The Orma Boran

A trypanotolerant East African breed

Fifteen years of research on Galana Ranch in Kenya

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The Kenya Trypanosomiasis Research Institute (KETRI) is a national research institute established in 1979. KETRI's mandate is to carry out research that would lead to effective control of human and animal trypanosomosis and effective reclamation of tsetse infested lands. KETRI's headquarters and laboratories are at Muguga, 35 kilometres from Nairobi. The institute operates a referral hospital for sleeping sickness patients at Alupe in Busia and field stations in tsetse and trypanosomosis endemic areas. These include Galana Ranch in Kilifi, Nguruman in Kajiado and Lambwe Valley in Suba District.

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Front cover: Orma cattle crossing the Tana River in the Tana River District in Kenya.
Photograph: Bernhard Sacher

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Summary

The Boran cattle of the Orma people in the Tana River District of Kenya, an indigenous *Bos indicus* breed, have been studied by the Kenya Trypanosomiasis Research Institute (KETRI) on Galana Ranch in the Coastal Province of Kenya since 1980. There they have been compared under tsetse challenge to the home bred Galana Borans, improved Kenya Borans that originated in the Kenya Highlands.

Orma Boran steers did better than Galana Boran steers under tsetse challenge. Trypanosome prevalence, particularly *Trypanosoma vivax* prevalence, was lower in the Orma Boran compared with the Galana Boran. Once detected parasitaemic the Orma Breed were less likely to suffer from severe anaemia and in some cases they recovered without treatment. Infection and mortality rates from the disease in the Orma are approximately half those observed in the Galana Borans. Under both prophylactic and treatment regimes the Orma cattle required fewer drugs. Acquired resistance to trypanosomosis was not an important factor in explaining the observed differences between the two Boran types. Comparisons between Orma and Galana calves showed that the differences existed in animals born in the same environment and exposed from birth to the same tsetse challenge.

However, the Galana Borans generally grew faster and reached a heavier mature body size than the Orma Borans. In 1983 KETRI embarked on a breeding and selection programme aimed at improving the beef production characteristics of the Orma Boran while at the same time maintaining their level of trypanotolerance. Males are selected for breeding on the basis of post weaning growth rate and bulls that reached 400 kg by four years of age have been sold to farmers in other tsetse infested areas of Kenya. Results are presented from data collected from this breeding herd between 1983 and 1996 and estimates of various genetic parameters are given.

1 General introduction

In 1913, Balfour described *Bos indicus* cattle in the Koalib area of Sudan which he claimed were immune to trypanosomosis (Balfour, 1913). Archibald (1927) referred again to these trypanotolerant Sudanese cattle. Trypanotolerance has also be described in the Mongalla, a small Zebu type in Southern Sudan (Coulombs et al 1978) and in the Lugware cattle, found in parts of Zaire, Uganda and Sudan (Joshi et al 1957). Cunningham (1966) reported the presence of large numbers of Zebu cattle thriving on the tsetse infested shores of Lake Victoria. Yet despite these reports in the literature little effort has been made to investigate the nature or extent of differential susceptibility to trypanosomosis amongst cattle breeds in East Africa and trypanotolerance in cattle continues to be associated almost solely with the *Bos taurus* breeds of West Africa.

The mistaken idea, arising from the West African situation, that trypanotolerance is in some way associated with the presence of humpless cattle rather than with the presence of tsetse flies has perhaps diverted attention from the possible existence of trypanotolerance in East Africa. Trypanotolerance clearly correlates with tsetse distribution and not with the presence or absence of a hump; one would not expect to find trypanotolerance in humped cattle in Europe and conversely some degree of resistance to trypanosomosis is likely in the humped cattle which inhabit tsetse infested lands of East Africa.

The indigenous cattle of Africa are the product of generations of natural selection and survival of the fittest. Trypanosomosis has taken its toll on cattle in tsetse areas over the centuries. It is only in this century that drugs or tsetse control have afforded some protection. The degree of trypanotolerance found amongst African cattle is a reflection of the severity of tsetse challenge to which they have been exposed and the length of time over which that exposure has taken place.

The tsetse distribution maps of Africa indicate a less concentrated distribution of tsetse flies in East Africa. The climatic conditions of large areas of East Africa are unsuitable for tsetse flies; in Kenya, for example, high temperature is the critical factor in controlling the spread of the fly in areas of low altitude while in the highlands cold becomes the limiting factor with tsetse flies rarely found above 6,000 feet. In contrast almost the entire landmass of the Congo has climatic conditions suitable for tsetse flies. Thus in Kenya the cattle keeping people have usually been able to find alternative grazing when tsetse challenge became severe and it seems likely that over the centuries natural selection for trypanotolerance has been less intense than in areas of West or Central Africa.

There is also evidence to support the hypothesis that *Bos taurus* cattle arrived in Africa many centuries before *Bos indicus* cattle (Epstein and Mason 1983). Thus exposure to trypanosomosis has been over a shorter period for the *Bos indicus* cattle of East Africa than that experienced by the *Bos taurus* cattle of West Africa. Nonetheless, observations of differential susceptibility between breeds and breed types have been made in East Africa and should be further investigated. The possibility of introducing trypanotolerant N'Dama cattle into East Africa is remote particularly as there are indications that their trypanotolerance is perhaps confined to specific strains of the trypanosome and breaks down under challenge from virulent East African strains (ILRAD 1992). Furthermore, N'Dama cattle have no experience of theileriosis, one of the major cattle killing diseases of East Africa, and have been shown to be extremely susceptible on primary challenge (Williams et al 1992). Cattle which have evolved with the parasites and diseases present in East Africa are likely to prove more suitable for the tsetse infested areas of East Africa.

The Orma Boran

The Boran cattle owned by the Orma pastoralists in the Tana River District of Kenya were first described by Mason and Maule (1960) under the name Tanaland Boran.

The Orma people, descendants of the Oromo, originated from the Borana Province in Ethiopia (Figure 1). Between 1400 and 1500 the Oromo migrations resulted in the Oromo people occupying lands far outside their previous boundaries (Ensminger 1996). In Kenya they occupied vast tracks of land in the tsetse infested coastal areas stretching into northern Tanzania. Pressure from the Somali people in the east and the Kamba in the west eventually restricted these nomadic pastoralists to the tsetse-infested lands of the Tana River District.

The south-eastern part of Garsen Division in Tana River District, between Garsen and the Indian Ocean encompassing the Tana Delta, is heavily tsetse infested (Figure 1). The majority of the Orma are concentrated in the Garsen Division of the District and they use the Tana Delta area as dry season grazing. In the wet season they move their herds out of the Delta and seek grazing to the north and the west as long as supplies of standing water persist. The tsetse distribution in the northern reaches of the district is primarily confined to the Tana River. The Orma people, who pride themselves on their cattle keeping abilities, maintain cattle primarily for milk. They trade in Somali cattle, buying Somali steers for fattening and then selling them. They do not use Somali cattle for breeding preferring their own bulls. In the last 20 to 30 years they have used trypanocides to protect their cattle from trypanosomosis. However, prior to the advent of trypanocides avoidance of the fly was their only means of reducing the risk of disease.

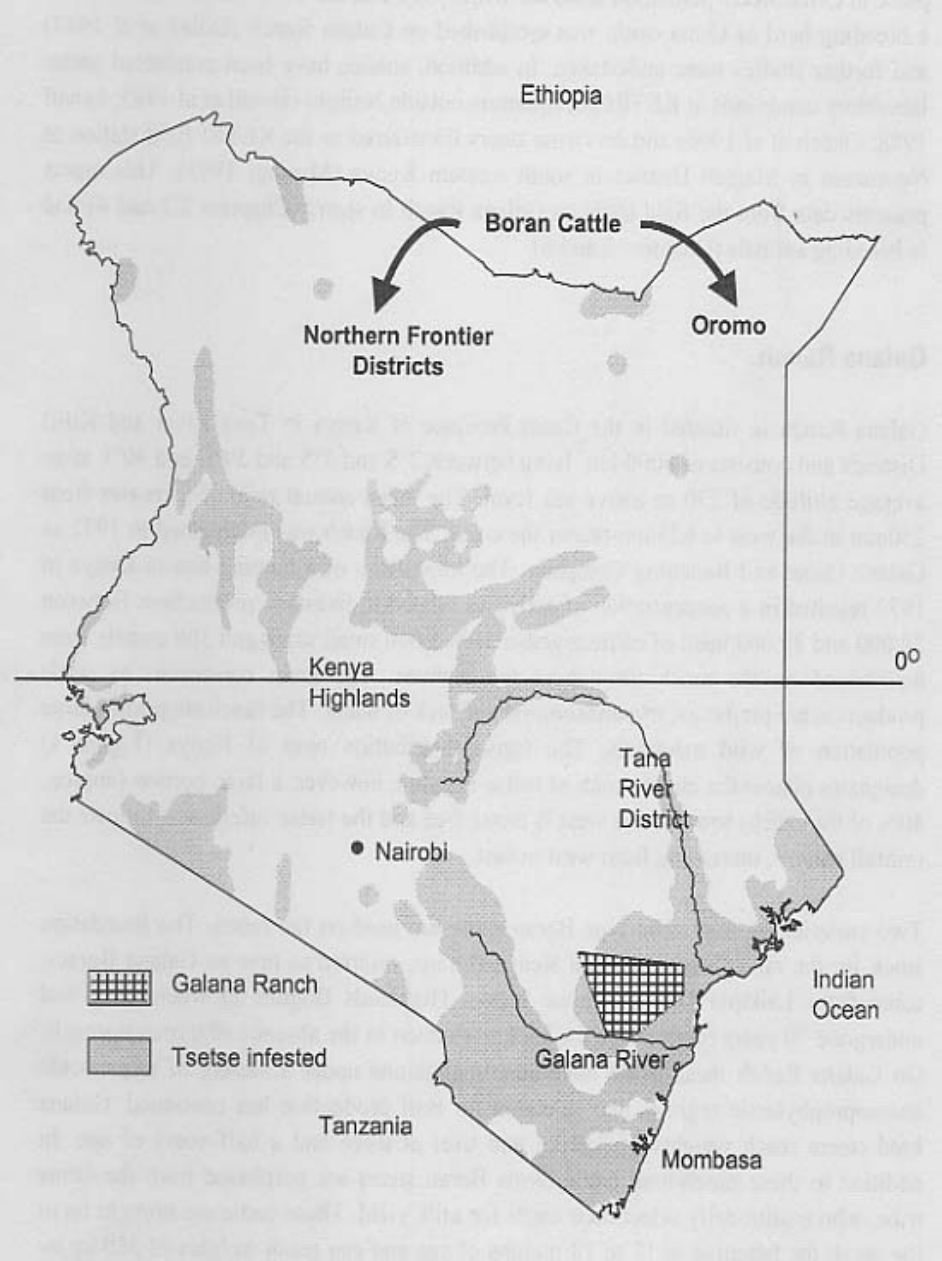


Figure 1: Kenya showing the tsetse distribution, Galana Rench, the Tan River District and the migration rotes of the Boran cattle used on Galana Ranch

The value of the Orma cattle in tsetse infested lands was first quantified in field studies undertaken by the Kenya Trypanosomiasis Research Institute (KETRI) on Galana Ranch in Kenya (Dolan et al 1985; Njogu et al 1985a, b). These first trials took place in Orma steers purchased from the Orma people in the Tana River District. Later a breeding herd of Orma cattle was established on Galana Ranch (Dolan et al 1987) and further studies were undertaken. In addition, studies have been conducted under laboratory conditions at KETRI headquarters outside Nairobi (Ismail et al 1985; Ismail 1988; Okech et al 1996) and on Orma steers transferred to the KETRI field station at Nguruman in Magadi District in south western Kenya (Mwangi 1993). This report presents data from the field trials on Galana Ranch in steers (Chapters 2,3 and 4) and in breeding animals (Chapters 5 and 6).

Galana Ranch

Galana Ranch is situated in the Coastal Province of Kenya in Tana River and Kilifi Districts and consists of 6000 km^2 lying between 2°S and 3°S and 39°E and 40°E at an average altitude of 270 m above sea level. The mean annual rainfall increases from 250mm in the west to 625mm nearer the coast. The Ranch was established in 1972 as Galana Game and Ranching Company. The imposition of a hunting ban in Kenya in 1977 resulted in a concentration of activities related to livestock production. Between 20,000 and 30,000 head of cattle together with 6,000 small stock and 300 camels were maintained on the ranch throughout the eighties. The main constraints to cattle production are predators, trypanosomosis and lack of water. The ranch supports a large population of wild mammals. The tsetse distribution map for Kenya (Figure 1) designates almost the entire ranch as tsetse-infested however, a large portion (approx. 40% of the ranch) towards the west is tsetse free and the tsetse infestation follows the rainfall pattern, increasing from west to east.

Two varieties of large zebu type Boran cattle are used on the ranch. The foundation stock for the ranch-bred improved Kenya Borans, referred to here as Galana Borans,

came from Laikipia District in the Kenya Highlands (Figure 1) where they had undergone 70 years of selection for beef production in the absence of trypanosomosis. On Galana Ranch these cattle have been maintained under a variety of trypanocide chemoprophylactic regimes and selection for beef production has continued. Galana bred steers reach weights of 350 kg and over at three and a half years of age. In addition to these ranch-bred cattle Orma Boran steers are purchased from the Orma tribe, who traditionally select their cattle for milk yield. These cattle are brought on to the ranch for fattening at 12 to 18 months of age and can reach weights of 350 kg by four years of age.

The cattle experiments described here took place in two tsetse-infested areas of the ranch: Kapanagani in the south-eastern part of the ranch and Kisiki further south, bordering the Galana River. The tsetse challenge in the Kapangani area was primarily from *Glossina pallidipes* with increasing numbers of *G. longipennis* found in the latter years. In Kisiki *G. pallidipes* was also the principle vector of the disease but *G. longipennis, G. brevipalpis* and *G. austeni* are also found.

Kenya Trypanosomiasis Research Institute

In 1979 KETRI established a field station on Galana Ranch. Field trials studying various aspects of trypanosomosis in cattle, camels, sheep and goats have been undertaken since then. In 1986 KETRI expanded its activities to evaluate the new tsetse control technology developed in Zimbabwe (Vale 1981) involving odour-baited, insecticide-impregnated targets (Opiyo et al 1987). The studies reported here were undertaken by KETRI on Galana Ranch between 1980 and 1996. The International Livestock Centre for Africa (ILCA) and later the International Livestock Research Institute (ILRI) provided facilities to the author for the analyses of the data and compilation of this monograph.

2 Differential response to tsetse challenge and trypanosomosis in two types of Boran steers

Introduction

Between 1980 and 1987 a number of 12-month trials were conducted by KETRI at its field station on Galana Ranch. In all of these trials Orma steers, purchased from the Tana River District, were compared with ranch-bred Galana steers (improved Kenyan Borans). Results from some of these trials have been published (Dolan et al 1985; Njogu et al 1985a, 1985b; Dolan et al 1990) and a summary of these published trials is presented here in Chapter 2. Other trials, that were published only in abstract form (Dolan and Njogu 1986), are presented in Chapter 3, and in Chapter 4 results from unpublished trials are presented.

Twelve-month drug trials

Prior to KETRI's involvement on Galana Ranch in 1979 the policy for the control of bovine trypanosomosis was to give all the cattle, each year, four doses of isometamidium chloride (Samorin, May & Baker) at 0.5 mg/kg bodyweight and two doses of diminazene aceturate (Berenil, Hoechst) at 3.5 mg/kg bodyweight. Wilson et al (1981), however, recommended an alternative strategic treatment regime based on the observation that the challenge varied within the ranch and that it was seasonal; there were several months of the year when the cattle were not at risk from trypanosomosis. There were also indications that there were differences in prophylactic drug requirement in the two cattle types maintained on the ranch.

Materials and Methods

Galana Ranch and the tsetse fly species found there have been described in Chapter 1. The two types of Boran cattle maintained on Galana Ranch, the Orma Boran and the improved Kenyan Boran, referred to here as Galana Boran, are for simplicity described here as breeds.

Between 1982 and 1985 prophylactic drug regimes involving isometamidium chloride at 0.5 mg/kg and 1 mg/kg bodyweight and pyrithidium bromide (Prothidium, Boots) at 1 mg/kg and 2 mg/kg bodyweight and other experimental compounds were evaluated by KETRI on Galana Ranch. In 1987 a further strategic treatment regime trial involving homidium bromide (Ethidium, CAMCO) at 1 mg/kg bodyweight was undertaken. These trials were conducted in two different tsetse-infested areas of the ranch described in Chapter 1, Kisiki and Kapangani, and involved Orma and Galana steers which varied between 18 and 36 months of age at the commencement of the trials.

The trials generally consisted of Orma and Galana steers in groups as follows:
(i) Sentinel groups (n =30) - treated immediately on detection of infection with diminazene aceturate (Berenil, Hoechst or Veriben, Sanofi) at 7 mg/kg bodyweight. Data from these animals provided a measure of the trypanosome challenge measured as the Berenil Index. The Berenil Index (infections per animal per annum) is calculated as (p/n x 365/d), where p is the number of cattle detected positive for trypanosomes during the month, and n the number of animals exposed to infection and d is the number of days in the month. To allow for a seven-day prophylactic effect of diminazene a slightly modified form of the Berenil Index was used as described by Njogu et al (1985a).
(ii) Prophylactic groups (n = 15, 30 or 45) - administered with one of the prophylactic drugs mentioned above when the infection rate in the sentinel herd reached a critical value (see below). Data from these animals provided information on efficacy of the prophylactic.
(iii) Untreated groups (n = 10) - in which mortality due to trypanosomosis was measured.

All animals were herded together and exposed to natural field challenge, involving primarily *G. pallidipes*. Trials commenced in September or October and ran for twelve months with the exception of two trials where pyrithidium bromide at 1.5 and 2 mg/kg was used. Both of these trials were terminated after four months. The packed cell volume (PCV) percent was estimated weekly and blood examined for trypanosomes following the haematocrit centrifugation and buffy coat technique of Murray et al (1977). Bodyweights were recorded monthly. Ticks were controlled by fortnightly spraying with acaricide, and anthelmintics were administered every six months. Wet blood smears were examined for other parasites when animals with low PCV values were trypanosome negative. Cases of anaplasmosis were treated with tetracycline.

The strategic treatment regime adopted for the Orma or Galana prophylactic herds depended on the trypanosome prevalence in the sentinel herd of that breed. As there was no evidence of diminazene resistance on the ranch, each detection of parasites in the sentinel herd was regarded as a new infection. To allow for a possible prophylactic effect of diminazene, animals were considered to be protected during the week following treatment and were therefore excluded when the prevalence for that week was calculated. Twenty percent of the sentinel herd detected positive over five weekly examinations was regarded as the critical level of challenge, at which point prophylaxis was administered to the prophylactic group of that breed. In each trial the first administration of drug in each prophylactic group depended solely on the infection rate in the sentinel herd for the breed. Subsequent prophylaxis depended on the number of infections observed in each prophylactic group (referred to as "breakthrough" infections) and on the trypanosome prevalence in the respective sentinel herd. If the third infection detected in a prophylactic group occurred at a time when challenge in the sentinel herd was at the critical level then prophylactic drug was re-administered. If the challenge was below the critical level prophylactics were not administered.

Results

Trypanosome prevalence

In Table 1 data from the sentinel herds are presented as mean monthly trypanosome prevalence rates in cattle for each of five years. Also shown are the number of *Trypanosoma vivax* and *T. congolense* infections detected in each group of 30 steers throughout the five years together with the "*vivax* ratio" (the ratio of the number of *T. vivax* infections to the number of *T. congolense* infections).

Table 1. *Mean monthly prevalence, number of Trypanosoma vivax and T. congolense infections and the vivax ratio (T. vivax:T. congolense infections) in groups of 30 Orma and 30 Galana steers exposed to tsetse challenge on Galana Ranch in five years.*

	Mean monthly prevalence		No. of *T. vivax* infections		No. of *T. congolense* infections		*Vivax* ratio	
Year	Orma	Galana	Orma	Galana	Orma	Galana	Orma	Galana
1982	0.05	0.12	1	7	17	41	0.06	0.17
1983	0.20	0.41	38	96	33	40	1.15	2.40
1984	0.17	0.37	27	86	32	38	0.84	2.26
1985	0.38	0.46	86	104	38	38	2.26	2.74
1987	0.05	0.19	3	28	13	30	0.23	0.93
Mean	0.17	0.31	31	64	27	37	1.17	1.72

An analysis of variance was used to test for differences among years and between breeds. There were significant differences among years and between breeds in mean monthly trypanosome prevalence ($P < 0.001$), in the number of *T. vivax* infections ($P < 0.05$) and in the *vivax* ratio ($P < 0.05$). The difference between breeds was also significant ($P < 0.05$) for the number of *T. congolense* infections but there was no difference between years. The *vivax* ratio appeared to be related to the degree of challenge being highest in the years when infection rates were highest. The observation of no significant difference among years in the *T. congolense* challenge

emphasises the fact that the differences in trypanosome challenge among years were related to variation in *T. vivax* challenge.

Mortality

Annual mortality was recorded in groups of 10 untreated steers of each Boran type in the first three years (1982-1984 in Table 2.). In 1986 and 1987 "mortality" was estimated in groups of 20 or 10 steers treated when their PCV fell below 16% so as to avoid death. This PCV value was chosen as the critical value from examination of the results in the previous three years; over ninety percent of animals whose PCV fell to 15% failed to recover.

Higher mortality was observed in the Galana than the Orma steers in all years,. Mean annual mortality in 60 Galana steers was 71% compared with 35% in comparable Orma steers. Also, mean time to detection of infection was longer in the Orma and the percentage of infected Orma animals which died (or required treatment) was less, as was time to death (Dolan et al 1985).

Table 2. *Annual mortality rates (%) in groups of untreated Orma and Galana steers in five yearly trials*

Year	No. of steers per breed group	Orma	Galana
1982	10	0	40
1983	10	60	70
1984	10	40	80
1986+	20	55	75
1987+	10	20	90
All years	60	35	71

+ To avoid death, animals were treated when their PCV fell below 16%.

Requirement for prophylactic drugs

Differences between breeds were recorded in requirement for prophylactics under different drug regimes as shown in Table 3. In the homidium bromide trial in 1987

(Dolan et al 1990) the critical level of challenge was never reached in the Orma sentinel herd so that the Orma prophylactic group received no prophylactics. In the other trials shorter periods of prophylactic activity and more breakthrough infections were observed in the Galana steers. Galana steers generally required twice as many treatments as their Orma counterparts (Table 3).

Table 3. *The number of prophylactic treatments required over a 12-month period and the number of breakthrough infections in groups of Orma and Galana steers under different drug regimes*

Year	Drug regime	No. of steers per breed group	No. of treatments		No. of breakthrough infections	
			Orma	Galana	Orma	Galana
1982	Prothidium (1.0 mg/kg)	30	1	3	0	6
1983	Prothidium (1.5 mg/kg)+	15	2	4	5	18
1983	Prothidium (2.0 mg/kg)+	15	3	5	9	19
1984	Samorin (0.5 mg/kg)	30	3	7	6	26
1884	Samorin (1.0 mg/kg)	30	3	5	5	19
1987	Ethidium (1.0 mg/kg)	45	0	2	0	5

+ Trial of four months' duration.

In 1982, during the first trial involving pyrithidium bromide, the trypanosome challenge was low (Table 1), however, resistant trypanosomes were detected. The drug was tested at two higher dose rates the following year. In this year the trypanosome challenge was 3-4 times higher and breakthrough infections were detected within two weeks of administration of pyrithidium bromide. Frequent dosing at 1.5 and 2 mg/kg resulted in toxicity problems; ten of the 15 Galana steers in the prophylactic group died and three deaths occurred among the Orma steers. The prophylactic groups were discontinued after four months but the sentinel herds and the control groups were maintained for the full year.

The growth rate information from these various trials will not be presented here as these data have been reported as part of an economic analysis (Ashley et al 1992).

Discussion

The trypanosome prevalence in the Galana sentinel herd in each year of these trials represented moderate to high challenge as defined by Whiteside (1962). Njogu et al (1985a) presented a measure of the tsetse challenge for two years from October 1981 to September 1983 in the areas where these trials were conducted. This was based on biconical trap catches and fly infection rates and correlated with the Berenil Index calculated for those years

Comparisons of the Orma Boran with the Galana Boran suggest that there were two separate components in the ability of the Orma cattle to withstand tsetse and trypanosome challenge. Firstly, the Orma Boran, when exposed to natural field challenge, were detected parasitaemic less often and secondly, once parasitaemic, they had a superior ability to control anaemia and were less likely to die of trypanosomosis.

The difference in trypanosome prevalence observed in the two Boran breeds has been a consistent feature throughout the field studies. The magnitude of the difference in the infection rates appeared to vary depending on the species of trypanosome involved and the intensity of challenge (Table 1). The superiority of the Orma Boran was more apparent under *T. vivax* challenge; a phenomenon also observed in N'Dama cattle when compared with a trypanosusceptible breed (Murray et al 1981).

Control of parasitaemia and anaemia, once trypanosomes were detected, was the second facet of the observed superiority of the Orma cattle. Mortality rates were lower in untreated cattle and time to death was extended (Dolan et al 1985). Also the prophylactic period for a number of different drugs was shown to be longer in Orma than in Galana steers.

It must be noted however, that the differences between the Orma and Galana Boran, summarised here, were all recorded in one-year trials in steers for which there was only limited information on previous exposure to trypanosomosis. The Galana Boran had been reared from birth on the ranch and exposed to the trypanosome serodemes circulating in the tsetse fly population on the ranch. However, any such exposure would have been under the protection of prophylactic drugs. The Orma steers had been exposed from birth to tsetse challenge in the Tana River District. No information was available on the nature or extent of this challenge. The Orma people use trypanocides both as prophylactics and curatives. On arrival on Galana Ranch, aged between 12 and 18 months, the Orma steers were maintained on similar drug regimes to the ranch-bred cattle for variable periods of time before the commencement of trials described here.

If previous exposure to trypanosomosis is important in determining subsequent response then it could be argued that the differences observed between the Orma and Galana Borans were simply the result of the Orma steers being exposed to more intense trypanosome challenge prior to their arrival on Galana Ranch. On the other hand, it could be argued that the Galana steers had been exposed to the array of serodemes present on the ranch while the previous exposure of the Orma steers may have been to a different assembly of serodemes in the Tana River District. The experiments described in the next chapter were thus designed to investigate the development of immunity to trypanosomosis in cattle under different treatment regimes and assess the importance of previous exposure to response to subsequent challenge.

3 Acquired and innate resistance to trypanosomosis under an immediate treatment regime

Introduction

The importance of previous exposure in determining response to trypanosomosis is unclear. In laboratory trials, using both bloodstream and metacyclic trypanosomes, it has been shown that infection with one strain or serodeme of trypanosome can induce immunity to reinfection with that particular strain or serodeme but not to heterologous challenge (Murray and Urquhart 1977; Nantulya et al 1980). The immunity conferred however was short lived and was no longer apparent in animals re-challenged with the same strain after six months. This appears to be the situation for *T. congolense* and *T. brucei* but not with *T. vivax*. Studies on *T. vivax* have been extensively reviewed by Gardiner (1989). In goats challenged with *T. vivax* stocks, immunity to homologous tsetse challenge was induced in a small number of animals in some experiments while no immunity was elicited in other experiments. However, extended pre-patent periods and lowered parasitaemia were observed on re-challenge.

From these laboratory findings it might be supposed that if the number of serodemes in any particular area was limited then, over time, resistance to those particular serodemes might develop. Field studies with N'Dama cattle in West Africa and *Bos indicus* cattle in East Africa (reviewed by Murray et al 1982) have reported that cattle which survive trypanosomosis, irrespective of the use of trypanocides, are more resistant to subsequent challenge. However, other studies have shown no evidence of immunity in cattle maintained in endemic areas over long periods (Hornby, 1941; Wilson et al 1975). Rushigajiki et al (1986), in a seven year field study on a herd in Lugala, Uganda reported that no significant resistance developed when the cattle were maintained on a diminazene aceturate treatment regime.

In many of these field studies an increase in the interval between drug treatments was considered indicative of the development of immunity. This assumption can only be made in situations where there is precise information on tsetse and/or trypanosome challenge which are determined independently of the cattle being monitored. A decrease in the challenge from one year to the next would also result in an increase in the interval between treatments.

In an attempt to investigate the role of previous exposure to trypanosome challenge, and to study the response of steers over longer time periods, groups of 30 Orma and 30 Galana steers were kept in the same area of the ranch and monitored weekly for 24 months from October 1983 to September 1985. For the second 12 months their response was compared with that of steers newly introduced into the same area.

Materials and Methods

The general management of the cattle and the recording of PCV, infecting trypanosome strains and bodyweights were previously described in Chapter 2. Infected steers were treated with diminazene aceturate at 7 mg/kg bodyweight immediately on detection of parasites. In the first year, 60 steers (30 Orma and 30 Galana) were monitored and, in the second year, these two groups of steers were again monitored together with two newly introduced groups of 30 Orma and 30 Galana steers. The Galana steers used in both years had been reared on the ranch and maintained on prophylactic drugs when exposed to tsetse fly. The Orma steers had been brought on to the ranch three to six months before the trial commenced and maintained under the same regime as the Galana steers from the time of their arrival on the ranch.

The impact of previous exposure on subsequent performance was assessed by estimating the regression of infection rates and PCV in Year 2 on infection rates and PCV in Year 1. This approach was preferred to a simple comparison between years of

the mean interval between drug treatments as it overcame the problems associated with different trypanosome challenge in the two years. In the regression analysis differences in challenge would be reflected in the magnitude of the regression coefficient while the sign of the regression coefficient would reflect the effect of previous exposure. If acquired immunity was more important then a negative regression coefficient would be expected; the animals with the most infections in Year 1 would be expected to have the least number of infections in Year 2. Conversely, if innate immunity was more important then the regression coefficient would be positive; those animals with the least number of infections in Year 1 would also have the least number of infections in Year 2. Both factors operating to an equal extent, or not at all, would result in a regression coefficient not significantly different from zero.

Results

The number of infections of each trypanosome species detected in the two groups of steers during the two years is shown in Table 4. The means of PCVs measured when no infections were detected are also shown together with the mean reduction in PCV recorded when animals were detected positive for *T. vivax* or for *T. congolense*.

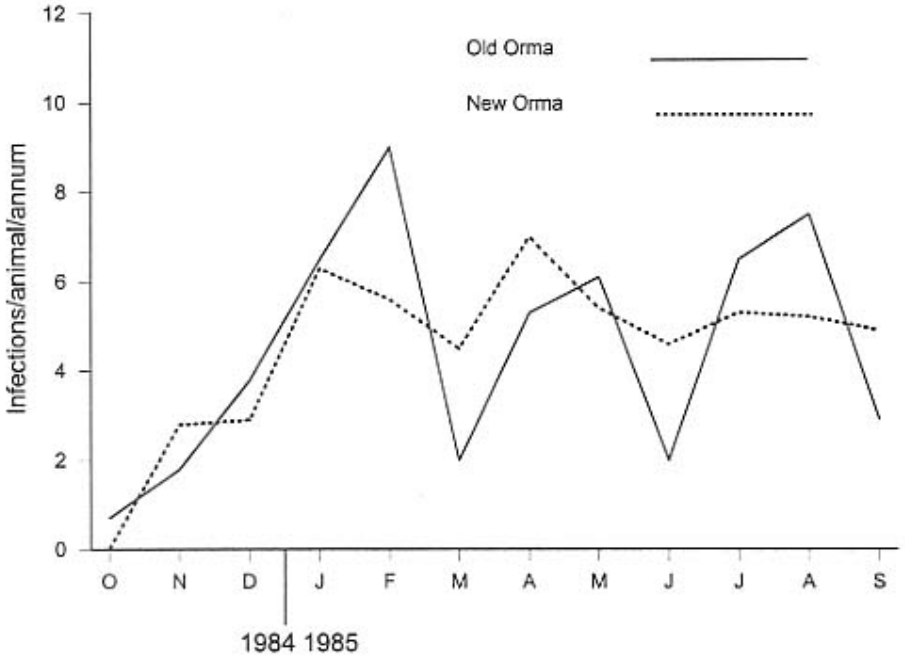
Table 4. *Mean packed cell volume (%), ± standard error, while not infected, and the fall in PCV when infected with Trypanosoma vivax (Tv) and T. congolense (Tc), measured in 30 Orma and 30 Galana steers.*

	Year 1	n+	Year 2	n+
Orma				
PCV when non-infected	30.5 ± 0.1	1501	28.0 ± 0.1	1436
Fall in PCV with Tv infection	5.6 ± 0.8	27	1.7 ± 0.6	45
Fall in PCV with Tc infection	6.8 ± 0.7	32	2.9 ± 0.5	79
Total no. of infections		59		124
Galana				
PCV when non-infected	28.9 ± 0.1	1439	26.1 ± 0.1	1373
Fall in PCV with Tv infection	7.8 ± 0.4	87	5.0 ± 0.3	119
Fall in PCV with Tc infection	5.7 ± 0.7	34	5.1 ± 0.4	68
Total no of infections		121		187

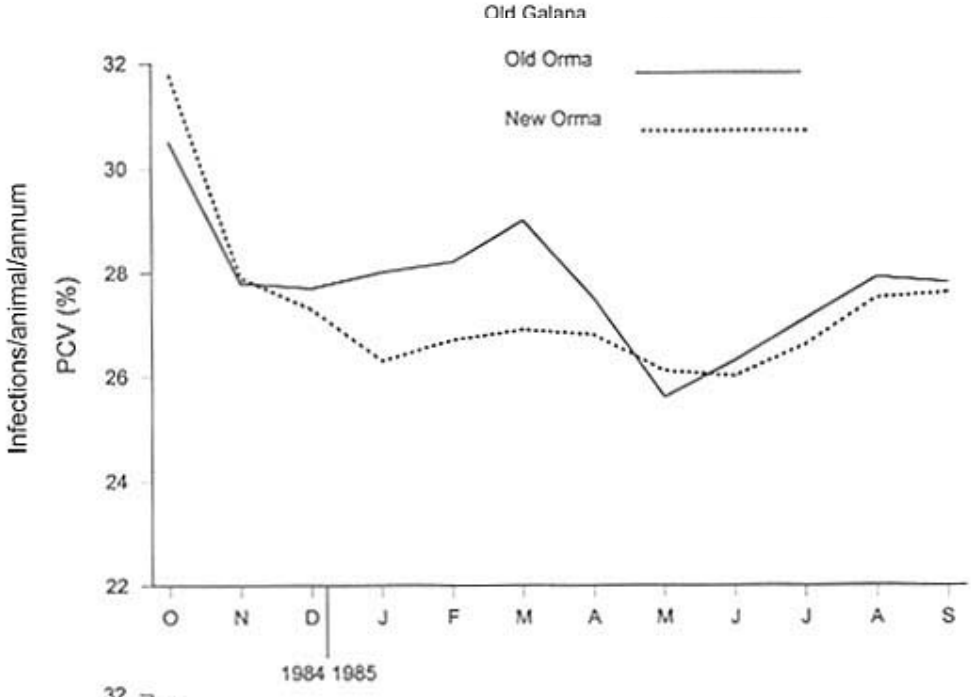
+ PCV was measured 52 times for each of 30 steers in each group. These 1560 observations (n) are divided into those where no parasites were detected and those were parasites were detected.

The trypanosome challenge was higher in the second year with more infections detected in both groups of steers. Packed cell volume was also significantly lower in Year 2. However, despite this higher challenge there was evidence of an improved control of anaemia in Year 2. The fall in PCV associated with infection in Year 2 was less than that observed in Year 1. This improved control of the anaemia was more apparent in the Orma steers than in the Galana steers for which the improvement in the control of anaemia in Year 2 was not significant in relation to *T. congolense* infections (Table 4).

Infection rates in the previously exposed (old) steers were compared with those in the newly introduced (new) steers in the form of the Berenil Index in Figure 2. The newly introduced Orma steers had 124 infections in the second year; the same number detected in the old Orma steers (Table 4). The newly introduced Galana steers had 142 infections in the second year. This was possibly an underestimate as these Galana steers had been mistakenly given isometamidium chloride**,** at 1mg/kg bodyweight, in August before commencing the trial in October. The prophylactic effect of the drug was apparent in the first two months of the trial (Figure 2). The previously exposed steers had 187 infections during the year. The infection rates were reflected in the mean monthly PCV values (Figure 3). There was little difference in the mean monthly PCV in the old and new Orma steers, although the old steers generally had higher PCVs than the new steers. The PCV values in the new Galana steers were consistently higher than that in the old Galana steers ($P < 0.05$).


Old Orma
New Orma
Infections/animal/annum
12
10
8
6
4
2
0
O N D J F M A M J J A S
1984 1985

łuced


Old Orma
New Orma
Infections/animal/annum
PCV (%)
32
30
28
26
24
22
O N D J F M A M J J A S
1984 1985

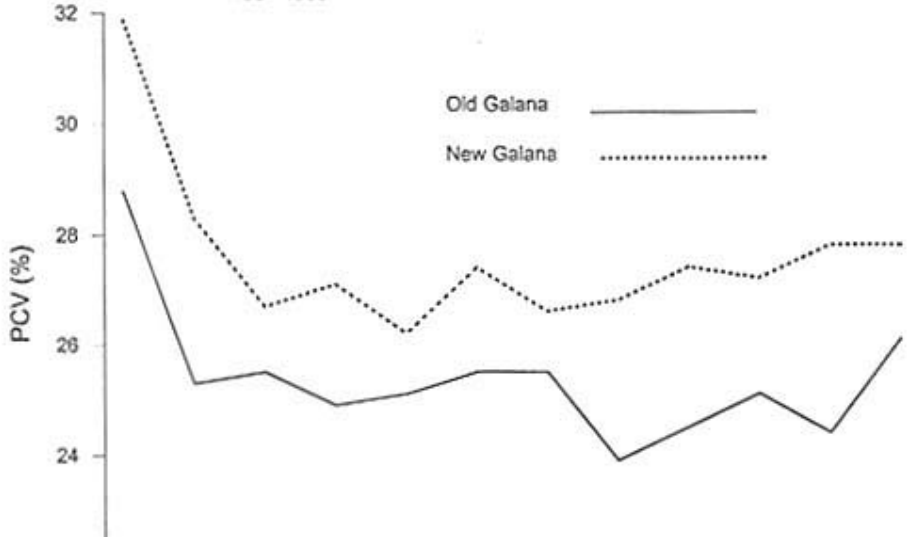

Old Galana
New Galana
PCV (%)
32
30
28
26
24

Figure 3 *Mean monthly PCV in 30 previously exposed (old) and 30 newly introduced (new) Orma and Galana steers.*

Although the numbers of infections detected in the new and old groups in Year 2 were similar, the species of infecting trypanosome were different. In the old Orma steers *T. congolense* infections predominated and 79 of the 124 infections were attributed to this species (*vivax* ratio of 0.57). The reverse was true in the new Orma steers in which 38 of the 124 infections were caused by *T. congolense* (*vivax* ratio of 2.26). The same pattern was observed in the Galana steers; the vivax ratio in the old Galana steers was 1.75 compared with 2.74 in the new Galana steers.

The regression of number of infections in Year 2 on number of infections in Year 1 for the 60 steers combined was 0.66 ± 0.11 (Figure 4) and the regression of mean PCV in Year 2 on mean PCV in Year 1 was 0.77 ± 0.06 (Figure 5). In both cases the regression was positive and significant ($P < 0.001$). This relationship was examined separately in each breed for *T. congolense* and *T. vivax* infections; a positive regression was detected in all cases and thus the data were combined. These data also provided estimates of repeatability. The repeatability of number of infections over the two years was 0.24. Weekly PCVs had repeatability estimates of 0.27 and 0.26 in Year 1 and Year 2 respectively.

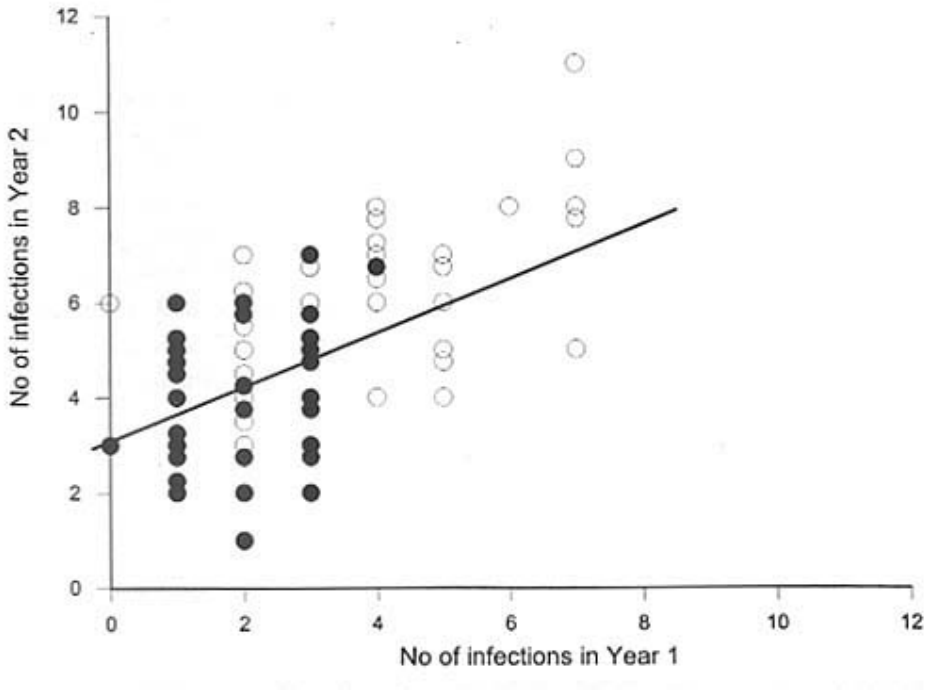


Figure 4. *The regression of number of infections in Year 2 on number of infections in Year 1 in 30 Orma (closed circles) and 30 Galana (open circles steers.*

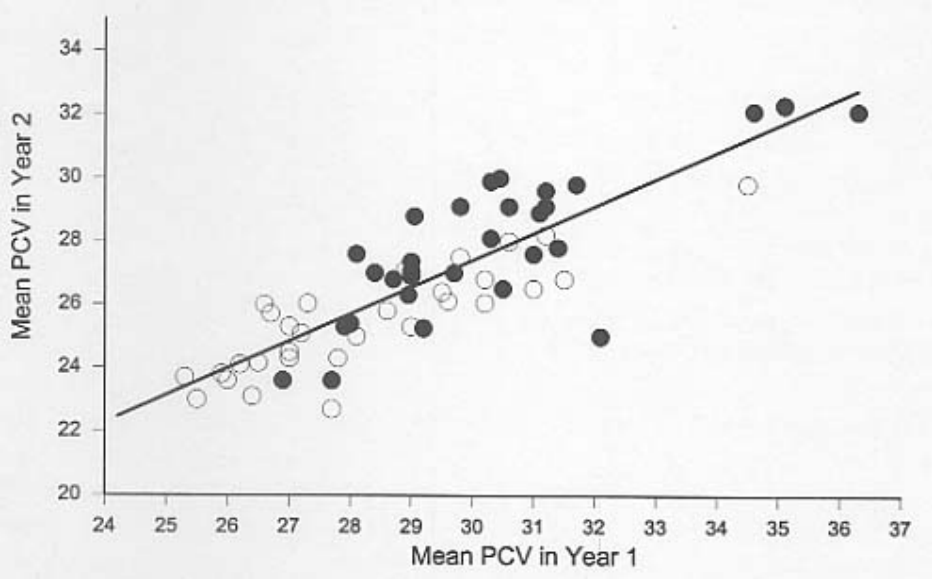


Figure 5. *The regression of mean PCV in year 2 on PCV in Year 1 in 30 Orma (closed circles) and 30 Galana (open circles) steers.*

Discussion

The results of this study provided little support for the suggestion that trypanosome prevalence decreases over time in animals under constant exposure to tsetse challenge (Murray et al 1982). In the trial reported here it was clear that differences in trypanosome challenge from one year to the next were more important than previous exposure in determining infection rates. Overall infection rates in the previously exposed steers were similar to those recorded in the newly introduced steers.

However, there was some evidence that the control of anaemia improved with exposure to trypanosomosis. The reduction in PCV associated with infection was less in Year 2 (Table 4) and mean PCV in infected animals was higher in the second year

despite a greater trypanosome challenge. This was more apparent in the case of the Orma steers and in relation to *T. vivax* infections. Furthermore, the *vivax* ratio was lower in the old than the new steers lending support to the idea that the previously exposed steers had acquired an immunity to some *T. vivax* infections but not to *T. congolense* infections.

This apparent acquisition of some immunity to *T. vivax* was not reflected, however, in a lower Berenil Index in the old steers when compared with the new steers. A possible explanation for this may be that while immunity may have developed to some *T. vivax* strains this simply resulted in the detection of *T. congolense* infections instead. This would result in no difference in the Berenil Index in old and new steers but a significant difference in the *vivax* ratio. *Trypanosoma vivax* infections are more easily detected than *T. congolense* infections; they generally give rise to higher parasitaemias and the *T. vivax* organism is more motile and more easily seen on examination of the blood slide. Thus, many mixed infections may have been recorded as *T. vivax* infections so that where *T. vivax* predominated the infection rate with *T. congolense* may have been underestimated.

The existence of a positive regression of response to trypanosomosis in Year 2 on that in Year 1, as measured both by infection rates and PCV, indicates that innate immunity to the disease was more important than acquired immunity. Thus, while there was some evidence of an acquired immunity to *T. vivax* following trypanosome challenge this was not reflected in the regression analyses as the innate responses were more powerful. The strength of the innate response was confirmed by the observation that a positive regression was detectable in each breed and for both trypanosome species. Furthermore, the repeatability both of number of infections and of PCV provides evidence of the existence of permanent factors, possibly genetic, which affect the animal's response to challenge.

4 Acquired and innate resistance to trypanosomosis under a delayed treatment regime

Introduction

There is some disagreement in the literature regarding the type of previous exposure necessary to elicit development of immunity to trypanosomosis. Wilson et al (1976) reported that animals treated with diminazene aceturate on detection of infection, as done in the experiment described in Chapter 3, developed a better immunity than those on other drug regimes. Welde et al (1979) drew a distinction between the development of immunity in animals re-challenged after self-cure and that in animals re-challenged after therepeutic cure. Vos et al (1988) suggested that challenge delivered after early cure was unlikely to provide the animal with exposure to all the different variable antigen types of the trypanosome and that prolonged infection was necessary if animals were to develop any degree of immunity. If prolonged exposure to the trypanosome is necessary then the immediate treatment regime adopted in the previous experiment (Chapter 3) may not have provided sufficient exposure to the parasites and may therefore have underestimated the importance of acquired immunity. This second experiment was designed to study the impact of previous exposure in groups of steers in which infection was prolonged and treatment was administered only if the PCV fell to a critical level of below 16%.

Materials and Methods

The groups of steers that were used to estimate mortality in the 1986 trials (Table 2, Chapter 2) were used in this experiment. In that year mortality rates were estimated in two different areas of the ranch each with ten Orma and ten Galana steers which were treated only when their PCV value, measured weekly, fell below 16%. These steers had been exposed to prolonged infections from the trypanosome serodemes present in two different areas of the ranch: Kapangani (referred to here as Area 1) and Kisiki (Area 2). In the following year (1987) these steers were monitored weekly under the

same regime but in this year both groups of steers were kept at Kapangani. In addition, in this second year, a third group of steers (10 Orma and 10 Galana) was introduced into Kapangani for the first time and herded with the previously exposed steers.

The results reported here are from the two years and consist of a comparison of the two Boran breeds in three groups as follows:
(i) Group 1 - Orma and Galana steers- monitored under the same tsetse challenge in Kapangani (Area 1) for two years and treated only if the PCV fell to below 16%.
(ii) Group 2 - Orma and Galana steers- monitored as for (i) but in Kisiki (Area 2) in the first year and in Kapangani (Area 1) in the second year.
(iii) Group 3 –Orma and Galana steers - newly introduced into Area 1 in the second year and monitored under the same regime as the other two groups.

This experimental design allowed for a number of comparisons in the second year. Steers with prolonged exposure to the serodemes in Area 1 were compared with steers with prolonged exposure to the serodemes in a different area of the ranch (Area 2). These two groups (Group 1 and Group 2) were also compared with steers, new to the area and not previously exposed to prolonged infections (Group 3). Furthermore, the response of Group 1 and Group 2 steers in Year 2 was compared with that in Year 1.

Results

The trypanosome challenge in both years was primarily caused by *T. congolense* but with a high percentage (22%) of mixed *T. congolense*/*T. vivax* infections. Under a regime of delayed treatment animals that became infected with one species of trypanosome and remained untreated were likely to become infected with another species. Forty six percent of the infections in the Orma steers and 50% of the infections in the Galana steers were caused by *T. congolense* alone. These proportions of the different trypanosome species were similar in the two areas in the two years;

infections caused by different trypanosome species were therefore not considered separately in the analysis.

Number of infections and packed cell volume

The proportions of cattle in each group that became infected are shown in Table 5. The numbers of infections detected in these infected cattle are also tabulated and each infection is classified as either treated, self-cured or on-going. An infection was considered to have self-cured if no parasites were detected over a three month period following infection and the PCV recovered to over 30%. Infections not treated or not self-cured as defined above were classified as on-going at the end of the year.

Table 5. Proportion of animals infected and the number of infections detected, number of infections treated, mean time to treatment (±standard error), number of infections self-curing and number of infections on-going at the end of the year in Orma and Galana steers in different areas of Galana Ranch during two years.

			Orma									
Year	Group	Area	Proportion of cattle infected	Infections detected	Infections treated	Mean time to treatment (weeks)	Infections self cured	Infections on-going	Proportion of cattle infected	Infections detected	Infections treated	Mean to trea
1	1	1	8/9	11	5	13.4±6.2	3	3	9/10	18	15	
1	2	2	7/10	8	6	5.5±1.4	1	1	9/10	18	11	
2	1	1	8/9	12	9	12.8±5.4	1	2	9/9	16	16	
2	2	1	6/9	6	2	6.5±1.5	3	1	9/9	13	12	
2	3	1	3/10	4	2	5.0±1.0	1	1	9/10	15	14	
	Total		32/47	41	24		9	8	45/48	80	68	

One of the Group 1 Orma steers was killed by lion in the third month of the first year of the trial; it was not infected at the time and was excluded from the analysis. During the first year two Galana steers (one Group 1 and one Group 2) died of trypanosomosis in spite of treatment. In the second year no Orma steers died and eleven remained uninfected throughout the trial; seven of these in the newly introduced Group 3 (Table 5). In the three Galana groups only one steer remained uninfected during the second year and two deaths were recorded, one was a suspected case of foot and mouth disease and the other animal suffered from Besnoitiosis.

There was no reduction in the number of infections detected in the second year in the previously exposed (Groups 1 and 2) compared with the newly introduced steers (Group 3). Indeed, in the Orma steers more infections were detected in the previously exposed steers. In Figures 6 the mean monthly PCV in Year 2 is shown for the three Orma and the three Galana groups. The PCV values reflected the infection rates; amongst the Orma Group 3 steers those with the least number of infections had the highest PCVs. The PCVs observed in the three Galana groups were very similar.

Figure 6

There were large differences in response between the two breeds (Table 5). Each of the Orma groups had, in each year, fewer infections and fewer treatments than the comparable Galana group. Overall there were twice as many infections detected in the Galana steers. Eighty five percent of the Galana infections required treatment compared with 59% of the Orma infections and 22% of the Orma infections self-cured by comparison with 5% of the Galana infections.

In Figure 6c the mean monthly PCV in the second year, in the three Orma groups combined, was compared with that of the three Galana groups combined. The mean PCV in the Galana steers was consistently lower than that in the Orma steers despite a total of 42 treatments administered to the three groups of Galana steers during the course of the year compared with 13 treatments in the three Orma groups.

Time to treatment

Time to treatment was very variable (Table 5) with some infections requiring treatment immediately while in other cases treatment took place several months after detection of parasites. An improvement in the control of the development of anaemia over time would have been reflected in an increase in the time to treatment in a group. This occurred only in Group 2 Galana steers in which time to treatment increased from 0.8 weeks in Area 2 in Year 1 to 12.6 weeks in Area 1 in the following year. A

separate analysis was undertaken to examine the differences between groups and between breeds in Year 2. Time to treatment was on average slightly longer in the Orma than in the Galana steers. It was also longer in the previously exposed steers than in the newly introduced steers. However, the differences were not statistically significant in either case.

Discussion

The regime adopted in this trial might have been expected to provide the animals with a better opportunity to acquire immunity than that used in the immediate treatment experiment (Chapter 3). This does not appear to have been the case. The regime adopted here of prolonged exposure to infection failed to cause any significant improvement in response to tsetse challenge in four groups of steers exposed for two years. The extended time to treatment observed in the Group 2 Galana steers in the second year seems likely to be related these to steers being in a different area in the second year. It is possible that the strains of trypanosomes present in Area 2 were more virulent than those in Area 1; this would explain the difference observed in both the Orma and Galana steers in the two areas in Year 1

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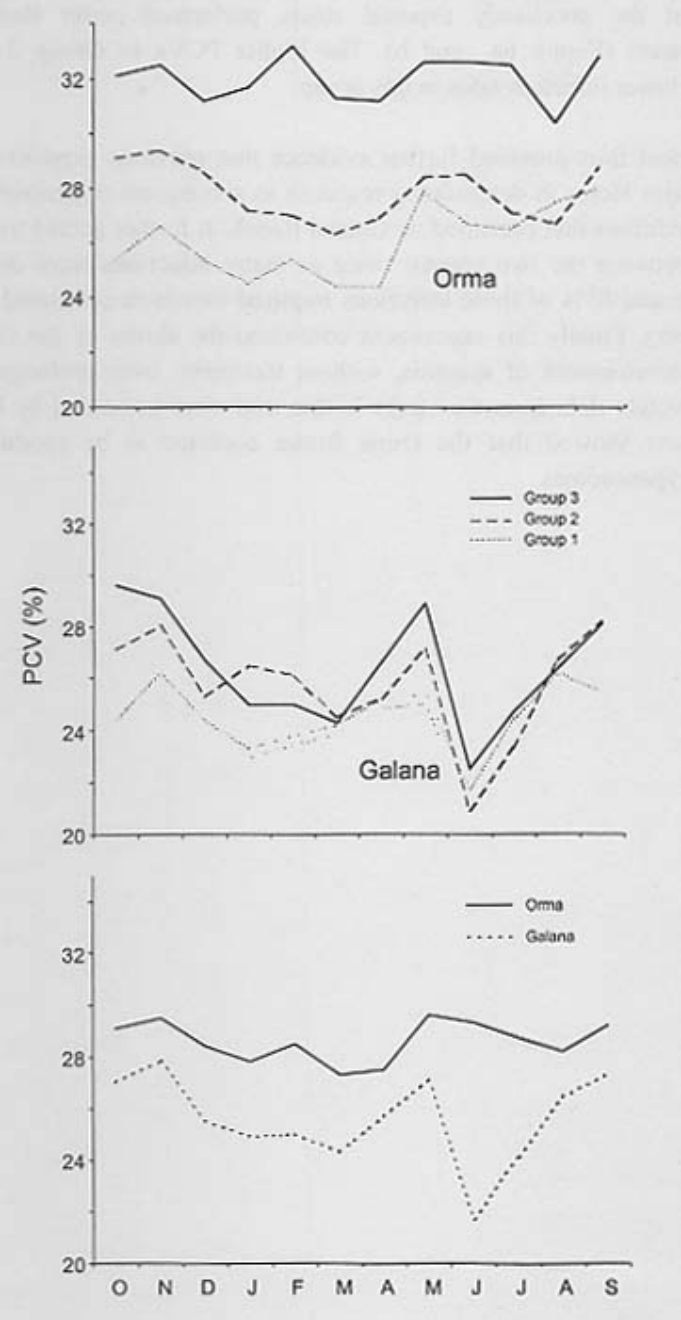


Figure 6. (a and b) *Mean monthly PCV in previously exposed (group 1 and 2) and newly introduced (group 3) Orma and Galana. 6c Mean monthly PCV in the three Orma groups combined and the three Galana groups combined.*

Comparison of the Group 3 steers with the Group 1 and 2 steers failed to provide evidence that the previously exposed steers performed better than the newly introduced steers (Figure 6). The higher PCV values in Group 3 Orma steers reflected the lower infection rates in this group.

This experiment thus provided further evidence that previous exposure to infection was not a major factor in determining response to subsequent trypanosome challenge under the conditions that prevail on Galana Ranch. It further served to highlight the differences between these two breeds; twice as many infections were detected in the Galana steers and 85% of these infections required treatment compared with 59% in the Orma steers. Finally this experiment confirmed the ability of the Orma cattle to control the development of anaemia, without treatment, over prolonged periods of time. Bodyweight data from the steers in this trial are presented in Dolan (1993) and the results showed that these cattle continue to be productive despite harbouring trypanosomes.

5 Differential response to tsetse and trypanosomosis challenge in Orma and Galana Boran calves

Introduction

The trials described in Chapter 2 demonstrated that the Orma Boran, originating from the Tana River District, was less susceptible to tsetse challenge than the ranch-bred Galana Boran. However, the data to substantiate this claim were gathered from animals born in different environments and which had, prior to the trials on Galana Ranch, undergone different exposure to tsetse challenge. Trials reported in Chapters 3 and 4, however, indicated that previous exposure did not play a significant role in determining the response of these two cattle types to tsetse challenge. But to demonstrate unequivocally that the differences between the two breeds were genetic, comparisons between calves born together in the same environment and exposed from birth to the same tsetse challenge were necessary.

In 1983 breeding herds of Orma Boran and Galana Boran cows were established on Galana Ranch under KETRI management (Dolan et al 1987). In addition to providing contemporaneous data for a breed comparison, the establishment of an Orma breeding herd aimed to provide information on other aspects of productivity previously unmeasured in this breed. Its decreased susceptibility to trypanosomosis could be studied in greater detail and the herd would provide the foundation stock for a long-term selection programme for improved productivity under trypanosome challenge.

Materials and Methods

The foundation cows

The Orma cows that formed the foundation stock for the Orma breeding herd were purchased from the Tana River District by the ranch management between August and December 1983. The Orma people were very reluctant to sell their breeding stock and, in general, only cull cows were offered for sale. These cows were trekked on to the ranch and held initially in the ranch quarantine area for a three to four month period. There they underwent various quarantine procedures. This area located close to the eastern boundary of the ranch was tsetse-infested. No trypanocide prophylactics

were administered; individual cows that showed clinical symptoms of trypanosomosis may have been treated with diminazene aceturate but no records were available for this period. The cows, some with calves at foot, were then moved into Kapangani and handed over to KETRI for ear-tagging. Recording commenced on the first group of 83 cows in October 1983. These were joined by a further group of 34 cows in February and March 1984 and a final group of 15 cows joined the recording system in November 1984.

The ages of the Orma cows were estimated through inspection of their teeth. For the younger animals, in which incisors were still erupting, two or three observations were taken to establish the time of eruption more accurately (Carles and Lampkin 1975). Thirty one of the Orma cows were culled before the end of 1984 either on the basis of age or reproductive defects (Table 6). Fourteen of these cows entered the recording system either pregnant or with calves at foot and were therefore included in the analysis; the other 17 which were culled in the first year had no calves and were excluded from the analysis. A further 18 cows were culled in 1985 and nine in-calf cows, selected on the basis of weight and good general health status, were transferred to KETRI headquarters at Muguga for experimental purposes. Twenty eight cows were culled in 1986 and six heifers born from the original stock had calves before the end of that year and were included in the analyses (Table 6).

Table 6. *Numbers of Orma and Galana cows present over the study period.*

	1984	1985	1986	1987
Orma				
Number of cows	117[+]	96	67	42
Number culled[++]	31[+]	18	28	0
Number transferred	0	9	0	0
Number new cows	15	0	6	0
Galana				
Number of cows	99	96	80	73
Number culled [++]	0	0	5	0

[+] 17 cows with no calves were excluded from analysis
[++] Mortality not included

The Galana cows, which were provided by the ranch management, were all born on Galana Ranch and consisted of 99 heifers, aged 27 to 36 months when recording commenced in October 1983. Prior to being transferred to KETRI management these animals had been given isometamidium chloride every three months if grazed in tsetse-infested areas. They were generally in a much healthier state than the Orma cows at the start of the experiment; no culling was required apart from five animals that failed to calve and were culled in 1986 (Table 6).

Herd management

The two breeding herds were grazed in the same area of Galana Ranch but in two separate herds. Animals were grazed during the day and kept in thorn bush bomas at night to protect them from predators.

Sire identity was required for the purpose of estimating genetic parameters. An experimental design with several sire groups within each breed producing offspring at the same time, whilst optimal, is not practical under extensive range conditions. A compromise system that was agreeable to the management and provided the necessary information had to be established. Normal ranch practice was to run four bulls with every two hundred cows. Initially the ranch management was reluctant to accept limiting this to one bull at a time and no information on sire identity was available for calves born in the first year. For calves born from the end of 1984 onwards, however, sire identity was known in most cases. A single Orma and Galana bull ran with their respective cow herds for six to eight weeks and another bull was introduced after a two week interval. This interval was later increased to four weeks. The calving intervals achievable under such a system were longer than those achieved under conventional ranch management. In addition to periods when there was no bull with the cows the calving intervals were further prolonged if the newly introduced bull did not function efficiently. Prophylactics were administered to bulls before they joined the cow herd to insure that they remained free of trypanosomes during the period they were with the cows.

In an effort to find an area with moderate tsetse challenge suitable for both herds the location of the trial was changed twice during the four years. From October 1983,

when recording first began, until the end of September 1985 the two herds were kept in Kapangani. Concerns about the high mortality rates in the Galana breeding herd resulted in the trial being moved to Kisiki an area with slightly higher rainfall and close to the Galana River. A tsetse control trial using insecticide impregnated odour baited targets was initiated in January 1986 (Opiyo et al 1987) in a 25km^2 area along the river, two kilometers from the Kisiki crush. These targets proved so effective that tsetse challenge was reduced in a much wider area and the breeding herds were transferred back to Kapangani in July 1987. The Galana breeding herd was disbanded in August 1987.

Tsetse challenge and rainfall

The tsetse challenge in the Kapangani area was primarily from *G. pallidipes* but with increasing numbers of *G. longipennis* as the trial progressed. *Glossina pallidipes* was also the principle vector of the disease in Kisiki but *G. longipennis, G. brevipalpis* and *G. austeni* were also found in significant numbers. Rainfall data were available from October 1984 onwards when a simple rain gauge was set up at both sites. The data were not collected directly by KETRI staff; the ranch provided staff for this purpose. Daily rainfall records were summed into monthly totals but there were some months in which no data were available. There were indications that on many occasions the rain was recorded and the gauge emptied every second or third day rather than daily. However, the monthly totals provided a good measure of rainfall over the month.

Packed cell volume and bodyweight recording

Blood from the ear vein was taken from all the cows and their calves every two weeks; packed cell volume (PCV) was estimated and the buffy coat examined for trypanosomes. Presence or absence of parasites and species of parasite were recorded. Diminazene aceturate, at a dose rate of 7 mg/kg bodyweight, was administered to animals in which parasites had been detected and the PCV dropped to below 26% or, at times when the challenge was considered low, below 21%.

In cases when the PCV dropped to below 26% and trypanosomes were not detected in the buffy coat additional blood smears were examined. Where *anaplasma* was

detected treatment with antibiotics was administered. If no parasites were detectable on either buffy coat or wet smear examination and the animal, in addition to having a low PCV, was considered to have clinical signs of trypanosomosis, then treatment for trypanosomosis was administered. Ticks were controlled by weekly spraying with acaricide and animals were treated with anthelmintics twice yearly.

Bodyweights were taken monthly and, in addition, birth weights and weaning weights of calves were recorded. Post mortem examinations and pregnancy diagnoses were also performed.

Data recording and analysis

All the data recorded were entered into recording books on Galana Ranch and copies of the data were sent to KETRI headquarters at Muguga at the end of every month. These records were entered into the computer at KETRI. PANACEA, a software package developed for livestock data by Pan Livestock Services Limited, Reading, UK, was used. The package incorporated standard statistical analyses and monthly and yearly summaries of the data were produced for KETRI internal reports. A full-scale analysis of the data required access to a general statistical least squares analysis computer programme. The African Trypanotolerant Livestock Network of The International Livestock Centre for Africa (ILCA) agreed to assist KETRI with the analyses. All the data, a total of 15,251 calf records and 46,449 cow records, were checked and verified against the original recording. Least squares analyses were undertaken using Harvey's least squares analysis program (Harvey 1990).

The statistical analyses concentrated on the calf data and the results from this data set on pre-weaning mortality, trypanosome infection and treatment rates, PCV, birth and weaning weights and average daily gain to weaning presented first. Births took place over 44 months between May 1983 and December 1986. Calves born in 1987 had not reached weaning age by August 1987 comparisons between Orma and Galana ceased; these calves were not included in the analyses. A total of 190 Orma calves and 169 Galana calves were born over the study period.

No birth weight data were available on 43 Orma calves, of which 40 were born before recording commenced. Age of dam and season (time) were confounded in the Galana cows, which all started the trial at two to three years of age. The impact of age of dam on the various traits analysed is considered in Chapter 6 in the analysis of the complete Orma data.

Comparisons of the performance of the Orma and Galana cows were more difficult as the two foundation groups were so different. However, some analyses were conducted to provide information of the impact of trypanosomosis on cows compared with calves. Data collected on cows, when they had calves at foot, were analysed. Trypanosome infection rates, PCV and change in cow weight during lactation were the variables considered.

Designation of seasons for breed comparison analyses

Seasons were designated as wet or dry according to rainfall. When no rainfall data were available for a month a measure of trypanosome challenge for that month was considered as an additional source of information in verifying the classification of seasons. Trypanosome challenge was measured by means of the Berenil Index in herds of 30 to 60 sentinel cattle monitored for parasitaemia on a weekly basis. These herds were not part of the breeding herds but were grazed in the same areas of the ranch.

Rainfall on Galana Ranch is bimodal with a brief dry spell in January and February followed by rain from March to May and a longer dry spell between from June to September. The rainfall information available for 1984 to 1987 (Table 7) indicated that each year could be divided into two seasons. The first "wet season" commenced in November (and included two dry months) and the "dry season" ran from June to October. The rains on Galana Ranch generally commence in October but there is little measurable impact either in terms of improved grazing or increased tsetse challenge until November. Years, for the purpose of the analyses, were considered to commence in November and end in October.

Calves were classified according to season of birth. The first season, in the classification in Table 7, May - October 1983, was before recording commenced but some Orma calves born during this period were included in the analyses and their season of birth was classified as dry. The last season in the classification covered births that took place in November and December 1986 only. Orma calves were born in all 44 months between may 1983 and December 1986 with the exception of July and November 1984 and July and December 1986. The distribution of births over time was more clumped in the Galana calves (Table 7). The first Galana calf was born in April 1984 and in that year the majority of Galana cows calved in June and July. The low trypanosome challenge in the 1986/87 wet season (see Berenil Index, Table 7) was a result of the tsetse control programme described earlier (Opiyo et al 1987).

Year and season of birth, classified as wet or dry, were used in some initial analyses but the presence of a year x season interaction indicated that seasonal effects were better accounted for by fitting each season separately for each year. Classification of calves by season of birth involved grouping together calves that experienced varying levels of tsetse challenge between birth and weaning. For example calves born in November of one year were grouped with calves born in May of the following year. The November born calves were weaned in July and therefore only overlapped for two pre-weaning months with the calves born in May. A classification of calves by month of birth might have been more meaningful but would have resulted in very small sub-class numbers.

Table 7. *Total rainfall (mm) and average monthly Berenil Index in different seasons between 1983 and 1987 and the distribution of births.*

	Season		Total rainfall (mm)	Berenil Index	No. Orma calves born	No. Galana calves born
1	May ‘83 - Oct ‘83	Dry	-	-	40	0
2	Nov. ‘83- May ‘84	Wet	-	5.2	33	1
3	June ‘84 - Oct ‘84	Dry	-	0.3	24	71
4	Nov. ‘84- May ‘85	Wet	303	6.2	28	12
5	June ‘85 - Oct ‘85	Dry	11	1.1	24	2
6	Nov. ‘85- May ‘86	Wet	419	3.9	30	63
7	June ‘86 - Oct ‘86	Dry	32	2.0	6	7
8	Nov. ‘86- May ‘87	Wet	345	0.9	5	13

Results

Orma and Galana calves

Pre-weaning mortality

Seven percent (14/190) of the Orma calves died before weaning (Table 8) compared with 17% (28/169) of the Galana calves ($\chi^2_1 = 7.32$, $P < 0.01$). Seven of the 14 deaths in the Orma calves resulted from inadequate mothering. Many of the Orma cows, being cull cows with ages varying from three to 14 years, had defective teats and their calves died of malnutrition. In the case of the Galana calves trypanosomosis was the principal cause of death. Ten calves died of trypanosomosis and three other calves died as a consequence of their dams suffering from the disease. Only one Orma calf died of trypanosomosis; the other two deaths attributed to trypanosomosis (Table 8) were associated with trypanosomosis in the dam.

Table 8. *Calf mortality and trypanosome infections and treatments between birth and weaning in Orma and Galana calves.*

	Orma	Galana
No. of calves born	190	169
No. of calf deaths	14	28
No. of calf deaths due to trypanosomosis [+]	3	13
No. of calves in which parasitaemia/PCV was recorded	184	160
No. of calves infected	55	67
No. of *T. vivax* infections detected	38	68
No. of *T. vivax* infections treated	25	66
No. of *T. congolense* infections detected	23	27
No. of *T. congolense* infections treated	19	25
Tv:Tc ratio	1.65	2.52
No. of NPS treatments	24	55
Total number of trypanosome infections (including NPS)	85	150
Average number of infections per calf.	0.46	0.94

[+] Trypanosomosis is cited as the cause of death where the calf itself had the disease or where the dam died of, or was unable to feed the calf because of, trypanosomosis and the calf died as a consequence

Trypanosome infection and treatment

Twenty nine percent (55/184) of the Orma calves in which parasitaemia was recorded (Table 8), compared with 42% (67/160) of the Galana calves, were detected positive for trypanosomes. The difference in infection rates between breeds was primarily due to a difference in the numbers of *T. vivax* infections ($\chi^2_1 = 13.7$; $P < 0.001$). The difference in the number of *T. congolense* infections was not significant. The Orma calves also required fewer treatments than the Galana calves particularly for *T. vivax* infections. Sixty six percent of *T. vivax* infections in the Orma calves were treated compared with 97% in the Galana ($\chi^2_1 = 19.6$; $P < 0.001$). Treatment of infected animals generally took place when the PCV fell to below 26%, or below 21% when the challenge was deemed to be low. However, in some cases where clinical symptoms of the disease warranted, treatment was administered earlier. Fifteen Galana calves and four Orma calves were treated with diminazene aceturate when parasitaemic but with a PCV of 25% or above.

In addition to the treatment of patent parasitaemia, some calves in which no parasites could be detected also received diminazene aceturate treatment; these were referred to as “no parasites seen” (NPS) treatments. Such treatments were administered if the PCV was below 26%, no other haemoparasites were detectable and the animal showed clinical signs of trypanosomosis. Twenty four NPS treatments took place in the 184 Orma calves compared with 55 in the 160 Galana calves and this difference was significant ($\chi^2_1 = 16.5$, $P < 0.001$). These treatments were administered to calves born in Seasons 2, 3 and 4 while the herd was in Kapangani and the trypanosome challenge high (Table 7). When each of these additional NPS treatments was regarded as a trypanosome infection then the total number of infections increased to 85 in the 184 Orma calves and to 150 in the 160 Galana calves.

Trypanosome infections were also analysed using logistical regression analysis. The number of infections per month was calculated for each calf and the effects of breed,

season of birth and sex were analysed. However, the data did not lend themselves easily to such analyses because the majority of calves never became infected and the distribution of calf births over time was different in the two breeds (Table 7). The results of these analyses are not presented in detail here but are given in Dolan (1993). In summary, there were large and significant seasonal effects, male calves had significantly more infections than female calves and the breed differences apparent in the simpler χ^2 analyses were confirmed.

Packed cell volume

Mean packed cell volume was calculated for all calves in which PCV was measured, excluding those calves which died within the first two weeks of life. Least squares analysis examined the effect of season of birth, breed, sex and weaning age and the first order interactions. There were no significant interactions. The final model included season of birth, breed and sex and accounted for 28% of the variation in PCV. There were significant differences among seasons ($P < 0.001$) and between breeds and sexes ($P < 0.05$). The mean PCV in the Orma calves was higher than that in Galana calves for all seasons of birth (Table 9).

Table 9. *Mean packed cell volume and standard error (SE) in Orma and Galana calves born in different seasons*

				Orma			Galana		
Season of birth			Location of herd	n	PCV	SE	n	PCV	SE
1	May ’83 - Oct ‘83	Dry	Tana/Galana	30	31.3	0.5	0	-	
2	Nov. ‘83- May ‘84	Wet	Kapangani	40	29.7	0.5	1	28.5	
3	June ’84 - Oct ‘84	Dry	Kapangani	24	28.1	0.6	68	27.8	0.4
4	Nov. ‘84- May ‘85	Wet	Kapagani	28	27.7	0.6	11	24.8	0.9
5	June ’85 - Oct ‘85	Dry	Kapagani	24	29.0	0.6	2	27.0	2.1
6	Nov. ‘85- May ‘86	Wet	Kisiki	28	32.0	0.6	59	31.6	0.4
7	June ’86 - Oct ‘86	Dry	Kisiki	6	28.1	1.2	7	27.5	1.1
8	Nov. ‘86- May ‘87	Wet	Kisiki	5	28.9	1.3	13	28.0	0.8
Sex									
	Male			97	29.1	0.4	75	27.7	0.6
	Female			88	29.6	0.4	86	28.6	0.6
Total mean				185	29.3	0.3	161	28.2	0.6

Mean PCV reflected the infection rates that were highest in calves born in Season 3 and 4; mean PCV values were low for these calves. The higher infection rates in the Galana calves compared with the Orma calves also corresponded with lower mean PCV values. Mean PCV was higher in female calves than in male calves (Table 9) for both breeds and the overall difference in PCV between breeds was significant ($P < 0.05$).

Birth weight

There was no birth weight information available for calves born prior to November 1983. Preliminary analyses indicated that there was a significant interaction between year of birth and season of birth and therefore seasons were fitted, as for the other variables, as seven consecutive time periods. The final model, run on 316 birth weights, fitted season, breed and sex as main effects, and accounted for 48% of the variation in birth weight. There were large and highly significant differences in the birth weights of calves born in different seasons ($P < 0.001$); season of birth alone accounted for 37% of the total variation in birth weight. However, there appeared to be no pattern related to wet or dry seasons (Table 10).

Table 10. *Least square means (x) and standard errors (SE) for birth and weaning weights in Orma and Galana calves born in different seasons*

Season of birth			Location of herd	Orma						Galana					
				Birth weight			Weaning weight			Birth weight			Weaning weight		
				n	x	SE	n	x	SE	n	x	SE	n	x	SE
1	May '83 - Oct '83	Dry	Tana River/ Galana	-	-		30	104.4	3.9	-	-		-	-	
2	Nov. '83- May '84	Wet	Kapangani	31	18.9	1.5	35	100.7	3.6	1	29.7		1	104.7	
3	June '84 - Oct '84	Dry	Kapangani	23	22.1	0.9	22	103.9	4.6	71	24.1	2.1	58	123.7	2.8
4	Nov. '84- May '85	Wet	Kapangani	28	19.4	1.4	27	98.4	4.2	12	25.2	2.8	8	138.5	7.6
5	June '85 - Oct '85	Dry	Kapangani	24	14.9	1.1	23	101.2	4.5	2	17.9	3.7	2	128.7	15.2
6	Nov. '85- May '86	Wet	Kisiki	30	15.4	1.4	27	112.4	4.2	63	17.1	1.1	55	148.1	2.9
7	June '86 - Oct '86	Dry	Kisiki	6	17.2	1.7	6	131.2	8.8	7	19.0	2.9	5	180.7	9.5
8	Nov. '86- May '87	Wet	Kisiki	5	21.9	2.3	5	131.4	9.5	13	25.1	2.7	12	164.0	6.2
Sex															
	Male			78	18.9	0.7	63	113.9	2.6	75	21.6	0.5	91	145.7	4.8
	Female			71	17.6	0.5	84	107.0	2.7	92	20.6	0.5	78	134.9	4.6
Total mean				149	18.3	0.3	147	110.4	2.0	167	21.1	0.4	169	140.3	4.3

There were also significant differences between breeds and between sexes. The Galana calves were significantly heavier by 2.8 kg at birth than the Orma calves ($P < 0.001$) and male calves were on average 1.2 kg heavier than female calves ($P < 0.05$).

Birth weight was also considered as a factor influencing survival. The least squares mean birth weight, corrected for season, breed and sex, of calves which survived to weaning was 19.9 ($\pm$ 0.3) kg compared to 18.2 ($\pm$ 0.6) kg in the non-survivors ($P < 0.01$).

Weaning weight and average daily gain to weaning

Calves were weaned at approximately eight months of age and weaning weights were recorded on 147 0rma and 169 Galana calves. Average daily gain (ADG) was calculated for 274 calves which survived at least one month and for which birth weights were recorded; 257 of these survived to weaning. Because of the different number of observations in the two data sets, analyses were performed on both weaning weight and ADG. Similar results were obtained in both cases and, to avoid repetition, the results for one or other variable only are presented.

Analysis of variance of weaning weight was undertaken in which season of birth, breed, sex and weaning age were fitted together with the first order interactions. There were no significant first order interactions. In the final model season of birth, breed, sex and weaning age were all significant ($P < 0.001$) and the model accounted for 50% of the total variation. The least squares means for weaning weights for the two breeds of calves born in different seasons are shown in Table 10. Calves born in Kisiki in the latter part of the study reached higher weaning weights. Weaning weights of the Galana calves were on average 30% higher than those in the Orma calves, and male calves were weaned 7% heavier than female calves (Table 10).

The impact of trypanosome challenge on growth of Orma and Galana calves

The number of infections per month (counting both patent and non-patent (NPS) infections) was included as a covariate in analyses of average daily gain in calves that survived at least one month. Season of birth, breed and sex were included as fixed effects in the model. There was a significant interaction between breed and number of infections ($P < 0.01$). Infection

significantly depressed ADG in the Galana calves but had no impact on the Orma calves. In the Galana calves there was a decrease in ADG of 16.5 (± 5.5) g for every 0.1 increase in the number of infections per month. In the Orma calves the regression was actually positive but not significantly different from zero (12.5 ± 9.6). There were also significant differences in ADG related to breed ($P < 0.01$), sex ($P < 0.01$) and season ($P < 0.001$) as already shown for weaning weight.

Orma and Galana cows

The 99 Galana Boran heifers began the trial between 27 and 36 months of age. The Orma cows commenced the trial aged between 3 and 12 years of age and many of them had to be culled (Table 6). A limited number of comparisons are presented here; more detailed results can be found in Dolan (1993).

Table 11. *Number of Orma and Galana cows with different numbers of calves born between 1983 and 1987 and the mean calving interval (days), ± standard error.*

	No. of calves born per cow					
	0	1	2	3	Total	Calving interval
No. of Orma cows	3	59	49	11	190	520 ±14
No. of Galana cows	7	25	57	10	169	532±10

The Orma cows gave birth to 190 calves and the Galana cows to 169 calves over the study period (Table 11). Cows that did not give birth were excluded from the analyses and only data collected during lactation were analysed. Three Orma calves and six Galana calves died within a week of birth; their dams were also excluded. The results presented in Table 12 are from data on 349 lactation records from 204 cows.

Table 12. *Cow mortality and number of trypanosome infections during lactation for Orma and Galana cows.*

	Orma	Galana
No. of cow years	332	348
No. of cow deaths	10	23
No. of cow deaths due to trypanosomosis	8	18
No. of lactation records analysed	186	163
No. of *T. vivax* infections detected	67	143
No. of *T. congolense* infections detected	99	80

Tv:Tc ratio	0.68	1.79
No. of mixed infections detected	6	2
No. of NPS treatments	18	47
Total number of trypanosome infections (including NPS)	190	272
No. of total infections/cow/lactation	1.0	1.7

Morality was higher in Galana than in the Orma cows (Table 12), however, more Orma cows were culled (Table 6). The mean number of infections per cow per lactation was 1.0 in Orma cows compared with 1.7 in Galana cows. There were significant differences in the number of *T. vivax* infections detected in the two breeds but no difference in the number of *T. congolense* infections. Trypanosome prevalence was considerably higher in cows (Table 12) than in their calves (Table 8). Thus, 190 infections were recorded in the Orma cows during lactation compared with 85 infections in their calves. Two hundred and seventy two infections were recorded in the Galana cows compared with 150 in their calves. There were also differences in the trypanosome species involved in these infections. The majority of infections in the Orma calves were *T. vivax* infections while *T. congolense* was the predominant species in the cows. The *vivax* ratio in the Orma calves was 1.65 compared to 0.68 in the cows. *Trypanosoma vivax* infections predominated in both Galana calves and cows but were again more frequent in calves where the *vivax* ratio was 2.50 compared with 1.79 in the cows.

Mean PCV during lactation was calculated for each cow. Breed and season of birth of calf were considered as fixed effects and cow within breed was included as a random effect. There were no significant differences between breeds but there were differences among cows (($P < 0.001$) and among seasons ($P < 0.001$). Change in cow bodyweight during lactation was also analysed; there was no significant difference between breeds.

Repeatability of cow traits

In cows with more than one lactation record it was possible to measure the repeatability of trypanosome infection rate, mean PCV and weight change during lactation derived from the least squares analyses. These estimates are presented in Table 13. The repeatabilities of *T. congolense* infection rate, mean PCV and body weight change averaged over breeds were all significant.

Table 13. *Repeatabilities of traits, averaged for Orma and Galana cows, during lactation (± standard error).*

T. vivax infections	0.06 (± 0.10)
T. congolense infections	0.26 (± 0.09)
Mean PCV	0.53 (± 0.06)
Bodyweight change	0.42 (± 0.08)

Discussion

The results presented here confirm that the differences in response to tsetse and trypanosomosis challenge in Orma and Galana steers reported in Chapters 2-4 of this monograph. Here, the difference in response between these two Boran types has been observed in calves born in the same environment and reared together under the same tsetse challenge.

The difference between the two Boran types in the trypanosome prevalence confirmed previous observations and again this difference was primarily in relation to *T. vivax* infections. In both the Orma calves and cows significantly fewer *T. vivax* infections were detected. Thus it is clear that the Orma cattle have a superior innate ability to deal with *T. vivax* infections. Many bites from *T. vivax* infected flies never give rise to detectable infections. Also the Orma cattle are less likely to suffer from anaemia when infected with *T. vivax*; 66% of the *T. vivax* infections detected in Orma calves required treatment compared with 97% in the Galana calves (Table 8). Murray et al (1981) compared N'Dama and Zebu cattle under natural field challenge in The Gambia and found no differences in the prevalence of *T. congolense* but significant differences in the prevalence of. *T. vivax.*

An increased susceptibility to trypanosomosis in males was observed in zebu cattle in Ghibe, Ethiopia (Rowlands et al 1993). A similar trend was observed in both the Orma and Galana calves and PCV values (Table 9) were significantly lower in male calves when compared with female calves. This difference between the sexes in susceptibility to the disease was confirmed in the larger data set for the Orma breeding herd (Chapter 6).

The low trypanosome infection rates, observed in calves when compared to cows, agrees with other reported studies. Fiennes (1970) suggested that young animals were more resistant to trypanosomosis than adults. Wellde et al (1981) demonstrated that this was the case in a laboratory study in cattle infected with *T. congolense* and showed that it did not involve specific maternal antibodies. Rowlands et al (1993) reported a similar phenomenon in East African Zebu in Ethiopia although this may have been partly due to differences in exposure of calves and adults to tsetse. Trail et al (1993) reported no difference in the trypanosome prevalence between N'Dama cows and calves but significant differences in the infecting trypanosome species. The majority of infections in calves were caused by *T. vivax* while the reverse was true in cows. This difference in the vivax ratio was also observed in both breeds on Galana Ranch. In the Orma *T. vivax* was the most frequently detected trypanosome in calves while *T. congolense* predominated in cows. In the Galana *T. vivax* predominated in both Galana cows and calves but the vivax ratio was higher in the calves.

This study also substantiated earlier observations on differences in productivity of these two Boran types (Dolan et al 1985). The Galana Borans gave birth to significantly heavier calves than the Orma Boran and these calves had superior growth rates and at eight months reached weaning weights which were 30 kg heavier on average than those recorded for the Orma Boran. The superior growth rates of the Galana calves must be set against the differences in mortality, requirement for treatment and impact of infection on average daily gain. Ashley (1992) presented results from an economic analyses of these two breeds and concluded that under high challenge the Orma cattle were a better option. Indeed the concern of the management of Galana Ranch about the productivity of the Galana Borans under high challenge in Kapangani resulted in the movement of the herds to Kisiki and in the eventual termination of the trial in 1987. The ranch management concluded that maintaining a breeding herd of Galana Borans in a heavily infested area of the ranch was not a viable proposition. However, the Orma breeding herd has been successfully maintained in Kapangani from 1987 until 1997 (see Chapter 6).

The impact of infection on average daily gain provides further evidence of the superiority of the Orma cattle under tsetse challenge. The ability to continue growing despite the presence of

infection is often considered to be a major component of the trypanotolerance trait defined by Trail et al (1994a). Most of the infections detected in the calves were treated and this treatment helped restore growth rates. Nevertheless, despite a significantly greater number of treatments in the Galana calves, infections in this breed resulted in a significant loss of 16.5 (±5.5) g per day in growth to weaning per 0.1 increase in infections per month. In contrast infected Orma calves continued to grow.

The stocking rates on the ranch are such that availability of grazing is rarely a constraint and while increased rainfall brings improved grazing this is offset by increased tsetse challenge. Seasonal fluctuations in tsetse challenge are of more importance than seasonal fluctuations in grazing. The highly significant seasonal effects on birth and weaning weights and the different patterns across years indicate the impact of the disease on productivity. The major difference between the two areas of the ranch in which these cattle were grazed was in the level of tsetse challenge. The installation of insecticide odour baited targets in this area reduced the tsetse challenge to almost zero and calf weaning weights increased significantly (Table 10).

The 160 Galana calves received 146 diminazene aceturate treatments during the study period (0.91 treatments per calf) compared with 68 treatments administered to 184 Orma calves (0.37 treatments per calf). Despite receiving over twice as many treatments, mean PCV between birth and weaning was consistently lower in the Galana calves. In both breeds mean PCV values were higher in females, than in males, which was consistent with the lower infection rates observed in females.

Definite conclusions cannot be drawn from the comparisons of performance of the Orma and Galana cows in this study. The foundation Orma cows were cull cows of varying ages, purchased from the Orma people and trekked on to ranch. The Galana breeding cows were a uniform group of 27-month old heifers born and reared on the ranch.

Repeatabilities (Table 13) were estimated using cows of both breeds. Repeatability sets an upper limit to the heritability and is often easier to estimate than heritability (Falconer 1981). Repeatabilities were estimated here for those cows that had produced more than one calf and

provided some indication of which traits had significant components of genetic variation within breeds. The repeatabilities for infection rates during lactation were lower than those estimated for mean PCV and bodyweight change indicating that selection of breeding animals on the basis of infection rates might not be successful. Chapter 6 provides further evidence to support this suggestion.

The studies reported in this chapter aimed to investigate the hypothesis that there were genetic differences in these two Boran types in response to tsetse challenge. The performance of calves of the two breeds born in the same environment and reared together from birth was compared over a four-year period. From the results presented here it can be concluded that there are genetic differences between the two breeds. Under natural tsetse challenge, trypanosome prevalence is lower in Orma calves than in Galana calves particularly in relation to *T. vivax* infections. Once infected, Orma calves maintain higher PCV and rarely die of trypanosomosis. While trypanosomosis reduces pre-weaning growth rate in both breeds the reduction in growth rate is significant only in the Galana calves. However, Galana calves are heavier at birth and grow faster to weaning than their Orma counterparts. The Orma cows were the foundation stock for the Orma Breeding herd and a selection programme aimed at improving the growth rate characteristics of the Orma Boran described in Chapter 6.

6 The Orma Boran Breeding Herd

Introduction

Previous chapters presented comparative data on the two types of Boran cattle maintained on Galana Ranch. The observation that the Orma Boran was superior to the Galana Boran in its response to trypanosomosis fuelled interest in these cattle and their possible role in livestock production in tsetse infested areas. Under low to medium tsetse challenge the faster growing Galana Borans could be maintained under trypanosome prophylactic regimes. However, drug resistance was already becoming a problem on Galana Ranch in the early 1980's and there was evidence that the Orma cattle had a lower drug requirement both under prophylactic and therapeutic drug regimes (Njogu et al 1985b; Dolan et al 1990; Dolan et al 1992). In addition

there were many areas of Galana Ranch where the intensity of challenge was such that the Galana Boran could not be economically maintained. The Galana Boran breeding herd was moved out of Kapangani and the breed comparison, presented in the previous chapter, was discontinued for this reason. The evidence of decreased disease susceptibility amongst the Orma Boran also led to interest in the nature of this trait and the possibility of genetic selection to improve it.

Altering the genetic constitution of domestic animals through selective breeding is a slow process requiring many generations of selection, preferably under controlled environmental conditions, and with accurate data. The information available to the breeder usually relates only to the phenotype. However, he needs to breed from the best genotype. Knowledge of the heritability of a trait provides information on the correlation between the phenotype and the genotype, and the rate of improvement to be expected depends, amongst other factors, on the strength of this correlation. Traditional animal breeding methods tend to be regarded as slow, however, there have been many outstanding successes. Egg production has increased from 165 eggs per year in the 1940s to over 350 eggs per year in the 1990s. Milk production in Friesian cattle has also doubled over the same time period. In addition improvements in nutrition and management have made huge contributions to these production increases. The advances in molecular genetics in the past two decades have highlighted the possibilities of identifying markers, or maybe even the genes themselves, associated with important production and disease resistant traits that will make the identification of superior genotypes easier and lead the way to the production of highly productive disease resistant transgenic animals. However, such achievements will also take time.

When the Orma breeding herd was established on Galana Ranch in 1983 KETRI did not have the resources to embark on the search for the gene or genes controlling trypanotolerance, nor was there any evidence to suggest that such an approach would be useful. In any case the N’Dama, which had a more highly evolved trypanotolerance was clearly a better candidate for such studies (Soller and Beckman 1987). KETRI thus choose to use traditional animal breeding methods but even this approach was difficult.

Selection for disease resistance is generally more complex than selection for production traits and this is particularly true in the case of trypanotolerance (Dolan 1987). Firstly, it is difficult to define precisely what is meant by trypanotolerance; there are usually many components to the trait. When a herd of cattle is exposed to tsetse challenge some animals become infected while others remain non-parasitaemic. Amongst the infected animals some become ill while others show little signs of disease. Amongst the diseased animals some develop a chronic wasting syndrome, others become severely anaemic, loose weight and die, while others control the anaemia and continue to be productive. There may be genetic differences between animals operating at each of these stages. To select for all the components of trypanotolerance animals must be exposed to natural field challenge and subsequent generations produced from the survivors. Such a drastic method of selection will entail very high losses.

Selection on one component of trypanotolerance is another alternative. Selection for ability to maintain PCV while infected has been suggested (Trail et al 1991a and 1993). This trait has been shown to have a medium heritability and to be associated with important production parameters. This association with productivity appears to exist in both trypanosome infected and non-infected animals. If the genetic correlation between maintenance of PCV in the non-infected animal and maintenance of PCV in the infected animal is high, then selection might be based on PCV in non-exposed animals and a correlated response achieved in their offspring when exposed. Such a selection method would involve minimum losses. However, as the association between PCV and productivity is more apparent in infected animals it is likely that exposure to the disease will be necessary to identify the more trypanotolerant animals. If this proves to be the situation then other components of trypanotolerance should be measured apart from PCV and an index of trypanotolerance developed as the basis for selection.

Another possible approach is to select for productivity under tsetse challenge. In many livestock production situations the farmer wishes to improve the productivity of his animals while at the same time enhancing their disease resistance. When improvement of the Orma breed on Galana Ranch was considered the ranch management expressed the view that, as the ranch was a beef production enterprise, improved disease resistance should not be emphasised

at the expense of beef production. This posed yet another quandary: would it be possible to select for these two different traits at once and satisfy the management's concern?

If two traits are to be improved together the situation becomes more complex. The animal that is the best for one trait may not necessarily be the best for the other trait, thus less progress will be made in the more important trait if a second trait is to be improved at the same time. In some situations it may actually be impossible to improve both traits. For example in poultry there is a negative genetic correlation between growth rate and egg production which means that the genes which increase egg production at the same time decrease growth rate. This has been overcome by having two poultry industries - broiler production and egg production. In the case of trypanotolerance there are suggestions of a possible negative phenotypic, and maybe also genetic, correlation between disease resistance and growth rate; all the West African livestock breeds purporting to be trypanotolerant are small or in some cases even dwarf.

Attempting to select for two traits, for one of which there is no clearly defined phenotype, is a daunting task. When the Orma breeding herd was established there were no estimates available on the heritability of trypanotolerance or of any of its component traits. There may have been no genetic variation in any of the traits associated with trypanotolerance. Traits that are important for survival are generally observed to have very low heritabilities. If there was a negative genetic correlation between beef production and trypanotolerance then improvement of both traits together might be impossible. Furthermore to obtain the necessary information on heritabilities and genetic correlations several years data would be required and production traits would have to be measured in both disease and disease free environments (Gavora and Spencer 1978).

In the absence of such data the best alternative appeared to be selection for growth rate under continuous exposure to tsetse challenge on the assumption that the animal that continues to be productive despite the presence of the disease will be the best choice economically for that environment. At the same time data could be collected to provide answers to some of the questions discussed above. The protocol for the Orma breeding herd selection programme was

thus devised using a single easily measurable trait. The trait of choice was post weaning growth rate, a trait with a medium to high heritability and frequently used in beef production breeding programmes.

Growth rate was chosen in preference to PCV for several reasons. Firstly, there are many estimates of heritabilities of growth rates in the literature and generally this trait responds well to selection. There are very few estimates of the heritability of PCV control when this programme was started in 1983. Secondly selection for PCV control is more complicated than selection for growth rate. Selection for growth rate between weaning and two years of age requires two weight measurements only. If selection is based on the maintenance of PCV between weaning and two years of age then PCV would have to be measured possibly monthly to ensure that episodes of disease are not missed. Both PCV and weight are monitored in the KETRI herds on Galana but this is not the case in most cattle breeding situations in Africa. Selection based on growth rate is simpler and more likely to be adopted in other situations. Furthermore, selection on PCV is considered worthwhile (Trail et al 1993) because of the association between PCV values and growth. If growth is the trait desired and PCV is important only because of its association with growth, then selection on PCV will be equally effective to selection on growth only if there is a perfect genetic correlation (r_g) between PCV and growth rate (i.e. $r_g = 1$), if the correlation is anything less than 1 then selection on PCV will be less effective in improving growth than direct selection on growth.

Post weaning growth rate was chosen in preference to weaning weight as weaning weight has a large maternal component and is often a more reliable indicator of the dam's milk production than the calf's ability to grow. Furthermore, response to trypanosomosis in calves (Chapter 5) is different than that observed in older animals and the reasons for the apparent decreased susceptibility of calves is not clearly understood.

Materials and Methods

The foundation stock

The Orma Breeding herd, as previously described in Chapter 5, was established through the purchase, from the Orma people, of cows, heifers and five bulls between 1983 and 1987 (Table 14).

Table 14. *Number and age of animals purchased to establish the Orma breeding herd on Galana Ranch.*

Month of 1st record	No. of animals	Mean age (years)
October 1983	83	5.1
February 1984	34	8.7
November 1984	15	4.0
January 1987	30	2.9
July 1987	8	not estimated
December 1987	99	2.5

Because many of the original 132 cows purchased in 1983/84 had to be culled a further group of 99 Orma heifers/young cows were purchased in December 1987 and were maintained in a separate breeding group which became known as the "heifer herd".

From 1986 onwards the original cow herd was expanded through the recruitment of Orma females born from the original stock. When these animals reached 27 months of age they joined the breeding cows. They were recorded as calves from birth to weaning at approximately eight months of age; at which time they were transferred to the ranch management and no records were kept until they were returned to the KETRI herd at 27 months of age. This policy of returning 27 month old heifers to the KETRI herd was not always adhered to particularly in later years when there was considerable pressure on the ranch management to sell stock.

The results presented here extend those already described in the previous chapter and are from data recorded on the cow and heifer herds and on calves born to dams in both herds between May 1983 and October 1995. Some of the analyses of genetic paramaters included data to October 1996. Male calves were retained by KETRI after weaning and maintained in a separate weaner/bull herd on the same regime as the cows and calves. Bulls were selected

from these weaners on the basis of post weaning growth rate. Results from analyses of data from the weaner/bull herd from 1985 to 1995 are also presented.

Recording was disrupted when the ranch management changed hands in September 1989 and no data were available for September and October 1989. Apart from these two months, blood samples were taken from all cows, weaners and calves every two weeks.

Breeding and Selection

A breeding scheme was devised, as described in Chapter 5, whereby bulls were introduced to the cows one at a time. A single bull ran with the herd for a period of six to eight weeks with a two to four week interval before the introduction of a new bull. For a period of six months, after the change in ranch management in September 1989, there were no bulls with the herds.

The original purchased bulls were used until selected bulls born on Galana were ready for breeding. Selected bulls were first used in July 1986 and the first calves sired by these bulls were born in April 1987. No castration of male weaners occurred at weaning. Growth rates were first assessed when males reached between 15 and 18 months of age. Males with below average post weaning growth rates were then returned to the ranch management. Further decisions on castration were made between 18 and 24 months of age and bulls for breeding were used first between 42 and 48 months of age.

Using one bull with a large group of cows has the disadvantage that all the calves from that bull were born over the same period and there was confounding between the effect of any one bull and the season and year. Selection for post weaning growth rate was applied within half sibs groups, all born within the same season and exposed to the same environment. When there were sufficient numbers of males weaned in the same month decisions on castration were based on comparing post weaning growth rate between animals weaned in the same month. Otherwise growth rates were assessed in comparison to weaners weaned in either the previous or the following month. No artificial selection was applied to the cows but under a limited treatment regime a degree of natural selection still operates through infection causing reduced fertility in cows and mortality.

Pregnancy diagnoses were performed approximately every three months from the beginning of the study in 1983 until the end of 1988. One further pregnancy diagnosis was performed in 1989 and one in 1990, but none thereafter. When pregnancy diagnoses were on a regular basis, accurate information on number of abortions was obtainable. The herdsmen also provided information on abortions and still-births observed.

Statistical analysis

Similar least squares models were used as those described in Chapter 5. Heritability analyses were undertaken primarily for the calf data set in which the pedigree information was fairly complete and there were a large number of animals. Estimates of variance components were obtained by Restricted Maximum Likelihood (REML) using a derivative-free algorithm and fitting an animal model throughout (Meyer 1992). The programme used, Derivative Free Restricted Maximum Likelihood (DfREML), was written specifically for animal breeding data where estimates of variance components are used to provide estimates of heritability (h^2) for traits of interest.

Results

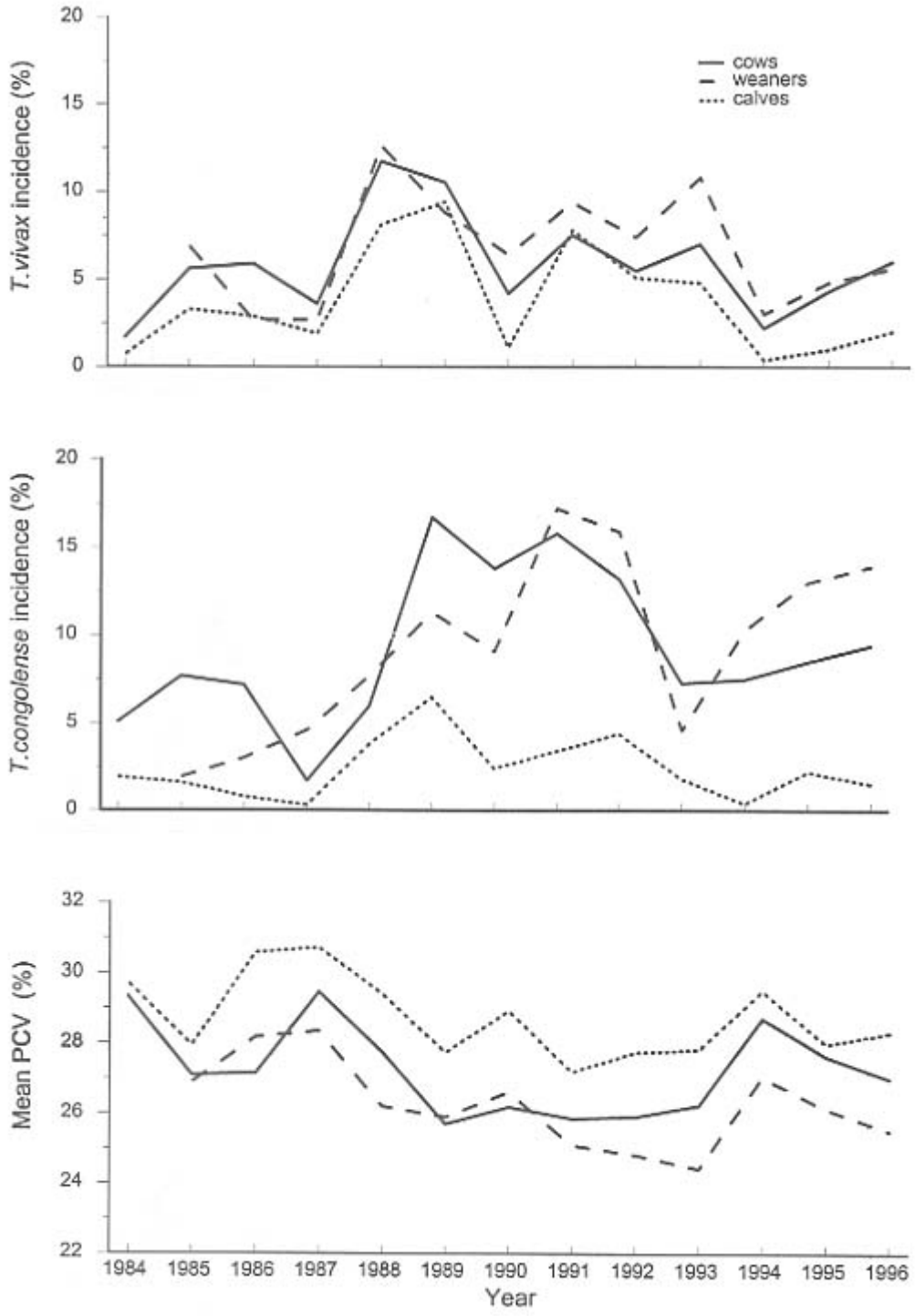
Rainfall data for Kisiki and Kapangani from 1984 to 1990 and from the KETRI weather station at the field station headquarters on Galana Ranch at Tank E from 1991 to 1995 were summarised in Dolan (1996). Data on tsetse number and infection rates of flies on Galana Ranch have been collected by KETRI since 1980. Some of these data were presented in Njogu et al (1985a). Data from trap catches taken in the Kapangani area between December 1989 and December 1995 were summarised in Dolan (1996).

Yearly and seasonal variation in the incidence of trypanosomosis

The purchased heifers, which entered the recording scheme in January 1988, were grazed separately from the cow herd, five to ten km. away. They were thus possibly exposed to a different tsetse challenge so trypanosome incidence was calculated separately for the two herds. Initial analyses, however, showed that there was no difference in the incidence of *T.*

vivax, *T. congolense* or total infections in the two herds. Thus data from the heifer herd were combined with those from the cow herd.

The mean monthly *T. vivax* and *T. congolense* incidence from 1984 to 1995 is presented in Figure 7 for cows, calves and weaners. There were only slight differences between the three groups of animals in the *T. vivax* incidence over the years. However, calves had a much lower incidence of *T. congolense* infections than older animals. *Trypanosoma brucei* incidence was very low. In Figure 8 the mean monthly PCV is presented for the three sections of the herd. The PCVs in cows and weaners were lower than observed in calves



Figures 7. *Trypanosoma vivax and T. congolense incidence and mean PCV in Orma cows, weaners and calves.*

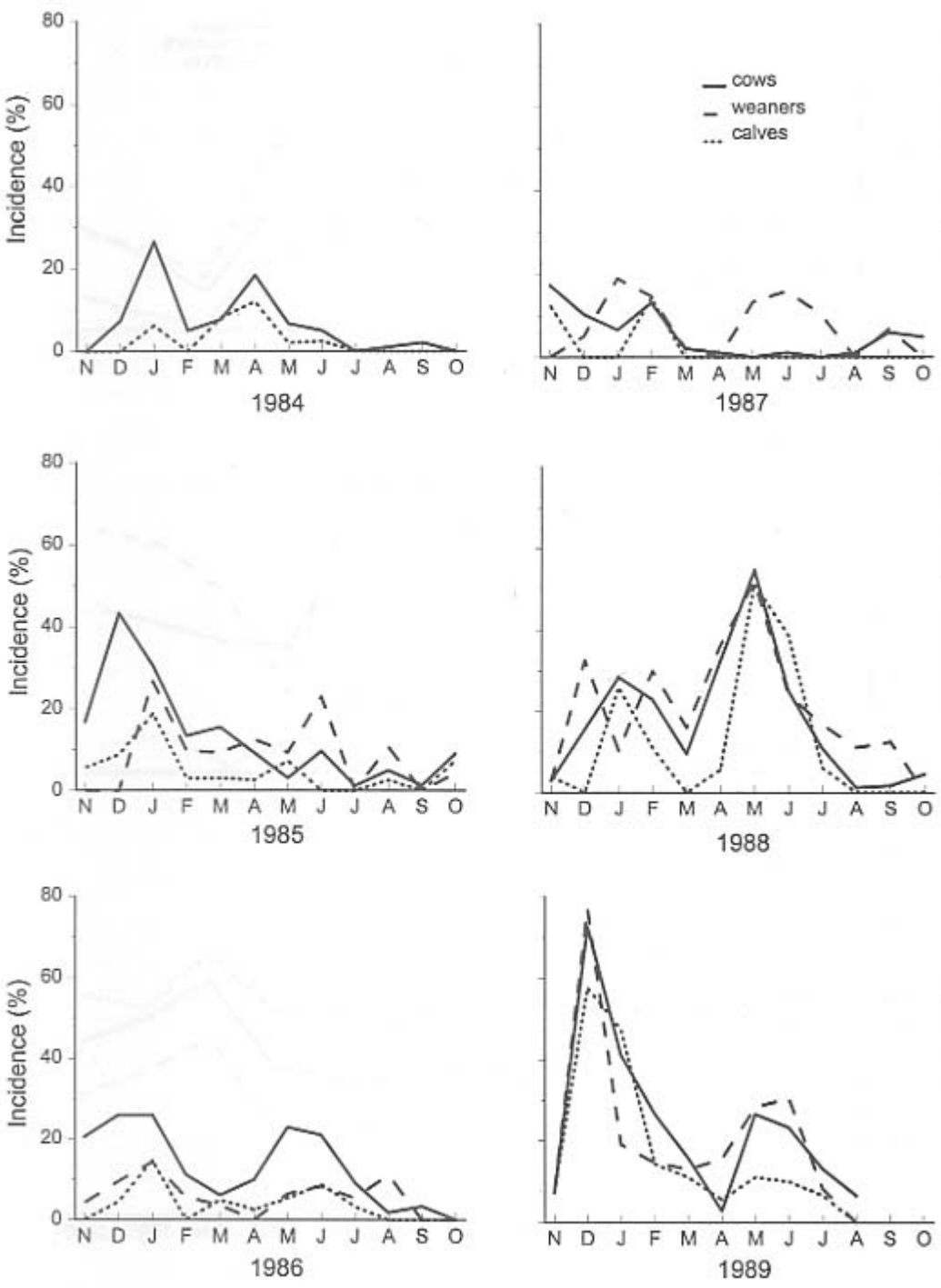
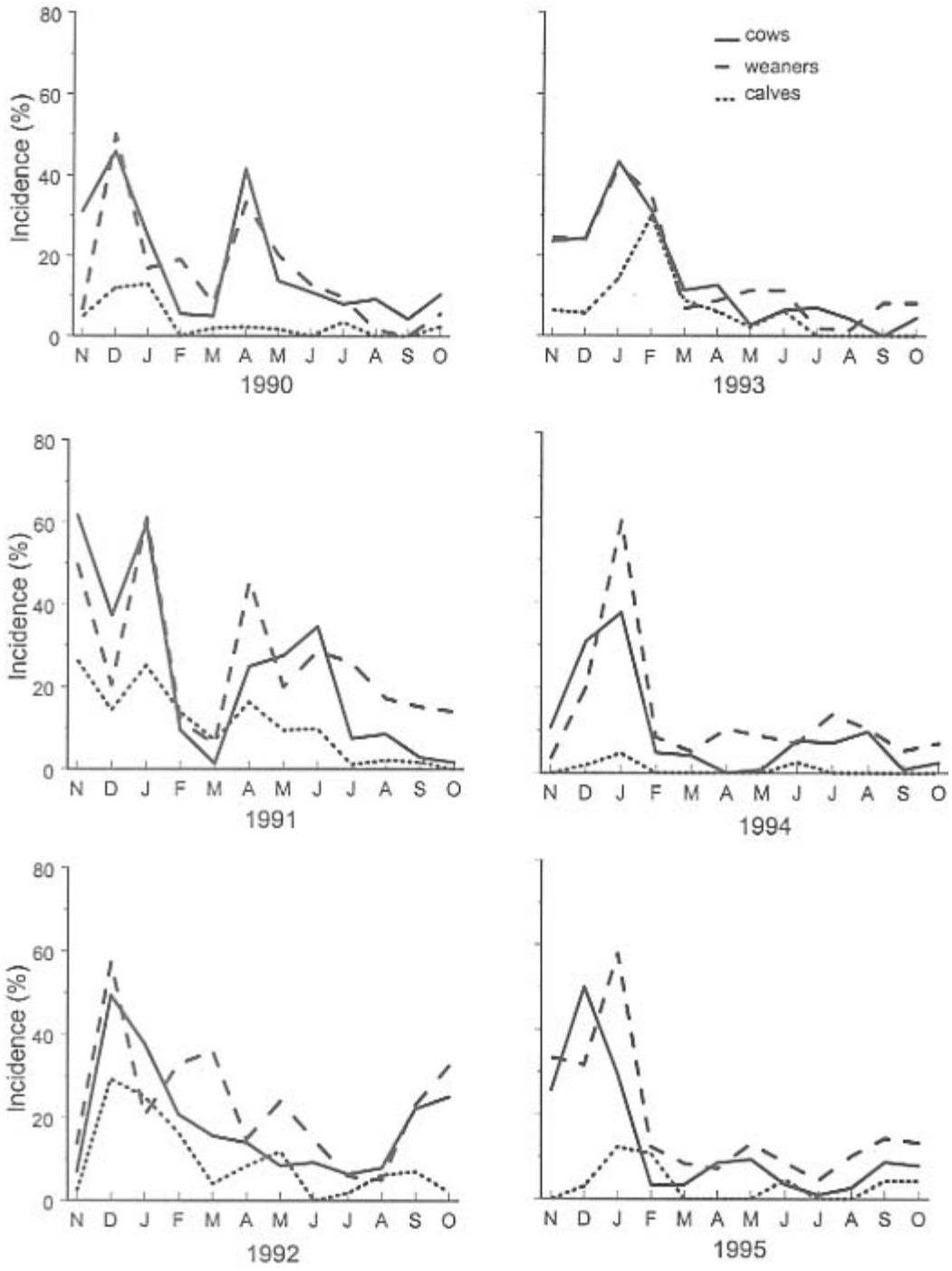

Incidence (%)
80
60
40
20
0
N D J F M A M J J A S O
1984
cows
weaners
calves
1987
1985
1988
1986
1989

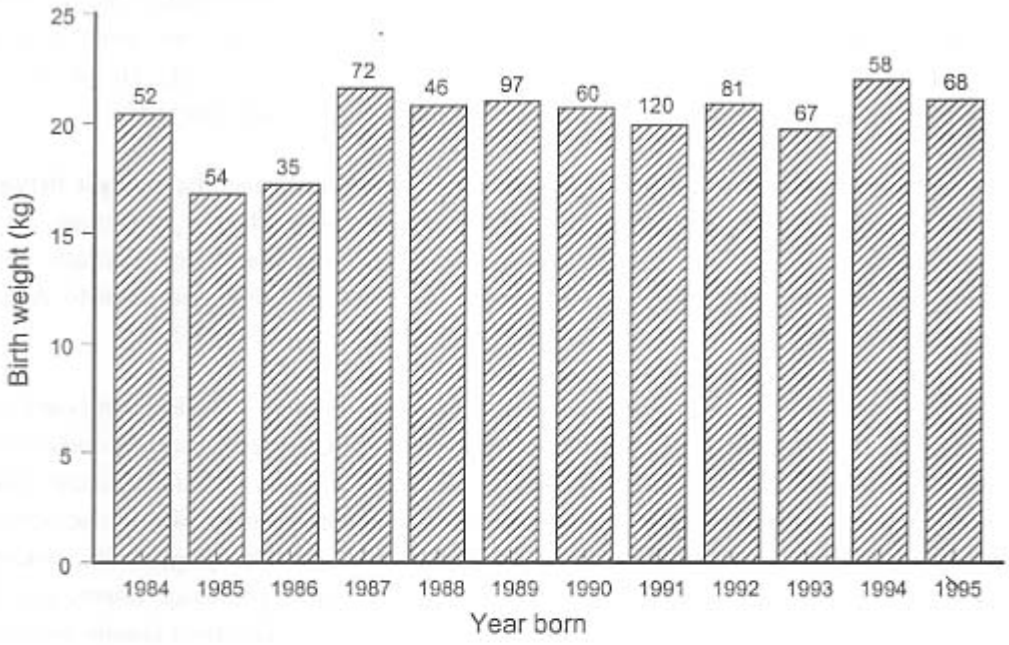
Figure 8. *Monthly trypanosome incidence in Orma cows, weaners and calves (1984-1989)*

The monthly incidence of all infections throughout each year in the three sections of the herd is shown in Figures 9 and 10. There was considerable variation in the trypanosome challenge over the twelve years with a lower average trypanosome incidence in the first four years. Trypanosome incidence was higher between 1988 and 1992; incidence rose dramatically in all sections of the herd in the second half of 1988 and again in early 1989 with peak incidences in the cows and weaners of 60-70%. In the last three years trypanosome incidence was high between November and January but low for the remainder of the year.

The seasonal pattern of trypanosome incidence, related to the bimodal rainfall, is apparent (Figures 9 and 10) throughout most years with an initial peak in December/January followed by a second, generally lower peak, between April and June.



Figures 9 . *Monthly trypanosome incidence in Orma cows,weaners and calves (1990-1995)*


Birth weight (kg)
25
20
15
10
5
0
52
54
35
72
46
97
60
120
81
67
58
68
1984
1985
1986
1987
1988
1989
1990
1991
1992
1993
1994
1995
Year born

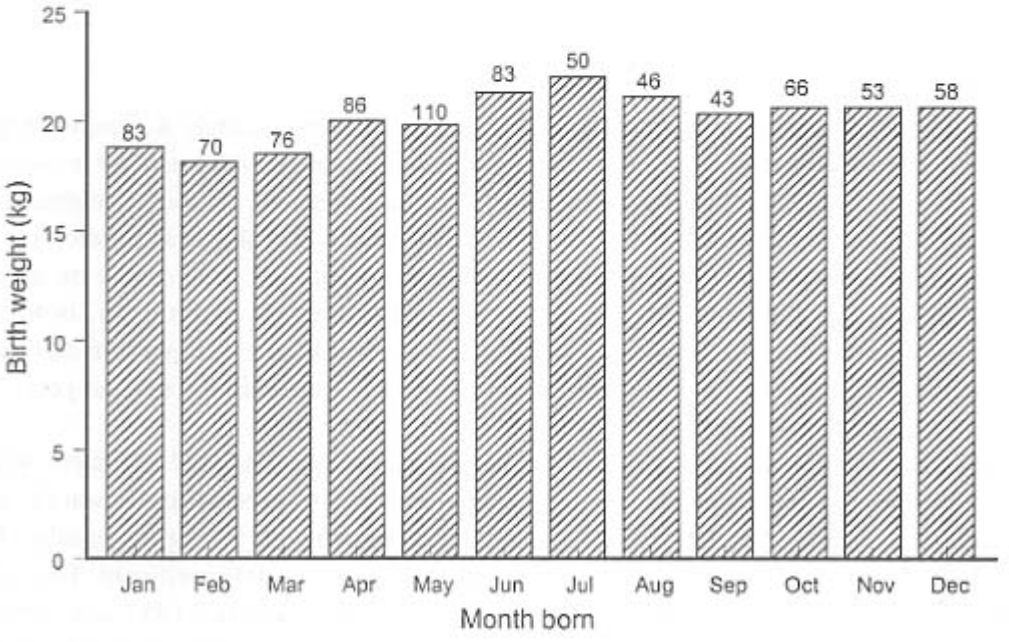

Birth weight (kg)
25
20
15
10
5
0
83
70
76
86
110
83
50
46
43
66
53
58
Jan
Feb
Mar
Apr
May
Jun
Jul
Aug
Sep
Oct
Nov
Dec
Month born

Figure 10 *Mean yearly (a) and monthly birth weights of Orma calves. The numbers on each graph are the number of calves born in each year and in each month.*

Calves

Birth weight

A total of 904 births were recorded in the herd from May 1983 to November 1995, 902 of these to identified dams. The least squares means for birth weight (corrected for sex and month of birth) are shown for calves born between 1984 and 1995 in Figure 11a, and Figure 11b presents the same data showing mean weights for each month of the year (corrected for sex and year). The differences between months and between years were both significant ($P<0.001$). Birth weights were greatest in 1987, 1994 and 1995. In 1986/87 the herd was in Kisiki where the grazing was better than in Kapangani. The first target trial to control tsetse was set up in Kisiki in 1986 and there was a dramatic effect on trypanosome challenge reducing it to close to zero in 1987 (Figure 9). The combination of good grazing and low challenge probably contributed to the increased birth weights. The birth of heavier calves in 1994 and 1995 may have been an indication of some improvement in productivity of the herd. However, the trypanosome incidence in these years was less than in some previous years.

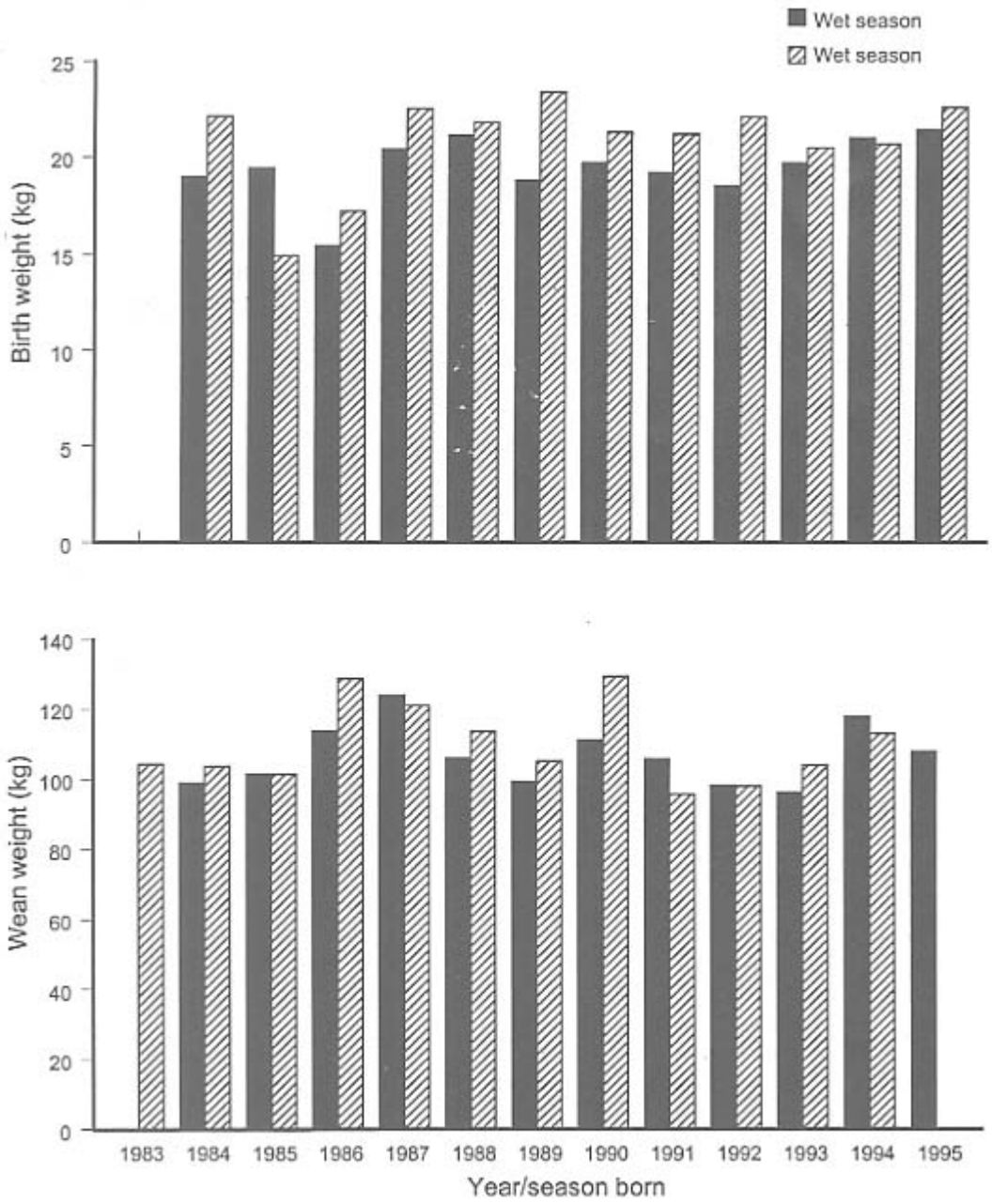


Figure 11 *Mean seasonal birth and weaning of calves. Rhe wet season wad from November to May and the dry season from July to October*

Calves were heaviest when born between June and August and the lightest between January and March. Trypanosome infection rate is probably the most likely explanation for this. Dams

that calve between January and March are generally under heavy trypanosome challenge in late gestation; for cows calving in June to August trypanosome challenge is generally lower in the months preceding calving.

Birth weights of 794 calves, for which age of dam was known, were also analysed with season of birth (using the classification described in Chapter 5) as 25 consecutive time, age of dam (3,4,5,6,7,8 and 9 years and over) and sex as fixed effects. There were significant differences in the birth weights of calves born in different seasons ($P < 0.001$). Generally calves born in the dry season were heavier (Figure 12), possibly because their dams were exposed to a lower trypanosome challenge during the dry season. Male calves weighed on average 20.9 kg, 1.4 kg heavier than female calves ($P < 0.001$). Older dams produced lighter calves but the effect was not statistically significant. Birth weights for calves born in different seasons are shown in Figure 12.

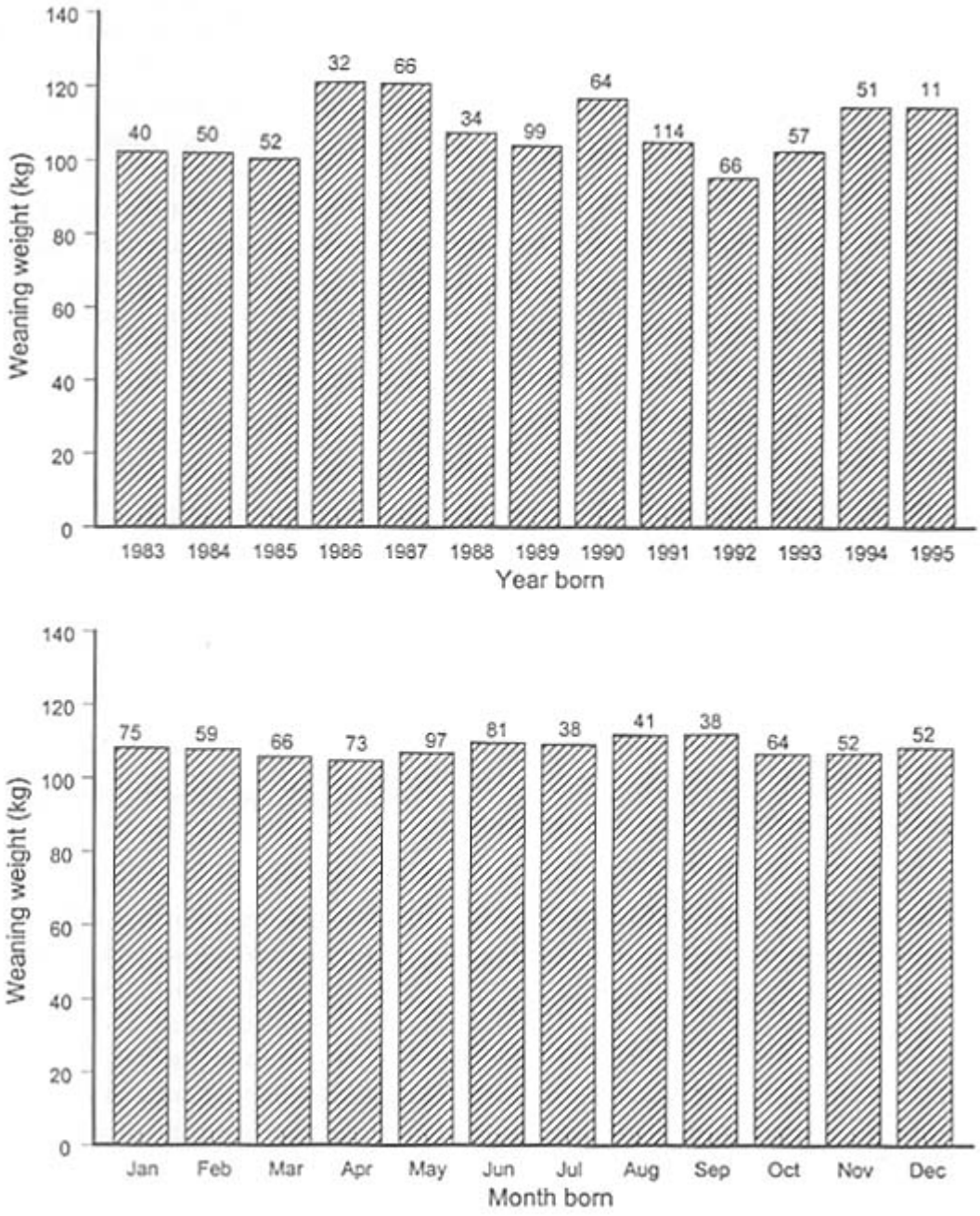


Figure 12. *Mean yearly (a) and monthly(b) weaning weights of Orma calves. The numbers on each graph are the number of calves born in each year and in each month on which the weaning weights are calculated.*

Weaning weight

Calves were weaned in the middle of each month at approximately 8 months of age. Until 1989 the ranch management weighed the calves at weaning and these weights were used in the

analyses. Under the new ranch management, weaning weights were not measured and the weight recorded by KETRI at the end of the previous month, two weeks before weaning, was taken as the weaning weight. The least square means for weaning weight (corrected for sex, age at weaning and month of birth) are shown for calves born between 1984 and 1995 in Figure 13a, and Figure 13b presents the same data showing mean weights for each month of the year (corrected for sex and year).

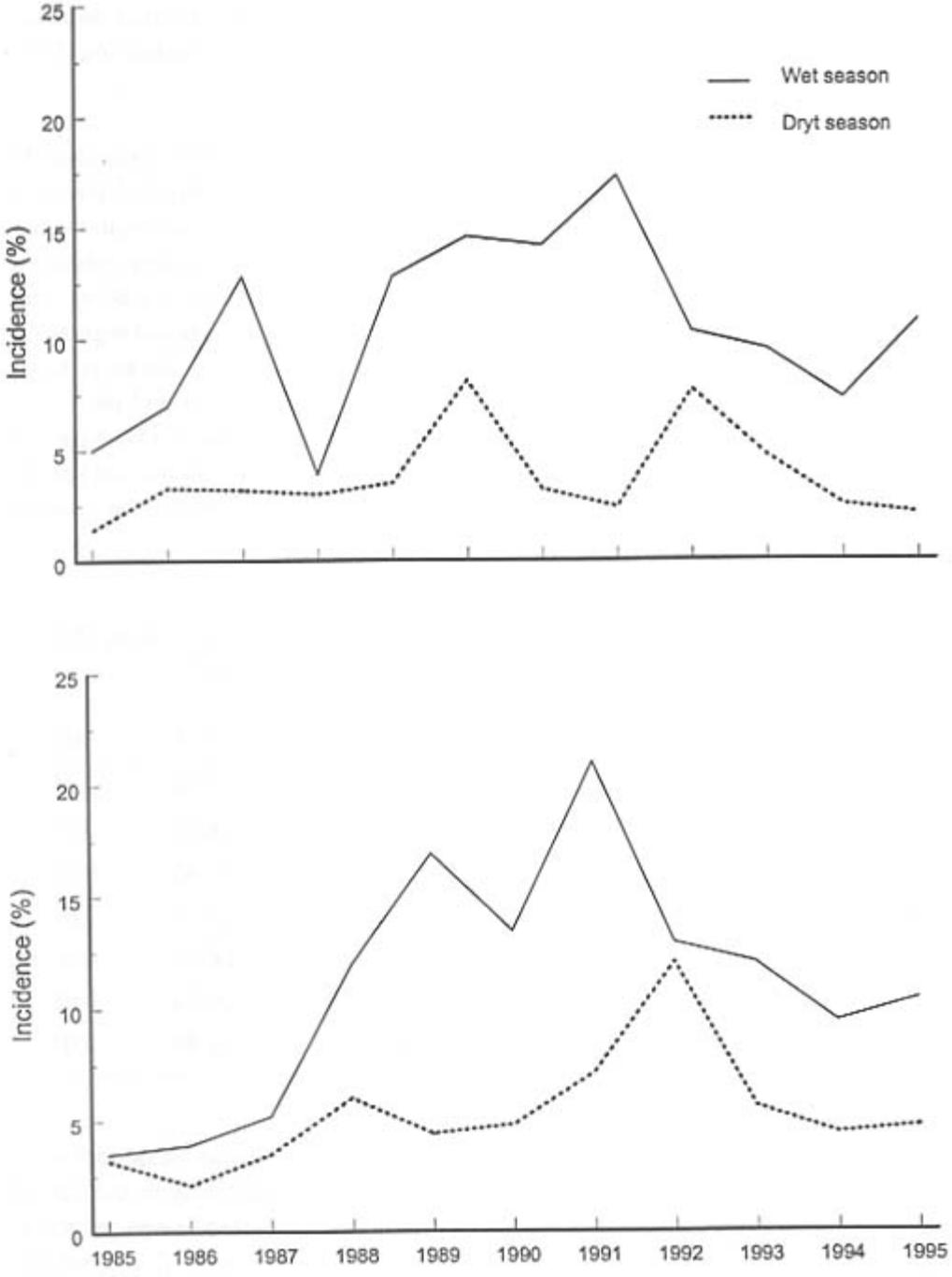


Figure 13. *Trypanosome incidence in cows (a) and weaners (b) during wet and dry seasons.*
Season of birth, sex, age at weaning and age of dam were considered as factors which might affect weaning weight. The mean weaning weight (corrected for season of birth and age at weaning) of male calves was 112 kg compared with 104 kg for females ($P < 0.001$). Older dams weaned lighter calves but the effect was not significant. The mean weaning weight of calves born in the different seasons between 1983 and 1995 is presented in Figure 12. The inclusion of season in the model removed a large proportion of the environmental variation, which was particularly important for later genetic analyses, but there was no clear pattern from year to year. This may be partly explained by the fact that birth and thereby weaning, takes place throughout the year. Thus a calf born in one season will have part of it growth to weaning take place in dry conditions and part in wet conditions.

Pre-weaning mortality

One hundred and two deaths were recorded in calves up to October 1995 (Table 15), 42 of these deaths were in female calves and 60 in male calves ($\chi^2_1 = 3.25$, $P < 0.10$). The overall pre-weaning mortality between 1983 and 1994 was 11.3%. Annual pre-weaning mortality could not be estimated for calves born in 1995 as most of them had not reached weaning before November 1995. The mean age at death for the 102 calves was 54 days but the distribution was skewed with 59% of deaths occurring in calves of less than one month of age. Post mortems were performed where possible.

Table 15. *Number of births and number of deaths in male and female Orma calves over twelve years.*

Year	Born	Deaths		Mortality (%)
		Female	Male	
1983/4	95	3	4	7.4
1985	54	3	1	7.4
1986	36	2	1	8.3
1987	64	2	2	6.3
1988	57	5	10	26.3

1989	105	5	9	13.3
1990	79	6	1	8.9
1991	114	2	9	9.6
1992	99	3	12	15.2
1993	63	4	5	14.3
1994	56	3	2	8.9
1995	83	4	4	-

Variation in pre-weaning mortality in the early years of the study appeared to be related to trypanosomosis challenge. In years of low to medium challenge it remained below 10% while in the years of heavy challenge, 1988 and 1989 (Figure 7), pre-weaning mortality rose. However, calves rarely died from trypanosome infections and the increased pre-weaning mortality recorded in years of high challenge was related to trypanosomosis in the dams rather than in the calves themselves (Table 16). For example, in 1988 when pre-weaning mortality was 26.3%, eleven of the 15 calf deaths were ascribed to trypanosomosis in the dam. The dams of ten of these calves had trypanosomosis at parturition or shortly afterwards. Their calves died either after the dams died of trypanosomosis or as a result of poor mothering from their sick dams. The eleventh calf was born prematurely, with a very low birth weight, to an infected dam and died within a few days of birth.

Table 16. *Different causes of pre-weaning mortality in Orma calves.*

Cause of death	No. of deaths
Trypanosomosis in calf	2
Trypanosomosis in dam	44
Mothering ability	19
Predators	13
"Other" +	24
Total	102

+ "Other" included congenital disorders, anaplasmosis and cases where no post mortem information was available or a definitive cause of death was not possible.

Many of the foundation Orma cows present in the early years of the study had defective teats and failed to feed their calves adequately. Where such calves died mothering ability is cited as the cause of death (Table 16). In some cases where a predator was responsible for the death of a calf other factors such as mothering ability or a sick dam may also have been implicated. Predators have always been considered a major cause of death in cattle by the ranch management but this study showed that while the predator may be the final cause of death, in many cases it is the weak or sickly animals that are taken.

In the later years of the study calf mortality was also high. Poor mothering may again have been a contributory factor in these years. Under the previous ranch management the herdsmen were provided with milking cows and the experimental cows were not milked. This policy was not continued by the new management and milking of the dams by herdsmen became a common practice; this together with trypanosomosis infection in the dam decreased the chances of survival in the calf.

Trypanosome infections and packed cell volume

Trypanosome incidence and PCV in calves over the twelve years is shown in Figures 8-11. Blood slides were examined for trypanosomes and PCV measured at least twice in 777 calves. The majority of calves, four hundred and ninety three (63%), were never detected positive for trypanosomes (Table 17). Seven hundred and forty four of these calves reached weaning and the infection rate between birth and weaning was 0.28 *T. vivax* infections per calf and 0.20 *T. congolense* infections per calf. Over 90% of infections with both trypanosome species were treated with diminazene aceturate. Treatments took place when the PCV fell to below 26%, or 21% when the challenge was deemed to be low. In addition 82 treatments were administered to calves with non-patent parasitaemias.

Table 17. *Least square means and standard errors for mean packed cell volume*

and standard error (SE) between birth and weaning Orma calves, classified according to number of infections, corrected for the effects season and sex.

Infection status	No. of calves	PCV	SE
Non-infected	493	29.3	0.2
One *T. vivax* infection	117	28.5	0.3
One *T. congolense* infection	100	27.8	0.3
Two or more *T. vivax* infections	28	26.9	0.6
Two or more *T. congolense* infections	11	27.5	0.8
Two or more infections (*T. vivax* or *T. congolense*)	28	26.4	0.5

Mean PCV was calculated for the 777 calves which survived at least one month. Season of birth, sex, birth weight, age of dam and trypanosome infection were included as factors in the analysis. Age of dam had no significant impact on calf PCV. Season of birth, sex, birth weight and trypanosome infection had significant effects on mean PCV ($P < 0.001$). Female calves had on average mean PCVs 1% higher than male calves (29.2 ± 0.2 compared with 28.2 ± 0.2). An increase of 1kg in birth weight resulted on average in an increase of 0.1% in mean PCV. The effect of trypanosome infection on mean PCV is shown in Table 17. Calves detected positive twice between birth and weaning had a lower average PCV than those detected positive only once.

The impact of trypanosome infection on calf productivity

Infection rate and mean PCV were each in turn examined for their effect on calf growth. Calves were classified as infected or non-infected. Infection in the calf had no significant impact on weaning weight when adjusted for other factors in the model. However, weaning weight was related to PCV ($P< 0.001$); calves with higher mean PCV had higher weaning weights; a 1% increase in PCV was associated with 1.7 kg increase in weaning weight.

Cows

Mortality and disposals

A number of the foundation cows were old and were culled within the first year of the trial (Table 18). Culling of cows based on age or reproductive performance continued throughout and, in addition, a total of 81 cows died during the study period; this constituted an annual

mortality of 6%. Mortality in the cows was divided into deaths from trypanosomosis and deaths from other causes (Table 18). In addition to cases where trypanosomosis was clearly the cause of death, other deaths were ascribed to trypanosomosis if the animal had a low PCV in the month before death and trypanosomes had been detected within three months of death. Trypanosomosis was considered to be the most likely cause of death in 58% of cases. Other causes of death included predators, anaplasmosis and complications at calving. Twenty two percent of the total mortality occurred in one of the eight years, 1988, a year of high challenge.

Table 18. *Mortality from trypanosomiasis and other causes in Orma cows.*

Year	Average no. of cows in herd	No. culled	No. of deaths	
			Trypanosomosis	Other causes
1983/4	90	31	3	2
1985	93	18	2	0
1986	70	28	3	0
1987	88	6	2	2
1988	177	13	13	5
1989	177	5	6	2
1990	171	27	5	5
1991	159	27	4	7
1992	145	11	3	0
1993	136	35	3	4
1994	121	40	3	4
1995	119	3	0	3
Mean over all years	129	18%	4%	2%

In addition to reasons of age and poor reproductive performance, sick cows were also returned to the ranch management. KETRI were required to pay the ranch management for any cows which died while on KETRI experiments. Thus sick cows with foot and mouth disease, besnoitiosis, lumpy skin disease or injured by predators were returned to the ranch management. They appear, together with old and reproductively defective cows, as cull cows. Because of this policy the deaths ascribed to other causes in Table 18 is likely to be an underestimate of the true figure.

Reproductive performance

A total of 448 cows entered the recording system over the twelve years and 381 of these cows produced live calves. The number of calves born per cow varied from one to seven, 135 cows had one calf and 29 cows gave birth to five or more calves. Average age at first calving for the heifer herd, purchased in 1987, was 4 years and for cows born on Galana was 3 years and 7 months.

Abortions, calving percentage and calving interval

Pregnancy diagnosis took place regularly until 1988, but not thereafter. It was not possible to obtain accurate estimates of the abortion rates without regular pregnancy diagnosis. Abortions occurring in the first few months of pregnancy may have gone unnoticed. Approximate estimates of the abortion rate and of the calving percentage from 1985 to 1995 is presented in Table 19. Still births were counted as abortions. Abortion rates were highest in 1988 and 1989 during periods of high trypanosome challenge (Figure 9).

The calving percentage up to and including 1988 was based on the cow herd only. Although recording for the heifer herd commenced in early 1988 no bull was introduced until August 1988 and the first calves were born in May 1989. There was no bull with either herd from September 1989, when the ranch management changed, until March 1990. The calving percentage for 1990 was thus based on calvings that took place during an eight month period: November 1989 to June 1990.

Table 19. *Abortion rate and calving percentage in Orma cows.*

Year	Abortions %	Calving %
1985	6	58
1986	7	51
1987	8	75
1988	28	63
1989	16	59
1990	7	46

1991	5	83
1992	3	57
1993	6	51
1994	7	64
1995	2	41
Mean	9	59

The herd management procedures were such that there were periods throughout each year when the cow and heifer herds had no bull. Thus the estimates presented here for calving percentage are lower than might be expected under normal ranch practice where a number of bulls are run with the cow herds on a continuous basis. Likewise the calving intervals presented in Table 20 are longer than might have been achieved had bulls been with the cows throughout. The calving intervals between 5th and 6th and 6th and 7th calves are not included in the table as they were based on very few observations. The median calving interval considering all calvings was 472 days.

Table 20. *Calving interval and standard deviation (SD) in Orma cows.*

	Calving intervals (days)			
	1st	2nd	3rd	4th
n	245	149	84	29
Mean	568	479	460	446
SD	152	107	111	107

Trypanosome infections and packed cell volume

Trypanosome incidence and PCV in cows over the twelve years is shown in Figures 8-11 where it is compared with that recorded in calves and weaners. Further analyses were undertaken to elucidate the factors influencing trypanosome incidence and PCV in cows.

The possible factors influencing trypanosome incidence in cows were examined in various ways. Seasons, in relation to trypanosome incidence, were divided into wet and dry, as described for rainfall but lagged by one month on the assumption that trypanosomosis incidence builds up some weeks after the rain. Cows were classified according to their

reproductive status as either lactating, gestating, lactating and gestating or resting. Trypanosome incidence was analysed using a logistical regression analysis, which included year, season, age of cow and reproductive status as fixed effects. Year, season and age of cow were all found to be significant and there was also a year x season interaction. The trypanosome incidence over the twelve years in wet and dry seasons is shown in Figure 14. The incidence was higher in the wet season than in the dry season, however, the magnitude of the difference was not the same in all years giving rise to the year x season interaction.

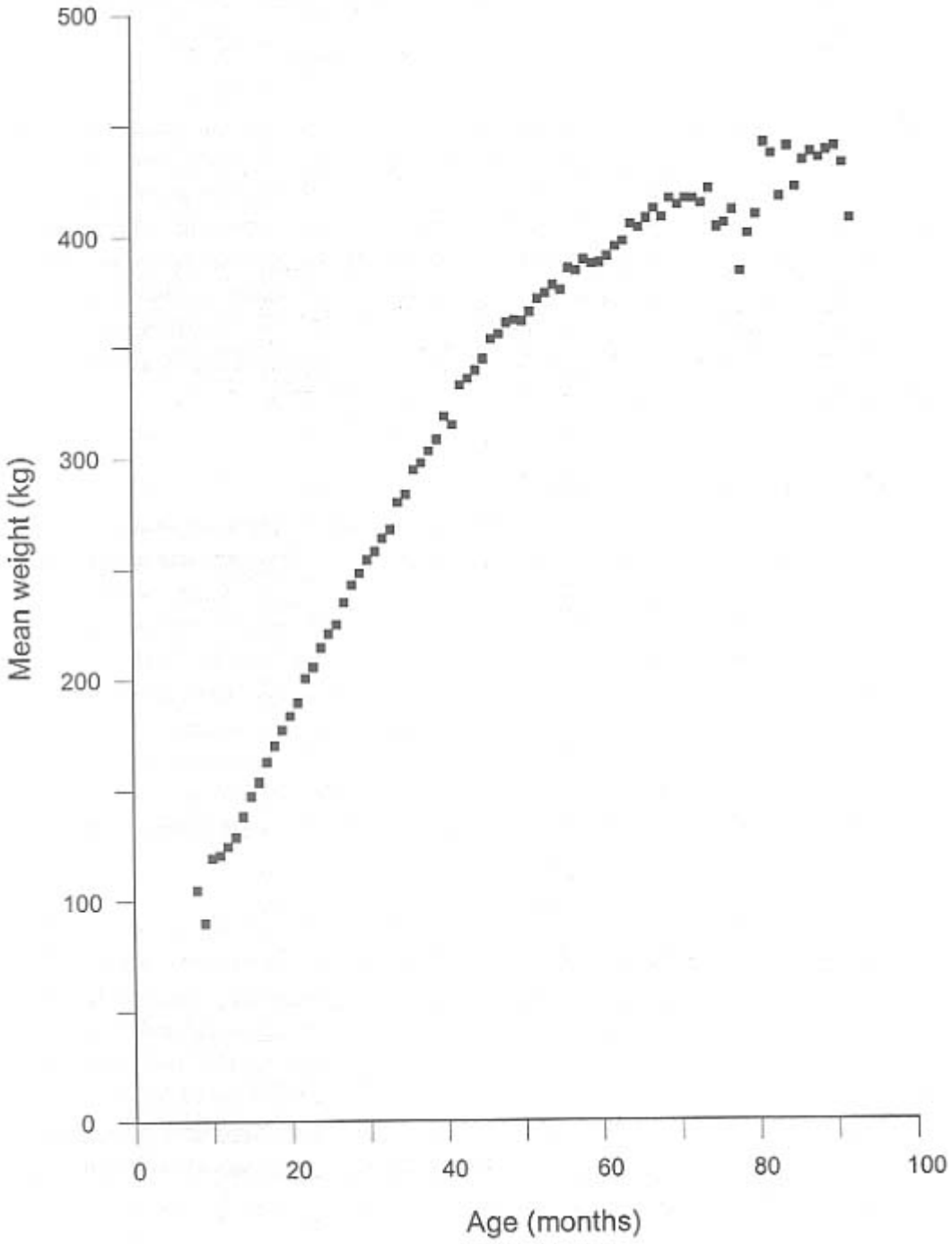


Figure 14 *The relationship between weigh and age in the Orma weaner/bull herd.*

Results from analyses in which *Trypanosoma vivax* and *T. congolense* incidence were considered separately are shown in Table 21. The model included season and age of cow and in analyses of PCV, infection status, coded as either infected or non- infected, was also

considered. Age had a significant effect on *T. vivax* and *T. congolense* infection rates and on PCV ($P < 0.001$).Younger cows, in the first two age categories (Table 21) were more susceptible to *T. vivax* infections while the youngest age group (<4 years) had the lowest infection rates with *T. congolense.* Packed cell volume gradually declined with age and was 5.4 % lower in samples taken from infected cows than in samples taken from non- infected cows ($P < 0.001$)

Table 21. *Trypanosome infection rates (%) and standard error for T. vivax (Tv) and T. congolense (Tc) and mean PCV and standard error in cows of different ages.*

Age group	*Tv*. infection	SE	*Tc*. infection	SE	PCV	SE
< 3 years	3.0	0.3	2.0	0.2	28.95	0.08
< 4 years	2.9	0.2	3.7	0.2	28.23	0.05
< 5 years	3.3	0.3	5.4	0.3	27.45	0.06
< 6 years	2.2	0.2	4.1	0.3	27.20	0.07
< 7 years	2.1	0.2	5.3	0.4	26.70	0.07
< 8 years	2.3	0.3	5.3	0.4	26.64	0.08
> 8 years	2.1	0.3	5.5	0.4	25.85	0.07

Weaners and Bulls

Male calves were separated from their dams and handed over to the ranch at weaning to undergo routine ranch weaning procedures. However, these males were not castrated and on return to KETRI management one to two months after weaning they joined the weaner/bull herd. Blood samples were examined for parasites and PCV measured every two weeks and weights were recorded each month.

Mortality and disposals

The constitution of this herd was continually changing; new weaners joined the herd each month and animals with poor post weaning growth or some abnormality that would have hindered their performance as bulls were returned to the ranch management for castration. The average number of animals in the herd in each year from 1985 to 1995 is shown in Table 22.

Table 22. *Disposals and deaths in the Orma weaner/bull herd*

Year	Average no. in herd	No. culled	No. of deaths	No. transferred
1985	28	14	2	2
1986	33	5	3	0
1987	41	11	3	0
1988	48	15	7	2
1989	46	31	1	5
1990	52	24	4	2
1991	39	20	2	5
1992	62	21	3	7
1993	57	19	4	0
1994	58	2	1	4
1995	62	1	2	4
Mean over all years	48	33%	6%	6%

The overall annual mortality in the weaner/bull herd over eleven years was 6% (Table 22) Eighteen of 32 deaths were ascribed to trypanosomosis and eight animals were taken by predators.

Bodyweight and growth rate

The relationship between bodyweight and age is shown in Figure 15. Analyses were performed on bodyweight at 15, 24, 36 and 48 months of age and on growth rate from weaning to each of these ages. The models used for the analyses of bodyweight at different ages (Table 23) included year of weaning, month of weaning and weaning weight as a covariate. In all cases the model accounted for over 60% of the variation in weight. Year of weaning had a significant effect on bodyweight at 15, 24 and 36 months of age but not on weight at 48 months. Month of weaning had a significant effect only on weight at 15 months. Calves weaned in the wet season between November and January were heavier at 15 months than those weaned at other times of the year. The older the animals became, the less important month of weaning became as a factor influencing weight.

Table 23.*Mean bodyweights at different ages and mean growth rate from weaning to different ages and standard deviation (SD) in Orma weaner/bulls.*

	Age (months)			
	15	24	36	48
Number of animals	227	165	115	63
Bodyweight (kg)	147	213	292	359
SD	27	36	37	31
Growth rate from weaning (g/day)	175	214	215	204
SD	98	66	38	22

There was a positive correlation between each of the four weights (15, 24, 36 and 48 months) and weaning weight. The regression was equally significant ($P < 0.001$) and of similar magnitude ($b = 0.65 - 0.77$) for the four weights. An increase of 1 kg at weaning resulted in an increase which varied between 0.65 and 0.77 kg in later in life.

Growth rates between weaning and 15, 24, 36 and 48 months of age were also analysed and means are shown in Table 23. A gradual decrease in growth rate as the animals grow older would normally be expected but here the slower growing animals were culled so the growth rates in the later stages were those for a selected group of animals. The decrease in the standard deviation in growth rate from 98 g/day at 15 months to 22 g/day at 48 months was a reflection of the selection process; the inferior growers were culled and the variation in the group decreased.

The impact of year of weaning, month of weaning and weaning weight on growth rates was examined. The results for year of weaning and month of weaning were similar to those described above for the four weights. The correlation between growth rate and weaning weight was negative. The animals with the lowest weaning weights grew fastest thereafter and this compensatory growth was still apparent up to 48 months of age. However, compensatory growth did not result in a change in the ranking of animals; the correlation between weaning weight and weight later in life was positive. Thus the lighter animals at weaning grew the fastest after weaning but on average they were still lighter at later stages.

Trypanosome infections and packed cell volume

Trypanosome incidence and PCV in weaners over the twelve years is shown in Figures 8-11 where it is compared with that recorded in calves and cows. Trypanosome incidence in weaners was analysed by logistical regression analyses using a similar model to that used for the cow data. There was significant differences between years and between wet and dry seasons (Figure 14) in incidence and there was no year x season interaction. *Trypanosoma vivax* and *T. congolense* infection rates were also examined separately and the incidence of the two trypanosome species in weaners of different ages is presented in Table 24. There was a very clear and significant increase with age (P < 001) in *T. congolense* incidence, with *T. vivax* the incidence was generally lower and the trend with age was in the opposite direction but the differences between the age groups were not statistically significant.

Packed cell volume was analysed using individual PCV values classified according to season and age of animal. There were significant differences between seasons and between age groups (P < 0.001). The PCV values for the different age groups are presented in Table 24, the differences although statistically significant were very small and there was no obvious trend. In bulls over 4 years of age the *T. congolense* infection rates were almost five times higher than in animals under two years of age yet PCV values in the two age groups were almost identical. It appears that these animals develop a certain level of tolerance to the effects of trypanosome infection and manage to maintain their PCV values despite being infected.

Table 24. *Trypanosome infection rates (%) and standard error for T. vivax (Tv) and T. congolense (Tc) and mean PCV and standard error (SE) in weaner/bulls of different ages.*

Age group	*Tv* infection	SE	Tc infection	SE	PCV	SE
< 2 years	3.3	0.2	2.1	0.2	26.7	0.1
2 - 3 years	3.6	0.3	5.7	0.4	26.2	0.1
3 - 4 years	3.3	0.4	6.2	0.5	26.4	0.2
> 4 years	2.7	0.4	10.0	0.7	26.6	0.2

+ n is the number of blood samples on which infection rates and PCV were based.

The relationship between PCV and bodyweight was also examined. Packed cell volume was added, as a covariate, to the analyses of bodyweight at various ages. Mean PCV between weaning and 15 months of age was positively correlated with weight at 15 months of age ($P < 0.05$). There was a similar, but non-significant trend in the relationship between PCV and bodyweight in older animals.

Estimation of genetic parameters

Repeatability and heritability estimates were obtained for various traits. Repeatability sets an upper limit to the heritability and is often more easily estimated than heritability because information on the relationships between the animals is not required. Repeatability analyses were undertaken on the cow data and a summary of the results from these analyses is presented here. Heritability analysis were undertaken primarily for the calf data set in which the pedigree information was fairly complete and there were a large number of animals

The repeatability of PCV in cows was estimated for both single PCV measurements and for mean PCV over seasons. For single PCV values the repeatability was 26% and when the mean PCV for a cow during a season was calculated the repeatability of this mean was 49%. Repeatabilities were also estimated for infection rates in cows. Infection rates for all species of trypanosome and for *T. vivax* and *T. congolense* separately were used and in all cases the repeatability estimates were less than 10%. The mean weight for each cow in each season was calculated and the repeatability was estimated as 50%. In the previous Chapter the repeatability of mean PCV, trypanosome infection rates and weight change in cows during lactation was estimated. The repeatability estimates for weight and PCV in those analyses were also higher than those obtained for infection rates.

For the heritability analyses information on dam identity was available for all but three of the calves and, in addition, sire identity was known for 610 of the 905 calves. There were 35 different sire groups, including the “sire unknown” group. The number of calves per known sire varied from two to 45 with a mean of 18. Estimates of variance components were obtained by Restricted Maximum Likelihood (REML) using a derivative-free algorithm and fitting an "animal model" throughout (Meyer 1992). The animal model used in the analyses includes a

random effect representing the additive genetic merit (or breeding value) for each animal and trait. Environmental effects detected as significant in previous least squares analyses for each variable were used as fixed effects. The h^2 estimates are summarised in Table 25.

Table 25. *Heritability estimates for traits measured between birth and weaning in Orma calves*

	Model 1			Model 3		
Trait	h^2	SE	$h^2(A)$	SE	$h^2(M)$	SE
No. *T. congolense* infections	0.03	0.06	0.01	0.05	0.04	0.04
No. *T. vivax* infections	0.09	0.06	0.10	0.07	0.00	0.04
Mean PCV	0.44	0.08	0.37	0.09	0.07	0.04
Birth weight	0.29	0.07	0.15	0.08	0.13	0.05
Weaning weight	0.31	0.08	0.04	0.06	0.29	0.05

h^2 is the total heritability, $h^2(A)$ the direct heritability estimate and $h^2(M)$ the maternal heritability.
Model 1: The animals' additive genetic effects is the only random effect; maternal influence is ignored.
Model 3: Maternal effects are attributed to the genotype of the dam and the maternal genetic effect becomes a second random effect.

Model 1 is a simple animal model with the animals' additive genetic effects as the only random effects; maternal influence is ignored in this model. Model 3 attributes all maternal effects to the genotype of the dam, fitting the maternal genetic effect as a second random effect for each animal. Weaning weight had a very large maternal component as expected; weaning weight is a reflection of the dam's milk production more than the calf's ability to grow. Birth weight too was affected by the maternal environment. The maternal component was not significant for any of the other traits. The estimates for birth weight and weaning weights were in line with those published elsewhere (Meyer 1992). None of the estimates of infection status were significant.

Repeatability and heritability were also estimated on some traits in male weaners. The repeatability of single PCV values was 22%; season and age were included in the model as fixed effects. Heritability was estimated for weight at 15 and 24 months of age using Model 1 only (Table 26). There was considerably less data available than in the calf data set resulting in

larger standard errors. There were too few observations for weight at 36 and 48 months of age to provide adequate measures of heritability.

Table 26. *Estimates of heritability for post weaning weight and growth rate in male Orma weaners.*

	No. of weaners	h^2	SE
Bodyweight at 15 months	227	0.37	0.23
Growth rate to 15 months	227	0.31	0.24
Bodyweight at 24 months	165	0.29	0.25
Growth rate to 24 months	165	0.27	0.25

The medium to high heritabilities estimates for weight traits and packed cell volume indicate that these traits may be more suitable than trypanosome infection rate as selection criteria. Post weaning growth rate under challenge, the trait on which selection was based, has a heritability of around 30%. This information confirms that some progress may be expected through selection. The policy of selection of weaners within weaning groups will help correct for a number of environmental factors (year of weaning, month of weaning, weaning weight) shown in the analyses to effect post weaning growth rate.

The selection programme

Artificial selection took place on the male side and bulls were selected for breeding based on post weaning growth rate. The first calves sired by Galana-bred selected bulls (Generation 1 calves) were born in April 1987 (Table 27). Generation 1 calves were born throughout an eight year period between April 1987 and May 1995. The first Generation 2 calves were born in November 1991

To assess the impact of the selection programme analyses of weight and growth rate traits in calves and male weaners were undertaken. Animals were classified according to generation. Season of birth was fitted as a random effect nested within generations. The birth weights and weaning weights of the calves in the three generations are shown in Table 27. An additional

year's data up until October 1996 was used in these analyses in order to increase the numbers of observations for Generation 2. Although there was no direct selection for increased birth or weaning weights the correlation between the trait under selection, post weaning growth rate, and birth and weaning weights will be positive so any improvement in birth or weaning weight might be ascribed to a correlated response in these traits.

Table 27. *Least square mean and standard error (SE) for birth and weaning weight (kg) in Orma calves from different generations.*

Generation	Birth dates	Birth Weight.			Weaning Weight		
		No. of calves	Mean	SE	No. of calves	Mean	SE
0	May 1983 - April 1987	164	18.1	0.7	192	106.9	3.3
1	April 1987 - May 1995	564	20.7	0.4	507	106.1	2.6
2	November 1991- October 1996	175	21.1	0.6	133	111.7	3.7

Birth weights improved over time; the difference between generations in birth weight was statistically significant ($P < 0.05$) but that in weaning weights was not. However, the extent to which these changes were related to real changes in the genetic constitution of the herd can not be ascertained. The analysis was such that the effects of generation and season/year were confounded. The confounding meant that it was not possible to distinguish differences among years from those among generations; trypanosome challenge was lowest in the four years when the Generation 0 calves were born (Figures 8-11) yet birth weights were lowest in these calves. Milk off-take for human consumption became a common practice from 1990 onwards; this will depress weaning weights. Many of the Generation 1 calves and all the Generation 2 calves (Table 27) were reared by dams that were milked.

It was more difficult to estimate response in the trait under selection because of the smaller numbers of animals. Using a similar model, with season nested within generations, growth rate to 15 months and to 24 months was analysed in 245 and 187 weaners respectively (Table 28). The differences between generations in growth rate to 15 months were not statistically

significant but the growth rate differences to 24 months of age were significantly lower in Generation 1 and 2 than in Generation 0.

Table 28. *Least squares mean and standard error (SE) for post weaning growth rates (g/day) in male Orma weaners from different generations.*

Generation	Growth rate to 15 months			Growth rate to 24 months		
	No. of weaners	Mean	SE	No. of weaners	Mean	SE
0	86	212	24	55	258	18
1	137	160	23	112	190	16
2	22	217	34	20	212	31

Here again season/year of birth was confounded with generation and it is possible that the better growth rates in the earlier years were a result of the lower trypanosome challenge. Post weaning growth rates were available for only 22 Generation 2 animals; more data from Generation 2 animals will be required to allow for any meaningful interpretation of these results.

Discussion

There are no data available for Orma cattle under traditional systems of management and little data for other East African breeds, apart from the Boran, under ranch conditions. Comparisons are therefore difficult. De Leeuw (1990) in a survey of several traditional cattle production systems in sub-Saharan African with a variety of disease constraints, reported average calf weights of 82kg at twelve months of age. Maloo et al (1988) studied East African Zebu in villages in the Coast Province of Kenya where trypanosomosis was the major disease constraint and three of the four tsetse species found on Galana ranch were present. Calf weight at 12 months was increased from 59 kg to 63 kg with the use chemoprophylaxis. The eight-month weaning weight of the Orma calves under the regime on Galana Ranch was 107kg.

Orma calves rarely die of trypanosomosis; annual calf mortality was 11.3%, compared with 16% in De Leeuw's (1990) study. On Mkwaja Ranch in Tanzania, improved Kenya Borans under constant chemoprophylaxis, had pre-weaning mortalities of 8% (Trail et al 1985). Calf

mortality of over 40% has been reported for N'Dama calves under high trypanosome challenge (Agyemang et al 1992)

The N'dama cattle of West Africa have come to be regarded as the "gold standard" for trypanotolerance. These *Bos taurus* type cattle have a longer history of exposure to the tsetse fly and the trypanosome than the *Bos indicus* type cattle of Africa. Thus, their trypanotolerance would be expected to be far greater than that observed in the Orma Boran. Similar studies, under ranch conditions, to those reported here for the Orma Boran have been reported for the N'dama. Comparisons between breeds maintained under challenge from different species of tsetse flies in different environments must be made with caution, however, some interesting points arise from the comparison. In one particular study on N'dama calves in Gabon (Trail et al 1993), the treatment regime was almost identical to that used here. Trail et al (1993) reported 0.19 *T. vivax* infections and 0.12 *T. congolense* infections per calf from birth to weaning under what was considered to be medium tsetse challenge. The fly challenge on Galana Ranch during the years of this study varied from medium to high (Njogu et al 1985a; E. Opiyo pers comm.). Infection rates in the Orma calves were higher than in the Gabon study; 0.28 for *T. vivax* and 0.20 for *T. congolense*. However, Trail et al (1993) gave no indication of pre-weaning mortality and the infection rates are based on calves that survived to weaning. The infection rates calculated in this study were for all calves in which PCV was measured at least once irrespective of whether or not they survived to weaning.

In both studies treatment of infected calves took place when the PCV value fell below 26%. Additional treatments were administered on the basis of detection of clinical symptoms of the disease. In the N'dama study 0.52 curative treatments per calf were given and the overall infection rate was 0.31 infections per calf. The Orma calves received an average of 0.54 curative treatments each and the infection rate was 0.48. In both studies there were a proportion of infections detected which were not treated and treatments were given to animals in which no infection was detected. However, in the Orma calves, the majority of treatments were based on detected parasitaemia while in the Gabon study, the majority of treatments were based on clinical symptoms and not on detected parasitaemia. The Orma calves were sampled once ever two weeks while the N'dama calves were sampled monthly. More infections will be

missed on monthly sampling leading to a lower recorded infection rate and possibly to more calves being treated on clinical symptoms only.

Treatment of the N'Dama calves, was based on detection of clinical symptoms of the disease, treatments were administered when "a clinical examination showed the animal to be badly affected". Thus it appears that many of the N'Dama calves were badly affected but parasitaemia remained undetected. More frequent sampling of animals might have ensured that the majority of infected animals were detected as parasitaemic. These comparisons emphasise the problems associated with the interpretation of results from studies where the protocol allows for treatments based on clinical judgement.

The PCV values of the N'Dama calves (Trail et al 1993) and the Orma calves also provide an interesting comparison. The mean PCV from birth to weaning of 458 N'Dama calves was 35.7%, considerably higher than that recorded for the Orma: 28.7%. In the N'Dama calves PCV was measured eight times between birth and weaning, in the Orma calves 16 measurements were recorded. A mean PCV of 35.7%, is very high when one considers that 132 infected calves were treated with PCV values of below 26% and 117 were treated on the basis on clinical symptoms. This implies that many calves had PCV values close to 40% or above. Such high PCV values are rarely recorded on Orma cattle even when protected from trypanosomosis or maintained in tsetse-free areas. Mean herd PCV values of below 30% are frequently recorded on Galana Ranch during the dry season in tsetse-free areas.

The infection rates in lactating Orma cows were considerably higher than those recorded in N'Dama cows in the Gabon study (Trail et al 1993). However, over half of the N'Dama cows were given prophylactic treatments during two of the five years of the study. Infection rates in the N'Dama calves, which received no prophylaxis, indicated that during these two years, the trypanosomosis challenge was two to threefold that observed in the other three years of the study. The use of prophylactic drugs in the Orma cows during the period of peak challenge in 1988/89 would have reduced overall infection rates substantially. An annual calving rate of 61% was reported in the N'Dama study where seasonal calving was practised. The overall annual calving rate for the Orma was 59%, under management conditions where a bull was not always present.

Annual cow mortality was probably underestimated in this study at 6%. Cow mortality in a variety of production systems rarely exceeded 10% (de Leeuw and Thorpe, 1996). However, cow mortality of 19% was reported in N'Dama under high challenge in one study in the Gambia (Agyemang et al 1992).

Lower infection rates in calves than in their dams has been a consistent feature of the Galana studies and observed in both Orma and Galana Boran (Chapters 5). Similarly, in Zaire, Trail et al (1994b) found that infection rates with both *T. vivax* and *T. congolense* were lower in N'Dama calves than in their dams. Rowlands et al (1993) also reported lower infection rates in calves in Ethiopia. In contrast infection rates in N'Dama calves and dams was very similar in Gabon (Trail et al 1993).

The change in the proportion of *T. vivax* and *T. congolense* infections with age appears to be a feature of many studies in both East and West Africa. The majority of infections observed in N'Dama calves in Gabon and Zaire were caused by *T. vivax* while *T. congolense* was the predominant trypanosome in cows (Trail et al 1993, 1994b).

Trail et al (1991b) in a study in N'Dama cattle reported a heritability estimate for average PCV of 0.35 ± 0.30, compared with 0.44 ± 0.08 for the Orma data. The N'Dama study had 29 sire groups and 5.1 progeny per sire. The present study had 34 known sire groups, with 18 progeny per sire. The larger progeny groups in the Orma data will have improved the precision of the heritability estimates. Furthermore, the DfREML procedure adopted here uses information for all relationships in the data set and thus better use is made of the available data and the estimates are more precise. However, the traits measured were slightly different. In the Orma study the estimates are based on 15 PCV measurements in calves over an eight month period from birth to weaning. A proportion of these calves (39%) became infected and most of these infections were treated. The N'Dama estimates were for 11 PCV measurements over three months in 12 month old animals. Twenty eight percent of the animals became infected but no treatments were administered. The genes controlling response to trypanosomosis may be different at different stages of the animal's life and non-genetic (i.e. environmental) influences may also be different. Nonetheless these studies indicate that mean PCV with or without treatment could be improved through selection.

In the N'Dama data the heritability estimate for PCV increased to 0.64 ± 0.33 when PCV was corrected for trypanosome infection. In the Orma study correction for infection made no significant change to the heritability estimate (Dolan 1996). This was probably because the infected Orma calves were treated when the PCV value dropped thus preventing any further decrease and thereby reducing the variance in PCV values. In non-treated animals the variance will be greater and taking the infection status and parasitaemia score into account will remove a large proportion of this environmental variation thereby increasing the heritability estimate.

The low heritability estimates obtained for trypanosome infection rates (Table 25) indicate that selection for this trait is unlikely to be successful. Traits that affect survival are often found to have low heritabilities because of the impact of natural selection. In the case of trypanosomosis one would expect infection rates to have a low heritability; the disease kills and exposure over many centuries will result in breeds with little genetic variation in disease resistance. Trypanocides, which will counteract the impact of natural selection, have only been available over the past fifty years. The low estimates of heritability are an indication of the long term exposure of the Orma cattle to trypanosomosis.

The results presented here can be used to provide an estimate of the expected response to selection for post weaning growth rate. The predicted change in the mean value of a trait from one generation to the next obtained by breeding from superior animals is given by the formula $R = h^2S$ where R is the predicted response, h^2 is the heritability of the trait under selection and S is the selection differential i.e. the difference between the mean of the parents selected for breeding and the herd mean (Falconer, 1981).

The heritability of the trait under selection, post weaning growth rate under trypanosomosis challenge, was estimated at around 30%. This is in line with other heritability estimates for growth rates found in the literature and indicates that the trait can be improved through selection.

The superiority of the animals used for breeding (S) can be estimated in advance and in the absence of precise measurements on the selected animals, provided the proportion to be used for breeding is known and the trait under selection can be assumed to be normally distributed. The superiority of the selected parents is then estimated in terms of standard deviations rather than in the units of measurement of the trait. The selection differential (S), measured in terms of the phenotypic standard deviation, (S/σ_p), is called the selection intensity, $i = S/\sigma_p$, thus $S = i\sigma_p$. The selection intensity depends only on the proportion of the herd to be selected and can be obtained from tables based on the properties of a normal distribution. For example if the best 50% of a herd is used for breeding then the mean of these selected parents will be $0.8\sigma_p$ above the herd mean.

Using the formula: $R = h^2 i\sigma_p$, the expected response in one generation can be estimated. The selection intensity, $i = (i_m + i_f) / 2$, where i_m is the selection intensity in males and i_f the selection intensity in the females. As there was only selection on the male side this becomes: $i = i_m / 2$. Twenty-nine selected bulls were used throughout the study from 283 male weaners in which post-weaning growth was measured (10%). However, many of the 283 weaners had not yet reached maturity by the end of 1995, thus the percentage, of those available, used for breeding was closer to 12%. In reality selection intensities varied between generations. There were many more Generation 1 weaners available for selection than in other generations (Table 27). Also the weaners used for breeding were not always those with the superior growth rates as explained below. Thus the mean growth rate of those used for breeding was not the mean of the best 12%. If it is assumed that those bulls used for breeding were from amongst the best 20% this gives a value for the selection intensity (from the tables of the normal distribution) of $i_m = 1.4$. The standard deviation in growth rate to 15 months of age was estimated in the analyses as 98 g/day (Table 23). Taking $\sigma_p = 100$ g/day and $h^2 = 0.30$ then response per generation = 0.30 x 1.4/2 x 100 = 21 g/day. An improvement in growth rate between weaning and 15 months of age of approx. 20 g/day in one generation could be expected. This does not appear to have been not achieved (Table 28).

The confounding effects of changes in trypanosome challenge with generations and the resultant difficulties in interpreting the changes in the herd mean from one generation to the next have been discussed. Nonetheless, it seems likely that the expected response to selection has not been achieved and the time taken to produce each generation has been too long. The predicted response to selection is often presented as the predicted response per year given by $R = h^2S/L$, where L is the generation interval. From this formula it can be seen how the rate of progress in a selection programme can be improved.

The fewer animals selected for breeding the greater is the selection intensity and the more progress that can be expected. However, in a closed herd care must be taken to avoid inbreeding. Genetic variation in future generations is decreased as fewer animals are used for breeding and as genetic variation decreases the h^2 decreases. In the Orma programme the selection intensity could be greatly improved without decreasing the genetic variation. Firstly if all the male calves weaned were returned from weaning then the proportion used for breeding would be decreased and a higher selection intensity could be achieved. Up until October 1996 only 283 of the 382 male calves weaned entered the selection programme.

Secondly the selection of breeding bulls must be based on post weaning growth rate and not on any other criteria. On occasion the ranch made the decision as to which animals to castrate, on other occasions a number of weaners of differing ages were considered together and selected or rejected on the basis of body size or conformation, irrespective of growth rate. Sometimes animals with above average growth rates were culled because they were frequently infected. During the change in ranch management in 1989 there was a period when all KETRI herds were kept under ranch management, the protocol for the herd was not adhered to during this period. Thirty of the 31 weaners culled in 1989 were culled on instructions from the ranch management, there was no indication of the basis on which these decisions were made. Also, during this time, conceptions took place and the bulls' identity was unknown. This resulted in 19 weaners being culled in 1991 because they had no sire identification.

The rate of response to selection can also be improved if the generation interval is decreased. The turn over of generations has been slow in the Orma selection programme and only 175 Generation 2 calves were born by October 1996 (Table 27). Some of the original cows purchased from the Orma people were still in the herd in 1995. If the older original cows had been culled earlier and all the 27 month-old heifers returned by the ranch management to KETRI on time and calves produced from these heifers, then the generation interval could have been much reduced.

7 General discussion

The first indications of trypanotolerance in the Orma Boran came from a series of field trials, summarised in Chapter 1, conducted in Orma steers purchased from the Orma people in the Tana River District. However, it was not possible to ascertain if the differences observed between the Orma Borans and the Galana Borans were indicative of real innate genetic differences between these breeds. There was the possibility that previous exposure to trypanosomosis was responsible for a superior acquired immunity observed in the Orma compared to the Galana Boran. The idea that an innate immunity is genetic while an acquired immunity is not is incorrect; animals can inherit a superior ability to acquire immunity. However, it seemed necessary to investigate the role of previous exposure to trypanosomosis in the response to subsequent challenge on Galana Ranch.

The importance of previous exposure to trypanosomosis in determining response to subsequent challenge depends on the variety of the different serodemes involved. A number of different serodemes of both *T. congolense* and *T. vivax* have been isolated from Galana Ranch over the years. However, no systematic attempt has been made to define the number and nature of the serodemes circulating in the tsetse fly population as was done in Nigeria (Gray 1970). Thus it was not possible to distinguish between the nature of the trypanosome challenge in different areas of the ranch nor to distinguish between homologous and heterologous challenge.

The similarities or otherwise between the trypanosome challenge on Galana Ranch and that in the Tana River District, from where the Orma Boran originated, is a matter of speculation. The tsetse distribution maps indicate the presence of *G. pallidipes, G. longipennis* and *G. brevipalpis* in the Tana River District mostly associated with the river. In the northern reaches of the river the tsetse population is confined almost entirely to an area a few kilometers wide

on either side of the river (Figure 1); here *G. longipennis* and *G. brevipalpis* are the only fly species found. However, the tsetse belt on Galana Ranch is contiguous with the tsetse belt in the Tana River District. Furthermore, it is likely than many of the trypanosomes circulating in the fly population on Galana Ranch have been introduced, over the years, in cattle trekked onto the ranch from the Tana River District. Until 1989, the ranch policy was to move new cattle into the quarantine area, close to the ranch boundary, to be treated with trypanocides. Such a policy would reduce, but not eliminate, the probability of introducing new trypanosome strains on to the ranch.

The contention that animals that survive trypanosomosis, with or without chemotherapy, are more resistant to subsequent challenge (Murray et al 1982) is not substantiated by the experiments presented here. While there appears to be some evidence of an improved ability to control the anaemia associated with infection, particularly in relation to *T. vivax* in the Orma cattle, there is no evidence for a reduction in the overall trypanosome prevalence. It seems likely that a large number of different serodemes of both *T. congolense* and *T. vivax* are circulating in the tsetse fly population on Galana Ranch. The probability of re-infection with the same serodeme within a six months period is perhaps low and even if this does occur the challenge on Galana Ranch is such that animals are constantly being re-challenged and the overall infection rate is little changed.

The possibility that the previous exposure of the Orma steers used in these trials may have contributed in some way to their superior response to trypanosomosis cannot be completely discounted. However, the experiments described in Chapters 3 and 4 that attempted to assess the role of previous exposure served to emphasise the differences between the two breeds rather than the role of previous exposure.

That the superiority of the Orma cattle under tsetse challenge was indeed innate was finally confirmed in studies in calves born in the same environment and reared together under the same tsetse challenge (Chapter 5). Considering the history of these two Boran breeds over that past 400 years the finding of genetic differences in response to trypanosomosis is hardly surprising. Trypanosomosis affects reproductive performance and survival; the two most important components of natural fitness. Minute differences in fitness result in major changes in the genetic constitution of a population over evolutionary time. The selection coefficients associated with the genes controlling the response to trypanosomosis will vary but are likely to have been larger in the past, before the advent of chemotherapy. These two Boran types have been geographically isolated for some 400 years. During that time the Orma cattle occupied tsetse infested lands while the Kenya (Galana) Boran, in the Borana Plateau of Ethiopia and the Kenya Highlands, was reared in a trypanosome free environment. It would be surprising to find that there were no differences in their response to trypanosomosis. In cattle exposed over generations, the evolution of a superior response to tsetse challenge in both its innate and acquired components is to be expected.

The mechanisms whereby the Orma cattle are less susceptible to field challenge remain to be elucidated. The difference in trypanosome prevalence observed in cattle exposed to the same tsetse challenge is an important and consistent feature of these studies. This could be due, in part, to the Orma cattle being bitten less often. The majority of the Orma Boran is white or fawn coloured while the predominant colour in the Galana Boran is red; the colour preferred by the beef breeders in the Kenya Highlands. The two sentinel herds used in 1982/83 were colour matched to provide an assessment of these colour differences; the results indicated that coat colour was not important. However, variation amongst species of wildlife in attractiveness to tsetse flies has been demonstrated (Grootenhuis 1998) and differences in attractiveness to other fly species have been observed both between and within cattle breeds (Warnes and Finlayson 1987; Brown et al 1992). Vale (1981; Vale and Hall 1985) has demonstrated that odour is of great importance in attracting the tsetse fly to its host and he has suggested that cattle suffering from loss of condition are less attractive to tsetse. However, Baylis and Nambiro (1993) reported that trypanosome infected cattle are bitten more often.

If biting rate alone was the key factor then one would expect the reduction in detected parasitaemias to be the same for both trypanosome species. In the trials involving steers (Table 1, Chapter 2) there were significant differences in the number of *T. congolense* infections detected in only two of the five years. In contrast the differences between the breeds in *T. vivax* prevalence were much larger and significant in all years. In the breeding herds the trypanosome prevalence differences in both cows and calves was associated with *T. vivax* infections and not *T. congolense* infections. It is unlikely that such observations could have arisen solely through the Orma cattle being bitten less often.

Differences in trypanosome prevalence in cattle exposed to the same challenge would also be observed if a higher dose of metacyclics was required to establish an infection in one breed than in the other. Very low numbers of metacyclic *T. vivax* trypanosomes are extruded by tsetse flies (Otieno and Darji 1979) and the variation in prevalence observed amongst the two breeds is primarily in the prevalence of this species. Thirdly, the differences could be explained by control of the parasitaemia to below detectable levels. Possibly all three factors could contribute to the reduced prevalence observed in the Orma cattle under field challenge.

The variation in response to an established infection observed between the two breeds seems to be related to both acquired and innate factors with innate factors appearing to be more important under the conditions prevailing on Galana Ranch. The Orma cattle seem to have a better capacity to improve their control of the anaemia over time particularly in relation to T. vivax infections. However, under the delayed treatment regime (Chapter 4) response to trypanosomosis did not improve over time and the newly introduced Orma steers had fewer infections and higher PCV values than the previously exposed steers. In these trials the immediate treatment regime appeared to induce greater immunity than delayed treatment or self cure.

The economic benefits that might accrue from the exploitation of the differences between the two breeds will vary in time and space (Ashley et al 1992). The Galana steers generally have

superior growth rates to the Orma steers but higher mortality rates. However, under intense challenge the growth rates observed in Orma steers are similar and can be superior. Furthermore their reduced requirement for prophylactics is likely to become increasingly important. The spread of drug resistant trypanosomes (Peregrine 1994) has serious implications for the future of cattle keeping in tsetse infested areas of East Africa. Isometamidium chloride was the prophylactic of choice on Galana until 1987 when decreasing periods of prophylaxis necessitated increased drug usage (Dolan et al 1992). This frequent dosing with trypanocides resulted in high mortality rates associated with liver toxicity (Stevenson et al 1993). With no new trypanocides under development and resistance reported to all the currently available drugs, cattle with reduced susceptibility to trypanosomosis and a lower requirement for drug therapy or prophylaxis may have a major contribution to make to future sustainable livestock production in the tsetse infested areas of East Africa.

The data collected from the Orma breeding herd since 1983 indicate that these cattle can achieve levels of productivity under constant tsetse challenge which are at least comparable and in many cases superior to other indigenous African breeds. The herd has been maintained in an area of Galana Ranch considered unsuitable for the Galana Borans. The results of the heritability analyses suggest that selection for post weaning growth rate may be a viable method of selecting for trypanotolerance. However, up to October 1996 it was not yet possible to demonstrate that the selection programme had had any significant impact. A concerted effort to adhere strictly to the protocols will be required to ensure the continued success of the programme.

Improvement of indigenous breeds is a major component of the drive to increase productivity from livestock in West and Central Africa. In East Africa the Orma Boran is an obvious candidate for improvement. It has some important advantages over many of the other indigenous breeds; it is faster growing, reaches a larger mature body size and has reduced susceptibility to trypanosomosis.

The conditions for field research that existed on Galana Ranch in the 1980s no longer prevail. An outbreak of CBPP on Galana Ranch in 1997 resulted in the loss of several Orma bulls.

Plans are currently underway to move the herd to a more accessible part of the country from where breeding stock can be supplied to farmers in other tsetse-infested areas of Kenya. In 1995 bulls from the Orma herd, no longer required for breeding, were sold to Maasai pastoralists in the Kajiado district of Kenya. There is currently a demand for more bulls. In addition a survey of the Orma cattle in the Tana River District is currently underway. Information will be gathered on the cattle husbandry practices of the Orma people particularly in relation to trypanosomosis control and some assessment will be made of the numbers of cattle in the District and their productivity at village level.

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