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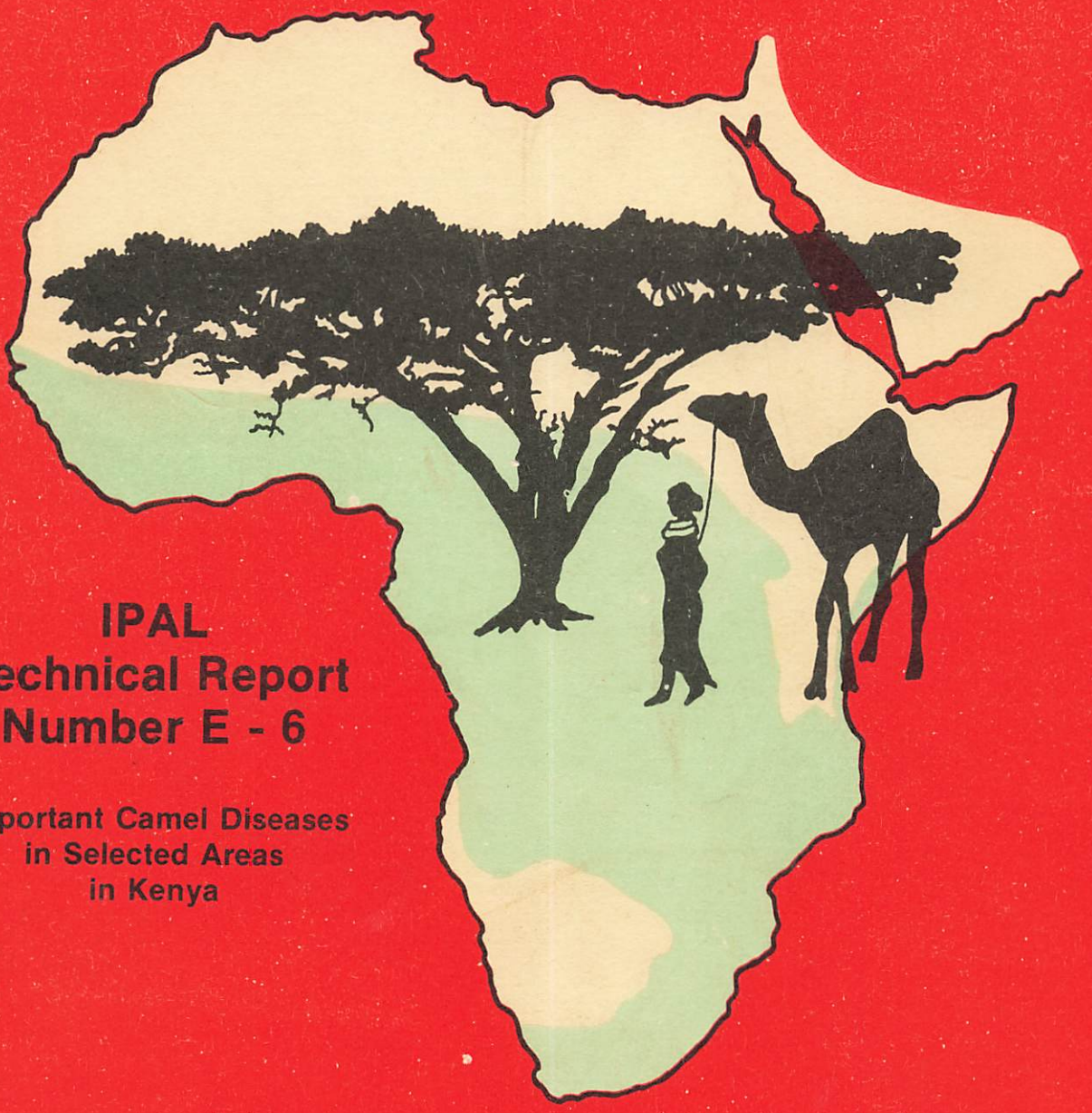
Integrated Project in Arid Lands (IPAL)



IPAL Technical Report Number E - 6

Important Camel Diseases in Selected Areas in Kenya

MAN AND THE BIOSPHERE PROGRAMME

Project 3: Impact of Human Activities and Land Use Practices on Grazing Lands

IPAL Technical Report Number E.6

IMPORTANT CAMEL DISEASES IN SELECTED AREAS IN KENYA

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UNEP - MAB Integrated Project in Arid Lands

SUMMARY INTRODUCTION TO IPAL AND THE TECHNICAL REPORT SERIES

The Integrated Project in Arid Lands (IPAL) was established by UNESCO with financial support from UNEP in 1976 with the aim of finding direct solutions to the most urgent environmental problems associated with desert encroachment and ecological degradation of arid lands. It forms part of the international UNESCO programme, Mand the Biosphere (MAB) which has links not only with UNEP's Desertification Unit but also with FAO, in response to the Plan of Action adopted by the 1977 United Nations Conference on Desertification. It is an example of the type of pilot activity that UNESCO and UNEP, together with other organizations and a number of governments, are trying to promote to provide the scientific basis for the rehabilitation and rational management of arid and semi-arid zone ecosystems, through integrated programmes of research (including survey, observation and experimentation), training and demonstration. Phase III of theproject, 1980-1983, is supported by funds in trust to UNESCO provided by the Federal Republic of Germany.

During the first phases of IPAL, a co-ordination unit was established in Nairobi and the initial field work started in the arid zone of northern Kenya in a working area of 22,500 km^2 situated between Lake Turkana and Marsabit Mountain. The project now operates five field stations at Mount Kulal, Olturot, Kargi, Korr and Ngurunit, with the project headquarters in Marsabit which is the administrative centre of the District. Since its establishment the project has researched several aspects of experimental management of the region, concentrating upon 'human ecology' of the nomadic pastoralists in dynamic inter-relationship with the animals, plants and the other resources of a drought-prone, uncertain environment.

During the next three years (1980-1983), the investigations in progress will be extended and intensified to develop resource management plans or models for the area, taking into account the increasing human population, the trend towards sedentarization, the degradation of primary productivity, and the increasing incidence of soil erosion, all of which are factors resulting in the necessity for constant famine relief measures in this region. Results obtained in the project are the subject of a number of training workshops and seminars in which Kenyan and regional scientists from the Sudano-Sahelian region participate.

This report is one of a series published by IPAL describing technical findings of the Project and, where appropriate, giving management recommendations relating to the central problems of ecological degradation in the arid zone. The reports are divided into the following categories distinguished by the base colours of their covers:

A. general, introductory and historical : white

B. climate and hydrology: blue

C. geology, geomorphology and soils: brown

D. vegetation: green

E. livestock and other animal life: red

F. social and anthropological: yellow

CONTENTS

1. SUMMARY 4
2. GENERAL PREAMBLE 4
3. INTRODUCTION 6
4. LITERATURE REVIEW 7
5. MATERIALS AND METHODS 14
   5.1 Study areas 14
   5.2 Herd Structures 17
   5.3 Herd management 17
   5.4 Recording procedures 17
   5.5 Sampling procedures 20
   5.6 Laboratory procedures 20
   5.7 Special laboratory procedures 23
   5.8 Field experiments 23
6. RESULTS 24
   6.1 Fertility and mortality 24
   6.2 Growth rates in all herds 24
   6.3 Haematology 27
      6.3.1 General 27
      6.3.2 Between herds comparison 31
      6.3.3 Differential leucocyte counts 31
   6.4 Trypanosomiasis 31
      6.4.1 Incidence rates 31
      6.4.2 Incidence rates with age 36
      6.4.3 IHA titre with age 38
      6.4.4 Point prevalence rates 38
      6.4.5 Categorisation of camel trypanosomiasis into types 42
      6.4.6 Stability of trypanosomiasis in study herds 44
   6.5 Internal parasitism 46
      6.5.1 Gastro-intestinal strongylosis 46
         (a) Herd incidence 46
         (b) Herd strongyle egg levels 46
         (c) Strongyle egg levels with age and sex 52
         (d) Larval cultures 57

6.5.2 Other internal parasites 57
6.5.3 Haemoparasites 60
6.6 External parasitism 60
6.6.1 Ticks 60
(a) Herd incidence levels 60
(b) Herd tick loads 60
(c) Species of tick and relative abundance 60
(d) Incidence of large and small mouthpart ticks 68
(e) Tick attachment sites 68
(f) Individual tick loads on camels over time 71
6.6.2 Mites 71
6.6.3 Camel fly 71
6.7 Virus diseases 71
6.7.1 Camel pox 71
6.7.2 Foot and mouth disease 71
6.7.3 Rinderpest 74
6.8 Bacterial diseases 74
6.8.1 Brucellosis 74
6.9 Miscellaneous diseases 74
6.10 Postmortem and mortality results 76
6.11 Effect of Panacur on strongyle egg levels in a small group of camels 76
7. DISCUSSION 80
8. RECOMMENDATIONS 86
9. ACKNOWLEDGEMENTS 88
10. REFERENCES 89

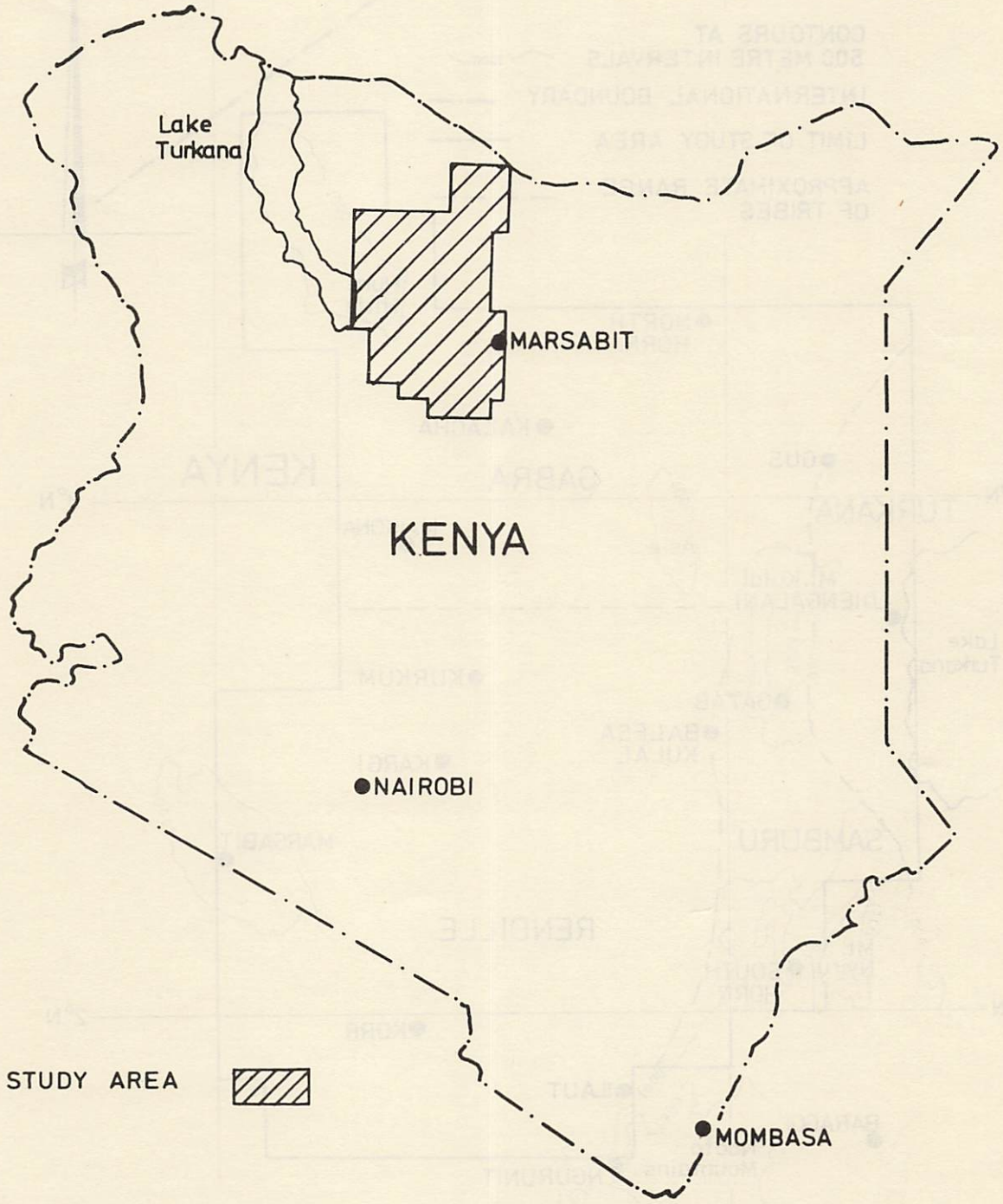
11. ADDENDA

1 Characterisation of camel trypanosomes by isoenzyme electrophoresis on starch gel 95

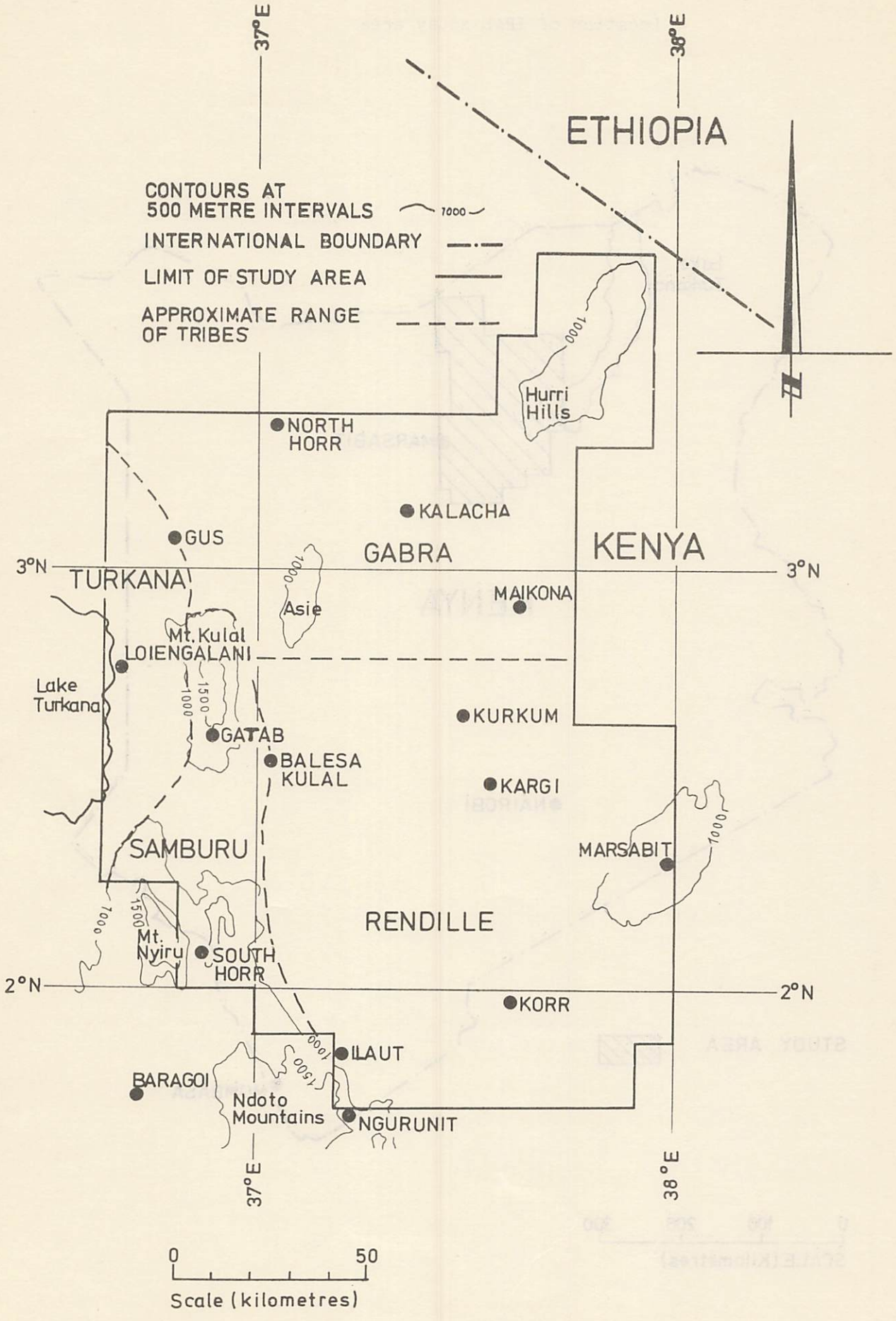
2 A description and results of drug tests conducted on trypanosomes isolated from camels in H3 97

3 Camel pox in Kenya, an examination of sera from four herds for antibody to the virus 99

Location of IPAL study area



The study area



1. SUMMARY

A study was conducted on the epidemiology of important camel diseases in four study herds in ecologically different areas of Kenya. The study showed that trypanosomiasis, malnutrition, internal parasitism, camel pox, external parasitism and brucellosis are important diseases which are constraints to production.

Control of these diseases can be achieved by good management and the incorporation of a simple drug package.

The epidemiology of these diseases and the importance of control strategies suggested are discussed.

A number of recommendations are made to maximise the potential of camels in the IPAL project area.

2. GENERAL PREAMBLE

Camels inhabit the more arid areas of the world where they demonstrate more efficient utilisation of pasture than most other species of livestock. The one humped camel (*Camelus dromedarius*) inhabits the arid and semi arid areas of North and East Africa and belongs to the ruminant family Camelidae (Mason, 1979).

About 600,000 camels are present throughout northern Kenya (KREMU, 1979) and are essential for the livelihood of the nomadic pastoralists throughout this area. They are mainly used for milk production and as draft animals. Little use is made of meat or skin products and there is little surplus for any export trade. Kenyan camels fall into 3 main types, the Somali, the Turkana and the Rendille.

Approximately 30,000 camels are present in the IPAL project area where their total biomass is roughly equal to that of goats and sheep (Field, 1979). Camels thus represent a vital component in the arid land ecosystem which is the IPAL study area.

The economic potential of camel production in Kenya is barely tapped (Fazil, 1977). There are only two private semi-commercial herds (Evans and Powys, 1979) and there is a scarcity of information on production figures among the nomadic herds. Dahl and Hjort (1976) have summarised the information which is available. One bull should be able to service 40-60 females. Females become mature around 4 years of age and should produce a calf every 2 years until the age of around 20 years. Males normally have one major "rutting" season of 2-3 months during the rainy season. Calves are then born during the following rainy season as gestation is approximately 1 year. Length of lactation varies according to nutrition but is generally longer than that found in cattle. Rendille camels lactate for approximately one year while Somali camels lactate for up to 18 months. The amount of milk produced per lactation also varies according to camel type. The Rendille camel can produce an average of 3.8 kg per day while a Somali type should be able to produce around 5 kg per day (Dahl and Hjort, 1976). Lactation figures for 4 camels in the IPAL study area are described in Field (1979). Models for growth rates in camel herds are given by Dahl and Hjort (1976) and Richard (1979).

Few figures are available on mortality in camel herds. Bremaud, quoted by Richard (1979) states that mortality can be between 30 and 50% during the first year of life. Mortality falls dramatically with age and is very low in camels over 3 years of age. The causes of this large calf mortality are uncertain. Competition with man for the dams milk is probably most important with disease another contributing factor.

Little information is available on the effects of disease on camel production as few systematic studies have been conducted. The camel is susceptible to a number of important diseases many of which are mentioned by Leese (1927) and Cross (1917) in India.

Burgmeister (1975), Fazil (1977), and Richard (1975), summarised a number of important diseases of camels in Africa. These included trypanosomiasis, a number of helminthiases, ectoparasitism, camel pox and a number of bacterial diseases. Environment and management have a large influence on the effects of disease in camels, and any study should be aimed at defining the constraints to production of all these factors.

3. INTRODUCTION

An attempt is made in this study to identify the major disease problems of camels in selected areas of Kenya and to suggest suitable strategies for their control.

The definitive aims can be summarised as follows:

1. To detect and examine the epidemiology of the important diseasesof camels in study herds maintained in different ecological areas and under different management systems in Kenya. Emphasis was to be placed on the identification of individual animals so that a systematic study could be conducted over a period of 12-18 months.
2. To ascertain the potential role of the camel in the maintenance and spread of zoonotic disease.
3. To ascertain the possible role of the camel in the maintenance and spread of important diseases of other livestock, notably cattle.
4. To suggest suitable remedial measures to counter the effects of disease on production.

An intensive study on a few herds was undertaken. This approach is more accurate than a superficial study on a large number of untraceable animals.

4. LITERATURE REVIEW

An attempt is made to summarise available information on the main disease conditions of camels. Disease will be discussed in a systematic manner according to causal agent.

4.1 Parasitic Disease

4.1.1 Trypanosomiasis

Two main forms of disease occur in camels, namely, that caused by *Trypanosoma evansi* which is transmitted mechanically by biting flies, and that caused by *T. brucei*, *T. congolense*, *T. vivax* and *T. simiae* which are biologically transmitted by the tsetse fly.

Disease caused by *T.evansi* is by far the most important as camels are rarely found in areas infested by the tsetse fly. Much of the early work on *T. evansi* in camels was undertaken in the Sudan. This classic work showed how the parasite could be detected, diagnosed and treated and the methods that were developed are relevant today (Knowles, 1924, 1925, 1927a, 1927b; Bennett, 1928, 1929, 1933; Hogan and Bennett, 1927).

Trypanosomiasis caused by *T. evansi* is considered by some authors to be the most important cause of abortion, mortality and loss of production in camels (Gatt-Rutter, 1967; Richard, 1975) and it occurs wherever camels are found. Several different types of disease have been reported, namely, hyperacute, acute and chronic. The factors which predispose to these types and their frequency of occurrence are uncertain. A review by Mahmoud and Gray (1980) has indicated the need for research on many aspects of this disease in camels.

Transmission of *T. evansi* is mechanical by blood sucking insects. Members of the family *Tabanidae* are mainly implicated (Gatt Rutter, 1967).

Eleven species of Tabanid flies are listed by Gruvel and Balis (1965) as vectors of camel trypanosomiasis in Chad. The possible role of ticks was investigated by Cross and Patel, quoted in Gatt Rutter (1967). Direct transmission to the foetus during acute disease has been reported (Sergent et al, 1919) and Leese (1927) states that transmission can occur when camels rub against one another.

Tsetse-borne trypanosomes can cause severe disease in camels. Bennett (1929) showed that *T. brucei*, *T. congolense* and *T. vivax* could cause pathogenic effects, and Pellegrini (1948) showed that *T. simiae* was pathogenic in camels. Tsetse-borne trypanosomes could become more important if camels are ranched in the proximity of the major tsetse belts of Africa.

The chemotherapy of *T. evansi* infections in camels was reviewed by Bennett (1933), and Leach (1961), and no major advances have been made since. Only two drugs are available for chemotherapy, namely, Antrycide (I.C.I) and Naganol (Bayer). The former is no longer produced and the latter is very expensive. Cattle trypanocides are either very toxic or ineffective (Balis and Richard, 1977).

A number of serological tests have been used to detect antibodies to *T. evansi* in camels. These include formol gel (Knowles, 1924), Mercuric chloride (Bennett and Kenny, 1928), indirect haemagglutination (Jatkar and Singh, 1971) fluorescent antibody, and enzyme-linked immunosorbent assay (Luckins et al, 1979). Little use has been made of serology in a detailed study of the epidemiology of camel trypanosomiasis.

4.1.2 Internal parasites

A large number of internal parasites have been recorded from the camel. Daynes and Richard (1974) reported the collection of 6 species of trematodes and 8 species of nematodes from camels in Ethiopia. Infection with strongyle worms is by far the most common.

4.1.2a Strongylosis

Infections with strongyle worms frequently occur in 90-100% of camels in herds. Their pathogenic effects are chronic and difficult to isolate, and include anaemia and emaciation. The most pathogenic strongyle worm belongs to the genus *Haemonchus*, which are voracious blood suckers.

Graber et al. (1967) found that infestations in the abomasum due to Haemonchus comprised 72% of 132 camels postmortemed in Chad and considered that this helminth was the most common and important in camels. Other strongyle nematodes which have been found in camels in Africa include *Trichostrongylus probolurus* and *Bunostomum trigonocephalum* (Daynes and Richard, 1974).
Transmission of the strongyles is direct and a number of anthelmintics are available for their destruction.

4.1.2b Strongyloidosis

Nematodes of the genus *Strongyloides* have been reported as relatively common in the camel (Steward, 1950; Graber et al, 1967; Richard, 1975). They are normally not pathogenic except with massive infestations in young animals.
Transmission is either direct or by skin penetration.

4.1.2c Trichurosis

The presence of worms of the genus *Trichuris* is very common in the large intestine of camels (Malek, 1959; Graber et al, 1967; Richard, 1975). Massive infestations can cause digestive disturbances and transmission is direct.

4.1.2d Cestodosis

Infestations of both the adult and larval stages of certain tape worms are common in camels.

Adult worms include those belonging to the genera *Moniezia*, *Stilesia* and *Avitellina* (Richard, 1975). Generally, these worms are of low pathogenicity.

The most important larval stages which parasitise camels are hydatid cysts from the adult dog tape worm *Echinococcus granulosus* (mainly in dogs), *Cysticerus bovis* the larval stage of the beef tape worm *Taenia saginata* and *Coenurus cerebralis* larval stage of *Taenia multiceps*.
Hydatid cysts and *C. bovis* occur in man and thus the camel could be important in maintaining these zoonoses in nature.
Hydatidosis has been reported in camels in Tunisia (Burgmeister, 1975), Sudan (Wilson, 1978), Nigeria (Dada, 1978), Ethiopia (Richard, 1979) and many other parts of Africa. It probably occurs wherever camels and dogs are kept. The incidence of cysticercosis in camels is generally low but it has been reported in Ethiopia in one animal (Richard, 1979) and in Chad (Graber et al, 1967). Coenurosis is also rare but has been reported in camels by Burgmeister (1975).

4.1.2e Coccidiosis

The presence of coccidial oocysts in camels is not common. According to Gruvel and Graber (1965), the most important camel coccidium is *Globidium cameli*. In general, they considered that it was not very pathogenic.

4.1.3 External parasites

External parasites comprise an important group of pests which affect the camel mainly causing intense irritation leading to loss of production. Other effects include anaemia, secondary infection, transmission of disease and the release of toxins.

4.1.3a Ticks

Richard (1975) records 11 species of tick which infest camels in Ethiopia.

These include *Rhipicephalus pulchellus*, *R. simus*, *R. pravus*, *Amblyomma variegatum*, *A. gemma*, *A. lepidum*, *Hyalomma excavatum*, *H. truncatum*, *H. dromedarii*, *H. impeltatum* and *H. rufipes*. He considered that quantitatively *R. pulchellus* and *R. simus* were the most important. However Curasson (1947) and Steward (1950a) consider *Hyalomma* to be the dominant species in the camel.

Little information is available on tick loads, preferential attachment sites and effect of age on tick load in the camel.

4.1.3b Mites

Mange (a disease of the skin caused by mites) is common in all ages of camel. Lesions usually commence on the face or flanks. Curasson (1947) considers Mange to be the most common disease of camels. The causal agent is *Sarcoptes scabei var cameli* which is spread by contact. Mange causes intense irritation and can result in rapid loss of condition.

4.1.3c Insects

Insect pests include the larvae of the nasal fly (*Cephalopsis titillator*) and the camel fly (family *Hippoboscidae*). Infestations with the larvae of the nasal fly are common (Leese, 1927; Steward, 1950a; Richard, 1975) and can cause inflammation of the nasal cavity which can lead to meningitis. Infestation with camel flies are also common causing irritation. Camel flies are also implicated in the transmission of *T. evansi* in camels.

4.2 Virus Diseases

4.2.1 Camel pox

Camel pox, the most important virus disease of camels, occurs mainly in young animals and is most common during the rainy seasons (Cross, 1917).

The characteristics of a Kenyan camel pox virus are described by Davies et al (1975) and this virus may be of some zoonotic importance as minor lesions have been reported in humans.

The disease can cause mortality in calves and is associated with the formation of crusts at many sites on the body although the head area is most severely affected. Recovery from infection generally confers a lasting immunity.

4.2.2 Rinderpest

It is still uncertain whether camels are fully susceptible to the rinderpest virus. The virus will cause a slight temperature rise and a stimulation of the immune response (Singh and Ata, 1967; Taylor, 1968). Clinical lesions have been reported in camels (literature reviewed in Richard, 1975); however it probably plays a minor role in the epidemiology of of the disease in cattle, although 15% serological positives were detected in sera from 538 camels in Ethiopia (Richard, 1975).

4.2.3 Foot and mouth Disease (FMD)

The virus of FMD does not appear to infect camels (Richard, 1979) although reports of FMD outbreaks in camels do appear in the literature.

4.2.4 Rift Valley Fever (RVF)

RVF antibodies were detected in the sera of camels originating in two widely separated areas in northern Kenya.
The incidence in various age groups was similar and large scale abortions were attributed to the outbreak (Scott et al, 1963).

4.3 Bacterial Diseases

A number of diseases of bacterial origin have been reported in camels. Many studies have been hampered by their distance from a laboratory capable of undertaking bacterial surveillance.

Acute bacterial infections, which have been confirmed include salmonellosis (Olitski and Ellenbogen, 1947; Zaki, 1956; Cheyne et al, 1977) and anthrax (Cross, 1917; Curasson, 1947). However the role of *Clostridia* and *Pasteurella* is uncertain and no confirmed cases have been reported (Richard, 1979). Camels are susceptible to various forms of pneumonia; however the precise causal agents have never been elucidated. The term "dromedary-respiratory-diseases-complex" has been used where a variety of organisms have been implicated. Brucellosis is an established disease of camels through serological surveys (Richard, 1975; Waghela et al 1978; Okoh, 1979) but the role of this disease in causing abortion is not certain. Tetanus has also been reported (Morcos, 1965).

Chronic bacterial infections of camels include skin necrosis caused by *Streptococcus* (Edelsten and Pegram, 1974), Corynebacteriosis (Domenech et al 1977), Tuberculosis and Leptospirosis (Gatt-Rutter and Mack, 1963).

4.4 General

The importance of disease in camel production is uncertain and is related to nutrition and husbandry. On the one hand, there is the opinion of Wilson (1978) that disease is of little consequence, and on the other the opinions of numerous authors that it is very important. However most camel diseases are easily controlled which combined with good management should lead to healthy and productive camel herds.

Richard (1979) classified camel disease according to economic impact as follows in descending order of importance:

- Trypanosomiasis
- Internal parasitism
- Mange
- Pyogenic diseases
- Nutritional deficiency
- Pulmonary disease
- Myiasis
- Camel pox
- Anthrax.

It is of interest that nutritional deficiency and camel pox are so low in order. However, this list is suggested as a starting point in this study.

4.5 Haematology

A study on the normal blood profiles for camels in the Sudan was reported by Salaheldin et al (1979). They quote the following values:

Total erythrocytes		$7.83 \pm 1.31 \times 10^6$/cumm
PCV		$30 \pm 2.64\%$
Total haemoglobin		11.1 ± 1.78 gm/100ml
Total leucocytes		$13.23 \pm 2.01 \times 10^3$/cumm
Differential leucocytes	neutrophils	51%
	lymphocytes	40%
	eosinophils	4%
	monocytes	4%
	basophils	0.5%

These values will be used in this study as normal values.

5. MATERIALS AND METHODS

5.1 Study areas

Four study areas in different areas of Kenya (designated 1-4) were selected according to availability of camels and vegetation type. The locations of the areas are shown on the map and general descriptions are given on table 1. Area 4 is the only area adjacent to a large tsetse fly belt. Two areas (1 and 2) are within the IPAL study area and represent northern Kenya. The other two areas (3 and 4) are further south and represent areas where camel production may occur to a much greater extent in the future. Herd 2 belongs to a group of nomadic pastoralists.

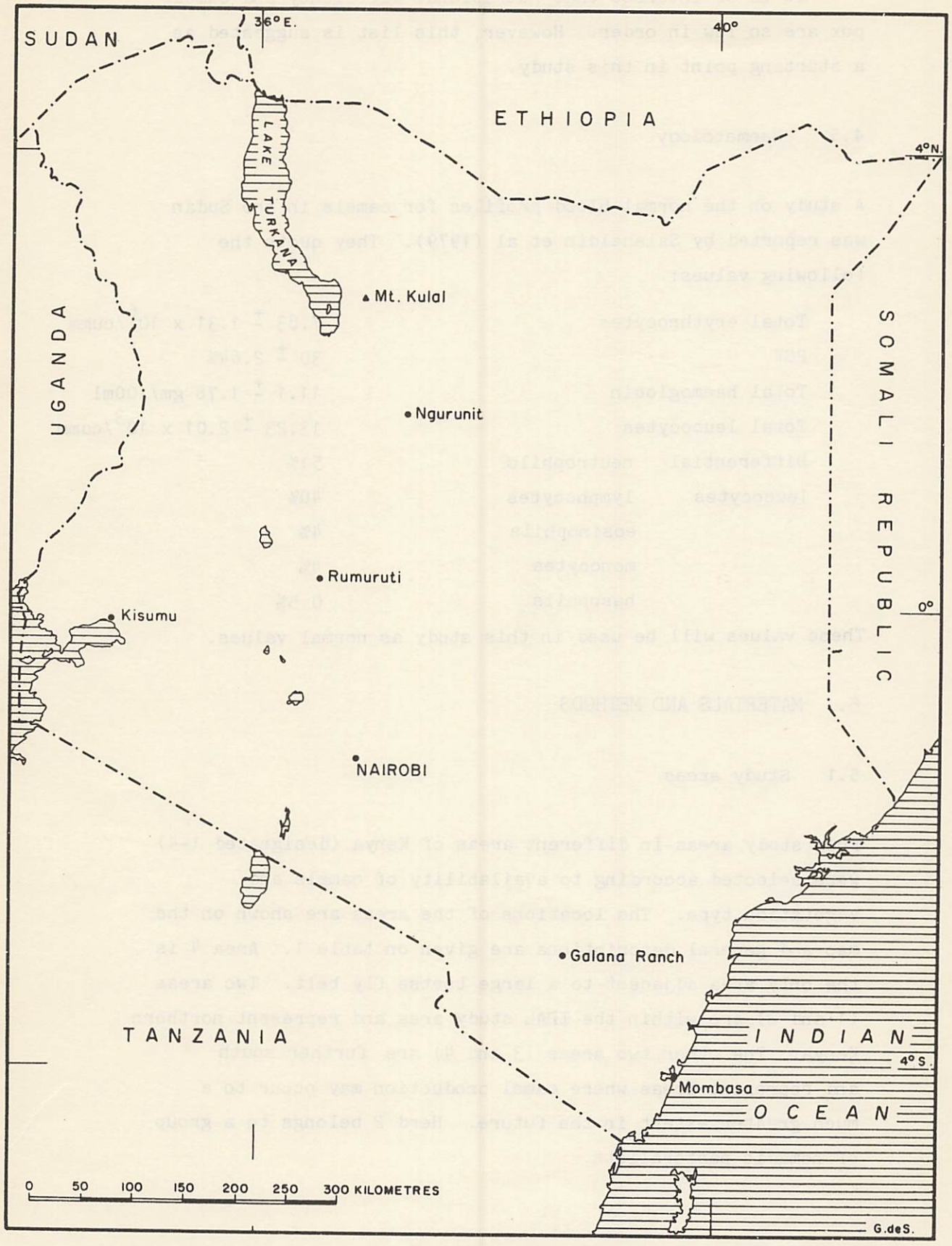

SUDAN
36° E.
40°
ETHIOPIA
4° N.
LAKE TURKANA
Mt. Kulal
Ngurunit
UGANDA
SOMALI REPUBLIC
Rumuruti
Kisumu
0°
NAIROBI
Galana Ranch
TANZANIA
INDIAN
4° S.
Mombasa
OCEAN
0 50 100 150 200 250 300 KILOMETRES
G. deS.

Table 1. General description of the four study areas.

	Area	Altitude (m)	Rainfall (mm)	Vegetation
1	Mount Kulal	700-2000	100-600	Evergreen forest leading into perennial grassland and thence into bushland and dwarf shrubland.
2	Ngurunit	700	300-700	Bushland, dwarf shrubland
3	Ol Maisor	1900	580	Grassland scattered widely with *Acacia* sp
4	Galana	270	300-500	Thick tsetse fly infested bush to the East with light *Acacia-Commiphora* to the West. Rain declines markedly East to West.

5.2 Herd Structures

Table 2 shows the number of camels studied in each area. Animals were tagged or branded and identified according to sex and then divided into 3 categories, (1) adults (>4 years), (2) immatures (<4 years>1 year) and (3) calves (<1 year).
The herd structures of each herd are also shown on the table. A total of 508 camels were studied of which 211 were in area 1, 174 in area 2, 138 in area 3 and 175 in area 4.

5.3 Herd management

The management of each herd in terms of ownership, grazing pressure, contact with other camels and veterinary input is summarised on table 3. Herds 3 and 4 are isolated from the main camel populations of Kenya, while herds 1 and 2 are in contact with other camels especially at watering points. Herds 1, 3 and 4 are privately owned and hence have some veterinary input. Herd 2 belongs to a group of nomadic pastoralists with no input. These herds will be referred to as H1, H2, H3 and H4.

5.4 Recording procedures

Each animal was assigned a specially designed sheet on which all relevant information was recorded (sex, age, sampling dates, samples taken and results). Obvious clinical disease was also recorded. Previous calving history and the calves born during the study period were recorded on the dams sheet, as were measurements to estimate body weight using the method of Schwartz (1980). The weight in kg is the product of 50, the shoulder height, the abdominal girth and the thoracic girth.

Table 2. The numbers of camels and their herd structures studied in each of the 4 areas. The time periods when each herd was examined are also included.

Herd (Study period)	ADULTS (4 years) male & male castrate	ADULTS (4 years) female	IMMATURES male	IMMATURES female	CALVES (1yr) male	CALVES (1yr) female	TOTALS
1 (Aug 79-Jan 81)	3	9	2	2	2	3	21
2 (Aug 79-Jan 81)	41	80	18	28	2	5	174
3 (June 80-April 81)	15	54	17	24	16	12	138
4 (June 80-April 81)	19	69	14	20	24	29	175
TOTALS	78	212	51	74	44	49	508

Table 3. A Summary of the management practices of the four study camel herds

Herd	Ownership	Grazing pressure	Veterinary inputs.		
			Acaracide	Anthelminthic	Antibiotics
1	UNESCO	+	monthly	2-monthly	*
2	TRADITIONAL	+	NIL		
3	PRIVATE	-	*	*	*
4	PRIVATE	-	*	*	*

* = as required on clinical evidence of disease

+ = severe competition for grazing at certain times of the year.

Herd 4 was under a 2-monthly treatment with the trypanocide Antrycide from late 1978 until April 1980 when supplies of the drug could no longer be obtained.

5.5 Sampling procedures

The frequency of sampling was different in each herd and is shown on table 4. This table also shows the number and type of samples taken from each animal and the reasons why they were obtained. An attempt was made to sample every animal in the study herd at each visit. However, this was not possible in every case, in particular with the traditional herd (H2).

Postmortems were conducted whenever possible by lay staff using a systematic schedule. A series of samples were obtained in formalin for subsequent histopathology.

5.6 Laboratory procedures

5.6a Haematology.

The packed cell volume (PCV), the total red cell count, the haemoglobin concentration and the total white cell count were measured using standard procedures on each sample. A differential white cell count was conducted on random samples from all herds.

5.6b Mouse inoculation

0.5 ml from each anticoagulant blood sample was inoculated into one or two mice. The tail blood from each mouse was examined for trypanosomes from day 3 postinoculation daily until day 60. If trypanosomes were detected, a stained blood film was prepared for identification, and the mouse discarded.

5.6c Trypanosome storage

A number of trypanosome isolates grown in mice from all herds were frozen in liquid nitrogen for subsequent detailed examination by iso-enzyme analysis (see addendum 1) and for drug resistence tests (see addendum 2).

Table 4. A summary of period, frequency and type of sampling undertaken on all animals in each herd. The reasons why each sample was taken are also given.

HERD	SAMPLING FREQUENCY AND PERIOD OF STUDY	
1	monthly	August 1979 - January '81
2	two-monthly	August 1979 - January '81
3	three-monthly	June 1980 - April '81
4	three-monthly	June 1980 - April '81

SAMPLES TAKEN FROM EACH ANIMAL AT EACH VISIT		REASONS
1	Anticoagulant blood sample	(Haematology (mouse inoculation (blood slides
2	Serum	(Serology for antibodies (against trypanosomiasis, (camel pox, brucellosis, (foot and mouth and (rinderpest.
3	Faeces	(Egg counts (larval culture
4	Ticks	(Total body count (identification.

5.6d Blood films

Thin blood films were prepared from selected samples from each herd at different times of year for examination for the presence of haemoparasites.

5.6e Serology

A number of serological tests were conducted to detect antibodies against specific diseases. The types of test varied according to the disease and are summarised as follows:

Trypanosomiasis (*Trypanosoma evansi*)	(Complement fixation (Mercuric chloride (Indirect haemagglu- (tination
Camel pox) Rinderpest)	Serum neutralisation
Foot and mouth disease	Metabolic inhibition test
Brucellosis	(Complement fixation (Rose Bengal (Serum agglutination

The description of these tests are beyond the terms of reference of this report.

5.6f Faecal egg counts and larval culture

All faecal samples collected were examined for the presence of eggs by the standard McMaster method and egg counts recorded. The type of egg was identified and larval cultures were conducted on random samples, containing over 1000 strongyle eggs per gram, from all herds.

5.6g Ticks

A total tick load was estimated on each animal at every sampling by counting all the ticks on one side of the animal and then doubling this figure. Samples of ticks were collected from camels at every visit in all herds. These were counted and identified accoring to stage, sex and species. A small study on the relationship of tick load and species to animal site was conducted using animals in herd 1.

5.7 Special laboratory procedures

5.7a Isoenzyme patterns

A number of trypanosome isolates from study areas were subject to isoenzyme pattern analysis using methods described by Gibson et al (1980). The methods and results are described in addendum 1.

5.7b Drug resistance

Trypanosome isolates from herd 3 were subject to drug resistance tests. The procedures and results are shown in addendum 2.

5.8 Field experiments

5.8a Effect of Panacur (Fenbendazol, Hoecst) on internal parasitism.

The animals of herd 1 were divided randomly according to sex and age into 2 groups of 10 and 11 animals on 30/1/80. The group of 10 were left untreated and the group of 11 were treated with Panacur (7.5ml of a 10% solution/100kg bw per os) at intervals of between 6 and 8 weeks from February 1980 to January 1981. The effects of the anthelmintic were monitored monthly accoring to faecal egg output.

6. RESULTS

6.1 Fertility and mortality

A summary of mean age of females at first calving, calving interval, abortion rates, calving percentage and calf mortality rates in all herds is shown on table 5. The data from H2 was estimated from 20 year personal history of the owner. The data from H1, H3 and H4 was calculated over a 3-4 year period between 1976 and 1981.

The production figures in the nomadic herd (H2) indicate that the camel population has remained static over the last 15-20 years. The mean age at first calving is estimated at 5 years with a calving interval of 30 months.

The comparable figures for H3 and H4 show a great increase in the productivity of both herds as compared with H2 and indicate that a 20-30% animal increase in numbers could be expected in both herds.

The figures for H1 are nearly as good as those for H3 and H4 and indicate that an increase in herd size could be expected in camel herds in the IPAL project area under good management. The results from H1 are a credit to the IPAL staff under the direction of Dr. Field.

6.2 Growth rates in all herds

A summary of mean weight (kg) in groups of animals of different ages in each of the herds is shown on table 6. This shows that the rate of growth in animals of H2 is much slower than those in H1, H3 and H4. The nomadic herd (H2) showed a stunted growth rate in all age groups.

Table 5. A Summary of mean age at first calving of adult females, calving intervals, abortion rates, calving percentage and calf mortality in the 4 study herds. Herds 1,3, and 4 are annual figures over the period 1976-1981. Data from herd 2 is obtained from a 20 year owner's history.

Herd No.	Mean age at first calving in adult females (years)	Calving interval (mean in months)	Abortion (A) Still birth (B) rates (%)	Calving percentage (% females calving)	Calf mortality %
1	5.1	18.7	NIL(A) 12(B) (8)	45	12.0
2	5*	30*	10*(A)	30*	50*
3	4.5	22.2	NIL(A)	45-50	NIL
4	4.5	20.0	NIL(A)	45-50	6.2

* Estimate

Data from H2-4 was obtained by courtesy of Dr H.J. Schwartz.

Table 6. A comparison of mean body weight (±SE) with age in the four study herds
Number of observations are in brackets.

Herd No.	Birth	1 year	2 years	3 years	Adults	
1	30.9 ± 4.3*(7)	190*	300*	+	500*	
2	+	97(17) ±5.6	160(14) ±13.9	226(11) ±19.6	♂ 450(28) ± 19.6	♀ 386(43) ±8.0
3	+	173(20) ±5.0	259(17) ±4.1	379(8) ±17.9	♂ 505(8) ±43.6	♀ 420(44) ±8.3
4	37.3 ± 3.9 (14)	247(14) ±9.1	331(15) ±13.6	330(10) ±27.7	♂ 573(14) ±24.4	♀ 567(57) ±9.1

* Data from Field (1979a)

\+ No data

6.3 Haematology

6.3.1 General

The results of the mean PCV, haemoglobin (Hb) concentration, total erythrocyte (RBC) and total leucocyte (WBC) counts from 153 samples (H1), 514 samples (H2), 83 samples (H3) and 296 (H4) over time are shown on table 7. The mean PCV and WBC levels are also shown on figure 1.

The PCV values in the animals of herd 1 were below normal throughout the study period with the exception of the bleed in January 1981. The total RBC counts and haemoglobin concentrations were normal throughout the period. These results indicate the presence of a generalised microcytic anaemia. The total WBC counts were elevated (leucocytosis) throughout the period with the exception of the bleed in January 1981.

The blood profiles of H2 were similar to H1 over the initial part of the study period (December 1979 - June 1980), except March 1980 when the total WBC count was normal.
However from this time to the end of the study the mean PCV, Hb and RBC parameters were normal, although the leucocytosis remained.

The June 1980 sampling from H3 showed a normal erythrocyte profile with the presence of a leucocytosis. Subsequent sampling measuring PCV values only showed anaemia developed in this herd (figure 1).
A normal red cell blood profile and a leucocytosis was observed in the samples from H4 except that normal WBC values were measured in January 1981.

Table 7. A summary of mean packed cell volume, and where relevant the haemoglobin concentrations, total red blood cell $X10^6$ and total white cell $X10^3$ ($\pm$SE) at each sampling date from all herds over the study period.

HERD NO.	Dec 1979 PCV	RBC	HB	WBC	February PCV	RBC	HB	WBC	March PCV	RBC	HB	WBC
1	26.60	8.11	11.29	18.42	27.10	8.30	10.80	21.25	25.40	8.24	11.63	19.03
	±1.01	±0.31	±0.44	±1.49	±1.11	±0.29	±0.38	±1.50	±0.82	±0.24	±0.40	±1.48
	n=15				n=20				n=19			
2	25.40	7.68	10.64	23.17					24.64	8.61	10.46	11.88
	±0.60	±0.17	±0.25	±1.44					±0.73	±0.32	±0.33	±1.55
	n=41								n=25			

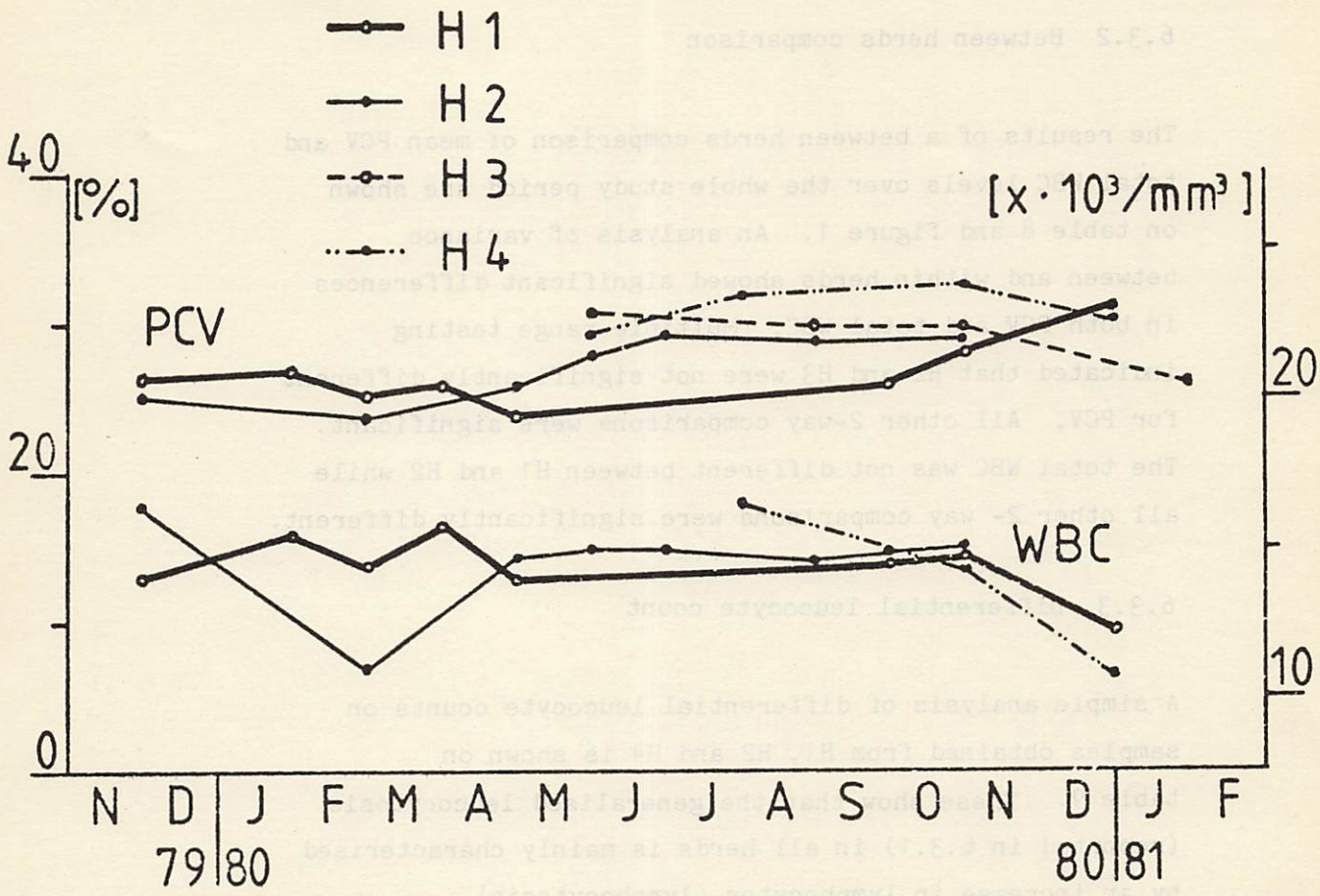
HERD NO.	April PCV	RBC	HB	WBC	May PCV	RBC	HB	WBC	June PCV	RBC	HB	WBC
1	24.2	7.83	11.01	19.52	24.10	8.26	10.52	18.35				
	±1.49	±0.4	±0.69	±2.10	±0.81	±0.19	±0.27	±1.4				
	n=19				n=21							
2					26.05	8.37	11.88	19.18	27.92	9.28	12.46	20.03
					±0.32	±0.09	±0.14	±0.82	±0.61	±0.16	±0.26	±1.56
					n=115				n=37			
3									30.31	10.19	13.66	22.14
									±0.32	±0.12	±0.25	±0.76
									n=83			

Table 7: (Continued)

HERD NO.	July PCV	July RBC	July HB	July WBC	August PCV	August RBC	August HB	August WBC	September PCV	September RBC	September HB	September WBC
1												
2	29.49 ±0.60	9.52 ±0.17	13.33 ±0.27	20.26 ±1.2					29.22 ±0.39	9.27 ±0.11	12.37 ±0.16	19.23 ±1.00
	n=99								n=110			
4					31.60 ±0.36	10.18 ±0.12	14.37 ±0.21	22.90 ±0.71				
					n=140							

HERD NO.	October PCV	October RBC	October HB	October WBC	November PCV	November RBC	November HB	November WBC	January PCV	January RBC	January HB	January WBC
1	25.90 ±0.86	8.31 ±0.20	11.10 ±0.36	19.30 ±1.57	28.10 ±0.78	8.84 ±0.24	12.32 ±0.38	19.50 ±1.60	31.12 ±0.83	8.97 ±0.19	12.93 ±0.26	14.74 ±1.78
	n=21				n=21				n=17			
2					28.29 ±0.51	8.99 ±0.14	12.45 ±0.23	16.70 ±1.02				
					n=87							
4					32.08 ±0.30	10.06 ±0.13	13.88 ±0.14	19.35 ±0.60	30.71 ±0.27	8.10 ±0.07	13.67 ±0.11	11.40 ±0.30
					n=141				n=160			

Figure 1: Mean packed cell volumes and mean total white blood cell counts in four selected herds of dromedaries



6.3.2 Between herds comparison

The results of a between herds comparison of mean PCV and total WBC levels over the whole study period are shown on table 8 and figure 1. An analysis of variance between and within herds showed significant differences in both PCV and total WBC. Multiple range testing indicated that H2 and H3 were not significantly different for PCV. All other 2-way comparisons were significant. The total WBC was not different between H1 and H2 while all other 2- way comparisons were significantly different.

6.3.3 Differential leucocyte count

A simple analysis of differential leucocyte counts on samples obtained from H1, H2 and H4 is shown on table 9. These show that the generalised leucocytosis (reported in 6.3.1) in all herds is mainly characterised by an increase in lymphocytes (lymphocytosis).

6.4. Trypanosomiasis

6.4.1 Incidence rates

The incidence rates of trypanosomiasis in all herds according to detection of patent parasitaemia, trypanosome species and detection of circulating antibody over the respective study periods are shown on tables 10 and 11. These results show that trypanosomiasis is endemic in all herds. The incidence of IHA antibody shows that most animals have had some contact with the disease. The incidence of patent disease, as judged by mouse inoculation (MI) is much higher in H1 (71.4%) than any of the other herds and this is reflected in the highest incidence of IHA antibody.

Table 8. An analysis of variance of PCV and total WBC between herds over the whole study period.

(A) PCV

Source	df	MS	
Between herds	3	1406.2	F = 75.0 ($p<0.001$)
Within herds	1470	18.8	

Herd means

H4	H2	H3	H1
30.89	27.80	27.71	26.43

(B) Total WBC

Source	df	MS	
Between herds	3	552.3	F = 42.81 ($p<0.001$)
Within herds	1213	12.9	

Herd means

H3	H1	H2	H4
22.14	19.14	19.01	17.60

Table 9. Mean of differential WBC counts undertaken on samples from herds 1,2 and 4.

Herd No.	No of Samples	MEAN PERCENTAGE OF DIFFERENT CELL TYPES				
		Neutrophils	Lymphocytes	Eosinophils	Basophils	Monocytes
1	109	25.4	69.8	3.9	0.1	0.8
2	220	18.9	74.6	5.1	0.2	1.2
4	109	30.5	61.2	7.3	0.1	0.9

Table 10. The incidence of camel trypanosomiasis in all herds over their respective study periods, as judged by mouse inoculation and the presence of IHA antibody.

Herd No	Incidence of trypanosomiasis (%)		Period of study
	Mouse inoculation	IHA antibody	
1	71.4	95.2	Nov '79 - Jan '81
2	9.7	79.4	Dec '79 - Jan '81
3	21.7	73.2	June '80 - Feb '81
4	5.9	80.6	July '80 - Feb '81

Table 11. A summary of trypanosome identification on all infected mice obtained from all herds over the study period.

Herd No.	PERCENTAGE OF ISOLATES DETECTED AS	
	Trypanosoma brucei group (probably *Trypanosoma evansi* - see Addendum 1.)	*Trypanosoma congolense*
1	100	NIL
2	100	NIL
3	97.4	2.6
4	83.4	16.6

Incidence of patent infection was also high in H3 (21.7%) and this herd had the lowest incidence of IHA antibody (73.2%). The disease showed low patency in both H2 and H4 and both herds had similar incidence rates of IHA antibody.

The most common type of trypanosome infecting camels in all herds belonged to the *Trypanosoma brucei* group (table 11). This trypanosome is probably *Trypanosoma evansi* although it is very difficult to differentiate this trypanosome from *Trypanosoma brucei* (see addendum 1). *Trypanosoma brucei* group trypanosomes were isolated in 100% of the infected mice inoculated with blood from herds 1 and 2. However the dangerous *Trypanosoma congolense* was isolated from both H3 and H4, being more common in H4 (16.6% of isolates). These results indicate that tsetse flies may be important in the transmission of camel trypanosomiasis in both H3 and H4 as *Trypanosoma congolense* is normally tsetse-dependent where biological development occurs. The disease is probably transmitted mechanically in both H1 and H2.

6.4.2 Incidence rates with age

Indidence rates of trypanosomiasis in the different age groups (adults, immatures and calves) as judged by MI and IHA over the study period are shown on table 12. The numbers of animals in H1 are too small for any deductions to be made; however IHA incidence was very high in all ages of the herd. The results from H2 and H4 indicate that a calf resistance to the disease is present. A nil MI incidence coupled with high IHA incidence was present in the calves of both H2 and H4.

Table 12. The incidence rates (%) of trypanosomiasis with age in all herds over their respective study periods, as judged by M1 and the presence of IHA antibody together with the χ^2 value for differences between observed and expected incidence rates (M1 only).

(A) Incidence on MI

Herd No.	Adults No	Adults Incidence	Immatures No	Immatures Incidence	Calves No	Calves Incidence
1	12	58.3	4	100	5	100
2	121	11.6	46	6.5	7	NIL
3	69	17.4	41	39.0	28	7.1
4	82	8.5	35	8.6	53	NIL
Overall No. positives (all herds)	Observed	40	26		6	$\chi^2_2 = 8.36$ p 0.05
	Expected	39.80	17.64		13.02	

(B) Incidence on IHA

Herd	Adults No	Adults Incidence	Immatures No	Immatures Incidence	Calves No	Calves Incidence
1	12	91.6	4	100	5	100
2	121	78.5	46	78.3	7	100
3	69	84.1	41	68.3	28	57.2
4	82	70.8	35	85.7	53	92.5

A lower IHA incidence together with a 7.1% incidence of patency was detected in the calves of H3 indicating that calfhood immunity was incomplete. A X^2 test indicated that the incidence on MI was significantly ($P<0.05$) lower in calves than other age groups in all study animals. No differences in MI incidence rates were detected between immatures and adults in H4. The immatures of both H1 and H3 showed a higher incidence of MI patency than the adults; however the reverse was the case in H2. IHA incidence decreased with age in H1, H2 and H4, the trend being most marked in H4. However in H3 IHA incidence increased with age.

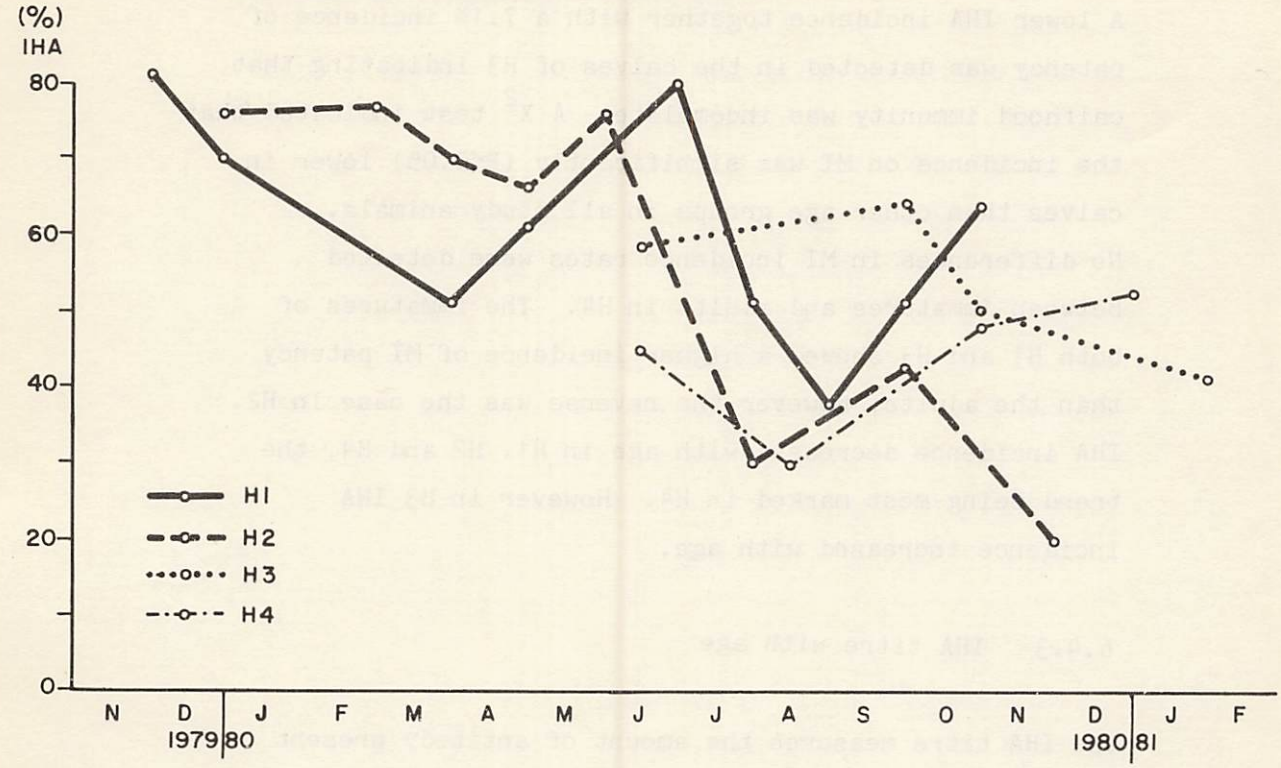
6.4.3 IHA titre with age

The IHA titre measures the amount of antibody present in the serum. A summary of IHA titre with age in all herds is shown on table 13. The number of calves is low in both H1 and H2 and these results should be examined with caution. There is a stronger antibody response in the calves of less than 3 months than in any other age group. This probably indicates the presence of maternal antibody. There are lowered antibody levels with age in H2 and H4. The immatures and adults of H1 and H3 show higher antibody levels than the corresponding age groups of H2 and H4.

6.4.4 Point prevalence rates (PPR)

The point prevalence rates of trypanosomiasis in all herds as judged by MI and IHA antibody at each sampling date are shown on figure 2. The PPR in H1 was much higher than any other herds as judged by MI over most of the study period.

FIGURE 2: TRYPANOSOMIASIS POINT PREVALENCE RATES JUDGED BY MI AND IHA IN FOUR SELECTED HERDS OF DROMEDARIES.



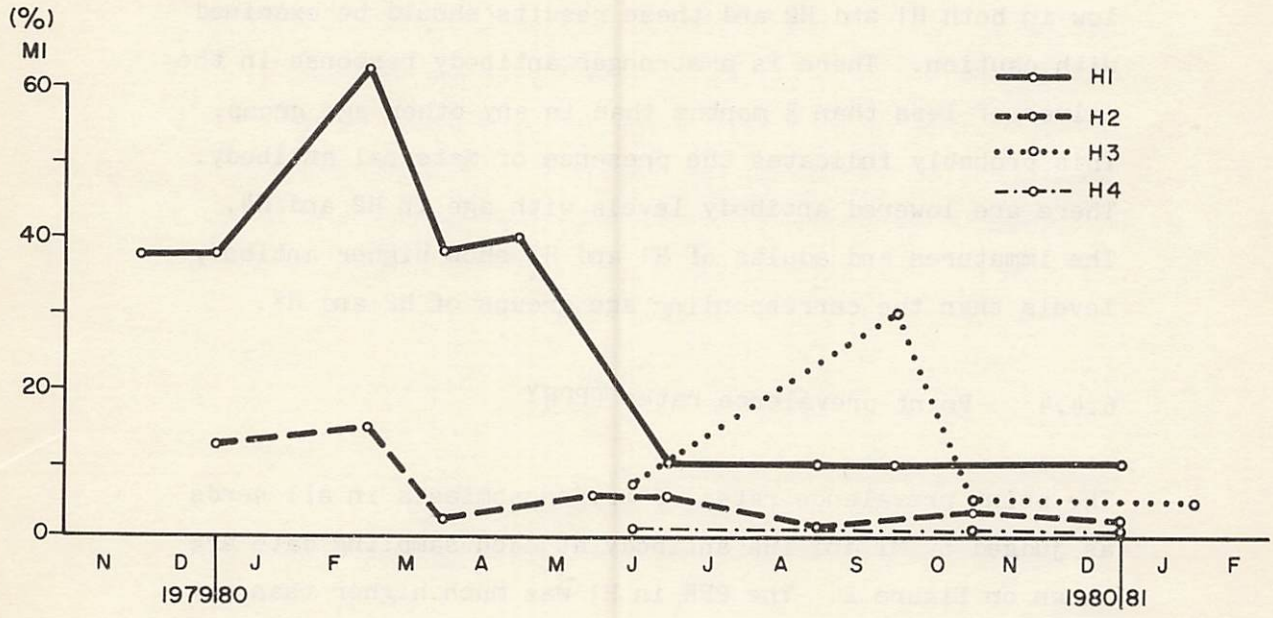


Table 13. Summary of the frequency of appearance of the different IHA titres with age in the 4 herds over the study period.

Herd No	Herd Structure/Animals	No	IHA TITRE							
			<40	40	80	160	320	640	1280	2560	5120
1	Calves <3m	0	-	-	-	-	-	-	-	-	-
	Calves 3-6m	2	0.09	0.18	0.18	0.05	0.32	0.05	-	-	-
	Calves >6m	3	0.30	0.27	0.21	0.12	0.03	0.06			
	Immatures	4	0.07	0.19	0.26	0.07	0.14	0.09	0.09	0.07	0.02
	Adults	12	0.54	0.17	0.16	0.06	0.03	0.02	0.02	-	-
2	Calves <3m	1	0.20	-	0.40	0.2	-	0.20	-	-	-
	Calves 3-6m	2	0.60	-	-	0.40	-	-	-	-	-
	Calves >6m	4	0.20	0.10	0.30	0.40	-	-	-	-	-
	Immatures	46	0.58	0.11	0.11	0.16	0.03	0.01	-	-	-
	Adults	121	0.69	0.08	0.10	0.08	0.03	0.01	-	-	-
3	Calves <3m	18	0.17	0.28	0.11	0.11	0.22	0.06	0.05	-	-
	Calves 3-6m	21	0.56	0.24	0.05	-	0.05	0.05	0.05	-	-
	Calves >6m	28	0.62	0.21	0.14	-	-	-	-	-	0.03
	Immatures	40	0.30	0.15	0.20	0.03	0.20	0.03	0.06	0.03	-
	Adults	67	0.38	0.15	0.15	0.06	0.16	0.06	0.03	0.01	-

continued

Table 13. Summary of the frequency of appearance of the different IHA titres with age in the 4 herds over the study period.

Herd No	Herd Structure	No Animals	IHA TITRE <40	40	80	160	320	640	1280	2560	5120
4	Calves <3m)		0.18	0.14	0.21	0.43	0.04	-	-	-	-
	Calves 3-6m)	53	0.38	0.44	0.09	0.09	-	-	-	-	-
	Calves >6m)		0.40	0.27	0.07	0.04	-	-	-	-	-
	Immatures	35	0.56	0.14	0.19	0.10	0.01	0.01	-	-	-
	Adults	82	0.66	0.18	0.08	0.06	0.02	-	-	-	-

However PPR steadily declined over the study period. The PPR's in both H2 and H4 were low throughout the study period indicating that the disease had stabilised at a low level of patency. The PPR in H3 showed a dramatic rise over the period June to October 1980 indicating that an epidemic was occuring; the PPR subsequently fell over the rest of the study period.

The PPR as judged by IHA antibody was high over most of the study period in all herds. A large fall in PPR was observed in H1, H2 and H4 over the period May to Sept, 1980. This pattern was not observed in H3 when the PPR rose during this period. The fall of PPR of IHA antibody could be related to the dry season and the reason that it did not occur in the animals of H3 was probably due to the epidemic that was occurring at this time. The cause of this epidemic could have been the introduction of infected animals.

6.4.5 Possible categorisation of camel trypanosomiasis (T. *evansi*) into types - H1

An attempt has been made to categorise camel trypanosomiasis into four types based on the results from animals in H1 which had the greatest sampling frequency. Mean PCV and the results of mouse inoculation are used to type the 21 animals. The results are shown on Table 14 and the animals have been divided into 4 distinct types. Type 1 (9.5%) is defined as acute trypanosomiasis followed by death and is characterised by a high patency rate on MI and a low PCV. Type 2 (47.6%) is defined as chronic trypanosomiasis showing clinical signs and characterised by a high patency rate on MI, and a low PCV.

Table 14. The differentiation of type of camel trypanosomiasis in herd 1, over the study period as judged by clinical picture, presence of circulating trypanosomes, and haematology.

Type	Clinical picture	No of animals	Percentage of samples positive on mouse inoculation	Mean PCV(%) $\pm$ SE	Mean WBC $\pm$ SE(10^3/cumm)	IHA antibody
1	Acute death	2	50.0.	24.13$\pm$0.62	25.3$\pm$ 1.9	+
2	Chronic showing some emaciation	10	39.0	24.79$\pm$0.58	21.8$\pm$ 1.3	+
3	Chronic showing no symptoms	3	21.2	30.56$\pm$0.22	17.0$\pm$3.6	+
4	Chronic showing no symptoms	6	NIL	27.48$\pm$1.84	13. 1$\pm$0.8	+

Type 3 (14.3%) is defined as chronic trypanosomiasis showing no clinical signs and characterised by a low patency rate on MI and a normal PCV. Type 4 (28.6%) is defined as chronic trypanosomiasis showing no clinical signs and characterised by a nil patency rate on MI, and normal PCV. All types showed the presence of IHA antibody. The differences between the 4 types on total leucocyte count was statistically significant ($F^{3}_{17} = 8.28$; p 0.01)

The levels of IHA titre in all 4 types of camel trypanosomiasis are shown on table 15. This clearly demonstrates that animals in types 1 and 2 have much higher and more frequent levels of antibody than those of types 3 and 4.

6.4.6 Stability of trypanosomiasis in all study herds

There is adequate epidemiological information to comment on the stability of trypanosomiasis in all study herds. The disease is stable at Ngurunit (H2) and Galana (H4) as the point prevalence rate is low and shows little monthly variation, a calfhood resistance is present and a high incidence of IHA antibody is present in all segments of the herds, the strength of which decreases with age.

The disease is unstable in the animals of both herds 1 and 3 as point prevalence rates fluctuate from month to month, calfhood immunity is not as absolute as in H2 and H4 and high levels of IHA antibody do not necessarily occur in all segments of the herd e.g. only 57.2% of the calves in H3.

Table 15. Summary of the frequency of appearance of the different IHA titres in the four categories of camel trypanosomiasis in Herd 1 over the year November 1979 - November 1980.

Type	Type of disease	No. of animals	IHA TITRES									No. obs
			<40	40	80	160	320	640	1280	2560	5120	
1	Acute - death	2	0.09	0.18	0.18	0.05	0.32	0.14	0.05	-	-	22
2	Chronic - positive MI low mean PCV	10	0.18	0.21	0.24	0.10	0.10	0.07	0.06	0.01	0.03	110
3	Chronic - positive MI. Normal mean PCV	3	0.71	0.03	0.23	0.03	-	-	-	-	-	31
4	Chronic-no positive MI. Normal mean PCV	6	0.56	0.25	0.11	0.06	0.02	-	-	-	-	64

6.5. Internal parasitism

6.5.1 Gastro-intestinal strongylosis

6.5.1a Herd incidence

Strongyle eggs were present at very high incidence levels in the adults and immatures of all herds where the majority of the animals were infected to a greater or lesser degree. Few of the calves showed eggs before 4-6 months of age with incidence increasing steadily until 1 year of age, when the majority of animals were infected.

6.5.1b Herd Strongyle egg levels

The mean levels of strongyle worms in each herd at each sampling date are shown on figure 3. These figures have to be accepted with caution due to the differences in herd structure and differences in levels in different age groups (see figures 4-7). Nevertheless, herd levels do represent a trend and do give a measure of pasture contamination

The highest herd mean strongyle egg levels were detected in H1 where a high output was detected between December 1979 and April 1980. After this period, the mean herd level remained between 150 and 450 strongyle eggs/gm until the end of study period. The levels in H2 showed a similar pattern at a lower level. Mean egg output in H3 and H4 remained at low levels throughout their respective periods of study.

Figure 3: Mean fecal egg counts (strongyle eggs / grm) on herd basis in four selected herds of dromedaries

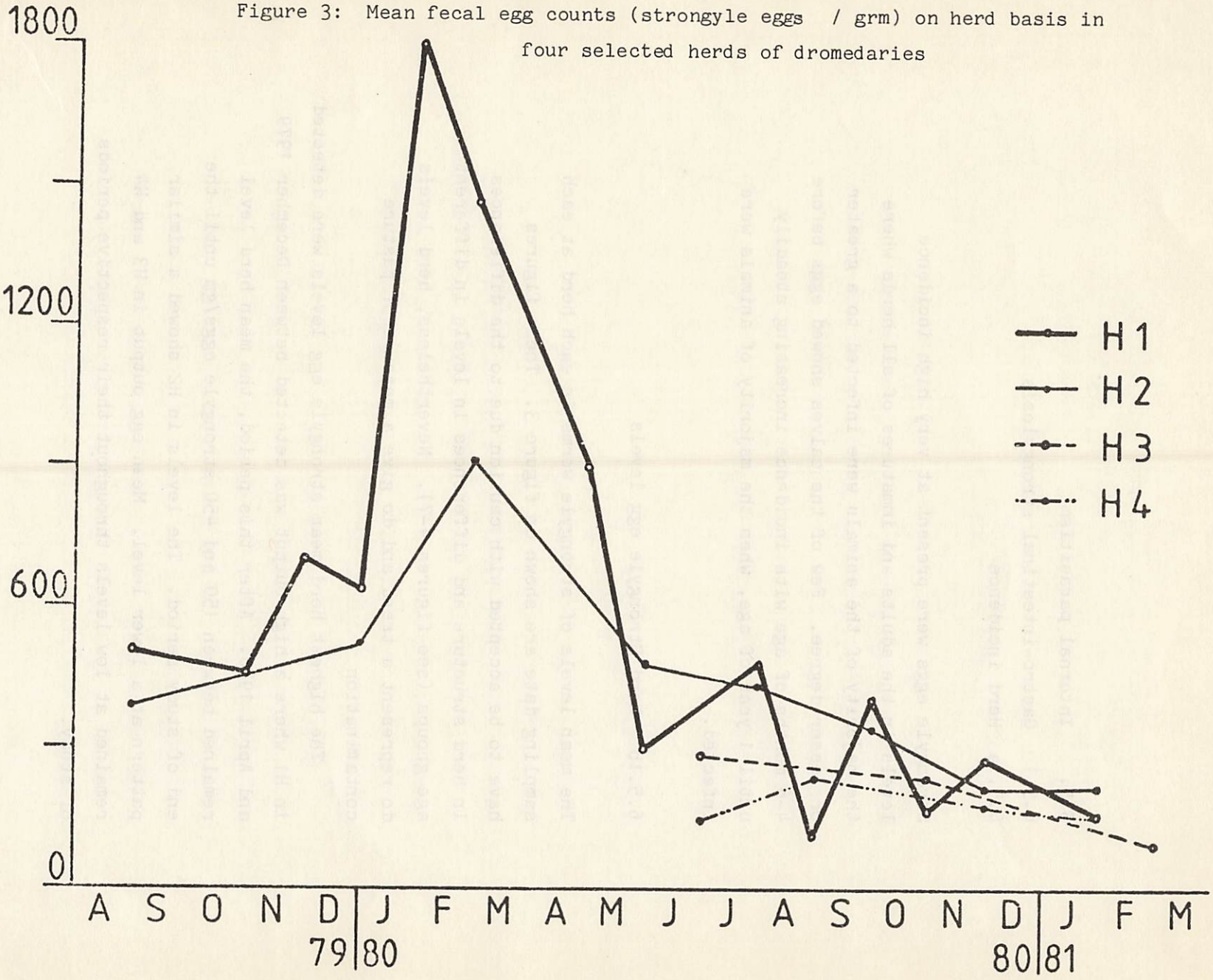


Figure 4: Mean fecal egg counts (strongyle eggs / grm) by age group in herd 1 (IPAL)

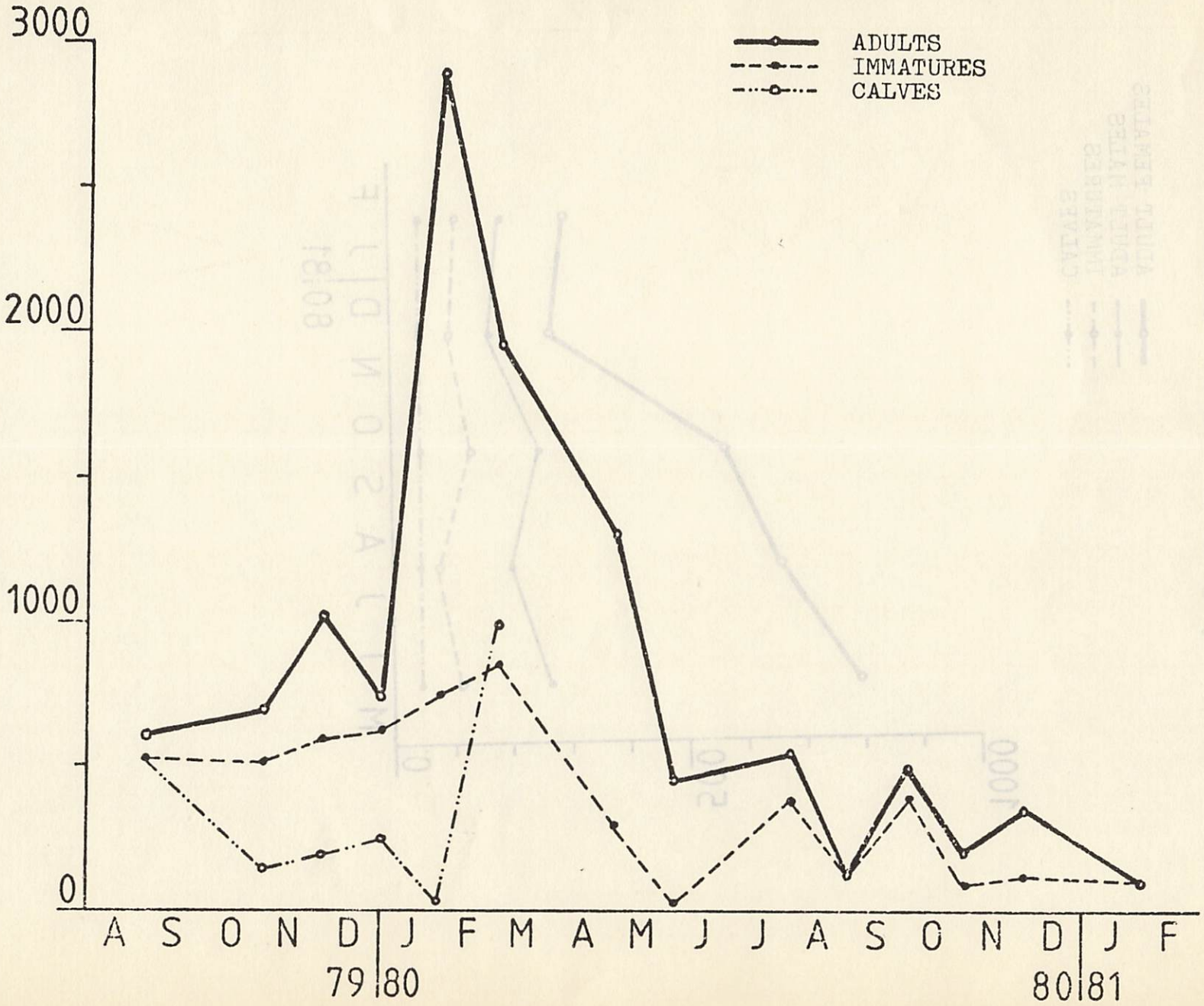


Figure 5: Mean fecal egg counts (strongyle eggs / grm) by age and sex in herd 2 (Ngurunit)

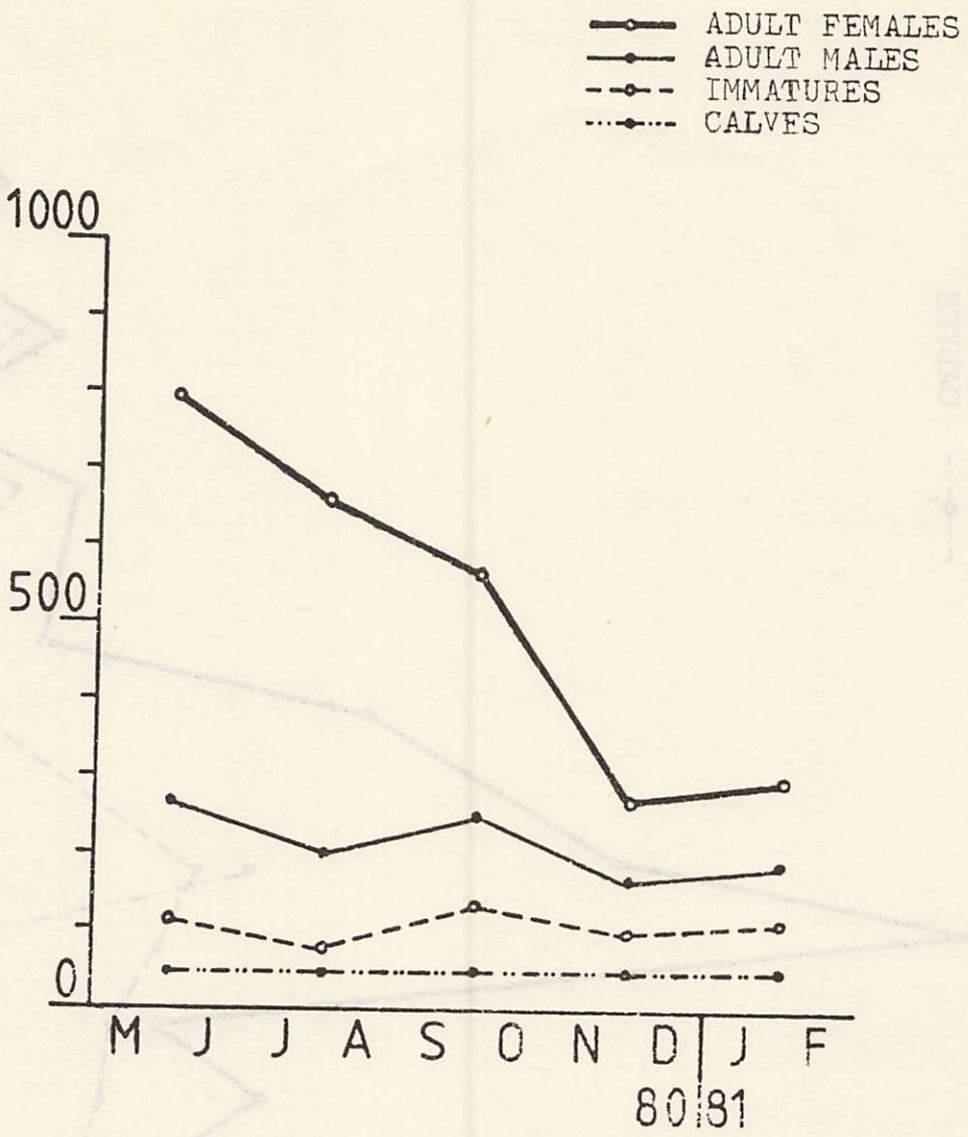


Figure 6: Mean fecal egg counts (strongyle egges / grm) by age and sex in herd 3

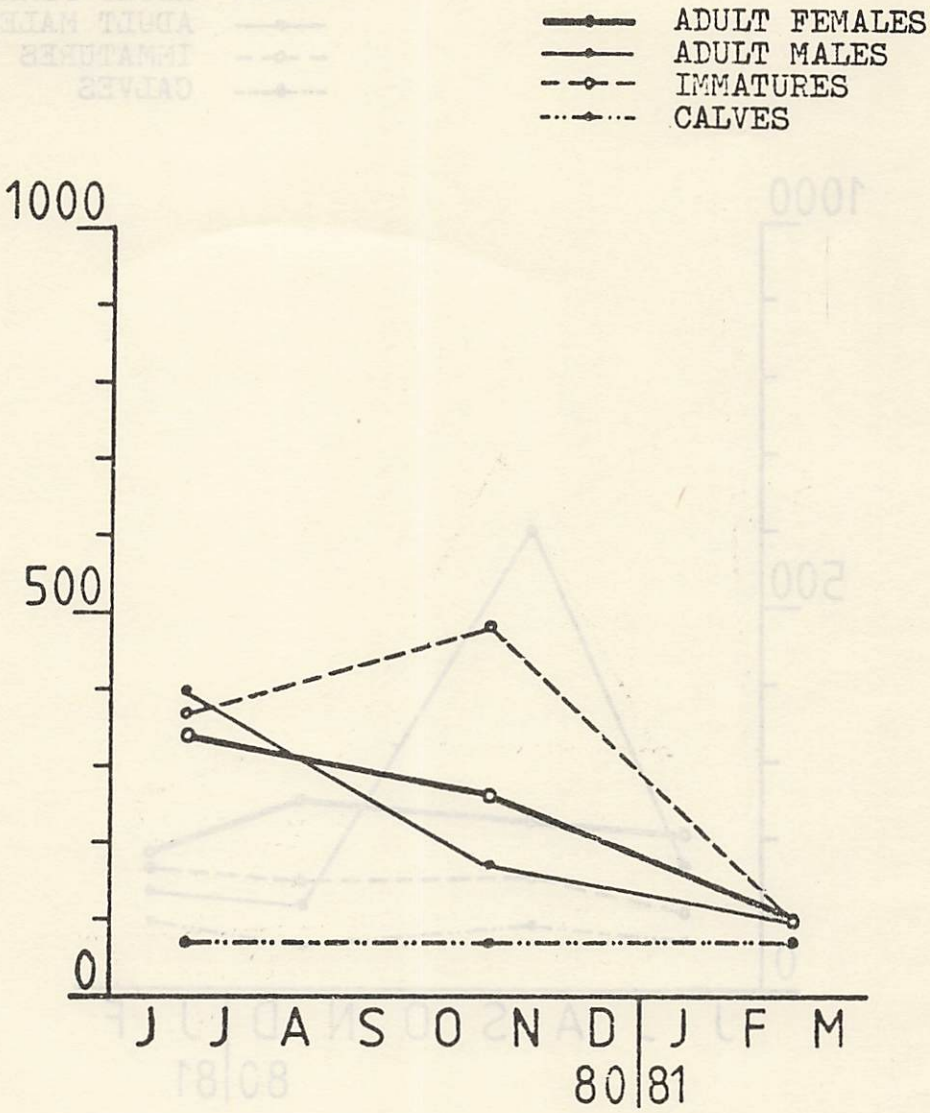
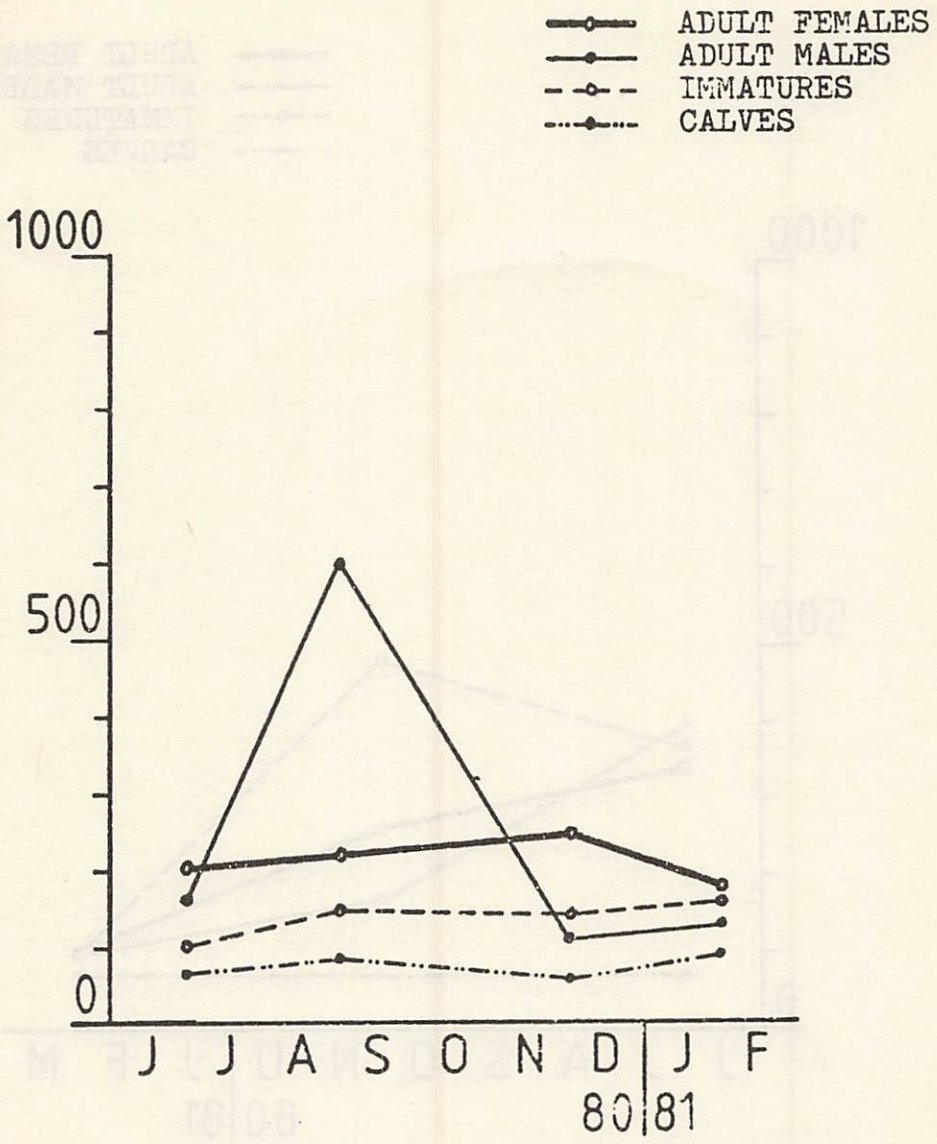


Figure 7: Mean fecal egg count (strongyle eggs / grm) by age and sex in herd 4



6.5.1c Mean strongyle levels with age and sex.

The results of mean strongyle levels in the different age groups in each herd are shown on tables 16-19 and figures 4-7.

Strongyle levels in adult and immature females and adult and immature males are also shown on tables 17-19.

The levels of strongyle eggs in the adults in H1 were much higher than those in the immatures or calves during the period December 1979 to April1980 after which time there was little difference in levels between adults and immatures (figure 4). The lowest levels were observed in the calves from August 1979 to February 1980 after which time they had become immatures.

Much higher levels of egg output were observed in the adult females of H2 over whole period than those observed in the adult males, immatures or calves. This was especially observed from May to November 1980 (figure 5). Egg levels in adult males were consistently higher than immatures, which were in turn higher than calves. No sex differences were observed in the immatures (table 17).

No differences in egg output were observed in the adult females, adult males and immatures of H3 (figure 6) over the whole study period. The calves commenced the period with lower egg levels than the other age groups; however their output showed no difference at the end of the study.

The results from H4 showed a similar pattern to that of H2 but at a much lower level of egg output (figure 7, table 19). An exception is the adult male mean output for August 1980 which was high (610.7 $\pm$ 241 eggs/gm). The high SE does make this value inaccurate.

Table 16. Mean strongyle levels (±SE) at each sampling date in the calves, immatures and adults of herd over the study period.

Age group	1979						1980							1981
	A	O	N	D	J	F	A	M	J	A	S	O	N	J
Calves	430 ±264 n=5	150 ±69 n=5	240 ±40 n=5	300 ±100 n=2	100 ± n=2	1150 ±150 n=2	A L L	C A L V E S		I M M A T U R E S				
Immatures	450 ±322 n=4	450 ±126 n=4	500 ±129 n=4	514 ±164 n=7	743 ±636 n=7	886 ±368 n=7	263 ±118 n=4	100 - n=4	375 ±277 n=4	100 ±35 n=4	325 ±159 n=4	112 ±63 n=4	150 ±61 n=4	137 ±88 n=4
Adults	596 ±206 n=11	655 ±101 n=11	1031 ±281 n=11	814 ±142 n=11	2927 ±984 n=11	1973 ±365 n=11	1317 ±364 n=6	450 ±183 n=6	550 ±169 n=6	108 ±58 n=6	475 ±201 n=6	200 ±72 n=6	383 ±138 n=6	133 ±40 n=6

Table 17. Mean strongyle levels (±SE) at each sampling date in the calves, immatures, adult males and adult females of herd 2 over the study period.

Age group	SAMPLING TIME (MONTHLY) OVER STUDY PERIOD				
	1980				1981
	May	July	September	November	January
Calves	<100	100	100	100	100
	n=7	n=7	n=5	n=6	n=4
Immature females	118	<100	153.1	<100	123
	±28		±79		±34
	n=25	n=19	n=16	n=19	n=15
Immature males	128	117	88	105	133
	±38	±60	±23	±35	±50
	n=16	n=9	n=12	n=10	n=9
Adult females	800	683	567	262	308
	±20	±17	±18	±9	±10
	n=74	n=46	n=52	n=42	n=51
Adult males	260	196	248	152	190
	±7	±8	±17	±5	±11
	n=39	n=25	n=23	n=24	n=24

Table 18. Mean strongyle levels (±SE) at each sampling date in the calves, immatures, adult males and adult females of herd 3 over the study period.

Age group	SAMPLING TIME (MONTHLY) OVER THE STUDY PERIOD		
	1980		1981
	June	October	February
Calves	<100	<100	<100
	n=16	n=18	n=15
Immature females	278	290	<100
	±46	±89	
	n=9	n=4	n=16
Immatures males	475	1187	1033
	±162	±785	±28
	n=6	n=4	n=15
Adult females	332	263	<100
	±45	±83	
	n=41	n=23	n=47
Adult males	406	125	175
	±156	±50	±70
	n=8	n=12	n=6

Table 19. Mean strongyle levels (±SE) at each sampling date in the calves, immatures, adult males and adult females of herd 4 over the study period.

Age group	SAMPLING TIME (MONTHLY) OVER THE STUDY PERIOD			
	1980			1981
	June	August	November	January
Calves	<100	<100	<100	<100
	n=21	n=23	n=31	n=35
Immature females	100	160	132	154
	±21	±48	±39	±34
	n=15	n=10	n=11	n=13
Immature males	106	162	164	181
	±26	±36	±39	±42
	n=16	n=17	n=18	n=13
Adult females	197	221	246	182
	±30	±32	±32	±17
	n=58	n=61	n=61	n=60
Adult males	167	611	109	133
	±35	±241	±19	±40
	n=18	n=14	n=11	n=12

6.5.1d Larval cultures

The results of larval cultures of 36 faecal samples containing high levels of strongyle eggs from all herds are shown on table 20. This shows that the abomasal worm *Haemonchus contortus* was the most common strongyle nematode observed in all camel herds. The small intestinal nematode *Trichostrongylus* and the large intestinal nematode belonging to the genus *Oesophogostomum* were also present at much lower incidence levels.

6.5.2 Other internal parasites

The incidence of the nematodes *Strongyloides* and *Trichuris*, the cestode *Moniezia* and the protozoan coccidia in all herds over their respective study periods is shown on table 21.

Trichuris is common in H1 and H2, is relatively uncommon in H3 and was not detected in H4. The nematode is found in all segments of the herd appearing to be less common in calves than other segments.

Strongyloides was found in all herds being more common in calves, although it occurred in all age groups except in H3.

The incidence of *Moniezia*, *Ascaris* and coccidia was low, occurred in all herd segments and in most of the herds. Coccidial oocysts and *Ascaris* eggs were not detected in H1, and *Moniezia* eggs were not detected in H4.

Table 20. Identification of strongyle nematodes from larval cultures of 36 faecal samples obtained from all herds.

Herd No.	No. of samples	PERCENTAGE INFECTED WITH		
		Haemonchus	Trichostrongylus	Oesophagostomum
1	15	93	33	20
2	10	90	30	20
3	5	100	NIL	NIL
4	6	100	NIL	NIL

Table 21. The incidence of the eggs of *Trichuris*, *Moniezia*, *Strongyloides* and coccidial oocysts in faecal samples from all herds over the study period.

		Adults				
		Male	Female	Immatures	Calves	Total
Trichurus	1	100.0	33.3	75.0	60.0	57.1
	2	56.1	57.5	23.9	14.3	46.6
	3	13.3	1.9	7.3	3.6	5.1
	4	0.0	0.0	0.0	0.0	0.0
Strongyloides	1	0.0	22.2	75.0	40.0	33.3
	2	7.3	7.5	10.9	14.3	8.6
	3	0.0	0.0	0.0	14.3	2.9
	4	10.5	1.4	5.9	31.4	12.0
Coccidia	1	0.0	0.0	0.0	0.0	0.0
	2	0.0	2.5	0.0	0.0	1.1
	3	6.7	0.0	2.4	0.0	1.5
	4	0.0	1.4	11.8	0.0	2.9
Moniezia	1	33.3	0.0	0.0	40.0	14.3
	2	2.4	0.0	5.5	0.0	2.3
	3	0.0	0.0	0.0	3.6	0.7
	4	0.0	0.0	0.0	0.0	0.0
Ascaris	1	0.0	0.0	0.0	0.0	0.0
	2	0.0	1.3	4.3	0.0	1.7
	3	0.0	0.0	0.0	0.0	0.0
	4	0.0	0.0	0.0	0.0	0.0

6.5.3 Haemoparasites

452 blood films were examined from camels in all herds. No haemoparasites were observed.

6.6 External Parasitism

6.6.1 Ticks

6.6.1a Herd incidence levels

All camels in all herds were infested with ticks. Differences were present in terms of tick loads, predominant species and preferential attachment sites.

6.6.1b Herd tick loads

The mean herd tick loads over the study period are shown on table 22 and figure 8. Very low tick loads were observed in H3 with the highest observed in H1. The loads in H2 and H4 fluctuated between those of H1 and H3. No obvious seasonal effects could be observed possibly reflecting the different tick control strategies used in each herd.

6.6.1c Species of tick and relative abundance

The identification of 6,502 tick samples obtained from all herds on the sampling dates are shown on tables 23-26. The most common ticks identifed in H1 were adult *Hyalomma dromedarii*, *H. rufipes* *Rhipicephalus pulchellus* and *Amblyomma gemma*. All species were using the camel as a food source as gravid females were present. The relative abundance of these ticks varied according to time of year with *Hyalomma dromedarii* being the most common from August - December 1979 and again in November 1980.

Table 22. Mean total tick body loads (± SE) on a herd basis over their respective study periods.

Herd No.	DATE OF COLLECTION BY MONTH													
	1979					1980							1981	
	J	F	M	A	M	J	J	A	S	O	N	D	J	F
1	53.5 ±5.8	59.9 ±8.0	57.5 ±5.3	35.6 ±4.1	59.9 ±10.0		72.7 ±6.6	100.3 ±10.2	90.3 ±6.6	120.9 ±8.4	83.2 ±5.3		62.8 ±8.0	
2			79.8 ±9.8		47.8 ±3.3	41.2 ±3.8	46.0 ±3.2		100.3 ±5.5				42.6 ±1.6	
3						3.7 ±0.5				4.4 ±0.6				7.8 ±0.6
4						42.5 ±2.8		11.5 ±0.7		21.5 ±1.6			63.1 ±2.5	

Figure 8: Mean total tick body counts on herd basis in four selected herds of dromedaries

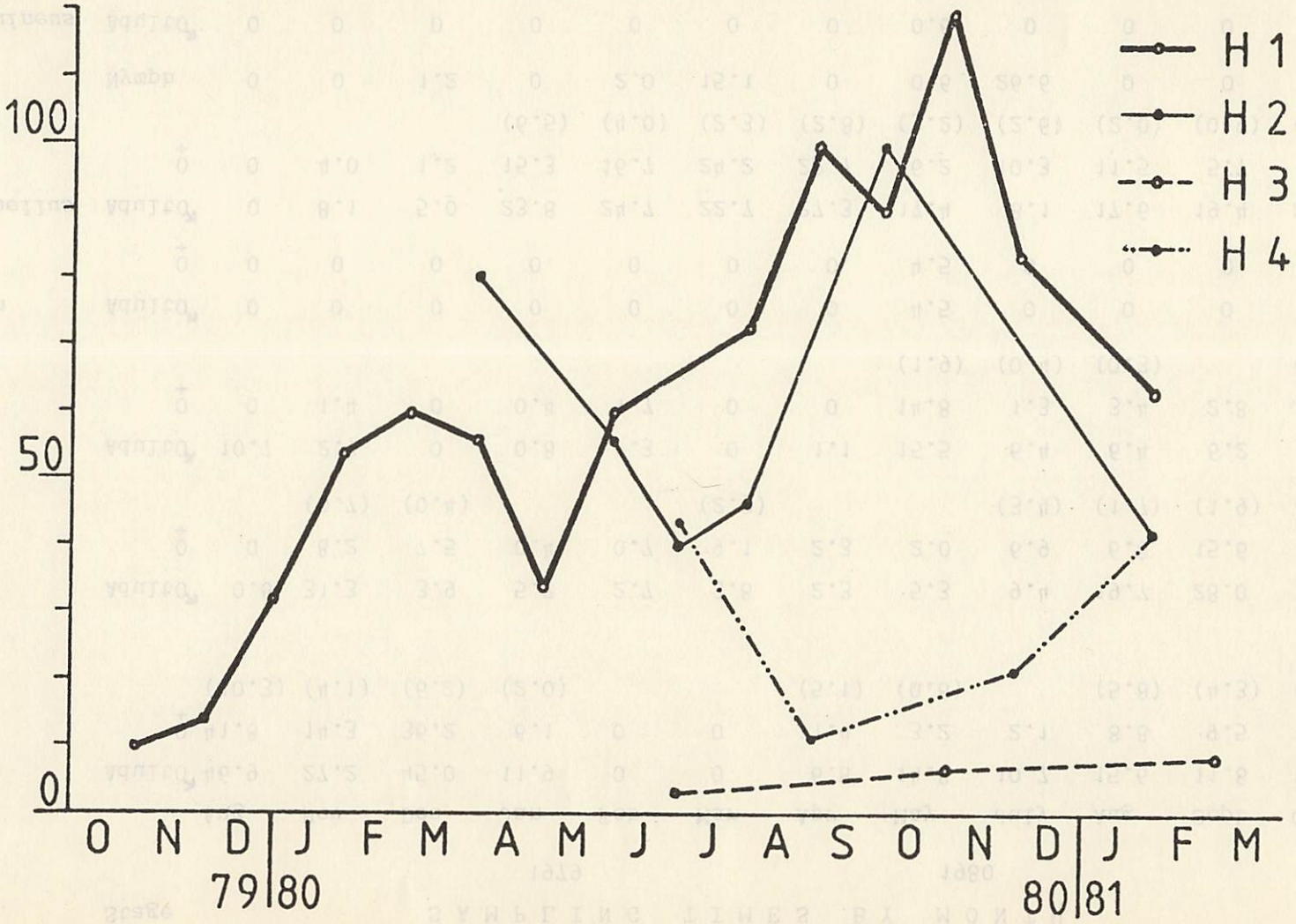


Table 23. Relative abundance of tick species and tick stage (%) of all ticks collected from animals of Herd 1 over the study period. Percentage of engorged females in brackets.

Tick species	Stage	SAMPLING TIMES BY MONTH												
		1979			1980									
		Aug	Nov	Dec	Jan	Feb	Mar	Apr	May	July	Aug	Sept	Oct	Nov
Hyalomma dromedarii	Adult♂	46.9	27.2	45.0	11.9	0	0	6.8	14.8	10.7	15.6	11.8	16.0	26.3
	♀	41.8	14.3	36.2	6.1	0	0	11.4	3.2	2.1	8.8	9.5	10.9	13.8
	+	(10.3)	(4.1)	(6.2)	(2.0)			(5.1)	(0.6)		(5.8)	(4.3)	(3.5)	(3.0)
Hyalomma rufipes	Adult♂	0.6	31.3	3.9	5.7	2.7	3.8	2.3	5.3	9.4	19.7	28.0	19.1	14.7
	♀	0	8.2	7.5	0.4	0.7	9.1	2.3	2.0	6.9	6.8	15.6	16.7	11.1
	+		(0.7)	(0.4)			(2.3)			(3.4)	(1.7)	(1.9)	(3.5)	(2.7)
Hyalomma truncatum	Adult♂	10.7	2.7	0	0.8	1.3	0	1.1	15.5	6.4	6.4	6.2	1.9	3.9
	♀	0	1.4	0	0.4	1.7	0	0	14.8	1.3	3.4	2.8	1.2	3.3
	+								(1.9)	(0.4)	(0.3)		(0.4)	(0.6)
Hyalomma marginatum	Adult♂	0	0	0	0	0	0	0	4.5	0	0	0	0	0
	♀	0	0	0	0	0	0	0	4.5	0	0	0	0	0
	+													
Rhipicephalus pulchellus	Adult♂	0	8.1	5.0	23.8	24.7	22.7	27.3	17.4	8.1	17.6	19.4	23.7	11.0
	♀	0	4.0	1.2	15.3	16.7	24.2	22.7	16.2	10.3	11.5	5.7	8.9	8.7
	+				(6.5)	(4.0)	(2.3)	(2.8)	(3.2)	(2.6)	(2.0)	(0.9)	(3.5)	(2.7)
	Nymph	0	0	1.2	0	2.0	15.1	0	0.6	26.6	0	0	0	0
Rhipicephalus sanguineus	Adult♂	0	0	0	0	0	0	0	0.6	0	0	0	0	0
	♀	0	0	0	0	0	0	0	0.6	0	0	0	0	0
	+													
Amblyomma gemma	Adult♂	0	1.4	0	21.7	27.3	12.9	18.2	0	8.1	3.0	0.5	1.2	2.4
	♀	0	1.4	0	13.9	24.0	12.2	7.9	0	9.9	1.4	0.5	0.4	4.5
	+				(4.1)	(2.0)	(2.3)	(1.1)		(3.4)	(0.3)	(0.5)		(0.6)
Nymphs (unidentified)		0	0	0	0	0	0	0	0	0	5.8	0	0	0
TOTAL NO IN SAMPLE		309	147	240	244	150	132	176	155	233	295	211	257	334

Table 24. Relative abundance of tick species and tick stage (%) of all ticks collected from animals of herd 2 over the study period. Percentage of engorged females in brackets.

Tick species	Stage	SAMPLING TIMES BY MONTH								
		1979			1980					1981
		Aug	Dec	Feb	Mar	May	July	Sept	Nov	Jan
Hyalomma dromedarii	Adult♂	37.1	39.0	34.6	32.8	44.8	38.9	11.5	31.5	29.6
	♀	10.2	15.6	11.9	21.1	12.1	8.7	5.5	18.5	12.4
		(1.3)	(3.1)	(1.5)	(8.6)	(2.7)	(1.6)	(2.7)	(2.5)	(4.4)
Hyalomma rufipes	Adult♂	40.2	29.5	35.5	23.4	36.5	33.3	52.4	28.5	25.2
	♀	10.0	14.4	14.1	20.3	3.8	14.1	25.7	15.5	12.8
		(1.0)		(2.5)	(4.7)	(1.4)	(1.0)	(4.4)	(3.5)	(1.8)
Hyalomma truncatum	Adult♂	1.0	1.5	1.0	0.8	2.0	2.0	3.3	3.5	1.3
	♀	0.5	0	0.9	0.8	0.5	0.3	1.6	1.5	0.4
Rhipicephalus pulchellus	Adult♂	1.0	0	1.0	0.8	0	1.3	0	1.0	2.6
	♀	0	0	1.0	0	0	0.7	0	0	5.3
										(1.8)
Rhipicephalus pravus	Adult♂	0	0	0	0	0.3	0	0	0	0
	♀	0	0	0	0	0	0	0	0	0
Amblyomma gemma	Adult♂	0	0	0	0	0	0	0	0	1.3
	♀	0	0	0	0	0	0	0	0	0.7
Nymphs (unidentified)					0	0	0.7	0	0	8.4
TOTAL NO IN SAMPLE		203	251	180	128	556	297	183	200	226

Table 25. Relative abundance to tick species and tick stage (%) on all ticks collected from the animals of herd 3 over the study period.

Tick species	Stage	SAMPLING TIMES BY MONTH		
		1980		
		June	October	February
Hyalomma dromedarii	Adult♂	0	0.9	0.5
	♀	0	0.9	0
Hyalomma rufipes	Adult♂	4.9	5.2	8.2
	♀	2.7	1.7	6.4
		(1.1)		(0.8)
Hyalomma truncatum	Adult♂	1.6	0	0.5
	♀	1.6	0	
Hyalomma marginatum	Adult♂	0.5	0	0
	♀	0	0	0
Rhipicephalus pulchellus	Adult♂	26.8	39.6	39.2
	♀	15.3	35.3	25.9
		(3.2)	(4.3)	(4.1)
	Nymph			1.0
Rhipicephalus evertsi	Adult♂	21.8	6.9	12.7
	♀	13.0	6.0	4.3
		(1.7)		(0.5)
Rhipicephalus simus	Adult♂	1.1	0.9	0
	♀	0.5	0	0

Table 25 continued:

Tick species	Stage	SAMPLING TIMES BY MONTH		
		1980		1981
		June	October	February
Amblyomma gemma	Adult♂	1.0	0	0.5
	♀	0	1.7	0
Amblyomma variegatum	Adult♂	1.0	0	0
	♀	0	0	0
Boophilus decoloratus	Adult♂	4.4	0	0
	♀	3.8	0.9	0.8
		(2.2)		
TOTAL NO TICKS IN SAMPLE		183	116	393

Table 26. Relative abundance of tick species and tick stage (%) on all ticks collected from animals of herd 4 over the study period.

Tick species	Stage	SAMPLING TIMES BY MONTH			
		1980			1981
		June	August	November	January
Hyalomma rufipes	Adult♂	23.1	15.4	21.0	34.4
	♀	11.2	10.3	12.0	11.5
	+	(1.7)	(1.7)		(2.7)
Hyalomma truncatum	Adult♂	3.3.	0	1.0	0.5
	♀	2.6	0	1.0	0.5
	+				
Rhipicephalus pulchellus	Adult♂	26.4	44.4	38.0	25.3
	♀	15.8	26.5	18.0	21.3
	+	(3.3)	(11.1)		(10.4)
Amblyomma gemma	Adult♂	10.2	2.6	7.0	3.8
	♀	5.6	0.8	2.0	2.2
	+				(0.5)
Amblyomma variegatum	Adult♂	1.8	0	0	0
	♀	0	0	0	0
	+				
Nymphs		0	0	0	0.5
TOTAL NUMBER IN SAMPLE		303	117	100	183

R. pulchellus was common throughout most of the study period being most prevalent from January to April 1980. *A. gemma* was only common over the period January to April 1980. *H. rufipes* was present throughout the study period but was more prevalent from August to November 1980. Nymphal stages were present in very low concentrations with nymphs of *H. dromedarii* and *R. pulchellus* being positively identified.

Two tick species only predominated in H2 over the study period, namely *H. dromedarii* and *R. pulchellus* both of which used the camel as a food source with the former predominating over most of the study period. Nymphal stages were detected only in July.

The tick species from the camels of both H3 and H4 show great diversity (tables 25 and 26) and most readily feed on cattle. The predominant species in both areas was *R. pulchellus* with *R. evertsi* and *H. rufipes* being important species in H3 and H4 respectively.

The toxin producing tick *H. truncatum* was detected in all herds at low levels.

6.6.1d Incidence of large and small mouthpart ticks on all herds.

The percentage of ticks with large or small mouthparts in total samples from all herds is shown on table 27. Large mouth-part ticks predominated in H1 and H2. They were in approximately equal proportions in H4 with small mouthpart ticks predominating in H3.

6.6.1e Tick attachment sites

The results of a small study on tick preferential attachment sites in H1 on one date is shown on table 28. *H. dromedarii* infests mainly the nose and ear. *R. pulchellus* attaches all over the body; *A. gemma* attaches mainly in the inguinal area; and *H. truncatum* was detected in significant quantities in the dewlap and inguinal area.

Table 27. The percentage of small and large mouthpart ticks collected over the whole study period in all the herds.

Herd No.	No. ticks in sample	% small mouthpart ticks	% large mouthpart ticks
1	2883	26	74
2	2224	3	97
3	692	82	18
4	703	51	49

Table 28. A study on attachment sites of tick species at one collection (23/4/80) on the camels of herd 1.

Site	No. in sample	Tick species	Percentage of total
NOSE	102	*Hyalomma dromedarii*	69.8
		Rhipicephalus pulchellus	30.3
		Amblyomma gemma	1.9
EAR	15	*Hyalomma dromedarii*	93.0
		Rhipicephalus pulchellus	7.0
INGUINAL AREA (including scrotum)	45	*Rhipicephalus pulchellus*	45.3
		Amblyomma gemma	25.0
		Hyalomma rufipes	17.2
		Hyalomma truncatum	12.5
FORE LEGS	49	*Rhipicephalus pulchellus*	75.5
		Hyalomma truncatum	13.2
		Hyalomma dromedarii	9.4
		Amblyomma gemma	2.1

6.6.1f Individual tick loads on camels over time

An analysis of mean tick loads over the study period on all the camels of H1 is shown on table 29. An age effect is indicated. This result was confirmed in the other herds with tick numbers increasing with age.

There is no evidence for any development of resistance to tick attachment in any animal.

6.6.2 Mites

Mange due to *Sarcoptes scabei var cameli* was common in all study herds. A quantitative study on this disease was not undertaken. However herd records showed that the disease predominated in the calf and immature segments of all herds.

6.6.3 Camel fly

The camel fly (family *Hippoboscidae*) was a common ectoparasite attacking the camels of H1 and H2 . A quantitative study on this parasite was not undertaken.

6.7 Virus diseases

6.7.1 Camel pox

The results of a special study on the epidemiology of this disease is shown in addendum 3 (see section 14.3)

6.7.2 Foot and Mouth Disease (FMD)

The results of assays for FMD antibodies from 275 sera collected from camels in H1-3 are shown on table 30. The point prevalence rates were very low with no reactors being detected in H1. Antibody to FMD virus types O, C and SAT2 were detected in H2, and to type SAT2 in H3. The antibody titres in the positive samples were low.

Table 29. The mean individual tick loads in the animals of Herd 1 over the study period.

Animal No	Age group	Mean tick load ± SE	Animal No	Age group	Mean tick load ± SE
20	Calf	37 ± 1*	7	Adult	89 ± 11
21	"	42 ± 8	8	"	81 ± 10
10	Immatures	60 ± 9	9	"	71 ± 9
11	"	76 ± 12	14	"	86 ± 13
12	"	49 ± 9	15	"	89 ± 19
13	"	65 ± 7	16	"	79 ± 10
17	"	43 ± 6	4	"	79 ± 12
18	"	59 ± 8	5	"	102 ± 16
19	"	89 ± 14	6	"	75 ± 8
2	Adult	60 ± 7	22	"	70 ± 7
3	"	103 ± 18			

Nos 17, 18 and 19 became Immatures during the study

* 3 observations

Table 30. The results of assays for FMD antibodies from sera collected from Herd 1, Herd 2 and Herd 3 using metabolic inhibition test.

Herd No.	No. samples	POINT PREVALENCE(%)	OF ANIMALS POSITIVE TO FMD VIRUS TYPE			
		O	A	C	SAT1	SAT2
1	39	0	0	0	0	0
2	151	0.66	0	0.66	0	1.32
3	85	0	0	0	0	2.35

Nevertheless, there is adequate evidence from these results that the virus could cycle in camels and pathogenesis studies would be required to examine susceptibility in this species.

6.7.3 Rinderpest

The analysis of 398 sera from the 4 herds showed that the incidence of rinderpest antibodies in camels is very low. Only one sample showed antibodies (in H2), the remaining 397 samples being negative.

6.8 Bacterial Diseases

6.8.1 Brucellosis

The point prevalence rates of camels having antibodies to *Brucella abortus* in H1-3 are shown on table 31. The antibody prevalence varied from 6% in H3 to up to 38% in H1 in April 1980. The prevalence in H2 varied between these two extremes. These results indicate that brucellosis is a relatively common disease in the camels of all 3 herds especially in the adult female segments. The importance of this disease in terms of loss of fertility and abortion will require further study.

6.9 Miscellaneous diseases

A number of other diseases have been observed. No quantitative studies have been undertaken and these diseases will merely be mentioned.

Table 31. Summary of point prevalence rates (%) of camels having antibodies to *Brucella abortus* in samples collected from herd 1, herd 2 and herd 3.

Herd No.	Sample No.	C O L L E C T I O N T I M E B Y M O N T H						
		1979			1980			
		December	February	March	April	June	July	August
1	21		33.3	33.3	38.0			9.5
2	116	36.5		34.0			14.5	
3	93					6.4		

Necrotic dermatitis
Pneumonia
Nasal muiasis (caused by *Cephalopsis titillator*)
Enteritis
Generalised swelling of lymph nodes

6.10 Postmortem and mortality results

6.10a Postmortems

Few postmortems were undertaken in this study. This was mainly due to low mortality rates in H3 and H4 and unavailability of animals in H2. Three postmortems were undertaken on camels in H1.

The results of postmortems and suspected causes of mortality in all herds over the study period are summarised on table 32. These indicate that the major causes of death appear to be calf mortality in H2 probably due to competition with man for milk, trypanosomiasis in all herds and predators in H2 and H4.

6.11 Effect of Panacur on strongyle egg levels in a small group of camels

The results of monthly faecal monitoring of the two group of camels in H1 (see section 6.11) to test the effects of Panacur on egg levels are shown on table 33. These show that Panacur every 6-8 weeks greatly reduces the level of faecal egg output.

Table 32. Summary of postmortem results and mortality causes in the camels of all herds over the study period.

Herd No.	Number of postmortems undertaken	Camel age and suspected cause of death	% Mortality
1	3	(Immatures - 2 Trypanosomiasis (acute)	9.5
		(Calves - 1 Enteritis	4.7
2	*	Number not recorded — Malnutrition; pneumonia; Trypanosomiasis; predators	
3	4+	(Adults - 3 Trypanosomiasis	2.2
		(Calf - 1 Camel pox	0.7
4	7+	(Adults - 2 Trypanosomiasis	1.1
		(Adults - 1 Mastitis	0.6
		(Calves - 2 Malnutrition	1.1
		(Calves - 2 Predators	1.1

* None undertaken; cause of death deduced from history.

\+ PM's undertaken by owners and results recorded.

Table 33. Mean faecal egg counts (strongyle eggs/gm) in both Panacur treated and untreated groups both before and after treatment.

Sampling period	No. of animals	Treatment	Mean strongyle eggs/gm (6 samples/animal) Before treatment	Mean strongyle eggs/gm (9 samples/animal) After treatment
August 1979 - January 1980	10	NONE	783	342
February 1980 - January 1981	11	PANACUR (7.5mg/kg monthly from 2/2/80-1/1/81)	957	<100

Table 34. The results of screening 398 camel sera for the presence of rinderpest antibodies by the serum neutralisation test.

Herd No	Number of sera	Number Positive	Percentage Positive
1	27	NONE	0
2	185	1	0.5
3	63	NONE	0
4	123	NONE	0

7. DISCUSSION

There have been few systematic studies on the epidemiology of diseases of camels. This study has shown some of the benefits of a long term study on a large group of camels of mixed age samples over a relatively long period of time.

A number of important diseases in camels in four different areas of Kenya have been identified. The economic effects of these diseases are difficult to measure due to their interactions with each other and the effects of climate, nutrition, management and many other possible factors. Richard (1979) attempted to classify camel diseases by economic effect (see section 4.4). This study confirms the importance of trypanosomiasis, internal parasitism and mange. However malnutrition, especially in calves, and camel pox are possibly more important than indicated by Richard. Malnutrition was probably the main cause of mortality in calves in H2. Camel pox also seems to be an important cause of calf mortality. The effects of ticks are uncertain and are not mentioned in Richard's classification. They are probably more important in H1 and H2 than in H3 and H4 as loads were heavier in the former which also contained a majority of large mouthpart ticks.

The studies on haematology indicated significant differences between herds (table 8). The animals of H4 showed normal PCV and total WBC counts indicating good herd health. The remaining herds showed anaemia and leucocytosis to a greater or lesser degree. The most anaemic herd was H1 and the herd with the highest mean WBC was H3. These results indicate that generalised disease was present in H1, H2 and H3 influencing the blood profiles.

The red blood cell profiles indicated the presence of a microcytic anaemia (low PCV, normal Hb and RBC levels). The white blood cell profiles showed the presence of a leucocytosis mainly characterised by a lymphocytosis and some eosinophilia. These findings indicate chronic parasitism associated with constant blood loss and sustained immunological stimulus. The former could be caused by internal or external parasites, and the latter by trypanosomiasis which because of antigenic variation constantly stimulates the immune response.

Trypanosomiasis has been identified as the most important disease of camels in all the study herds. The disease was endemic in all areas at varying degrees of stability. The most important trypanosome identified was *T. evansi* as expected, with *T. brucei* and *T. congolense* also present. The latter two trypanosomes are tsetse fly dependent. Tsetse flies occurred in the study area of H4 and could play a much more important role in camel trypanosomiasis if camels continue to move south. The herds in which the disease was stable were 2 and 4 having a calfhood immunity, a low, non-fluctuating, point prevalence rate and a high incidence of IHA antibody in all age groups of the herd. The disease was unstable in H1 and H3. The optimum for any potentially dangerous disease is to create a stable situation in which the parasite lives in harmony with its host. Some diseases can be manipulated so that a stable situation is created (Mahoney and Ross, 1972). No treatment or control of such diseases occurs within the stable areas, but takes place only at the periphery.

This approach is not entirely applicable to camel trypanosomiasis as the parasite itself is so unstable. Nevertheless block treatment of herds such as in H2 and H4 may be contra-indicated as it may upset stability. Individual treatment of clinically ill animals is suggested as the alternative strategy. This approach is being conducted at present in H4 with beneficial results. The treatment philosophy is different in unstable herds with fluctuating high PPR's (H1) or when an actual epidemic is occurring (H3). In these circumstances, block treatment may be justified to bring the disease under control in susceptible animals. It is thus important to recognise disease stability or instability in camel herds before drugs are administered.

The categorisation of camel trypanosomiasis into type (table 14) is important in terms of estimating potential economic effect. In H1, type 2 was identified as being most common which combined with type 1 were 57% of the animals. Severe economic effects of the disease could be expected in this group, namely, mortality, loss of weight and abortions. In herds where the disease is stable, the % of types 3 and 4 would be expected to be much higher as is the case in H2 and H4, and the economic effects would be expected to be correspondingly less.

The results in addendum 2 clearly shows that only 2 drugs are active against *T. evansi* namely Antrycide and Suramin. The former is no longer manufactured and the latter is the oldest trypanocide available having been in use for 60 years. The situation for *T. congolense* is little better as again only 2 drugs are active namely Antrycide and Samorin. Berenil is very toxis for camels.

The drug situation is therefore bleak and great care should be exercised in their use as drug resistance is an ever present danger.

The results from the studies on internal parasites show that the voracious blood sucker *Haemonchus contortus* is by far the most important pathogen. Strongyle egg levels were very low in H3 and H4 and haemonchosis is not a problem in these herds. The disease is however a problem in both herds within the IPAL project area where significant age effects were observed. The high levels of eggs in the adult female segment of H2 suggest a stress factor caused by nomadic management where there is great demand for camel milk. A periparturient egg rise has been recorded in other animals (Darvill *et al* 1978) and this also probably occurs in camels. The suckling time of camel calves reflects in their lower Strongyle egg and high *Strongyloides* levels. Seasonal effects on egg levels were minimal which is surprising and may reflect the lack of rain throughout the study period.

A number of drugs are available to treat internal parasites of domestic livestock. The strategy of treatment is crucial to maximise economic return. Strategies should be made for each area by monitoring faecal egg output and thereby ascertaining frequency rate for treatment.

Biologically, the camel is an acarologists paradise. However, this is beyond the terms of reference of this report. Ticks were present in significant numbers on the animals of all herds except H3. The most important tick, economically, is probably *H. dromedarii* which infests mainly the nose causing intense irritation and inflammation. The failure to find haemoparasites in the animals of any herds demonstrates that the tick effect mainly mechanical.

The control of ticks is simple but laborious requiring constant application of acaracide. Monthly application (as in H1) is of limited value whereas weekly application (as in H3) reduces tick load to acceptable levels.

Mange is also easily controlled and is an important disease to eliminate from a herd. Acaracide has to be rubbed into the affected area. The disease occurs principally in young stock and attention should be paid to this age group.

The studies on camel pox clearly again demonstrate the benefits of using serology to understand epidemiology. The disease is endemic in all the study herds and animals are probably under continuous low grade virus attack. Recovered animals are thought to carry the virus for long periods. Vaccination of calves in herds where the disease is endemic is recommended as this herd segment is more susceptible. An attempt should be made to persuade a biological company to prepare a camel pox vaccine.

Camel pox is the most important virus disease of camels causing calf mortality and severe clinical disease. The presence of rinderpest antibodies in one animal of H2 confirms earlier work (Singh and Ata, 1967; Taylor 1968; Richard 1975). However the very low point prevalence rate indicates that camels are not important in the epidemiology of rinderpest. The level of positive reactors to foot and mouth disease indicates that further laboratory studies are required to examine the possible role of this species as a virus reservoir. The camel probably plays a minor role in the epidemiology of these important cattle diseases.

The large number of reactors in all herds to *B. abortus* indicates that the camel maybe an important reservoir of this zoonosis. The role of this disease in camel abortion is uncertain and would require careful experimental work to elucidate. Abortions in nomadic camel herds are common (see H2, table 5) and the most likely causes are trypanosomiasis or brucellosis.

Mortality rates were only high in H2 especially in the calf segment. However trypanosomiasis was identified as a cause of death in all herds. Predators are a significant cause of death in camels confirming the observations of Kruuk (1980). Unless high abortion and calf mortality rates can be controlled, productivity of camel herds will be severely restrained (as in H2).

The main factors in the control of the principal camel diseases identified in this study are management and nutrition. The management of H4 shows that the effects of disease can be easily controlled by good management incorporating a simple veterinary input, sound grazing practice and an effective calf milking system. Similar practices are undertaken in H3 where trypanosomiasis is a temporary problem. Both these herds are productive and increasing 20-30% per annum. The management of H1 by Dr. Field shows that similar results should be possible in the IPAL study area and thus the main challenge is to create productive pastoral camel herds.

The main failures in this study are the lack of postmortem material due to minimal surveillance and a failure to collect samples for bacterial culture as the study areas were too far from a suitable microbiology laboratory. Some important diseases were therefore probably not identified. Nasal myiasis, lung worms, hydatidosis, salmonellosis and pasteurollosis could fall into this category.

8. RECOMMENDATIONS

8.1 A major study must be conducted on restraints to offtake of domestic stock in the IPAL study area. The success of IPAL is dependent on the production of an overall management programme for the whole study area. One of the most important components of such a programme is efficient offtake of livestock to remove the excessive pressure on the pasture which exists in some key areas. The findings of this report are of limited value unless they can be applied in an environment where production potential is not inhibited by an excess stocking rate. The concept of producing greater numbers of unthrifty animals is untenable. Offtake studies are especially important with respect to sheep, goats and cattle. Recommendations 8.2 - 8.6 do not seem justified unless 8.1 is thoroughly undertaken.

8.2 There is little doubt that camel productivity can be increased in nomadic herds with suitable management and veterinary inputs. A study on a nomadic herd under such a system is therefore suggested. Suitable regimes to control trypanosomiasis, haemonchosis, ticks, mange, camel pox and brucellosis should be incorporated. Management practices such as mating, calf milk supply and grazing pressure should be examined and improved. The epidemiology of disease over a period (minimum of 2 years, preferably longer) could be studied in a control herd using the techniques described in this report. Production parameters, such as abortions, calf birth weight, calf mortality, immature and adult mortality, growth rates in all herd sectors and milk production in females should also be measured. This type of study should provide information as to whether increased production in pastoral herds can be achieved in a manner which is economical and proactical.

This study would require the establishment of a simple diagnostic laboratory in the project area. Ngurunit would seem the obvious location. The laboratory should be capable of monitoring the presence of trypanosomiasis, strongyle egg levels and packed cell volume in the study herd.

Other procedures would include tick identification, total worm counts and serum storage. This laboratory would alleviate the necessity of transporting samples to Nairobi and thus save expense. A veterinarian would be required for the laboratory which could also service other livestock sectors of the project.

8.3 Trypanosomiasis has been identified as the most important disease of camels in the project area, and is a serious problem in well managed herds (H3,H4). The difficulties in the treatment of this disease have already been outlined (section 7).
A study on the economics of block treatment of herds with Naganol is suggested. Naganol is expensive and drug resistance may occur. It is therefore important to ascertain its efficacy. Such a study would require the assistance of another organisation and either the Kenyan Trypanosomiasis Institute, Kikuyu or the Veterinary Research Laboratories, Kabete are suggested.

8.4 The widespread use of drugs in the project area is not recommended until the results of studies as recommended in 8.2 and 8.3 are completed. The widespread use of drugs without adequate supervision and information lead to dangers which are self evident. This is especially the case with Naganol where such use could lead to drug resistance.

8.5 A study on the marketing of camels is suggested. There is, at present, little data on this subject and it would seem essential for adequate information on the potential of camels as a possible foreign currency earner for Kenya.

8.6 The use of improved breeding stock may improve overall productivity of camels. This is a long term study and may not be within the terms of reference of IPAL. Nevertheless, it may be worthwhile, initiating a programme attempting to identify a small herd of improved breeding stock in the project area. The use of Somali camels could be encouraged.

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10. REFERENCES

Balis, J. and Richard, D. (1977): Action trypanocide du chlorohydrate de chlorure d'isometamidium sur *Trypanosoma evansi* et essai de traitement de la trypanosomiase du dromodaire. Revue Elévage Veterinaire Pays Tropicaux, 30, 369-372.

Bennett, S.C.J. (1928). Mercuric chloride as a diagnostic agent for trypanosomiasis in camels. Journal of Comparative Pathology, 41, 341-353.

Bennett, S.C.J. (1929). Camel trypanosomiasis in the Sudan, Pan African Agricultural and Veterinary Conference, Pretoria.

Bennett, S.C.J., and Kenny, P.A.C. (1928). Mercuric chloride as a diagnostic agent for trypanosomiasis in camels. Journal of Comparative Pathology, 41, 341-353.

Bennett, S.C.J. (1933). The control of camel trypanosomiasis. Journal of Comparative Pathology, 46, 67-77, 174-185.

Burgmeister, R. (1975). Elevage de chameaux en Afrique du Nord. GTZ, Eschborn, W. Germany.

Cheyne, I.A., Pegram, R.G., and Cartwright, C.F. (1977). An outbreak of Salmonellosis in camels in Somalia. Tropical Animal Health and Production 9, 238-240.

Cross, H.E. (1917). The camel and its diseases. Baillière, Tindal and Cox, London.

Curasson, G. (1947). Le chameau et ses maladies - quoted in Richard (1975).

Dada, B.J.O. (1978). Incidence of hydatid disease in camels slaughtered at Kano abbatoir. Tropical Animal Health and Production 10, 204.

Dahl, G., and Hjort, A. (1976). Having Herds. Stockholm studies in Social Anthropology. 2, Stockholm, Sweden.

Darvill, F.M., Arundel, J.H. and Brown, P.B. (1978). The effect of anthelmintic treatment on maiden ewes in the periparturient period. Australian Veterinary Journal, 54, 575 - 584.

Davies, F.G., Mungai, J.N., and Shaw, T. (1975). Characteristics of a Kenyan camel pox virus. Journal of Hygiene, Cambridge, 75, 381-385.

Davies, F.G., and Atema, C. (1978). The antibody response in sheep to a Kenyan sheep and goat pox virus. Journal of Comparative Pathology, 88, 205-210.

Daynes, P., and Richard, D. (1974) Note about helminths (and some other parasites) of the dromedary in Ethiopia. Revue Elevage Veterinaire Pays Tropicaux, 27, 53-56.

Domenech. J., Guidot, G. and Richard, D. (1977). Les maladies pyogènes du dromodaire en Ethiopie. Revue Elèvage Mèdecin Veterinaire Pays Tropicaux. 30, 251-258.

Edelsten, R.M. and Pegram, R.G. (1974). Contagious skin necrosis of Somali camels associated with *Streptococcus agalactiae*. Tropical Animal Health and Production. 6, 255-256

Evans, J.O., and Powys, J.G. (1979). Camel husbandry to increase the productivity of ranchland. In 'Camels', International Foundation for Science, Sweden, Report 6, pp 241-250.

Fazil, M.A. (1977). The camel. Bulletin of Animal Health and Production in Africa. 25, 435-442.

Field, C.R. (1979). Preliminary report on ecology and management of camels, sheep and goats in northern Kenya. IPAL Technical report Series E.1a, UNESCO, Nairobi.

Field, C.R. (1979a). Camel growth and milk production in Marsabit District, Northern Kenya, preliminary report. In 'Camels' IFS, Sweden, Report 6, pp 215-240.

Gatt-Rutter, T.E. (1967). Diseases of camels. II. Protozoa diseases. Veterinary Bulletin. 37, 611-617.

Gatt-Rutter, T.E. and Mack, R. (1963). Diseases of camels. I. Bacterial and Fungal diseases. Veterinary Bulletin., 33, 119-124.

Gibson, W.C., Marshall, T.F., and Godfrey, D.G. (1980). Numerical analysis of enzyme polymorphism: a new approach to the epidemiology and taxomomy of trypanosomes of the *Subgenus Trypanozoon*. Advances in Parasitology, 18, 175-246.

Gruvel, J. and Balis, J. (1965). La trypanosomiase à *Trypanosoma evansi* chez le dromodaire du Tchad et ses principes vecteurs, Revue Elevage Mèdecin Veterinaire du Pays Tropicaux, 18, 435-439.

Gruvel, J. and Graber, M. (1965). Quelques resultats d'enquêtes récentes sur la globidiose du dromodaire au Tchad. Revue Elevage Mèdecine Veterinaire Pays Tropicaux, 18, 423-428.

Graber, M., Tabo, R., and Service, J. (1967). Enquêtes sur les helminthes du dromodaire tchadien. Etude des Strongyloses gastro-intestinales et de l' haemonchose à *Haemonchus longistipes*. Revue Elévage Médecine Pays Tropicaux, 20, 227-254.

Horgan, E.S. and Bennett, S.C.J. (1927). The mercuric chloride test for trypanosomiasis in camels, mechanism of the reaction. Journal of Comparative Pathology, 40, 188-196.

Jatkar, P.R. and Singh, M. (1971). Diagnosis of surra in camels by the passive haemogglutination test. British Veterinary Journal, 127, 283-288.

Knowles, R.H. (1924). The formol-gel test as applied to animals affected with *Trypanosoma soudanense*. Journal of Comparative Pathology, 37, 37-44.

Mahoney, D.F. and Ross, D.R. (1972). Epizootiological factors in the control of bovine babesiosis. Australian veterinary Journal 48, 292-298.

Mason, I.L. (1979). Origin, history and distribution of domestic camels. In 'Camels', International Foundation for Science, Sweden, Report 6, pp 21-34.

Morcos, M.B. (1965). Treatment of tetanus in the camel. Veterinary Medical Review, 2, 132-134.

Okoh, A.E.J. (1979). A survey of brucellosis in camels in Kano, Nigeria. Tropical Animal Health and Production, 11, 213-214.

Olitski, L., and Ellenbogen, V. (1947) A Salmonella strain isolated from camels in Palestine. Journal of Comparative Pathology, 57, 47-51.

Pellegrini, D. (1948). *Trypanosoma simiae* (Bruce) infection in the camel. East African Agricultural Journal, 13, 207-209.

Richard, D. (1975). Study on the pathology of the domedary in Borana Awraja (Ethiopia). Thesis I.E.M.V.T., Paris.

Richard, D. (1979). Pathology and production of camels. In 'Camels', International Foundation for Science, Sweden, Report 6, pp 409-430.

Salaheldin, E et al (1979). A note on the haematology of adult Sudanese camels. In 'Camels', IFS, Sweden, Report 6, pp 347-354.

Schwartz, J.H (1980). personal communication.

Scott, G.R., Coackley, W., Roach, R.W. and Cowdy, N.R. (1963) Rift Valley Fever in camels. Journal of Pathology and Bacteriology, 86, 229-231.

Sergent, E, Sergent E, and Lherritier, A. (1919). Dromadaires immunises contre la trypanosomiase 'Debab'. Bulletin Société Pathologie Exotique, 12, 86.

Singh, K.V. and Ata, F. (1867). Experimental rinderpest in camels: A preliminary report. Bulletin of Epizootic Diseases, Africa, 15, 19-23.

Steward , J.S. (1950). Trichostrongylosis and haemonchosis in the camel: Their recognition and response to Phenothiazine. Veterinary Record, 62, 837-839.

Steward, J.S. (1950a). Notes on some parasites of camels in the Sudan. Ibid, 62, 835-837.

Taylor, W.P. (1968). The susceptibility of the one humped camel to infection with rinderpest virus. Bulletin of Epizootic Diseases, Africa, 16, 405-410.

Waghela, S., Fazil, M.A. and Gathuma, J.M. (1978) A serological survery of brucellosis in camels in north-eastern province of Kenya. Tropical Animal Health and Production, 10, 28-29.

Wilson, R.T. (1978). Studies on livestock of southern Darfur, Sudan. v. Notes on camels. Tropical Animal Health and Production, 10, 19-25.

Zaki, O.A. (1956). The incidence of Salmonella infections in camels. Journal of Egyptian Public Health Association, 31, 75-79.

Knowles, R.H. (1925). Treatment of camels affected with *Trypanosoma soudanense* with Bayer 205 and further observations on the formol gel test. Journal of Comparative Pathology, 38, 42-46

Knowles, R.H. (1927a). Trypanosomiasis of camels in the Anglo-Egyptian Sudan: diagnosis, chemotherapy and immunity. Ibid, 40, 118-134.

Knowles, R.H. (1927b). Trypanosomiasis of camels in the Anglo-Egyptian Sudan: diagnosis, chemotherapy and immunity. Ibid, 40, 59-71.

Kremu (1979). Livestock and wildlife populations in Kenya 1977 compared to 1978. Stellfox, J.W., Kufwafwa, J.W., Mbugua, S.W., and Peden, P.G. Kremu Technical Report Series No. 6.

Kruuk, H. (1980). The effects of large carnivors on livestock and animal husbandry in Marsabit District, Kenya.

Leach, T.M. (1961). Observations on the treatment of *Trypanosoma evansi* infection in camels. Journal of Comparative Pathology, 71, 109-117.

Leese, A.S. (1927). A treatise on the one humped camel, in health and in disease. Haynes & Son, Stanford, Lincolnshire, U.K.

Luckins, A.G. et al (1979). Serdiagnosis of infection with *Trypanosoma evansi* in camels in the Sudan. Tropical Animal Health and Production, 11, 1-12.

Mahmoud, M.M. and Gray, A.R. (1980). Trypanosomiasis due to *Trypanosoma evansi* (Steel, 1885) Balbiani, 1888. A review of recent research. Tropical Animal Health and Production, 12, 35-42.

ADDENDUM 1. Characterisation of camel trypanosomes by isoenzyme electrophoresis on starch gel.

Introduction

Classically *Trypanosoma evansi* may be described as a minimally pleomorphic trypanosome that is transmitted by bloodsucking flies other than tsetse and occurs outside the tsetse belts of Africa. Before the introduction of biochemical means of trypanosome characterisation, the differentiation of *T. evansi* from other *brucei*-group trypanosomes depended for practical purposes on geographical origin alone, since it was almost impossible to distinguish *T. evansi* from *T.brucei* satisfactorily if both were found in animals in tropical Africa.

In a previous survey of *Trypanozoon* stocks by isoenzyme electrophoresis (Gibson *et al.*, 1980), *evansi* stocks from South America and Nigeria were found to be identical, although much variation was found among *brucei* stocks just in Africa; these *evansi*'s were electrophoretically very similar to *brucei*'s from West Africa.

It was hoped that an isoenzyme study of trypanosome isolates from Kenyan camels might serve to improve understanding of the concept of *T. evansi* in general, as well as to clarify the taxonomic position of these camel stocks in particular.

Methods

Procedures were essentially as described by Gibson *et al.* (1980), screening for the following 11 enzymes:- alanine aminotransferase, aspartate aminotransferase, glucose phosphate isomerase, phosphoglucomutase, isocitrate dehydrogenase, malate dehydrogenase, malic enzyme, nucleoside hydrolase, threonine dehydrogenase and 2 peptidases.

The following stocks were examined:- 20 isolations from Herd 1 (Kulal); 7 isolations from Herd 2 (Ngurunit); 3 isolations from Herd 3 (Rumuruti) and 8 from Herd 4 (Galana).

Results

The majority of isolates examined were identical or very similar to the *evansi*'s from South America and Nigeria screened in the previous study (Gibson *et al.*, 1980). However, 3 isolates from Ngurunit and one from Rumuruti were distinct from this group electrophoretically and may be *brucei*'s, that is to say tsetse transmissible; 2 of the 4 were seen to be pleomorphic in giemsa-stained blood films taken at the time of harvest, a further indication that they may be *brucei*'s

Conclusion

There can be little doubt that the majority of the Kenyan camel trypanosome stocks are of the *evansi* type electrophoretically, although it is surprising how closely they resemble stocks from South America and Nigeria, considering the large amount of isoenzyme variation found among *brucei*-group stocks in Africa alone.

Tsetse transmission experiments will decide whether the 4 electrophoretically distinct trypanosome stocks are *brucei*'s or not; for the moment, it is only possible to say that these 4 stocks are different to the majority of Kenyan camel isolates.

ADDENDUM 2. A description and results of drug tests conducted on trypanosomes isolated from camels in H3.

Methods

Trypanosomes (*T. evansi*) isolated in mice from camels in H3 were frozen in capillaries in liquid nitrogen and stored. 75 mice were inoculated with trypanosomes at a dose rate of 10^3/kg body weight. 6 hours after infection groups of 5 mice were each inoculated with one of 5 trypanocidal drugs at a dosage rate shown in the table. Each mouse was examined for the presence of trypanosomes in tail blood daily from days 3-60 post inoculation. Every mouse was postmortemed and the size of the spleen examined after day 60. The effect of each drug at each dose rate was measured by the ability to eliminate trypanosomes from the blood and the size of the spleen. If the spleen was enlarged, trypanosome activity is indicated.

Results

A summary of the results are shown on the table. Naganol and Antrycide cured the infection in mice at dose rates of 3 and 10 mg/kg but a failure occurred at 1 mg/kg. Prothium and Berenil failed to cure infection at any of the doses tested. 49/150 cured the infection at 10 mg but failed to cure at 1 and 3 mg/kg. Only mice treated with Naganol at 3 mg/kg showed no enlargement of the spleen. The remaining mouse groups showed splenic enlargement to a greater or lesser degree.

Conclusion

Of the 5 drugs tested only Naganol and Antrycide were effective in eliminating trypanosomes (*T. evansi*) from the mouse host. 49/150 showed trypanocidal activity at a high dose rate (10 mg/kg), the latter is a toxic dose. Berenil and Prothidium were ineffective.

Table A2. A summary of the methods and results in the testing of the sensitivity of *T. evansi* from H3 to 5 drugs.

Drug	Dose (mg/kg)	Ratio of No animals infected / No treated	Ratio of No animals showing splenic enlargement/ No treated
NAGANOL	1	3/5	5/5
	3	0/5	0/5
	10	0/5	2/5
BERENIL	1	5/5	5/5
	3	5/5	5/5
	10	3/5	4/5
ANTRYCIDE	1	4/5	5/5
	3	0/5	3/5
	10	0/5	2/5
49/150 experimental drug	1	5/5	5/5
	3	4/5	5/5
	10	0/5	2/5
PROTHIDIUM	1	5/5	5/5
	3	5/5	5/5
	10	5/5	4/5

ADDENDUM 3. Camel Pox in Kenya, an examination of sera from four herds for antibody to the virus.

Introduction

Camel pox in fections are regularly encountered in the herds kept by the nomadic pastoralists in northern Kenya. The strains of virus which have been isolated and studied have been shown to be identical with those strains found in the northern parts of the continent and in the middle East. (Davies, Mungai and Shaw, 1975). The mortality which is produced in the herds by the infection, is usually confined to the younger animals and influenced by management and other factors, such as intercurrent disease; losses of 8-15% have been seen on occasion.

Methods

The sera were tested by a microserum-neutralisation test similar to that described by Davies and Atema (1978) for the Kenyan sheep and goat pox virus. The end point of the test was taken where 50-70% of the virus challenge dose was neutralised. The method has not been used for a sequential study of the development and persistence of antibody after infection and the results can only be used as an indication of the presence of antibody to camel pox virus or another closely related orthopox virus in the sera.

Results

The results shown on the table indicate that there has been a high level of challenge in all the herds examined, and suggest an endemic situation.

Conclusion

Camel Pox is an important disease in all the study herds. The disease is present in an enzootically stable condition as serum antibodies were detected in all age groups.

Table A3: The results of serum neutralisation tests carried out with various camel sera and the +H 520 strain of camel pox virus.

Origin	Serum	Titres*						Totals Negative	Positive
	0	10	20	40	80	160	320		
Kulal	10	1	3	5	2	0	0	10	11
Ngurunit	10	13	15	28	14	18	4	10	91
Ol Maisor	17	4	6	7	21	15	16	17	69
Galana	71	6	16	14	14	10	5	71	65
								108	236

+ Kenya strain

* Assayed by microserum-neutralisation against 100 TC 11/50 virus, end point determined at approximately 60% reduction of entopathic effects.

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