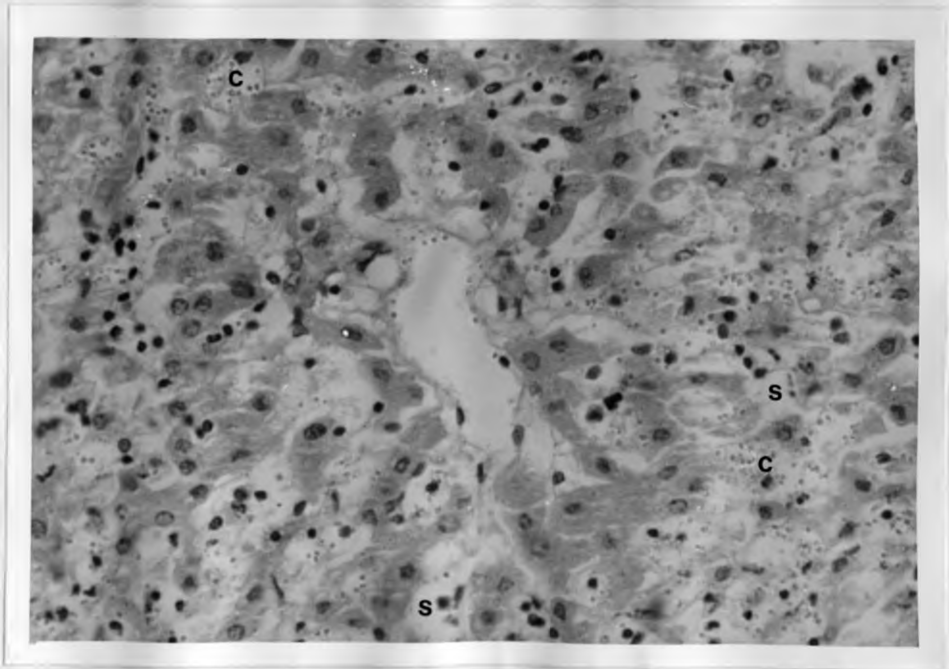


Fig. 28. Liver of Group III goat (428) showing enlarged sinusoidal spaces (S) infiltration with mononuclear cells (arrows) and congestion (C).

H & E x 400

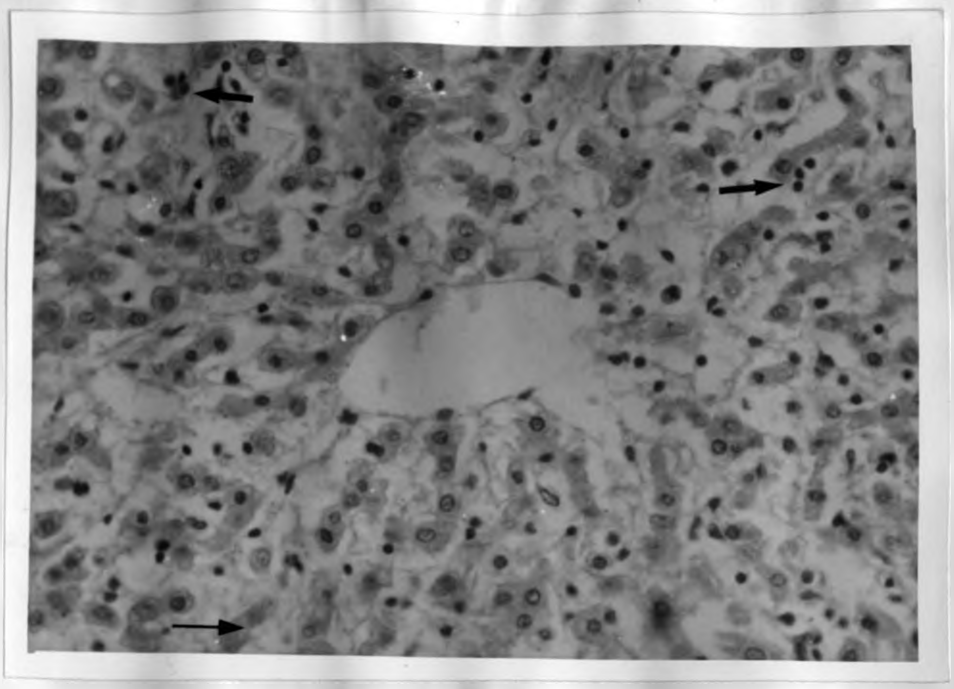


Fig. 29. Liver of Group III goat (447) showing necrosis (Fine arrow) of hepatocytes and mononuclear cell infiltration (thick arrow).

H & E x 400

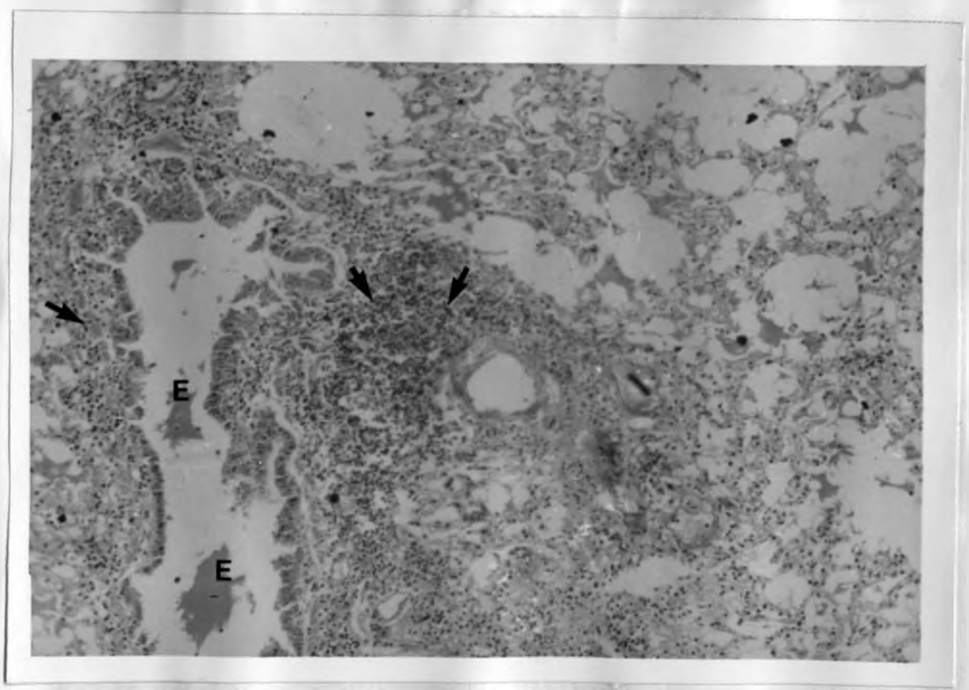


Fig. 30. Lung of Group III goat (445) showing infiltration with inflammatory cells (arrows), oedema (E) as signs of pneumonia.

MB x 100

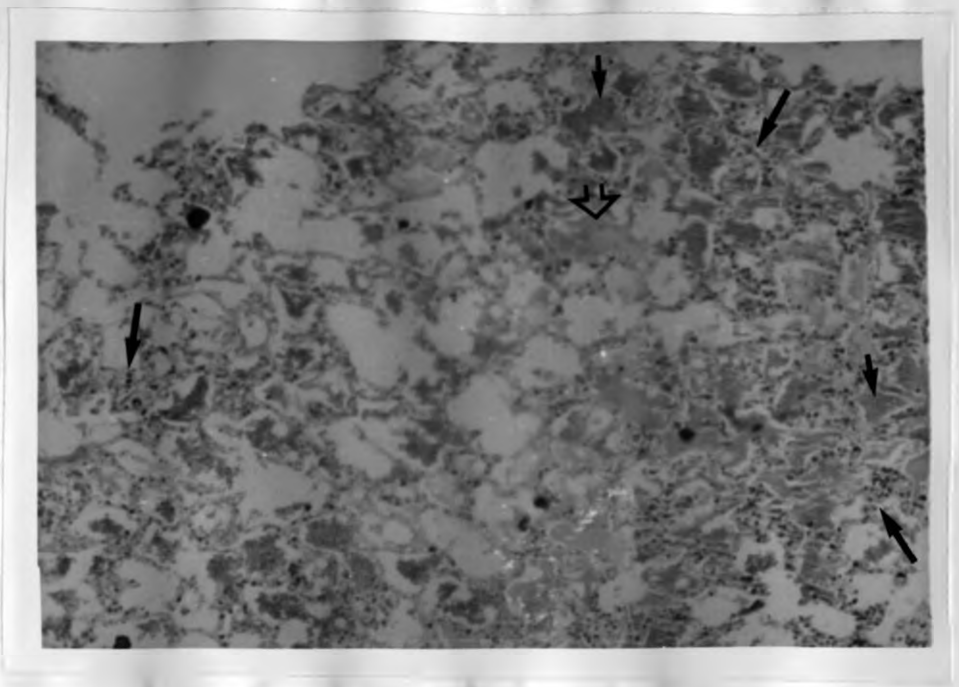


Fig. 31. Lung of Group III goat (437) showing marked pulmonary oedema (open arrows), congestion (long arrows) and marked inflammatory cell infiltration (short thick arrows).

MB x 100

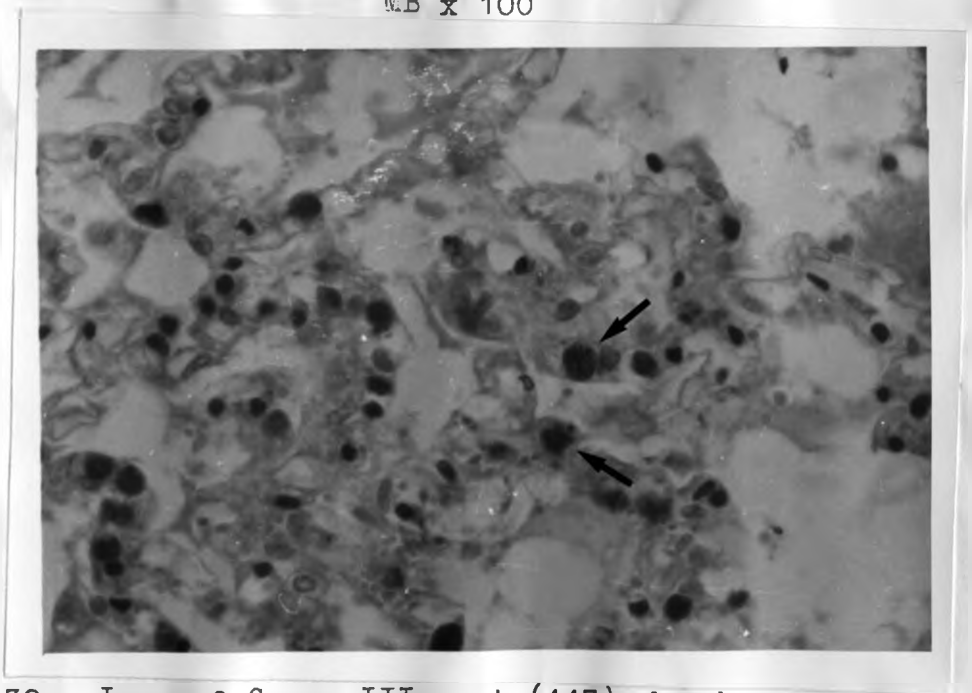


Fig. 32. Lung of Group III goat (447) showing numerous macrophages with phagocytised erythrocytes (arrow).

MB x 630

Blood.

There was marked predominance of large lymphocytes with large nuclei, rare chromatin material and large basophilic cytoplasms in Group III goats. Some lymphocytes were seen in division (Figure 33). Trypanosomes were readily observed in the blood smears of peripheral blood (Figure 34).

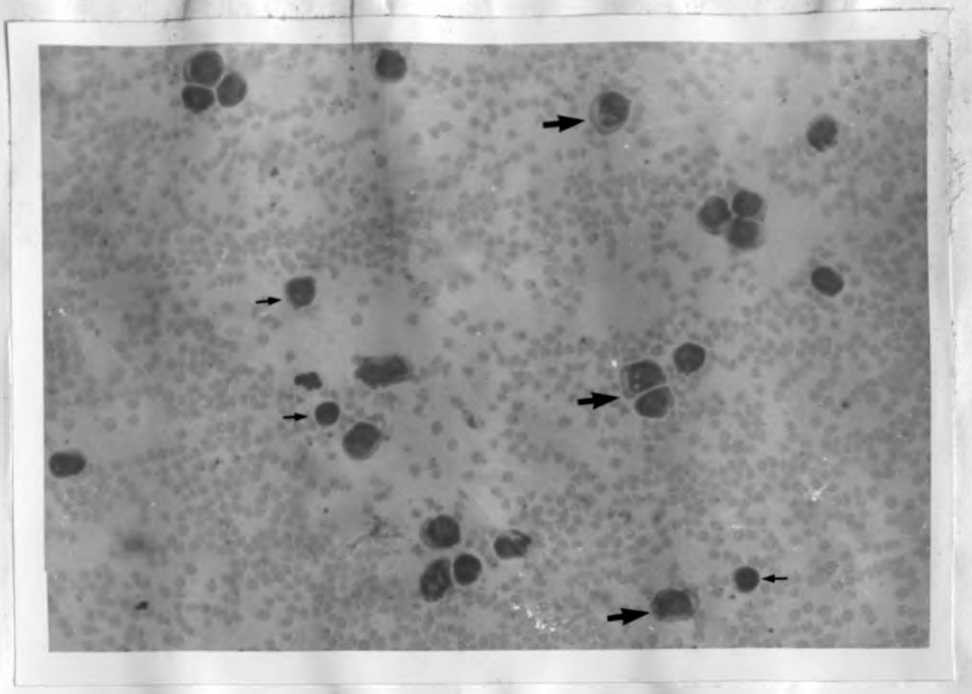


Fig. 33. Blood smear of Group III infected goat (446) showing large lymphocytes (thick arrow), with large cytoplasm and rare nuclear material, and normal lymphocytes (fine arrows).

Giemsa x 630

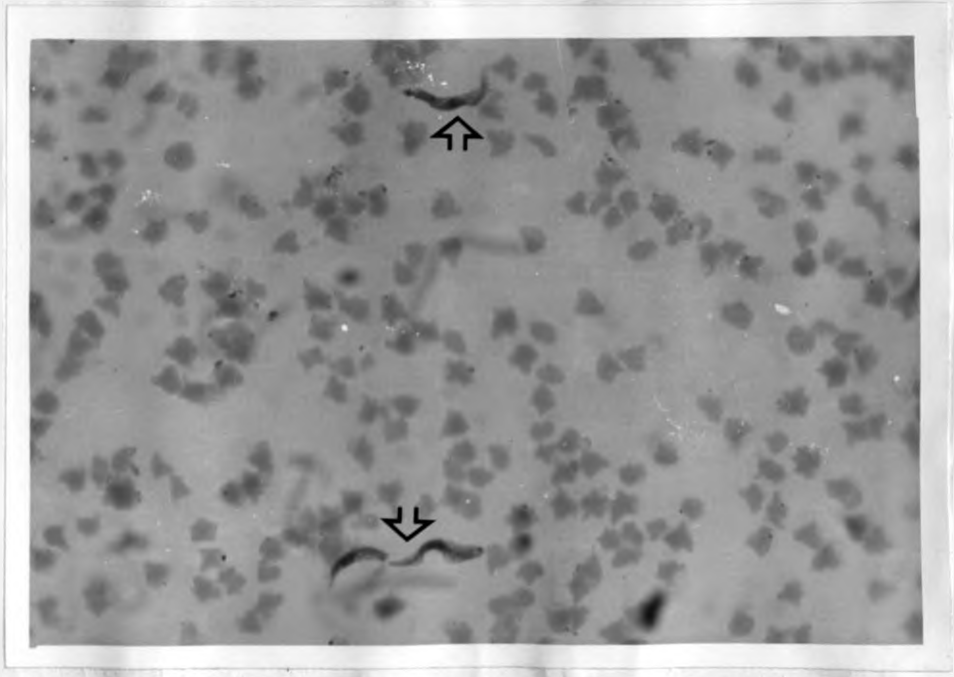


Fig. 34. Blood smear of Group III infected goat (447) showing trypanosomes (arrowed).

Giemsa x 1000

5. DISCUSSION.

Results indicate that goats infected with T. congolense (Group III) had significantly lower anti-anthrax humoral antibody response to vaccination with Bacillus anthracis vaccine 10 days PID than control vaccinated non-infected goats of Group II. The state of immuno-suppression observed in this study is in agreement with that of other workers (Scott et al., 1977; Whitelaw et al., 1979; Griffin et al., 1980; Sharpe et al., 1982 and Rurangirwa et al., 1983).

Trypanocidal therapy with Veriben on days 14 and 21 PID of Group IV infected goats enabled them to elicit a normal immune response to anthrax vaccination. Group IV goats developed anthrax antibody levels which were not significantly ($P > 0.1$) different from Group II control non-infected goats. The restoration of immune competence by trypanocidal therapy in trypanosome infected hosts is in agreement with findings of other workers (Rurangirwa et al., 1979; Whitelaw et al., 1979; Griffin et al., 1980; and Malu and Tabel, 1986).

This finding, however, differs with that of Sharpe et al., (1982) using T. congolense infected cattle. Reasons for this difference are not known although it may be associated with the trypanosome strain, extent of lymphoreticular damage at time of vaccination, levels of parasitaemia or lack of good adjuvant in the vaccine to increase the duration of the antigen stimulus into the period when immune competence was restored (Whitelaw

et al., 1979). Sharpe et al. (1982), used Friesian steers in their study which are more susceptible to T. congolense infection than the zebu cattle used by Whitelaw et al. (1979) and East African goats used by Griffin et al. (1980).

Group III infected goats developed significantly higher parasitaemia levels than Group IV infected treated goats throughout the experimental period. By day 30 PID Group IV goats were negative by blood smear examination following Veriben treatment. This finding highly suggests that patency, level and duration of parasitaemia are essential in induction and persistence of immunosuppression as earlier suggested by Scott et al. (1977), Whitelaw et al. (1979) and Tizard et al. (1980) in T. congolense infected cattle and Griffin et al. (1980)in T. congolense infected goats.

The levels of parasitaemia in infected goats were inversely correlated to the packed cell volume and red blood cell counts which are indicators of levels of parasitaemia. These finding agree with earlier work done by Naylor (1971) in T. congolense infected cattle, Kaaya et al. (1977) in T. congolense infected goats, and Mackenzie et al. (1978) in T. congolense infected sheep.

All vaccinated goats showed a rise in WBC. In Group III goats the rise may have been due to trypanosomiasis since it was mainly a lymphocytosis. The rise in WBC in Group II and Group IV goats may have been due to the anthrax vaccination since it was primarily due to both a neutrophilia and a lymphocytosis. This indicates the Group III goats had a hyporeactive immune system to anthrax vaccine. Group IV infected goats had a transient leucopenia in the early course of the disease which agrees with the findings of Losos et al. (1973) and Wellde et al. (1974) in T. congolense infected cattle.

There was no marked variation in MCV and MCHC of infected goats and the resultant anaemia was normochromic normocytic which could be due to depressed erythropoiesis and general depression of the bone marrow (Kaaya, 1975).

The total plasma proteins of infected goats was lower than that of control goats. This is in agreement with the findings reported by Fiennes (1970); Naylor (1971) and Wellde et al. (1974) in T. congolense infected cattle but differs with Kaaya (1975) in T. congolense infected goats. This observed difference could be due to strain differences

of T. congolense and that goats used by Kaaya (1975) had a more chronic type of trypanosomiasis. The observed decrease in total plasma proteins could be due to increased catabolism, tissue damage or hemodilution.

Pathological and histopathological findings were similar to those of other workers in T. congolense infected cattle (Losos et al., 1973; Kaliner, 1974), T. congolense infected goats (Kaaya, 1975) and in T. congolense and T. brucei infected laboratory animals (Murray et al., 1974; Brown and Losos, 1977; Kar et al., 1981 and Morrison et al., 1982). It is notable that the B-cell dependent areas of lymph nodes of Group III infected goats were severely depleted which agrees with the findings by Brown and Losos (1977) in T. congolense and T. brucei infected rats and Morrison et al. (1982) in T. congolense infected mice. In the lymph nodes of infected goats (Group III) there were very few germinal centres which were formed. Proliferation of some lymphocytes and an increase in mononuclear and plasma cells as reported by previous workers in T. congolense infected goats (Kaaya, 1975; Kanyari, 1981) was observed.

The spleens of Group III infected goats had severely depleted follicular areas, few germinal centres and proliferating lymphocytes. Macrophages and plasma cell numbers were increased especially in the red pulp and follicular areas. There was also hemosiderin in

macrophages of the spleen. This agrees with the findings of Murray M et al. (1974) and Roelants et al. (1979) in T. brucei infected mice and Morrison et al., (1982) in T. congolense infected mice. T. congolense infected cattle showed atrophy of lymph nodes (Kaliner, 1974) with a diffuse cortex. Murray M. et al. (1974) reported atrophy of the thymus in T. brucei infected mice as did Rurangirwa et al. (1980a) in T. congolense infected cattle. Group IV infected treated goats showed adequate population and hyperplasia of lymphoid cells in both the spleen and lymph nodes. Numerous active germinal centres were also evident indicating that trypanocidal therapy would restore immune competence as observed by Roelants et al. (1979) in T. congolense infected mice.

It is observed from the findings reported here that the spleen is the most severely affected in infected goats probably due to its ready access to trypanosome parasites. The pathology observed in these lymphoid organs could either be due to direct damage or immunopathology which could directly or indirectly contribute to the general state of immunosuppression that was observed.

The marked changes observed in the liver of infected goats, enlarged sinusoidal spaces, marked cellular infiltration, hepatic congestion and necrosis could result in hepatic failure especially in protein synthesis.

Pneumonia observed in Group III infected goats has also been reported by other workers (Hull et al., 1971 and Kanyari, 1981), in T. vivax infected mice and T. congolense infected goats respectively. Maxie et al (1979) reported high incidence of salmonellosis and abscesses in infected cattle. This observed secondary bronchopneumonias and bacterial infections may be due to trypanosome induced immunosuppression.

From the above observations, several mechanisms of immunosuppression in goats could be drawn. The findings that immunosuppression is correlated to levels of parasitaemia, drop in RBC and PCV and the fact that after Veriben treatment immune competence could be restored, a possible mechanism is antigenic competition as suggested by Goodwin et al. (1972); Musoke et al. (1981) and Nantulya et al. (1982). Antigenic competition could be mediated through various pathways, mainly at the macrophage level (Vickerman and Barry, 1982). Furthermore, there could be a physical block and poor antigen presentation and processing (Vickerman and Barry, 1982; Rurangirwa et al., 1983). From this study it is evident that the macrophage population and entire mononuclear phagocytic system is overloaded with hemosiderin, erythrophagocytosis and phagocytosis of trypanosomes and tissue debris. This could be

the physical block leading to poor antigen presentation and processing. Production of suppressor macrophages in the spleen as suggested by Nantulya et al. (1982) and the release of factors to suppress T and B-lymphocytes by macrophages (Liacopoulos and Ben Efraim, 1973) are possible mechanisms of immunosuppression. Production and secretion of interleukin-2 by T-lymphocytes could be blocked by suppressor macrophages thus mediating a state of immunosuppression (Sileghem et al., 1985).

Suppressor cells (T-lymphocytes and macrophages) generated in the spleen of infected animals (Jayawardena and Waksman, 1977; Terry et al., 1980; Grosskinsky and Askonas, 1981) could lead to immunodepression at least in infected mice. Since Veriben treated goats responded normally to the vaccine, it can be concluded that if suppressor cells play a role in immunodepression in large animals, it is not long-lived (Rurangirwa et al. 1979). It is also indeed possible that suppressor cells are generated later during infection.

A block in the maturation series of B-lymphocytes to plasma cells (antibody producing) as well as a block in antibody secretion at plasma cell level has been suggested (ILRAD, 1984). In this study, there was a marked increase in both large lymphocytes and plasma cells in infected goat The large number of plasma cells in Group III infected goats could either be specific for trypanosome antibodies

or there could be a block in anthrax antibody secretion at the plasma cell level.

The pathological findings observed in the lymphoid organs, lymphoid depletion, proliferative changes, and expansion of mononuclear phagocytic systems coupled with atrophy of thymus observed in T. congolense infected cattle (Kurangirwa et al., 1980a) and T. brucei infected mice (Murray M et al., 1974), could either lead to immunodepression or could be one of its manifestations.

Other possible mechanisms of immunodepression have been suggested including increased catabolism of immunoglobulins (Nielsen et al., 1978), polyclonal B-cell stimulation leading to clonal exhaustion (Greenwood, 1974; Hudson et al., 1976; Assoku et al., 1979), and hypocomplementaemia (Nielsen et al., 1978; Malu and Sabah, 1986). Furthermore complement 3 (C_3) plays a major role in induction of antibody synthesis and generation of B-memory cells (Nielsen et al., 1978). Vickerman and Barry (1982) suggested that low C_3 contributes to predisposition of infected animals to secondary infections.

There is high possibility that all these mechanisms could act alone or most commonly in concert to mediate the marked immunodepression observed in African trypanosomiasis.

It has been shown that trypanosomes possess mitogenic factors (Esuruoso, 1976, Mansfield et al., 1976; Assoku and Tizard 1978; Greenwood and Oduloju 1978; Hazlet and Tizard 1978; Masake et al., 1981) which alone could induce a state of immunodepression. It is indeed possible that these mitogenic factors are produced in large quantities during high parasitaemias. The mitogenic effects of trypanosomes would lead to increased activation of lymphocytes and eventual exhaustion.

Trypanosome plasma membrane fractions, complex lipids, trypanosome derived saturated fatty acids and proteins have been shown to induce immunosuppression (Assoku et al., 1979; Clayton et al., 1979; Albright and Albright 1981; Sacks et al., 1980a, and 1982; and Yamamoto et al., 1985). This indicates that the presence of living trypanosomes is not neccessary for induction of immunosuppression.

The intrinsic immunosuppressive activity of different trypanosome strains varies with parasite virulence (Sacks et al., 1980a). This is probably due to their differences in production of mitogenic factors, variable surface antigens and the extent of lymphoreticular damage.

Since in this study it was observed that Veriben treated infected Group IV goats elicited a normal humoral response, it might be advisable to consider

trypanocidal therapy in trypanosomiasis endemic areas during vaccination campaigns. Antimicrobial and antibiotic therapy in cases of trypanosomiasis should be essential due to the possible role of secondary bacterial infections in the pathogenesis of trypanosomiasis.

6. CONCLUSIONS.

1. There was marked depression of humoral immune response to anthrax vaccine in T. congolense infected goats.
2. Infected goats treated with trypanocidal drug (Veriben) elicited a normal anthrax antibody response.
3. The state of immunosuppression in infected goats was associated with high parasitaemia levels, and low PCV and RBC.
4. Infected goats showed marked pathological changes in the spleens, lymph nodes, and liver which could contribute to the state of immunodepression.
5. Infected goats which showed immunosuppression failed to show a leucocytosis after anthrax vaccination.
6. In the infected immunosuppressed goats, there was high incidence of secondary bacterial pneumonia.
7. Trypanocidal therapy in trypanosomiasis endemic areas is essential when carrying out vaccination campaigns.

8. Due to high incidence of secondary bacterial infections, use of antimicrobial and antibiotic drugs in cases of trypanosomiasis could be necessitated.

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APPENDIX I - TABLES

Table 2a

TEMPERATURES (°C) OF GROUP I CONTROL GOATS.

	Goat No.	403	406	408	MEAN
	-4	38.3	38.0	38.4	38.2
	-3	38.4	38.1	38.5	38.3
D	-2	38.2	38.9	38.3	38.5
A	-1	38.5	38.7	38.3	38.5
Y	0	38.0	38.6	38.6	38.4
S	1	38.6	38.4	38.7	38.6
	2	38.2	38.8	38.5	38.5
P	3	38.3	38.0	38.6	38.3
O	4	38.4	38.0	38.8	38.4
S	5	38.6	38.1	38.7	38.5
T	6	38.5	38.2	38.5	38.4
	7	38.6	38.1	38.6	38.4
I	8	38.5	38.3	38.5	38.4
N	9	38.8	38.4	38.4	38.5
F	10	38.0	38.7	38.8	38.5
E	11	38.0	38.1	38.5	38.2
C	12	38.2	38.5	38.4	38.4
T	13	38.4	38.7	37.7	38.3
I	14	38.7	38.3	38.3	38.4
O	15	38.5	38.2	37.8	38.2
N	16	37.8	38.7	38.3	38.3
	17	38.2	38.2	38.6	38.3
	18	37.8	38.4	37.7	38.0
	19	38.5	38.5	38.3	38.4
	20	38.1	38.8	38.7	38.5
	21	38.3	38.3	37.8	38.1

Table 2a(cont.).

TEMPERATURES (°C) OF GROUP I CONTROL GOATS.

22	38.4	38.5	37.5	38.1
23	37.7	38.3	37.8	37.9
24	38.1	38.1	38.0	38.1
25	38.2	37.7	37.5	37.8
26	38.0	38.7	37.4	38.0
27	37.8	38.5	37.6	38.0
28	37.8	38.3	37.4	37.8
29	37.8	38.4	37.2	37.8
30	37.5	38.2	37.4	37.7
31	37.8	37.8	37.2	37.6
32	38.4	38.2	37.9	38.2
33	38.2	37.9	38.6	38.2
34	38.0	38.7	37.9	38.2
35	37.9	38.6	38.1	38.2
36	38.4	38.3	38.3	38.3
37	38.3	37.9	37.7	38.0
38	38.2	38.0	37.9	38.0
39	37.9	38.4	37.6	38.0
40	38.0	38.6	38.1	38.2
41	37.8	38.1	38.2	38.0
42	37.8	38.3	37.9	38.0
43	37.8	38.5	38.0	38.1
44	38.0	38.2	38.1	38.1
45	38.4	38.4	38.6	38.5
46	38.1	38.5	38.0	38.2
47	37.8	38.5	37.0	37.8
48	38.5	37.6	37.6	37.9

Table 2a (Cont.).

TEMPERATURES (°C) OF GROUP I CONTROL GOATS.

49	38.5	38.7	38.0	38.4
50	38.2	38.8	37.8	38.3
51	38.2	38.8	37.4	38.1
52	38.3	38.5	37.5	38.0
53	38.5	38.2	37.8	38.2
54	38.1	38.5	37.4	38.0
55	38.3	38.3	37.7	38.1
56	38.6	38.7	37.8	38.4
57	38.5	38.5	38.2	38.4
58	38.4	38.4	37.8	38.2
59	37.9	38.4	38.2	38.2
60	38.0	38.5	37.9	38.1

Table 2b.

TEMPERATURE (°C) OF GROUP II CONTROL VACCINATED GOATS.

Goat No.	429	430	431	433	435	443	444	MEAN
-7	38.5	37.9	38.4	37.6	37.7	38.6	38.7	38.2
-2	38.3	38.0	38.6	37.8	37.6	38.5	38.4	38.2
-1	38.1	38.2	38.3	37.3	37.5	38.4	38.6	38.1
0	38.9	38.3	38.5	37.0	37.1	39.0	38.7	38.2
1	38.0	38.2	38.0	38.1	38.3	38.1	37.8	38.1
2	38.7	38.0	38.1	38.0	38.5	38.4	38.1	38.3
3	38.2	38.5	37.8	38.0	38.3	38.3	38.5	38,3
4	38.0	38.5	37.8	38.0	38.6	38.3	38.2	38.2
5	38.4	38.5	38.7	38.0	38.4	38.3	39.0	38.5
6	38.7	38.6	38.3	38.5	38.4	38.4	38.5	38.4
7	38.0	38.0	38.5	38.4	38.5	38.5	38.3	38.2
8	38.2	38.3	38.5	38.9	38.3	37.9	37.9	38.2
9	38.1	37.7	38.5	37.8	38.6	38.0	38.5	38.0
10	38.0	38.0	38.6	38.3	-	38.8	38.4	38.3
11	38.1	38.2	38.7	37.8	-	38.0	38.0	38.1
12	38.9	38.1	38.6	38.9	-	38.5	38.4	38.6
13	38.8	38.8	38.8	37.9	-	38.9	38.7	38.6
14	38.8	38.5	38.6	38.0	-	39.0	39.3	38.7
15	38.7	38.8	37.6	38.7	-	38.9	39.5	38.7
16	-	37.6	37.7	38.5	-	39.0	39.4	38.4
17	-	38.7	37.8	37.9	-	38.2	38.0	38.1
18	-	37.9	37.9	37.8	-	38.5	38.8	38.2
19	-	37.5	37.8	37.6	-	38.5	38.5	38.0
20	-	37.8	37.9	38.5	-	38.4	38.5	38.2
21	-	37.6	37.8	37.6	-	38.8	38.3	38.0

DAYS FOLLOWING INFECTION

Table 2b (cont.).

TEMPERATURE (°C) OF GROUP II CONTROL VACCINATED GOATS.

Goat No.	429	430	431	433	435	443	444	MEAN
46	-	38.4	38.3	38.0	-	37.9	38.0	38.1
47	-	38.4	37.9	38.3	-	38.0	38.2	38.2
48	-	38.1	37.8	38.0	-	38.1	38.2	38.0
49	-	38.0	37.7	38.0	-	38.3	38.5	38.1
50	-	38.6	37.8	38.1	-	38.2	38.3	38.2
51	-	38.3	38.1	38.2	-	38.3	38.4	38.3
52	-	38.5	38.0	38.1	-	38.4	38.6	38.3
53	-	38.4	38.3	37.9	-	38.7	38.6	38.4
54	-	38.5	38.7	37.8	-	38.4	38.5	38.4
55	-	38.2	38.0	37.9	-	38.1	38.0	38.0
56	-	38.3	38.4	38.1	-	38.3	38.1	38.2

Table 2b(cont.).

TEMPERATURE (°C) OF GROUP II CONTROL VACCINATED, GOATS.

Goat No.	429	430	431	433	435	443	444	MEAN
22	-	37.7	37.9	37.7	-	38.5	38.2	38.0
23	-	37.8	37.8	37.6	-	38.4	38.7	38.1
24	-	38.4	38.6	38.3	-	38.0	38.5	38.3
25	-	38.6	37.7	38.5	-	38.5	38.3	38.2
26	-	37.8	37.9	38.6	-	38.9	38.1	38.3
27	-	37.6	37.5	38.6	-	38.5	38.7	38.2
28	-	38.5	38.5	37.9	-	39.0	39.1	38.6
29	-	38.3	38.4	38.1	-	38.6	38.8	38.4
30	-	38.0	37.8	37.9	-	38.5	38.0	38.0
31	-	37.9	38.1	38.0	-	38.7	38.3	38.2
32	-	38.3	37.8	37.7	-	38.5	38.1	38.1
33	-	38.2	38.2	38.8	-	38.2	38.6	38.4
34	-	38.1	38.4	38.3	-	38.0	38.1	38.2
35	-	38.0	38.3	38.5	-	38.8	38.3	38.4
36	-	38.3	38.4	38.6	-	38.6	38.3	38.4
37	-	38.2	38.2	38.3	-	38.6	38.5	38.4
38	-	38.6	38.5	38.4	-	38.8	38.4	38.5
39	-	38.2	38.0	37.9	-	38.8	39.0	38.4
40	-	38.1	38.0	38.0	-	38.6	38.5	38.2
41	-	38.1	38.1	37.8	-	38.4	38.2	38.1
42	-	38.3	38.2	37.9	-	38.4	38.3	38.2
43	-	38.4	38.3	38.0	-	38.6	38.5	38.4
44	-	38.1	38.6	38.5	-	38.2	38.4	38.4
45	-	38.5	38.0	38.2	-	38.0	38.1	38.2

Table 2c.

TEMPERATURES (^{o}C) OF GROUP III INFECTED VACCINATED GOATS.

	Goat No.	426	428	436	437	445	446	447	448
	-2	38.0	38.3	37.6	38.2	38.2	39.2	38.8	39.0
D	-1	37.3	38.8	36.8	38.4	38.3	38.9	38.5	39.0
A	0	38.2	38.5	37.5	38.0	39.0	39.0	38.2	39.8
Y	1	37.0	37.5	37.0	37.2	38.4	38.3	38.2	38.8
S	2	38.3	37.0	37.8	37.5	38.5	38.5	38.9	38.5
	3	39.5	37.5	37.8	37.3	38.7	39.0	38.9	39.7
P	4	38.7	37.5	37.7	37.8	38.8	38.7	38.7	39.2
O	5	38.0	37.0	37.7	37.5	39.0	39.1	39.0	39.4
S	6	38.2	37.5	38.0	37.5	38.5	39.1	38.9	39.0
T	7	38.5	38.0	38.3	38.9	38.9	39.5	39.2	39.5
	8	38.3	38.5	38.0	38.7	37.5	38.9	38.5	38.3
I	9	37.8	37.5	38.0	38.4	38.6	38.8	38.7	39.0
N	10	38.0	36.9	38.0	38.9	39.0	38.5	38.7	39.1
F	11	37.5	36.9	39.0	38.0	39.4	38.3	38.5	38.1
E	12	38.3	37.8	39.0	38.6	39.6	39.3	39.0	39.1
C	13	38.0	38.6	39.0	38.6	39.4	39.7	39.6	39.1
T	14	38.6	39.7	37.9	39.8	39.4	38.9	40.0	40.1
I	15	37.4	39.1	36.5	38.3	39.1	39.7	39.3	39.2
O	16	37.0	38.3	-	38.1	39.1	38.7	39.9	39.5
N	17	-	37.6	-	38.4	38.7	39.1	38.0	39.7
	18	-	37.4	-	38.2	38.7	38.9	38.5	39.0
	19	-	37.0	-	38.0	39.3	39.6	39.3	39.0
	20	-	36.9	-	37.5	38.6	40.6	39.5	38.7
	21	-	-	-	37.9	38.6	38.5	38.8	39.1
	22	-	-	-	37.3	38.8	39.1	38.8	38.8

Table 2c (cont.).

TEMPERATURES (°C) OF GROUP III CONTROL GOATS.

Goat No.	426	428	436	437	445	446	447	448
23	-	-	-	37.1	38.5	38.7	38.0	38.5
24	-	-	-	37.0	38.7	39.3	38.6	39.2
25	-	-	-	36.9	38.5	39.2	38.7	38.9
26	-	-	-	36.9	38.6	39.4	38.7	39.0
27	-	-	-	36.0	38.5	38.8	38.2	38.5
28	-	-	-	-	38.2	39.0	38.5	38.7
29	-	-	-	-	38.0	39.1	38.7	38.5
30	-	-	-	-	38.4	38.0	38.0	39.0
31	-	-	-	-	38.5	38.4	38.3	38.7
32	-	-	-	-	38.5	37.3	36.7	38.3
33	-	-	-	-	38.0	37.2	35.0	38.6
34	-	-	-	-	37.6	39.3	36.7	38.9
35	-	-	-	-	36.0	39.0	35.0	38.6
36	-	-	-	-	-	39.8	-	39.0
37	-	-	-	-	-	39.2	-	39.5
38	-	-	-	-	-	38.9	-	39.2
39	-	-	-	-	-	38.7	-	39.1
40	-	-	-	-	-	38.8	-	39.3
41	-	-	-	-	-	38.3	-	39.0
42	-	-	-	-	-	37.9	-	38.6
43	-	-	-	-	-	38.0	-	38.4
44	-	-	-	-	-	39.0	-	38.3
45	-	-	-	-	-	36.9	-	38.5
46	-	-	-	-	-	-	-	38.2

Table 2c (cont.).

MEAN	GROUP III TEMPERATURES (°C)		
-2	38.3	28	38.6
-1	38.4	29	38.6
0	38.8	30	38.4
1	37.8	31	38.5
2	38.1	32	37.9
3	38.6	33	37.2
4	38.4	34	38.1
5	38.3	35	37.2
6	38.4	36	39.4
7	38.9	37	39.4
8	38.3	38	39.1
9	38.4	39	38.9
10	38.4	40	39.1
11	38.4	41	38.7
12	38.8	42	38.3
13	39.0	43	38.2
14	39.3	44	38.7
15	38.6	45	37.7
16	38.7	46	38.2
17	38.6	47	
18	38.5	48	
19	38.8	49	
20	38.6	50	
21	38.6	52	
22	38.6	53	
23	38.2	54	
24	38.6	55	
25	38.4		
26	38.7		
27	38.0		

Table 2d.

TEMPERATURES (^{o}C) OF GROUP IV GOATS.

	Goat No.	401	402	404	405	407	MEAN
	-4	38.1	38.1	38.3	38.5	38.8	38.4
D	-3	38.2	38.1	38.2	38.3	38.4	38.2
A	-2	38.1	38.0	38.0	38.2	38.3	38.1
Y	-1	38.3	38.2	38.3	38.4	38.3	38.3
S	0	38.3	38.3	38.4	38.4	38.4	38.4
	1	38.5	38.4	38.5	38.5	38.5	38.5
P	2	38.4	38.4	38.8	38.4	38.5	38.5
O	3	38.6	38.2	38.7	38.7	38.7	38.6
S	4	38.6	38.6	38.8	38.6	38.6	38.6
T	5	38.7	38.5	39.1	38.8	38.8	38.8
	6	38.8	38.5	39.0	38.8	39.0	38.8
I	7	39.4	38.4	39.2	39.0	39.0	39.0
N	8	39.6	38.7	39.5	39.0	38.9	39.1
F	9	39.8	38.8	39.5	39.0	38.9	39.2
E	10	40.0	38.9	39.7	39.4	38.9	39.4
C	11	37.8	38.2	38.7	38.5	38.4	38.3
T	12	38.3	38.7	38.2	38.6	39.5	38.7
I	13	38.0	39.0	39.5	39.2	39.7	39.1
O	14	38.7	40.5	39.4	39.0	39.3	39.4
N	15	38.2	40.5	39.4	38.7	39.2	39.2
	14	38.8	40.5	39.4	39.0	39.3	39.4
	15	38.2	40.5	39.4	38.7	39.2	39.2
	16	38.8	40.0	39.9	38.9	38.5	39.2
	17	39.0	40.5	39.3	38.5	39.5	39.4
	18	39.7	40.3	39.3	38.9	39.7	39.6

Table 2d(cont.).

TEMPERATURES (°C) OF GROUP IV GOATS.

Goat No.	401	402	404	405	407	MEAN
19	38.0	39.0	38.8	38.5	38.7	38.6
20	38.2	38.5	38.7	38.0	38.4	38.4
21	38.0	38.5	38.7	38.6	38.5	38.5
22	39.3	38.2	39.4	38.7	39.1	38.9
23	39.0	39.5	39.2	39.7	39.5	39.4
24	38.7	39.5	38.8	38.5	39.0	38.9
25	38.8	38.2	39.2	39.0	39.4	38.9
26	39.3	39.5	38.8	39.1	38.7	39.1
27	39.2	39.7	39.5	39.3	39.0	39.3
28	38.5	39.0	39.2	39.0	39.4	39.0
29	38.0	38.4	39.0	38.5	38.7	38.4
30	38.0	37.3	38.5	38.9	38.1	38.2
31	37.2	36.8	38.3	38.7	38.5	37.9
32	35.0	-	37.8	38.0	37.6	37.1
33	-	-	39.0	38.6	38.7	38.8
34	-	-	38.4	38.2	38.3	38.3
35	-	-	38.3	38.0	37.9	38.1
36	-	-	38.6	38.7	38.5	38.6
37	-	-	38.4	38.3	38.7	38.5
38	-	-	38.3	38.2	38.6	38.4
39	-	-	38.6	38.1	38.4	38.4
40	-	-	34.7	38.0	38.0	38.2
41	-	-	38.6	38.6	38.1	38.4
42	-	-	38.1	38.4	38.1	38.2
43	-	-	37.9	38.5	38.3	38.2
44	-	-	39.5	39.0	38.8	39.1
45	-	-	38.4	39.0	39.0	38.8

Table 2d (cont.).

TEMPERATURES (°C) OF GROUP IV GOATS.

Goat No.	401	402	404	405	407	MEAN
46	-	-	37.6	38.5	38.3	38.1
47	-	-	38.4	39.1	39.0	38.8
48	-	-	38.4	38.2	38.5	38.4
49	-	-	38.6	38.5	39.4	38.8
50	-	-	37.5	38.8	38.3	38.2
51	-	-	38.3	38.5	38.4	38.4
52	-	-	37.5	38.0	38.3	37.9
53	-	-	38.4	38.5	38.6	38.5
54	-	-	38.3	38.5	38.7	38.5
55	-	-	38.0	38.4	38.2	38.2
56	-	-	38.5	38.2	38.5	38.4
57	-	-	38.0	38.4	38.2	38.2
58	-	-	38.2	38.4	38.1	38.2
59	-	-	37.5	38.3	38.5	38.1
60	-	-	37.9	38.6	38.8	38.4

Table 3a. Parasitaemia (x 10^4/ul) in infected and vaccinated goats (Group III)

Goat Number	Days post infection													
	0	3	7	10	14	17	21	24	31	35	38	45	49	52
426	0	3.96	80.5	121	1170	DEAD								
428	0	0	11.0	52	1600	251	DEAD							
436	0	0	284	122	1490	DEAD								
437	0	0	182	330	983	38.6	158	37.6	DEAD					
445	0	0	0	179	39.5	35.8	69.4	62.6	330	9.8	2.29	DEAD		
446	0	0	50.8	133.	39.0	22.4	0	75.8	148	517	65.4	146	77.5	820
447	0	0	0	0	10.1	0	97.5	82.4	1140	2070	627	26	DEAD	
448	0	0	20.6	0	11.4	0	65.5	0	191	11.6	8.69	64.5	66.5	76.8
MEAN	0	0.5	91	102	668	58	78.1	119	452	652	176	79	72	448
Log_{10} Mean		3.7	6.0	6.0	6.8	5.8	5.9	6.1	6.7	6.8	6.2	5.9	5.9	6.7

Parasitaemia expressed as trypanosomes/ml of blood.

Table 3b. Parasitaemia ($x10^4$/ul) in infected, vaccinated treated goats (GROUP IV)

	Days post infection															
→ Goat Number	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52
401	0	0	18.7	26.9	0	51.6	10.3	8.66	17.1	0	-	-	-	-	-	-
402	0	0	0	13.2	0	0	0	29.3	3.12	-	-	-	-	-	-	-
404	0	0	0	6.4	0	0	0	5.8	18.4	0	0	0	0	0	0	0
405	0	0	0	9.4	6.88	20.5	7.7	3.97	7.67	0	0	0	0	0	0	0
407	0	0	34.5	59.8	0	113	126	40.5	6.58	0	0	0	0	0	0	0
MEAN	0	0	10.6	23.1	1.38	37.0	28.8	11.8	10.6	0	0	0	0	0	0	0
Log_{10} MEAN	-	-	5.0	5.4	4.1	5.6	5.5	5.2	5.0	-	-	-	-	-	-	-

Parasitaemia expressed as trypanosomes/ml of blood.

Table 4a. PCV (%) in Control goats (Group 1)

	Days post infection																		
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
403	36	35	36	37	35	33	33	30	29	30	30	30	29	27	30	22	22	20	22
406	32	30	31	30	33	30	28	27	24	27	29	30	30	29	25	25	25	24	23
408	34	33	34	34	35	33	32	31	29	31	30	30	29	26	29	23	23	22	21
MEAN	34	32.7	33.7	33.7	34.3	32.0	31.0	29.3	27.3	29.3	29.7	30.0	29.3	27.3	28.9	23.3	23.3	22.0	22.0
SE	0.94	1.19	1.19	1.66	0.54	0.82	1.25	0.98	1.36	0.98	0.27	0	0.27	0.72	1.25	0.72	0.72	0.94	0.47

Table 4b. PCV(%) in control vaccinated goats (Group II)

	Days post infection																			
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
409	36	39	36	34	35	34	33	SACRIFICED											
430	33	32	33	32	30	29	30	30	32	31	30	30	32	29	30	31	30	29	29
431	32	30	34	31	30	34	30	26	30	30	30	31	32	31	31	29	28	27	28
433	34	36	38	36	34	32	31	30	31	32	30	29	28	28	27	29	30	29	28
435	29	28	30	31	32	SACRIFICED													
443	29	33	30	30	28	27	27	25	26	27	23	24	23	24	25	25	24	25	24
444	28	30	29	30	29	27	27	24	25	27	26	25	27	23	25	26	25	26	25
MEAN	31.6	32.6	32.9	32.0	31.1	30.5	29.7	27.0	28.8	29.4	27.8	27.8	28.4	27.0	27.6	28.0	27.4	27.2	26.8
SE	1.05	1.34	1.19	0.78	0.91	1.22	0.87	1.13	1.25	0.92	1.28	2.24	1.51	1.36	1.12	0.98	1.12	0.72	0.87

Table 4c. PCV (%) in infected. vaccinated goats (Group III).

	Days post infection																		
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
426	36	35	38	34	37	32	29	DEAD											
428	36	35	36	35	35	32	26	25	DEAD										
436	33	30	32	28	28	25	22	DEAD											
437	31	32	33	28	29	26	23	22	22	20	DEAD								
445	24	26	22	28	20	20	17	12	13	11	11	11	9	DEAD					
446	28	27	28	29	27	23	21	18	16	14	12	12	11	10	11	9	DEAD		
447	30	31	31	31	29	28	25	21	19	18	16	15	15	DEAD					
448	31	30	28	29	26	25	22	17	18	20	20	19	22	18	17	17	18	20	19
MEAN	31.1	30.8	31.0	30.3	28.9	26.4	23.1	19.2	17.6	16.6	14.8	14.3	14.3	14.0	14.0	13.0	18	20	19
SE	1.33	1.09	1.67	0.93	1.74	1.38	1.19	1.69	1.34	1.59	1.78	1.56	2.48	2.83	2.12	2.83			

Table 4d. PCV (%) in infected. vaccinated. treated goats (Group IV).

	Days post infection																			
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
401	34	33	33	32	31	34	34	33	29	28	24	22	DEAD						
402	35	36	35	36	34	34	31	28	24	27	22	DEAD							
404	36	37	37	38	33	32	30	29	27	26	22	24	23	21	20	19	17	17	16
405	34	35	34	36	35	31	33	31	28	28	24	25	28	26	23	20	20	23	23
407	34	34	33	33	33	32	34	29	25	24	22	25	26	24	23	23	23	23	24
MEAN	34.6	35.0	34.4	35.0	33.2	32.6	32.4	30.0	26.6	26.6	22.8	26.5	25.7	23.7	22.0	20.7	20.0	21.0	21.0
SE	0.36	0.63	0.67	0.98	0.59	0.54	0.73	0.78	0.83	0.67	0.44	1.60	1.03	1.19	0.82	0.98	1.41	1.63	2.03

Table 5a. RBC counts ($x10^6$/ul) of control goats (Group I).

	Days post infection																			
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56	
403	26.1	25.5	21.5	28.0	22.7	19.9	16.1	20.0	15.2	15.1	16.0	15.3	15.5	14.1	12.3	12.7	17.8	11.1	16.3	
406	20.8	20.5	19.1	29.5	19.5	17.7	15.9	16.6	16.8	15.7	16.3	16.3	17.7	16.4	13.6	15.5	15.0	14.1	14.0	
408	20.6	20.5	20.2	20.0	19.3	19.2	17.8	20.5	16.7	17.7	17.2	16.3	15.9	14.9	13.2	13.5	17.5	12.2	15.3	
MEAN	22.5	22.2	20.3	25.8	20.5	18.9	16.6	19.0	16.2	16.2	16.5	16.0	16.4	15.1	13.0	13.9	16.8	12.5	15.2	
SE	1.47	1.36	0.57	2.41	0.90	0.53	0.49	1.00	0.42	0.64	0.29	0.27	0.55	0.55	0.31	0.68	0.72	0.72	0.54	

Table 5b. RBC counts ($x10^6$/ul) of control vaccinated goats (Group II).

Goat Number	Days post infection																		
	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
429	23.5	23.9	22.4	22.0	21.9	20.8	21.0	SACRIFICED											
430	19.4	19.3	19.4	19.2	19.1	19.0	19.0	19.3	20.0	20.9	21.0	20.1	19.8	19.4	19.5	18.9	18.4	18.3	17.9
431	18.0	17.6	17.5	15.8	15.8	16.9	17.2	21.0	16.0	16.8	17.8	17.5	17.4	17.6	16.9	17.0	17.2	16.9	17.1
433	20.2	20.3	23.0	21.7	20.8	20.2	20.6	21.0	20.7	21.0	20.8	20.3	20.5	21.0	20.1	19.8	19.0	18.8	18.9
435	18.4	18.4	18.5	18.1	18.0	DEAD NATURAL													
443	19.7	24.4	19.2	20.2	18.7	20.4	20.9	19.2	20.3	20.1	20.3	20.6	18.5	18.5	17.0	16.7	16.0	13.7	16.3
444	21.4	20.4	19.5	20.0	20.3	19.9	20.8	21.5	22.1	20.4	21.3	22.1	21.5	21.9	21.9	22.8	18.5	16.9	14.7
MEAN	20.1	20.6	19.9	19.6	19.2	19.5	19.9	20.4	19.8	19.8	20.2	20.1	19.5	19.7	19.1	19.0	17.8	16.9	17.0
SE	0.66	0.91	0.71	0.75	0.70	0.53	0.57	0.43	0.91	0.70	0.56	0.66	0.65	0.71	0.85	0.99	0.49	0.80	0.64

Table 5c. RBC counts ($x10^6$/ul) of infected. vaccinated goats (Group III).

	Days post infection																			
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56	
426	22.0	22.1	21.1	20.2	19.6	20.1	19.8	DEAD												
428	22.3	22.5	24.2	21.8	21.7	18.6	17.3	18.5	DEAD											
436	17.9	17.8	18.7	15.8	15.8	15.4	14.6	DEAD												
437	18.6	18.8	18.4	16.5	16.8	17.3	14.5	13.8	10.7	10.2	DEAD									
445	25.8	18.8	18.9	15.6	14.1	13.5	10.8	9.0	10.7	7.9	7.5	8.3	6.0	DEAD						
446	17.8	19.1	20.0	17.5	18.5	18.3	16.2	12.5	12.1	8.9	9.0	9.2	8.9	9.7	7.6	6.7	DEAD			
447	21.7	25.7	22.6	25.4	21.8	21.1	18.1	15.7	17.4	14.6	13.3	12.3	10.3	DEAD						
448	17.8	23.5	21.4	19.4	20.6	20.4	16.1	14.1	17.5	13.4	15.1	15.3	11.4	15.9	13.0	13.1	8.5	11.8	12.0	
MEAN	20.5	21.0	20.7	19.0	18.6	18.1	15.9	13.9	13.7	11.0	11.2	11.3	9.2	12.8	10.3	9.9				
SE	0.98	0.93	0.68	1.12	0.94	0.86	0.90	1.18	1.40	1.16	1.54	1.38	1.01	7.19	1.91	2.26				

Table 5d. RBC counts ($x10^6$/ul) of infected, vaccinated and treated goats (Group IV).

	Days post infection																		
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
401	20.2	20.5	20.4	20.5	20.3	16.8	22.2	17.2	15.0	13.8	12.4	16.1	DEAD						
402	22.0	23.0	20.1	25.1	20.4	20.8	17.8	19.9	16.4	16.9	12.8	DEAD							
404	18.4	18.0	17.9	18.0	18.3	19.9	16.0	18.4	14.5	13.8	12.0	12.3	12.9	11.1	10.5	10.2	12.9	9.3	8.4
405	22.4	19.5	20.7	26.5	24.6	17.6	15.8	20.2	16.0	14.4	13.4	14.1	18.2	13.1	11.4	12.1	16.3	13.3	12.2
407	21.6	22.0	18.0	20.5	22.4	17.5	18.2	18.8	14.8	13.0	11.8	12.5	14.2	12.4	12.1	14.5	12.1	12.1	11.9
MEAN	20.9	20.6	19.4	22.1	21.2	18.5	18.0	18.9	15.3	14.4	12.5	13.8	15.1	12.2	11.3	12.3	13.8	11.6	10.8
SE	0.65	0.79	0.54	1.42	0.96	0.69	1.03	0.48	0.33	0.60	0.26	0.76	1.30	0.48	0.38	1.02	1.05	0.97	1.00

Table 6a. MEAN CELL VOLUMES (MCV) OF GROUP I CONTROL GOATS

DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
403	13.8	13.7	16.7	13.2	15.4	16.6	20.7	15.0	19.1	19.9	18.8	19.6	19.2	24.4	17.4	12.9	18.0	13.5	19.0
406	15.4	14.6	16.2	10.2	16.9	16.9	17.6	16.3	14.3	17.3	17.8	18.4	17.7	18.4	16.2	16.7	17.0	16.4	15.0
408	16.5	16.1	16.8	17.0	18.2	17.2	18.0	15.1	17.4	17.5	17.4	18.4	17.4	22.0	17.1	13.1	18.1	13.7	18.8
MEAN	15.2	14.8	16.5	13.5	16.8	16.9	18.8	15.5	16.7	18.2	18.0	18.8	18.1	21.6	16.9	14.2	17.7	14.5	17.6
SE ±	0.64	0.57	0.14	1.61	0.66	0.14	0.79	0.34	1.39	0.68	0.34	0.33	0.45	1.42	0.29	1.01	0.29	0.76	1.06

Table 6b. MEAN CELL VOLUMES (MCV) OF GROUP II CONTROL VACCINATED GOATS.

DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
429	15.3	16.3	16.1	15.5	16.0	16.3	15.7												
430	17.1	16.5	17.6	16.7	15.7	15.3	15.8	15.5	16.0	14.8	14.3	14.9	16.2	14.9	15.4	16.4	16.3	15.8	16.2
431	18.2	17.0	19.4	19.7	19.8	20.2	17.4	12.3	18.8	17.9	16.9	17.7	18.4	17.6	18.3	17.0	15.3	16.0	16.4
433	16.7	18.2	16.6	16.6	16.3	15.8	15.1	14.3	15.0	15.2	14.9	14.3	13.7	14.8	13.4	14.6	16.8	15.4	14.8
435	15.8	15.2	16.0	17.1	17.8														
443	14.7	13.6	15.6	14.9	15.0	13.3	12.9	13.0	12.8	13.5	11.3	11.7	12.4	13.0	14.7	15.0	15.0	18.2	14.7
444	13.1	14.7	14.9	15.0	14.3	13.6	13.0	11.2	11.3	13.3	12.2	11.3	12.6	10.5	11.4	11.4	13.5	15.4	17.1
MEAN	15.8	15.9	16.6	16.5	16.4	15.8	15.0	13.3	14.8	14.9	13.9	14.0	14.7	15.8	14.6	14.9	15.4	16.2	15.8
SE ±	0.59	0.54	0.52	0.58	0.65	0.93	0.65	0.61	1.16	0.74	0.89	0.96	1.03	0.58	1.02	0.87	0.51	0.47	0.42

Table 6c. MEAN CELL VOLUME (MCV) OF GROUP III INFECTED VACCINATED GOATS.

DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52
426	16.4	15.8	18.0	16.9	18.9	16.0	14.6	DIED										
428	16.1	15.6	14.9	16.1	16.1	17.2	15.1	13.5	DIED									
436	18.4	16.8	17.2	17.8	17.8	16.2	15.1	DIED										
437	16.7	17.0	17.8	17.0	17.3	15.1	15.9	15.9	20.5	19.6	DIED							
445	9.3	13.9	11.6	18.0	14.2	14.9	15.7	13.3	12.2	13.9	14.8	13.3	15.1	DIED				
446	14.6	14.1	14.0	16.6	14.6	12.6	13.0	14.5	13.3	15.6	13.4	13.0	12.4	10.4	14.5	13.4	DIED	
447	12.9	12.1	13.7	12.2	13.3	13.3	13.8	13.4	11.0	12.3	12.1	12.2	14.6	DEAD				
448	15.2	12.8	13.1	14.9	12.6	12.3	13.7	12.1	10.3	14.9	13.3	12.4	19.3	11.4	13.1	13.0	21.2	
MEAN	15.0	14.8	15.0	16.2	15.6	14.7	14.6	13.8	13.5	15.3	13.4	12.7	15.4	10.9	13.8	13.2	21.2	
SE ±	0.92	0.60	0.79	0.62	0.75	0.59	0.34	0.48	1.64	1.09	0.48	0.22	1.25	0.35	0.49	0.14		

Table 6d. MEAN CELL VOLUME (MCV) OF GROUP IV INFECTED, VACCINATED TREATED GOATS.

DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
401	16.8	16.1	16.2	15.6	15.3	20.2	15.3	19.2	19.3	20.4	19.4	19.9	DIED						
402	15.9	15.7	17.4	14.3	16.7	16.3	17.5	14.1	14.6	16.0	17.2	DIED							
404	19.6	20.6	20.7	21.1	18.1	17.5	18.8	15.8	18.7	18.8	18.4	19.6	18.9	19.0	18.6	13.2	18.3	19.0	18.1
405	15.2	17.9	16.4	13.6	14.2	17.6	20.9	15.3	17.6	19.5	18.0	17.7	19.8	20.3	16.6	12.3	17.3	18.9	18.3
407	15.7	15.4	18.3	16.1	14.7	18.3	18.7	15.4	16.9	18.5	18.6	20.0	19.4	19.0	15.9	19.0	19.0	20.2	20.5
MEAN	16.6	17.1	17.8	16.1	15.8	18.0	18.2	16.0	17.4	18.6	18.3	19.3	19.4	19.4	17.0	19.0	18.2	19.4	18.9
SE ±	0.70	0.87	0.73	1.18	0.64	0.57	0.82	0.50	0.73	0.61	0.32	0.47	0.21	0.35	0.60	1.71	0.45	0.34	0.54

Table 7a. HAEMOGLOBIN VALUES (Hb mg/100 ml) OF GROUP II CONTROL VACCINATED GOATS.

DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
429	13.0	13.9	12.4	12.7	12.9	12.5	12.3												
430	11.6	11.7	11.1	11.0	12.0	11.9	11.8	11.9	11.8	11.6	11.5	11.2	10.8	10.1	10.3	10.6	10.2	9.9	10.3
431	11.4	11.3	11.8	11.0	11.3	11.8	11.3	12.4	10.5	10.9	10.8	10.6	10.7	10.3	10.0	10.4	10.3	10.5	10.4
433	11.6	11.8	12.6	11.8	11.6	11.4	11.5	11.8	11.6	11.7	11.8	10.9	11.0	11.3	11.2	10.7	10.3	10.6	10.7
435	10.0	9.5	11.0	11.3	11.2														
443	11.2	8.5	11.1	14.0	9.6	10.8	10.3	10.3	13.9	9.9	9.8	10.0	9.0	9.3	9.4	9.1	7.7	8.8	8.5
444	11.0	9.0	10.9	10.9	9.5	10.3	9.8	9.7	9.2	9.6	9.5	9.6	9.7	7.5	9.2	9.5	8.3	8.7	8.4
MEAN	11.4	10.8	11.6	11.2	11.3	11.2	11.2	11.4	10.7	10.7	10.5	10.2	9.7	10.0	10.1				
SE ±	0.31	0.67	0.25	0.40	0.43	0.36	0.36	0.46	0.70	0.38	0.41	0.26	0.34	0.57	0.32	0.29			

Table 7b.

HAEMOGLOBIN VALUES (Hb mg/100 ml) OF GROUP III INFECTED GOATS.

DAYS POST-INCTION

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
426	13.0	13.2	13.3	12.0	12.9	12.7	12.3	-											
428	12.0	12.5	12.8	12.1	12.6	10.8	10.2	10.7											
436	10.8	10.2	10.6	9.0	9.3	9.0	8.9	-											
437	11.6	11.7	11.3	10.5	11.1	10.1	9.3	9.4	7.9	7.6									
445	9.1	9.8	8.1	8.8	7.2	6.4	7.1	4.8	4.7	4.2	4.1	4.7	3.6	-					
446	10.5	11.3	10.5	10.6	9.7	9.4	7.9	7.1	6.4	5.1	4.5	4.8	4.3	3.9	4.2	3.5	-		
447	11.0	12.6	11.5	11.8	10.4	9.6	9.4	7.6	7.1	6.7	6.2	5.8	5.4	-					
448	8.8	10.7	10.3	9.5	9.4	9.1	8.1	6.2	6.4	7.1	7.8	4.7	5.7	6.8	6.3	6.0	5.6	6.6	6.1
MEAN	10.9	11.4	11.1	10.5	10.3	9.6	9.2	7.6	6.5	6.1	5.7	5.0	4.8	5.4	5.3	4.8			
SE ±	0.47	0.39	0.53	0.44	0.62	0.59	0.53	0.80	0.47	0.57	0.74	0.23	0.42	1.03	0.74	0.88	5.6	6.6	6.1

Table 8a. MEAN CORPUSCULAR HAEMOGLOBIN-CONCENTRATION (MCHC) OF GROUP II CONTROL VACCINATED GOATS, DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
429	36.1	35.6	34.6	34.4	37.3	36.9	36.8	37.3	SACRIFICED										
430	35.2	36.6	34.4	33.6	34.4	40.0	41.0	39.3	39.7	36.9	37.4	38.3	37.3	33.8	34.8	34.3	34.2	34.0	34.1
431	35.6	37.7	34.1	34.7	35.5	37.7	34.7	37.7	47.7	35.0	36.3	36.0	34.2	33.4	33.2	32.3	35.9	36.8	38.8
433	34.1	32.8	32.4	33.2	32.8	34.1	35.6	37.1	39.3	37.4	36.6	38.1	37.6	39.2	36.5	41.5	36.9	34.3	36.6
435	34.5	33.9	35.0	36.7	36.5	35.0	SACRIFICED												
443	38.6	25.8	37.0	46.7	34.3	40.0	38.1	41.2	53.5	36.7	42.6	41.7	39.1	38.8	37.6	36.4	32,1	35.2	35.4
444	39.3	30.0	37.6	36.3	32.8	38.1	36.3	40.4	36.8	35.6	41.3	38.4	35.9	32.6	36.8	36.5	33.2	33.5	33.6
MEAN	36.2	33.2	35.0	36.5	34.8	37.4	37.0	38.8	43.4	36.3	38.8	38.5	36.8	35.6	35.8	36.2	34.5	34.8	35.7
SE ±	0.70	1.45	0.62	1.64	0.61	0.79	0.83	0.65	2.79	0.40	1.16	0.82	0.74	1.27	0.71	1.37	0.78	0.52	0.84

Table 8b.

MEAN CORPUSCULAR HAEMOGROBIN-CONCENTRATION (MCHC) OF GROUP III INFECTED, VACCINATED GOATS. DAYS POST-INFECTION.

Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
426	36.1	37.7	35.0	35.3	34.9	39.7	42.4	DIED											
428	33.3	35.7	35.6	34.6	36.0	33.8	39.2	42.8	DIED										
436	32.7	34.0	33.1	32.1	33.2	36.0	40.5	DIED											
437	37.4	36.6	34.2	37.5	38.3	38.8	40.4	42.7	35.9	38.0	DIED								
445	37.9	37.7	36.8	31.4	22.7	32.0	41.8	40.0	36.2	38.1	37.3	42.7	40.0	DIED					
446	40.4	41.8	37.5	36.6	35.9	40.9	37.6	39.4	40.0	36.4	37.5	40.0	39.1	39.0	38.2	38.9	DIED		
447	39.3	40.6	37.1	35.8	35.9	34.3	37.6	36.2	37.2	37.2	38.8	38.7	36.0	DIED					
448	32.6	36.7	36.8	32.8	36.1	36.4	36.8	36.5	35.6	35.5	39.0	24.7	22.7	37.8	37.1	35.3	31.1	33.0	32.1
MEAN	36.2	37.5	35.8	34.5	34.1	36.5	39.5	39.6	37.0	37.0	38.2	35.1	34.5	38.4	37.7	37.1	31.1	33.0	32.1
SE ±	1.01	0.86	0.52	0.73	1.60	1.03	0.69	1.07	0.72	0.44	0.38	3.78	3.47	0.42	0.39	1.27			

Table 9a. WBC ($x10^3$/ul) counts in control goats (Group I).

Goat Number	Days post infection																		
	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
403	14.8	16.0	15.9	13.3	11.4	10.89	10.9	11.4	12.27	11.58	11.35	9.42	9.97	11.2	9.14	10.5	11.1	9.77	9.9
406	15.2	17.0	17.0	12.3	15.6	14.4	12.3	11.8	12.6	12.25	7.2	12.17	12.77	19.6	11.84	11.75	10.9	11.37	10.7
408	11.2	10.9	11.2	10.6	10.5	8.89	5.6	7.1	6.67	6.88	14.53	6.24	7.24	7.3	8.71	6.70	7.0	7.47	7.2
MEAN	13.7	14.6	14.7	12.1	12.5	11.4	9.6	10.1	10.5	10.2	11.0	9.28	10.0	12.7	9.90	9.65	9.67	9.54	9.3
SE	1.04	1.54	1.45	0.64	1.28	1.31	1.67	1.23	1.57	1.38	1.73	1.40	1.30	2.96	0.80	1.24	1.09	0.92	0.86

Table 9b. WBC ($x10^3$/ul) counts in control vaccinated goats (Group II).

	Days post infection																		
Goat Number	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
429	20.8	23.9	29.0	24.8	25.0	25.5	23.6	SACRIFICED											
430	24.0	26.2	18.0	15.0	17.5	19.5	18.7	17.5	20.0	22.0	20.0	16.2	15.0	16.1	14.9	15.5	11.9	12.7	12.1
431	9.8	8.75	13.1	10.8	8.21	7.32	9.47	18.7	14.8	15.6	17.0	14.3	13.8	14.6	12.9	13.2	12.6	12.6	13.3
433	13.8	12.3	15.5	20.0	19.7	16.9	15.9	16.4	17.8	18.0	22.0	18.0	14.1	15.6	12.7	14.9	15.6	13.8	16.9
436	30.0	31.0	26.0	24.0	26.0	SACRIFICED													
443	10.2	13.3	9.48	8.56	11.0	8.73	12.2	12.7	9.2	13.1	18.7	12.4	11.7	16.1	10.8	13.1	6.3	6.2	7.2
444	12.0	11.6	12.7	15.3	14.9	13.6	21.4	9.27	15.00	16.3	30.7	14.2	13.2	12.8	11.7	12.5	10.4	7.2	10.0
MEAN	17.2	18.5	17.6	16.9	17.5	15.3	16.9	14.9	15.4	17.0	21.7	15.0	13.6	15.0	12.6	13.8	11.4	10.5	11.9
SE	2.73	3.04	2.54	2.19	2.34	2.54	2.01	1.55	1.62	1.32	2.15	0.86	0.49	0.56	0.61	0.51	1.36	1.41	1.45

Table 9c. WBC ($x10^3$/ul) counts in infected, vaccinated goats (Group III)

Goat Number	Days post infection: -14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
426	12.1	10.8	12.1	10.9	9.31	11.5	16.7	DEAD											
428	29.0	21.1	29.0	25.2	32.0	30.9	31.0	37.6	DEAD										
436	23.6	14.6	23.6	17.9	16.9	11.9	12.6	DEAD											
437	23.3	22.8	23.3	27.2	29.3	35.9	29.5	34.0	36.0	40.0	DEAD								
445	6.66	11.5	7.19	7.83	5.54	6.28	4.69	7.16	6.94	8.76	10.7	9.8	6.66	DEAD					
446	10.9	10.7	8.97	12.8	9.0	8.4	7.74	11.2	14.4	13.4	10.3	8.86	6.54	19.6	7.1	6.36	DEAD		
447	16.5	16.7	13.4	13.6	12.8	10.9	14.2	18.7	19.5	21.7	26.2 (25.2)	23.1	15.1	DEAD					
448	22.3	23.0	22.2	25.9	18.0	13.9	11.7	17.0	13.1	15.2	22.8	17.8	11.3	37.4	21.3	14.7	16.0	12.9	16.7
MEAN	17.9	16.4	17.5	17.7	16.6	16.2	16.0	20.9	18.0	14.8	17.3	14.9	9.9	-	14.2	10.5			
SE	2.52	1.76	2.65	2.49	3.18	3.62	3.16	4.57	4.41	2.32	3.40	2.94	1.78	-	5.02	2.95			

Table 9b. WBC ($x10^3$/ul) counts in infected, vaccinated, treated goats (Group IV).

Goat Number	Days post infection																		
	-14	-7	0	3	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
401	10.0	9.3	11.3	10.0	10.2	10.9	9.4	10.2	7.37	8.66	15.19	26.22	DEAD						
402	16.8	17.9	19.0	17.0	17.6	14.43	9.4	7.4	7.16	10.75	13.05	DEAD							
404	18.6	20.8	20.0	16.2	14.7	12.84	7.6	8.9	12.19	12.17	13.6	19.3	19.9	18.9	14.85	12.45	11.3	11.62	9.8
405	14.9	16.1	15.2	12.7	12.5	10.85	11.0	11.4	7.55	7.93	15.65	17.03	12.18	13.9	10.41	9.25	8.9	9.51	9.5
407	13.9	14.1	14.67	10.5	11.6	11.29	9.4	7.1	5.2	5.27	9.97	18.75	19.08	18.5	14.49	14.71	10.8	10.99	13.7
MEAN	14.8	15.6	16.0	13.3	13.3	12.1	9.4	9.0	7.89	8.96	13.5	20.3	17.1	17.1	13.3	12.1	10.3	10.7	10.9
SE	1.30	1.73	1.41	1.28	1.16	0.62	0.48	0.73	1.03	1.06	0.89	1.75	2.00	1.31	1.16	1.29	0.60	0.51	0.86

Table 10a.

DIFFERENTIAL LEUCOCYTE COUNTS OF CONTROL GOATS (GROUP I).

	Goat No.	TWBC	L	E	N	M	B
			71%	1%	25%	3%	0%
	403	15900	(11289)	(159)	(3975)	(477)	0
			70%	0%	28%	2%	0%
Day 0	406	17000	(11900)	0%	(4760)	(340)	0
			63%	2%	33%	2%	0%
	408	11200	(7056)	(224)	(3696)	(224)	0
			68%	1%	28.7%	2.3%	0%
	MEAN	14700	(10082)	(128)	(4143)	(347)	0
			70%	0%	24%	6%	0%
	403	11400	(7980)	(0)	(2736)	(684)	0
			68%	0%	30%	2%	0%
Day +7	406	15600	(10608)	(0)	(4680)	(312)	0
			54%	0%	46%	0%	0%
	408	10500	(5670)	(0)	(4830)	0	0
			64%	0%	33.3	2.7%	0%
	MEAN	12500	(8086)	(0)	(4082)	(3327)	0
			87%	0%	12%	1%	0%
	403	10890	(9474)	(0)	(1307)	(109)	0
			75%	0%	24%	1%	0%
Day 10	406	14380	(10785)	(0)	(3451)	(144)	0
			63%	1%	34%	2%	0%
	408	8890	(4712)	(89)	(3023)	(178)	0
			75%	0.3%	23.3%	17%	0%
	MEAN	11387	(8324)	(30)	(2594)	(144)	0

Table 10a (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF CONTROL GOATS (GPI).

	Goat No.	TWBC	L	E	N	M	B
			75%	0%	24%	1%	0%
	403	10900	(8175)	(10)	(2616)	(109)	0
			58%	4%	36%	2%	0%
Day 14	406	12300	(7134)	(492)	(4428)	(246)	0
			77%	0%	22%	1%	0%
	408	5600	(4312)	(0)	(1232)	(56)	0
			70%	1.3%	27.3%	1.4%	0%
	MEAN	9600	(6540)	(174)	(2759)	(137)	0
			76%	0%	23%	1%	0%
	403	12270	(9325)	(0)	(2822)	(123)	0
			47%	0%	53%	0%	0%
Day 21	406	12600	(5922)	(0)	(6678)	(0)	0
			67%	0%	32%	1%	0%
	408	6670	(4469)	(0)	(2134)	(67)	0
			63.3%	0%	36%	0.7%	0%
	MEAN	10513	(6572)	(0)	(3878)	(63)	0
			73%	0%	27%	0%	0%
	403	11580	(8453)	(0)	(3127)	(0)	0
			54%	1%	45%	0%	0%
Day 24	406	12250	(6615)	(123)	(5512)	(0)	0
			70%	0%	30%	0%	0%
	408	6880	(4816)	(0)	(2064)	(0)	0
			65.7%	0.3%	34%	0%	0
	MEAN	10237	(6628)	(41)	(3568)	(0)	0

Table 10a (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF CONTROL GOATS (GPI).

	Goat No.	TWBC	L	E	N	M	B
			70%	0%	30%	0%	0%
	403	11350	(7945)	(0)	(3405)	(0)	0
			57%	0%	42%	1%	0%
Day 28	406	7200	(4104)	(0)	(3024)	(72)	0
			77%	0%	21%	2%	0%
	408	14530	(11188)	(0)	(3051)	(291)	0
			68%	0%	31%	1%	0%
	MEAN	11027	(7746)	(0)	(3160)	(121)	0
			77%	1%	22%	0%	0%
	403	9420	(7253)	(94)	(2073)	(0)	0
			64%	0%	35%	1%	0%
Day 31	406	12170	(7789)	(0)	(4259)	(122)	0
			75%	0%	24%	1%	0%
	408	6240	(4680)	(0)	(1498)	(62)	0
			72%	0.3%	27%	0.7%	0%
	MEAN	9277	(6574)	(31)	(2610)	(62)	0
			72%	0%	27%	1%	0%
	403	9970	(7178)	(0)	(2692)	(100)	0
			60%	0%	37%	3%	0%
Day 35	406	12770	(7662)	(0)	(4725)	(383)	0
			52%	1%	47%	0%	0%
	408	7240	(3765)	(72)	(3402)	(0)	0
			61.3%	0.3%	37%	1.4%	0%
	MEAN	9993	(6202)	(24)	(3607)	(0)	0

Table 10a (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF CONTROL GOATS (GPI).

	Goat No.	TWBC	L	E	N	M	B
			53%	0%	44%	3%	0%
	403	11200	(5936)	(0)	(4928)	(336)	0
			45%	0%	54%	1%	0%
Day 38	406	19600	(8820)	(0)	(10584)	(196)	0
			75%	0%	25%	0%	0%
	408	7300	(5475)	(0)	(1825)	(0)	0
			57.7%	0%	41%	1.3%	0%
	MEAN	12700	(6744)	(0)	(5779)	(177)	0
			70%	0%	30%	0%	0%
	403	9140	(6398)	(0)	(2742)	(0)	0
			66%	2%	32%	0%	0%
Day 42	406	11840	(7814)	(237)	(3789)	(0)	0
			70%	0%	30%	0%	0%
	408	8710	(6097)	(0)	(2613)	(0)	(0)
			68.7%	0.7%	30.6%	0%	0%
	MEAN	9897	(6770)	(79)	(3048)	(0)	(0)
			71%	1%	28%	0%	0%
	403	12450	(8840)	(124)	(3486)	(0)	(0)
			63%	0%	36%	1%	0%
Day 45	406	9250	(5828)	(0)	(3330)	(92)	(0)
			65%	2%	31%	2%	0%
	408	14710	(9562)	(294)	(4560)	(294)	(0)
			66.3%	1%	31.7%	1%	0%
	MEAN	12137	(8077)	(139)	(3792)	(129)	(0)

Table 10a (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF CONTROL GOATS (GPI).

	Goat No.	TWBC	L	E	N	M	B
			67%	0%	32%	1%	0%
	403	11300	(7571)	(0)	(3616)	(113)	(0)
			66%	2%	31%	1%	0%
Day 49	406	8900	(5874)	(178)	(2759)	(89)	(0)
			44%	0%	54%	2%	0%
	408	10800	(4752)	(0)	(5832)	(216)	(0)
			59%	0.7%	39%	1.3%	0%
	MEAN	10333	(6066)	(106)	(4069)	(139)	(0)
			71%	1%	27%	1%	0%
Day 52	403	11620	(8250)	(116)	(3138)	(116)	(0)
			44%	0%	54%	2%	0%
	406	9510	(4185)	(0)	(5135)	(190)	(0)
			57%	0%	43%	0%	0%
	408	10990	(6264)	(0)	(4726)	(0)	(0)
			57.3%	0.3%	41.3%	1%	0%
	MEAN	10707	(6233)	(39)	(4333)	(102)	(0)
			62%	1%	36%	1%	0%
Day 56	403	9800	(6076)	(98)	(3528)	(98)	(0)
			52%	2%	45%	1%	0%
	406	9500	(4940)	(190)	(4275)	(95)	(0)
			54%	0%	46%	0%	0%
	408	13700	(7398)	(0)	(6302)	(0)	(0)
			56%	1%	42.3%	0.7%	0%
	MEAN	11000	(6138)	(96)	(4702)	(64)	(0)

Table 10b.

DIFFERENTIAL LEUCOCYTE COUNTS OF CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
			54%	0%	45%	1%	0%
Day 0	429	29000	(15600)	(0)	(13050)	(290)	(0)
			61%	1%	38%	0%	0%
	430	18000	(10980)	(180)	(6840)	(0)	(0)
			46%	2%	49%	3%	0%
	431	13100	(6026)	(262)	(6419)	(393)	(0)
			39%	0%	61%	0%	0%
	433	15500	(6045)	(0)	(9455)	(0)	(0)
			61%	0%	38%	1%	0%
	436	26000	(15860)	(0)	(9880)	(260)	(0)
			65%	0%	33%	2%	0%
	443	9480	(6162)	(0)	(3128)	(190)	(0)
			48%	0%	50%	2%	0%
	444	12700	(6096)	(0)	(6350)	(254)	(0)
			53.4%	0.4%	44.9%	0.3%	0%
	MEAN	17683	(9538)	(63)	(7874)	(198)	(0)
			56%	0%	43%	1%	0%
Day +7	429	25000	(14000)	(0)	(10750)	(250)	(0)
			65%	1%	34%	0%	0%
	430	17500	(11375)	(175)	(5950)	(0)	(0)
			52%	2%	46%	0%	0%
	431	8210	(4269)	(164)	(3777)	(0)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
			45%	0%	55%	0%	0%
	433	19700	(8865)	(0)	(10835)	(0)	(0)
			55%	0%	44%	1%	0%
	435	26000	(14300)	(0)	(11440)	(260)	(0)
			48%	1%	51%	0%	0%
	443	11000	(5280)	(110)	(5610)	(0)	(0)
			55%	0%	43%	2%	0%
	444	14900	(8195)	(0)	(6407)	(298)	(0)
			53.7%	0.6%	45.1%	0.6%	0%
	MEAN	17472	(9469)	(64)	(7824)	(115)	(0)
			54%	0%	45%	1%	0%
Day -10	429	25500	(13770)	(0)	(11475)	(255)	(0)
			60%	1%	39%	0%	0%
	430	19500	(11700)	(195)	(7605)	(0)	(0)
			52%	0%	47%	1%	0%
	431	7320	(3806)	(0)	(3441)	(73)	(0)
			45%	0%	54%	1%	0%
	433	16900	(7605)	(0)	(9126)	(169)	(0)
	435	-	-	-	-	-	-
			57%	0%	42%	1%	0%
	443	8730	(4976)	(0)	(3667)	(87)	(0)
			47%	2%	50%	1%	0%
	444	13600	(6392)	(272)	(6800)	(136)	(0)
			52.7%	0.5%	46%	0.8%	0%
	MEAN	15258	(8042)	(78)	(7019)	(120)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
			51%	0%	48%	1%	0%
Day +14	429	23600	(12036)	(0)	(11328)	(236)	(0)
			58%	1%	41%	0%	0%
	430	18700	(10846)	(187)	(7667)	(0)	(0)
			50%	1%	49%	0%	0%
	431	9470	(4735)	(95)	(4640)	(0)	(0)
			43%	0%	57%	0%	0%
	433	15900	(6837)	(0)	(9063)	(0)	(0)
	435	-	-	-	-	-	-
			47%	0%	52%	1%	0%
	443	12200	(5734)	(0)	(6344)	(122)	(0)
			49%	0%	51%	0%	0%
	444	21400	(10486)	(0)	(10914)	(0)	(0)
			51.7%	0.3%	48.7%	0.3%	0%
	MEAN	16878	(8446)	(47)	(8326)	(59)	(0)
Day +17	429	-	-	-	-	-	-
			56%	1%	43%	0%	0%
	430	17500	(9800)	(175)	(7525)	(0)	(0)
			49%	0%	50%	1%	0%
	431	18700	(9163)	(0)	(9350)	(187)	(0)
			46%	0%	54%	0%	0%
	433	16400	(7544)	(0)	(8856)	(0)	(0)
	435	-	-	-	-	-	-

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L 50%	E 1%	N 48%	M 1%	B 0%
	443	12700	(6350)	(127)	(6096)	(127)	(0)
			47%	0%	53%	0%	0%
	444	9270	(4357)	(0)	(4913)	(0)	(0)
			49.6%	0.4%	49.6%	0.4%	0%
	MEAN	14914	(7443)	(50)	(7348)	(63)	(0)
Day +21	429	-	-	-	-	-	-
			55%	2%	42%	1%	0%
	430	20000	(11000)	(400)	(8400)	(200)	(0)
			48%	0%	51%	1%	0%
	431	14800	(7104)	(0)	(7548)	(148)	(0)
			45%	0%	55%	0%	0%
	433	17800	(8010)	(0)	(9790)	(0)	(0)
	435	-	-	-	-	-	-
			56%	0%	44%	0%	0%
	443	9200	(5152)	(0)	(4048)	(0)	(0)
			47%	1%	52%	0%	0%
	444	15000	(7050)	(150)	(7800)	(0)	(0)
			51.2%	0.6%	47.8%	0.4%	0%
	MEAN	15360	(7663)	(110)	(7517)	(70)	(0)
Day + 24	429	-	-	-	-	-	-
			52%	1%	47%	0%	0%
	430	22000	(11440)	(220)	(10340)	(0)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
	431	15600	46% (7176)	0% (0)	34% (8424)	0% (0)	0% (0)
			44%	0%	56%	0%	0%
	433	18000	(7920)	(0)	(10080)	(0)	(0)
	435	-	-	-	-	-	-
			42%	0%	56%	2%	0%
	443	13100	(5502)	(0)	(7336)	(262)	(0)
			59%	0%	41%	0%	0%
	444	16300	(9617)	(0)	(6683)	(0)	(0)
			48.6%	0.2%	50.8%	0.4%	0%
	MEAN	17000	(8331)	(44)	(8573)	(52)	(0)
Day +28	429	-	-	-	-	-	-
			50%	1%	49%	0%	0%
	430	20000	(10000)	(200)	(9800)	(0)	(0)
			49%	2%	48%	1%	0%
	431	17000	(8330)	(340)	(8160)	(170)	(0)
			46%	0%	54%	0%	0%
	433	22000	(10120)	(0)	(11880)	(0)	(0)
	435	-	-	-	-	-	-
			45%	0%	54%	1%	0%
	443	18700	(8415)	(0)	(10098)	(187)	(0)
			32%	2%	65%	0%	0%
	444	30700	(19648)	(614)	(19955)	(0)	(0)
			46.5%	1%	52.0%	0.4%	0%
	MEAN	21680	(11302)	(231)	(11979)	(71)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
Day +31	429	-	-	-	-	-	-
			54%	0%	44%	2%	0%
	430	16200	(8748)	(0)	(7128)	(324)	(0)
			52%	1%	47%	0%	0%
	431	14300	(7436)	(143)	(6721)	(0)	(0)
			50%	0%	50%	0%	0%
	433	18000	(9000)	(0)	(9000)	(0)	(0)
	435	-	-	-	-	-	-
			45%	3%	51%	1%	0%
	443	12400	(5580)	(372)	(6324)	(124)	(0)
			54%	1%	45%	0%	0%
	444	14200	(7668)	(142)	(6390)	(0)	(0)
			51%	1%	47.4%	0.6%	0%
	MEAN	15000	7686	131	7113	90	0
Day +35	429	-	-	-	-	-	-
			56%	1%	43%	0%	0%
	430	15000	(8400)	(150)	(6450)	(0)	(0)
			49%	0%	51%	0%	0%
	431	13800	(6762)	(0)	(7038)	(0)	(0)
			53%	1%	46%	0%	0%
	433	14100	(7473)	(141)	(6486)	(0)	(0)
	435	-	-	-	-	-	-
			40%	0%	60%	0%	0%
	443	11700	(4680)	(0)	(7020)	(0)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
			58%	1%	41%	0%	0%
	444	13200	(7656)	(132)	(5412)	(0)	(0)
	MEAN	13600	57.2% 6994	0.6% 65	48.2% 6481	0% 0	0% 0
Day +38	429	-	-	-	-	-	-
			54%	0%	45%	1%	0%
	430	16100	(8694)	(0)	(7245)	(161)	(0)
			48%	0%	52%	0%	0%
	431	14600	(7008)	(0)	(7592)	(0)	(0)
			46%	2%	52%	0%	0%
	433	15600	(7176)	(312)	(8112)	(0)	(0)
	435	-	-	-	-	-	-
			42%	1%	56%	1%	0%
	443	16100	(6762)	(161)	(9016)	(161)	(0)
			52%	1%	47%	0%	0%
	444	12800	(6656)	(128)	(6016)	(0)	(0)
	MEAN	15000	7259	120	7596	644	0
Day +42	429	-	-	-	-	-	-
			50%	0%	50%	0%	0%
	430	14900	(7450)	(0)	(7450)	(0)	(0)
			47%	0%	53%	0%	0%
	431	12900	(6063)	(0)	(6837)	(0)	(0)
			55%	1%	44%	0%	0%
	433	12700	(6985)	(127)	(5588)	(0)	(0)
	435	-	-	-	-	-	-

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
			47%	1%	50%	2%	0%
	443	10800	(5076)	(108)	(5400)	(216)	(0)
			54%	3%	43%	0%	0%
	444	11700	(6318)	(351)	(5031)	(0)	(0)
			50.6%	1%	48%	0.4%	0%
	MEAN	12600	6378	117	6061	43	0
	429	-	-	-	-	-	-
			51%	1%	47%	1%	0%
Day +45	430	15500	(7905)	(155)	(7285)	(155)	(0)
			56%	0%	44%	0%	0%
	431	13200	(7392)	(0)	(5808)	(0)	(0)
			55%	1%	44%	0%	0%
	433	14900	(8195)	(149)	(6556)	(0)	(0)
	435	-	-	-	-	-	-
			53%	0%	47%	0%	0%
	443	13100	(6943)	(0)	(6157)	(0)	(0)
			51%	1%	46%	2%	0%
	444	12500	(6375)	(125)	(5750)	(250)	(0)
			53.2%	0.6%	45.6%	0.6%	0%
	MEAN	13800	7362	66	6837	81	0
Day +49	429	-	-	-	-	-	-
			49%	0%	50%	1%	0%
	430	11900	(5831)	(0)	(5950)	(119)	(0)
			48%	1%	51%	0%	0%
	431	12600	(6048)	(126)	(6426)	(0)	(0)
			53%	0%	46%	1%	0%
	433	15600	(8268)	(0)	(7176)	(156)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL VACCINATED GOATS (GROUP II).

	Goat No.	TWBC	L	E	N	M	B
	435	-	-	-	-	-	-
			50%	0%	48%	2%	0%
	443	6300	(3150)	(0)	(3024)	(126)	(0)
			58%	0%	52%	0%	0%
	444	10400	(6032)	(0)	(5408)	(0)	(0)
			49.6%	0.2%	49.4%	0.8%	0%
	MEAN	11400	5866	25	5597	80	0
Day +52	429	-	-	-	-	-	-
			46%	0%	58%	1%	0%
	430	12700	(5842)	(0)	(6731)	(127)	(0)
			47%	1%	52%	0%	0%
	431	12600	(5922)	(126)	(6552)	(0)	(0)
			52%	0%	48%	0%	0%
	433	13800	(7176)	(0)	(6624)	(0)	(0)
	435	-	-	-	-	-	-
			56%	0%	43%	1%	0%
	443	6200	(3472)	(0)	(2666)	(62)	(0)
			59%	3%	38%	1%	0%
	444	7200	(4248)	(216)	(2736)	(72)	(0)
			57.4%	0.8%	47.4%	0.6%	0%
	MEAN	10500	5332	68	5062	52	0
Day +56	429	-	-	-	-	-	-
			50%	1%	49%	0%	0%
	430	12100	(6050)	(121)	(5929)	(0)	(0)
			56%	2%	42%	0%	0%
	431	13300	(7448)	(266)	(5586)	(0)	(0)

Table 10b (cont).

DIFFERENTIAL LEUCOCYTE COUNTS CONTROL

VACCINATED GOATS (GROUP II).

Goat No.	TWBC	L	E	N	M	B
		48%	0%	52%	0%	0%
433	16900	(8112)	(0)	(8788)	(0)	(0)
435	-	-	-	-	-	-
		52%	0%	48%	0%	0%
443	7200	(3744)	(0)	(3456)	(0)	(0)
		60%	0%	40%	0%	0%
444	10000	(6000)	(0)	(4000)	(0)	(0)
		53.2%	0.6%	46.2%	0%	0%
MEAN	11900	6339	77	5552	0	0

Table 10c.

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			57%	3%	40%	0%	0%
	426	12100	(6897)	(363)	(4840)	(0)	(0)
			61%	0%	39%	0%	0%
	426	29000	(17690)	(0)	(11310)	(0)	(0)
			60%	1%	37%	0%	0%
	436	23600	(14632)	(236)	(8732)	(0)	(0)
			68%	7%	30%	0%	0%
Day 0	437	23300	(15844)	(466)	(13980)	(0)	(0)
			52%	0%	48%	0%	0%
	445	7190	(3739)	(0)	(3451)	(0)	(0)
			58%	2%	40%	0%	0%
	446	8970	(5203)	(179)	(3588)	(0)	(0)
			54%	0%	44%	0%	0%
	447	13400	(7236)	(0)	(6164)	(0)	(0)
			51%	1%	48%	0%	0%
	448	22200	(11322)	(222)	(10656)	(0)	(0)
			57.9%	1.8%	40.6%	0%	0%
	MEAN	17470	(10320)	(183.25)	(7840)	(0)	(0)
			60%	1%	39%	0%	0%
	426	10900	(6540)	(109)	(4251)	(0)	(0)
			42%	0%	68%	0%	0%
	428	25200	(10584)	(0)	(14616)	(0)	(0)
			60%	0%	40%	0%	0%
	436	17900	(10740)	(0)	(7160)	(0)	(0)

Table 10c (cont).

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			85%	0%	12%	3%	0%
Day +3	437	27200	(23120)	(0)	(3264)	(816)	(0)
			56%	1%	42%	1%	0%
	445	7830	(4385)	(78)	(3289)	(78)	(0)
			60%	0%	39%	1%	0%
	446	12800	(7680)	(0)	(4992)	(128)	(0)
			53%	1%	46%	1%	0%
	447	13600	(7208)	(136)	(6120)	(136)	(0)
			47%	2%	50%	1%	0%
	448	25900	(12173)	(518)	(12950)	(259)	(0)
			57.9%	0.6%	40.6%	0.9%	0%
	MEAN	17666	(10304)	(105)	(7080)	(177)	(0)
			67%	0%	33%	0%	0%
	426	9310	(6338)	(0)	(3072)	(0)	(0)
			40%	0%	58%	2%	0%
	428	32000	(12800)	(0)	(18540)	(640)	(0)
			65%	0%	35%	0%	0%
	436	16900	(10985)	(0)	(5915)	(0)	(0)
			85%	0%	15%	0%	0%
	437	29300	(24905)	(0)	(4395)	(0)	(0)
			48%	0%	51%	1%	0%
Day +7	445	5540	(2659)	(0)	(2825)	(55)	(0)
			41%	5%	52%	2%	0%
	446	9000	(3690)	(450)	(4680)	(180)	(0)

Table 10c(Cont).

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			49%	0%	54%	1%	0%
	447	12800	(6272)	(0)	(6400)	(128)	(0)
			45%	3%	51%	.1%	0%
	448	18000	(8100)	(540)	(9180)	(180)	(0)
			55.5%	1%	42.6%	0.9%	0%
	MEAN	16606	(9456)	(124)	(6878)	(132)	(0)
			62%	0%	32%	6%	0%
	426	11500	(7130)	(0)	(3680)	(690)	(0)
			60%	0%	36%	4%	0%
	428	30900	(18540)	(0)	(11124)	(1236)	(0)
			70%	0%	30%	0%	0%
	436	11900	(8330)	(0)	(3570)	(0)	(0)
			88%	0%	12%	0%	0%
Day 10	437	35900	(31592)	(0)	(4308)	(0)	(0)
			43%	1%	54%	2%	0%
	445	6280	(2700)	(63)	(3391)	(126)	(0)
			64%	2%	31%	3%	0%
	446	8400	(5376)	(168)	(2604)	(252)	(0)
			64%	1%	35%	0%	0%
	447	10900	(6976)	(109)	(3815)	(0)	(0)
			48%	3%	48%	1%	0%
	448	13900	(6672)	(417)	(6672)	(138)	(0)
			62.4%	0.9%	34.7%	2%	0%
	MEAN	16210	(10915)	(44)	(4896)	(305)	(0)
			66%	0%	33%	1%	0%
	426	16700	(11022)	(0)	(5511)	(167)	(0)

Table 10c(cont)

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			65%	1%	34%	0%	0%
	428	31000	(20150)	(310)	(10540)	(0)	(0)
			72%	0%	26%	2%	0%
	436	12600	(9072)	(0)	(3276)	(252)	(0)
			66%	0%	34%	0%	0%
	437	29500	(19470)	(0)	(10030)	(0)	(0)
			59%	1%	36%	4%	0%
Day +14	445	4690	(2767)	(47)	(1688)	(188)	(0)
			59%	2%	38%	1%	0%
	446	7740	(4567)	(155)	(2941)	(77)	(0)
			51%	0%	48%	1%	0%
	447	14200	(7242)	(0)	(6816)	(142)	(0)
			50%	1%	48%	1%	0%
	448	11700	(5850)	(117)	(5616)	(117)	(0)
			61%	0.6%	37%	0.2%	0%
	MEAN	16016	(10018)	(79)	(5802)	(117)	(0)
	426	-	-	-	-	-	-
			64%	1%	33%	2%	0%
	428	37600	(24064)	(376)	(12408)	(752)	(0)
	436	-	-	-	-	-	-
			68%	0%	31%	1%	0%
	437	34000	(23120)	(0)	(10540)	(340)	(0)
			58%	3%	37%	2%	0%
	445	7160	(4153)	(215)	(2649)	(143)	(0)
			65%	1%	32%	2%	0%
	446	11200	(7280)	(112)	(3584)	(224)	(0)
				1%			

Table 10c (cont).

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			60%	1%	37%	2%	0%
	447	18700	(11220)	(187)	(6919)	(374)	(0)
			52%	0%	46%	2%	0%
	448	17000	(8840)	(0)	(7820)	(340)	(0)
			61.2%	1%	36.3%	15%	0%
	MEAN	20943	(13113)	(148)	(7260)	(362)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
	436	-	-	-	-	-	-
Day +21			69%	1%	30%	0%	0%
	437	36000	(24840)	(360)	(10800)	(0)	(0)
			28%	0%	71%	1%	0%
	445	6940	(1943)	(0)	(4927)	(70)	(0)
			57%	3%	39%	1%	0%
	446	14400	(8208)	(432)	(5616)	(144)	(0)
			56%	0%	43%	1%	0%
	447	19500	(10920)	(0)	(8385)	(195)	(0)
			65%	1%	34%	0%	0%
	448	13100	(8515)	(131)	(4454)	(0)	(0)
			55%	1%	43.4%	0.6%	0%
	MEAN	17988	(10885)	(185)	(6436)	(82)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
Day +24	436	-	-	-	-	-	-

Table 10c (cont).

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			79%	1%	18%	2%	0%
	437	40000	(31600)	(400)	(7200)	(800)	(0)
			41%	1%	54%	4%	0%
	445	8760	(3592)	(88)	(4730)	(350)	(0)
			61%	2%	33%	3%	0%
	446	13400	(8174)	(268)	(4422)	(402)	(0)
			66%	0%	34%	0%	0%
	447	21700	(14322)	(0)	(7378)	(0)	(0)
			58%	3%	34%	5%	0%
	448	152000	(8816)	(456)	(5168)	(750)	(0)
			61%	1.4%	36.2	1.4%	0%
	MEAN	19812	(12085)	(277)	(7173)	(277)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
	436	-	-	-	-	-	-
Day +28	437	-	-	-	-	-	-
			40%	1%	57%	2%	0%
	445	10700	(4280)	(107)	(6099)	(214)	(0)
			59%	0%	39%	2%	0%
	446	10300	(6077)	(0)	(4017)	(206)	(0)
			53%	0%	47%	0%	0%
	447	25200	(13356)	(0)	(11844)	(0)	(0)
			58%	2%	35%	5%	0%
	448	22800	(13224)	(456)	(7980)	(1140)	(0)

Table 10c (cont).

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			52.5	0.8%	44.5%	2.2%	0%
	MEAN	17250	(9234)	(141)	(7485)	(390)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
	436	-	-	-	-	-	-
Day +31	437	-	-	-	-	-	-
			48%	0%	50%	2%	0%
	445	9800	(4704)	(0)	(4900)	(196)	(0)
			77%	2%	20%	1%	0%
	446	8860	(6822)	(177)	(1772)	(89)	(0)
			57%	0%	43%	0%	0%
	447	23100	(13167)	(0)	(9933)	(0)	(0)
			63%	2%	32%	3%	0%
	448	17800	(11214)	(356)	(5696)	(534)	(0)
			61.3%	1%	36.2%	1.5%	0%
	MEAN	14890	(8977)	(133)	(5575)	(205)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
	436	-	-	-	-	-	-
Day +35	437	-	-	-	-	-	-
			70%	0%	30%	0%	0%
	445	6660	(4662)	(0)	(1998)	(0)	(0)
			57%	4%	36%	3%	0%
	446	6540	(3728)	(262)	(2354)	(196)	(0)
	447	-	-	-	-	-	-

Table 10c (cont).

DIFFERENTIAL LEUCOCYTE COUNTS INFECTED, VACCINATED GOATS (GROUP III).

	Goat No.	WBC	L	E	N	M	B
			67%	3%	29%	1%	0%
	448	11300	(7571)	(339)	(3277)	(113)	(0)
			(62.3)	2.3%	34.1%	1.3%	0%
	MEAN	9900	(6067)	(150)	(3568)	(115)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
	436	-	-	-	-	-	-
	437	-	-	-	-	-	-
Day +42	445	-	-	-	-	-	-
			60%	0%	39%	3%	0%
	446	7100	(4260)	(0)	(2627)	(213)	(0)
	447	-	-	-	-	-	-
			69%	1%	29%	1%	0%
	448	21300	(14697)	(213)	(6177)	(213)	(0)
			64.5%	0.5%	34%	2%	0%
	MEAN	14200	(9479)	(107)	(4402)	(213)	(0)
	426	-	-	-	-	-	-
	428	-	-	-	-	-	-
	436	-	-	-	-	-	-
Day +45	437	-	-	-	-	-	-
	445	-	-	-	-	-	-
			63%	0%	35%	2%	0%
	446	6360	(4007)	(0)	(2226)	(127)	(0)
	447	-	-	-	-	-	-
			69%	2%	30%	0%	0%
	448	14700	(9996)	(294)	(4410)	(0)	(0)
			65.5%	1%	32.5%	1%	0%
	MEAN	10530	(7001)	(147)	(3318)	(64)	(0)

Table 10d.

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED, VACCINATED AND TREATED GOATS (GROUP IV).

	Goat No.	TWBC	L	E	N	M	B
			68%	1%	31%	0%	0%
	401	11300	(7884)	(113)	(3503)	(0)	(0)
			59%	2%	38%	1%	0%
	402	19000	(11210)	(380)	(7220)	(190)	(0)
			58%	3%	38%	1%	0%
Day 0	404	20000	(11600)	(600)	(7600)	(200)	(0)
			70%	3%	26%	1%	0%
	405	15200	(10640)	(456)	(3952)	(152)	(0)
			47%	2%	51%	0%	0%
	407	14670	(6895)	(293)	(7482)	(0)	(0)
			60.4%	2.2%	36.8%	0.6%	0%
	MEAN	16034	(9606)	(368)	(5951)	(108)	(0)
			83%	0%	17%	0%	0%
	401	10200	(8466)	(0)	(1734)	(0)	(0)
			76%	2%	17%	5%	0%
	402	17600	(13376)	(352)	(2992)	(880)	(0)
			76%	1%	23%	0%	0%
	404	14700	(11172)	(147)	(3381)	(0)	(0)
Day +7			77%	0%	22%	1%	0%
	405	12500	(9625)	(0)	(2750)	(125)	(0)
			65%	0%	35%	0%	0%
	407	11600	(7540)	(0)	(4060)	(0)	(0)
			75.4%	0.6%	22.8%	1.2%	0%
	MEAN	13320	(10035)	(19.0)	(2983)	(201)	(0)

Table 10d (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED, VACCINATED AND TREATED GOATS (GROUP IV).

Goat No.	TWBC	L	E	N	M	B
		71%	0%	28%	1%	0%
401	10900	(7739)	(0)	(3052)	(109)	(0)
		71%	1%	25%	3%	0%
402	14430	(10245)	(144)	(3600)	(432)	(0)
		83%	0%	16%	1%	0%
404	12840	(10657)	(0)	(2054)	(128)	(0)
		76%	1%	17%	5%	0%
405	10850	(8246)	(109)	(1845)	(543)	(0)
		65%	0%	34%	1%	0%
407	11290	(7339)	(0)	(3839)	(113)	(0)
		73.2%	0.4%	24%	2.2%	0%
MEAN	12062	(8845)	(51)	(2878)	(265)	(0)
		63%	0%	35%	1%	1%
401	9400	(5922)	(0)	(3290)	(94)	(94)
		71%	0%	29%	0%	0%
402	9400	(6674)	(0)	(2726)	(0)	(0)
		86%	0%	13%	1%	0%
404	7600	(6536)	(0)	(988)	(76)	(0)
		72%	0%	28%	0%	0%
405	11000	(7920)	(0)	(3080)	(0)	(0)
		88%	0%	11%	1%	0%
407	9400	(8272)	(0)	(1034)	(94)	(0)
		76%	0%	23.2%	0.6%	0.2%
MEAN	9360	(7065)	(0)	(2224)	(34)	(0)

Day +14

Table 10d (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED; VACCINATED AND TREATED GOATS (GROUP IV).

	Goat No.	TWBC	L	E	N	M	B
Day 21			77%	0%	22%	1%	0%
	401	7370	(5675)	(0)	(1621)	(74)	(0)
			76%	0%	18%	6%	0%
	402	7160	(5442)	(0)	(1289)	(430)	(0)
			76%	0%	24%	0%	0%
	404	12190	(9264)	(0)	(2926)	(0)	(0)
			70%	0%	27%	3%	0%
	405	7550	(5285)	(0)	(2039)	(227)	(0)
			63%	0%	34%	3%	0%
	407	5200	(3276)	(0)	(1768)	(156)	(0)
			72.4%	0%	25%	2.6%	0%
	MEAN	7894	5788	(0)	(1929)	(177)	(0)
Day +24			76%	0%	19%	5%	0%
	401	8660	(6582)	(0)	(1645)	(433)	(0)
			72%	0%	26%	2%	0%
	402	10750	(7740)	(0)	(2795)	(215)	(0)
			67%	0%	31%	2%	0%
	404	12170	(8154)	(0)	(3773)	(243)	(0)
			78%	0%	21%	1%	0%
	405	7930	(6185)	(0)	(1665)	(79)	(0)
			52%	0%	46%	2%	0%
	407	5270	(2740)	(0)	(2424)	(105)	(0)
			69%	0%	28.6%	2.4%	0%
	MEAN	8956	(6280)	(0)	(2460)	(215)	(0)

Table 10d (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED: VACCINATED AND TREATED GOATS (GROUP IV).

	Goat No.	TWBC	L	E	N	M	B
			62	0	37	2	0
	401	15190	(9418)	(0)	(5620)	(304)	(0)
			80%	0%	19%	1%	0%
	402	13050	(10440)	(0)	(2480)	(130)	(0)
			53%	0%	45%	2%	0%
	404	13600	(7208)	(0)	(6120)	(272)	(0)
Day 28			60%	0%	37%	3%	0%
	405	15650	(9390)	(0)	(5790)	(470)	(0)
			47%	0%	49%	4%	0%
	407	9970	(4686)	(0)	(4885)	(399)	(0)
			60.4%	0%	30%	2.4%	0%
	MEAN	13492	(8228)	(0)	(4979)	(315)	(0)
			68%	0%	31%	1%	0%
	401	26220	(17830)	(0)	(8123)	(262)	(0)
Day 31	402	-	-	-	-	-	-
			58%	0%	37%	3%	0%
	404	19300	(11194)	(0)	(7527)	(579)	(0)
			61%	1%	38%	0%	0%
	405	17030	(10388)	(170)	(6471)	(0)	(0)
			43%	0%	56%	1%	0%
	407	18750	(8062)	(0)	(10500)	(188)	(0)
			57.5%	0.2%	41%	1.25%	0%
	MEAN	20325	(11869)	(43)	(8156)	(257)	(0)

Table 10d (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED: VACCINATED AND TREATED GOATS (GROUP IV).

	Goat No.	TWBC	L	E	N	M	B
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
			75%	0%	25%	0%	0%
	404	19300	(14475)	(0)	(4825)	(0)	(0)
Day 35			65%	0%	12%	3%	0%
	405	17030	(9367)	(0)	(7153)	(510)	(0)
			42%	0%	56%	2%	0%
	407	18750	(7875)	(0)	(10500)	(375)	(0)
			57.3%	0%	41%	1.7%	0%
	MEAN	18360	(10572)	(0)	(7493)	(295)	(0)
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
Day 38			52%	0%	46%	2%	0%
	404	18900	(9828)	(0)	(8694)	(378)	(0)
			83%	0%	17%	0%	0%
	405	13900	(11537)	(0)	(2363)	(0)	(0)
			35%	0%	65%	0%	0%
	407	18500	(6475)	(0)	(12025)	(0)	(0)
			56.7%	0%	42.7%	0.6%	0%
	MEAN	17100	(9280)	(0)	(7694)	(126)	(0)
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
Day 42			80%	0%	20%	0%	0%
	404	14850	(11880)	(0)	(2970)	(0)	(0)

Table 10d (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED: VACCINATED AND TREATED GOATS (GROUP IV).

	Goat No.	TWBC	L	E	N	M	B
			64%	0%	35%	1%	0%
	405	10410	(6662)	(0)	(3644)	(104)	(0)
			33%	0%	66%	1%	0%
	407	14490	(4782)	(0)	(9563)	(145)	(0)
			59%	0%	40.3%	0.7%	0%
	MEAN	13250	(7775)	(0)	(5392)	(83)	(0)
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
Day 45			77%	0%	21%	2%	0%
	404	12450	(9587)	(0)	(2615)	(249)	(0)
			77%	0%	22%	1%	0%
	405	9250	(7123)	(0)	(2035)	(92)	(0)
			43%	0%	56%	1%	0%
	407	14710	(6325)	(0)	(8238)	(147)	(0)
			65.7%	0%	33%	1.3%	0%
	MEAN	12137	(7678)	(0)	(4296)	(163)	(0)
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
			76%	0%	23%	1%	0%
	404	11300	(8588)	(0)	(2599)	(113)	(0)
Day 49			76%	0%	22%	2%	0%
	405	8900	(6764)	(0)	(1958)	(178)	(0)
			61%	0%	32%	7%	0%
	407	10800	(6588)	(0)	(3456)	(756)	(0)

Table 10d (cont).

DIFFERENTIAL LEUCOCYTE COUNTS OF INFECTED: VACCINATED AND TREATED GOATS (GROUP IV).

	Goat No.	TWBC	L	E	N	M	B
			71%	0%	25.7%	3.3%	0%
	MEAN	10333	(7313)	(0)	(2671)	(349)	(0)
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
Day 52			69%	0%	29%	2%	0%
	404	11620	(8018)	(0)	(3370)	(232)	(0)
			54%	1%	43%	2%	0%
	405	(9510)	(5135)	(95)	(4089)	(190)	(0)
			34%	2%	63%	1%	0%
	407	10990	(3737)	(220)	(6924)	(109)	(0)
			52.3%	1%	45%	1.7%	0%
	MEAN	10707	(5630)	(105)	(4794)	(177)	(0)
	401	-	-	-	-	-	-
	402	-	-	-	-	-	-
Day 56			76%	0%	23%	1%	0%
	404	9800	(7448)	(0)	(2254)	(98)	(0)
			62%	0%	38%	0%	0%
	405	9500	(5890)	(0)	(3610)	(0)	(0)
			53%	0%	45%	2%	0%
	407	13700	(7261)	(0)	(6165)	(274)	(0)
			63.7%	0%	35.3%	1%	0%
	MEAN	11000	(6866)	(0)	(4060)	(124)	(0)

Table 11a.

INDIRECT HAEMAGGLUTINATION TEST TITRES (RECIPROCAL OF DILUTION) OF CONTROL GOATS (GROUP I).

Goat	DAYS POST INFECTION																
Number	-7	0	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
403	4	4	4	0	4	4	4	0	4	-	4	0	4	4	8	8	4
406	4	4	4	0	4	4	4	0	4	-	4	8	8	8	16	8	8
408	0	0	0	0	0	4	4	0	4	-	4	4	4	0	4	4	4
MEAN	2.67	2.67	2.67	0	2.67	4.0	4.0	0	4.0	-	4.0	4.0	5.33	5.47	6.67	6.67	5.33
$\log_{10}$	0.42	0.42	0.42	-	0.42	0.60	0.60	-	0.60	-	0.60	0.60	0.72	0.74	0.82	0.82	0.72
SD ±	0.36	0.36	0.36	-	0.36	0	0	-	0	-	0	0.60	0.36	0.96	0.36	0.36	0.36

Table 11b.

INDIRECT HAEMAGGLUTINATION TEST TITRES (RECIPROCAL OF DILUTION) OF CONTROL VACCINATED, GOATS (GROUP II).

Goat	DAYS POST INFECTION																
Number	-7	0	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
429	0	0	0	0	0	-											
430	4	4	4	8	8	8	16	16	64	64	64	32	64	32	64	32	32
431	8	8	8	4	4	16	16	64	64	64	128	128	64	64	128	64	128
433	8	4	8	8	4	16	16	32	32	128	64	64	128	64	64	64	64
435	0	0	0	-	-												
443	0	4	0	4	0	8	8	16	8	8	8	16	16	8	16	16	16
444	4	4	4	4	0	16	16	16	16	32	32	16	16	16	32	16	16
MEAN	3.43	3.43	3.43	4.66	2.67	12.8	14.4	28.8	36.8	59.2	59.2	51.2	57.6	36.8	60.8	36.0	51.2
Log_{10}	0.54	0.54	0.54	0.67	0.43	1.11	1.16	1.46	1.57	1.77	1.77	1.71	1.76	1.57	1.78	1.56	1.71
SD $\pm$	0.56	0.44	0.56	0.48	0.51	0.64	0.55	1.32	1.42	1.65	1.65	1.67	1.66	1.42	1.63	1.30	1.67

Table 11c.

INDIRECT HAEMAGGLUTINATION TEST TITRES (RECIPROCAL OF DILUTION) OF INFECTED VACCINATED, GOATS (GROUP III).

Goat	DAYS POST INFECTION																
Number	-7	0	7	10	14	17	21	24	28	31	35	38	42	45	49	52	56
426	8	8	4	8	0	-	-	-	-	-	-	-	-	-	-	-	
428	0	4	4	4	0	8	8	-	-	-	-	-	-	-	-	-	
436	8	8	4	8	0	-	-	-	-	-	-	-	-	-	-	-	
437	0	4	4	0	0	0	0	16	-	-	-	-	-	-	-	-	
445	4	4	4	4	4	16	16	16	8	8	8	-	-	-	-	-	
446	0	0	0	0	0	4	0	4	0	0	4	4	4	4	-	-	
447	0	0	0	0	0	4	4	4	0	0	4	-	-	-	-	-	
448	0	0	0	4	0	8	0	8	8	8	8	8	8	16	16	16	
MEAN	2.5	3.5	2.5	3.5	0.5	6.7	4.7	9.6	4.0	4.0	6.0	6.0	6.0	10.0	16	16	
Log_{10}	0.40	0.48	0.30	0.54	-	0.90	0.72	0.90	0.60	0.60	0.78	0.78	0.78	1.00			

Table 11d.

INDIRECT HAEMAGGLUTINATION TEST TITRES (RECIPROCAL OF DILUTION) OF INFECTED VACCINATED, TREATED GOATS (GROUP IV).

Goat	DAYS POST INFECTION															
Number	-7	0	7	10	17	21	24	28	31	35	38	42	45	49	52	56
401	4	4	4	4	16	16	32	32	-	-	-	-	-	-	-	-
402	0	4	4	4	8	16	16	-	-	-	-	-	-	-	-	-
404	4	4	4	4	16	32	16	32	-	32	32	16	16	16	32	32
405	0	0	0	0	8	8	16	32	-	32	32	16	16	32	32	32
407	4	0	4	4	32	32	32	64	-	32	32	64	64	128	128	128
MEAN	2.4	2.4	3.2	3.2	16	20.8	22.4	40	-	42.7	32.0	37.3	32.0	58.7	64	64
Log_{10}	0.38	0.38	0.51	1.51	1.20	1.32	1.35	1.60	-	1.63	1.51	1.57	1.51	1.76	1.81	1.81
SD $\pm$	0.34	0.34	0.25	0.25	0.99	1.03	0.94	1.20	-	1.27	0.0	1.39	1.44	1.78	1.74	1.74

Table 12.

MEAN TOTAL PLASMA PROTEIN (g/100ml) OF INFECTED AND CONTROL GOATS. (Mean ± SE).

Days post infection	GROUP I	GROUP II	GROUP III	GROUP IV
-14	7.5 ± 0.43	6.8 ± 0.46	7.2 ± 0.29	7.7 ± 0.14
-7	7.4 ± 0.27	6.6 ± 0.60	7.1 ± 0.31	7.7 ± 0.13
0	7.5 ± 0.33	6.7 ± 0.43	7.2 ± 0.36	7.8 ± 0.13
3	7.7 ± 0.45	6.5 ± 0.55	6.6 ± 0.30	7.8 ± 0.13
7	7.3 ± 0.35	6.4 ± 0.38	6.6 ± 0.28	6.9 ± 0.16
10	6.9 ± 0.31	6.8 ± 0.43	6.7 ± 0.25	6.9 ± 0.20
14	6.8 ± 0.35	6.8 ± 0.40	6.3 ± 0.26	7.0 ± 0.20
17	6.6 ± 0.30	6.9 ± 0.33	5.4 ± 0.16	6.8 ± 0.08
21	6.6 ± 0.19	7.7 ± 0.34	6.0 ± 0.25	6.9 ± 0.07
24	7.2 ± 0.27	7.5 ± 0.28	5.9 ± 0.33	7.0 ± 0.13
28	7.3 ± 0.33	7.7 ± 0.20	5.6 ± 0.32	6.2 ± 0.23
31	7.3 ± 0.21	7.3 ± 0.22	5.5 ± 0.20	6.6 ± 0.57
35	7.6 ± 0.48	7.1 ± 0.19	5.8 ± 0.36	7.4 ± 0.17
38	7.1 ± 0.33	7.0 ± 0.31	5.8 ± 0.29	7.3 ± 0.23
42	6.7 ± 0.28	6.9 ± 0.18	5.7 ± 0.25	6.5 ± 0.19
45	7.0 ± 0.47	7.0 ± 0.18	5.6 ± 0.21	6.9 ± 0.33
49	6.3 ± 0.31	7.0 ± 0.21	5.7	7.0 ± 0.34
52	6.9 ± 0.36	7.0 ± 0.12	6.2	6.3 ± 0.37
56	6.3 ± 0.22	6.9 ± 0.13	6.3	6.2 ± 0.45

APPENDIX II.

1). Phosphate buffered saline pH 7.2.

Solution A: Sodium phosphate (dibasic) (Na_2HPO_4) - 9.47 g/l in distilled water.

Solution B: Potassium acid phosphate ($K H_2 PO_4$) 9.07 g/l of distilled water.

Solutions stored at $4^{o}C$

To prepare Phosphate buffer pH 7.2

72 mls of solution A was added to 28 mls of solution B.

2). Phosphate saline glucose (PSG pH 8.0).

Disodium hydrogen phosphate	13.48 g.
Sodium dihydrogen phosphate (hydrous)	0.78 g.
Sodium chloride	4.25 g

Added up to 1 litre with distilled water.

Added glucose to final concentration of 1%.