

**FACTORS ASSOCIATED WITH DISEASES AND MORTALITY OF CALVES  
IN MOROGORO, TANZANIA**



**BY**

**LESAKIT SIPIRA BERNARD MELLAU**

**A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT FOR THE  
AWARD OF THE DEGREE OF MASTER OF VETERINARY MEDICINE OF  
SOKOINE UNIVERSITY OF AGRICULTURE**

**1997**

**ABSTRACT**

This study was aimed at elucidating the influence of management and other factors associated with diseases and mortality of calves managed under intensive, semi intensive and free range systems practised by state farms, parastatal farms and small scale dairy producers in the region.

Management factors include bucket feeding versus suckling of colostrum, administration of post colostrum feeds, housing and disease control schemes. Other factors were breeds of cattle and season of the year. A total of 368 calves were involved in the study over a period covering one rain season and one dry season. 138 calves were from Kingolwira Prisons farm (KPF), 127 from Mlali, 57 from Azam Estate, 22 from Magadu dairy Unit and 24 from SUA farm. All these farms are within a radius of 50 kilometers from Morogoro town and were selected on the basis of differing management systems, herd size and access to the farm by road.

The study was conducted through questionnaires, clinical examination and laboratory analysis for hematological, parasitological and pathological parameters.

Prevalence of diarrhoea was significantly high in bucket fed calves than suckled calves ( $P < 0.001$ ). Bovine coronavirus, rotavirus and *Escherichia coli* K 99 antigens were detected in 61% of faecal samples collected, and mixed infection of 2 or all of these organisms was frequent.

Helminthosis was observed in both weaned and unweaned calves but calves less than 1 month old were relatively less affected as compared to older calves  $(P < 0.001)$ .

*Haemonchus spp*, *Trichostrongylus spp*, *Strongyloides papillosus*, *Oesophagostomum spp* and *Cooperia oncophora* were the most frequently isolated worms, where they accounted for 35.5%, 15.5%, 15.5%, 31.1% and 2.2% respectively. *Toxocara vitulorum* infestation was detected in only 4 calves of less than 28 days at Mlali farm where water buffaloes graze with cattle. Other diseases were, pneumonia, cutaneous mycosis which subsided with increasing age, bovine parasitic otitis, cases of Beta hemolytic *E. coli* otitis and alopecia of unknown cause.

Mortality rate among calves was 25%, 17.4%, 13.6%, 8.7% and 7.8% for SUA farm, KPF, Magadu dairy unit, Azam Estate and Mlali farms respectively. Weaned and unweaned calf mortality contributed equally to total calf mortality. Helminthosis was the cause of mortality in weaned calves, whereas, pneumonia and diarrhoea were the main causes of death in unweaned calves. Coccidiosis was not a serious disease in calves managed under semi intensive and free range systems as it was in intensively managed calves.

**DECLARATION**

I LESAKIT SIPIRA BERNARD MELLAU do hereby declare to the Senate of Sokoine University of Agriculture that, this dissertation is my own original work and has not been submitted for a degree award in any other University.

Signature.....*B. Mellaau*  
Date.....*10.06.1997*

**COPYRIGHT**

No part of this dissertation may be reproduced, stored in any retrieval system or transmitted in any form or by any means, electronic, photocopying, recording or otherwise without the prior permission of the author or Sokoine University of Agriculture in that behalf.

## ACKNOWLEDGEMENT

I wish to thank my Supervisors, Associate Professor J.A. Matovelo of the Department of Veterinary Pathology and Associate Professor U.M. Minga of the Department of Microbiology and Parasitology, Sokoine University of Agriculture (SUA) for their useful support during the course of this study. I also wish to express my thanks to Professor R.J. Jorgensen, S.M. Thamsborg and K. Nielsen all of the Department of Internal Medicine, Royal Veterinary and Agricultural University (RVAU) Denmark, for their great initiative at introducing me to the conduct of scientific work.

I wish to thank Professor A.A. Kassuku the Head of Department of Veterinary Microbiology and Parasitology and Leader of Ruminant Helminths Research Project, for his keen interest and guidance especially on the parasitological aspects of my work.

My gratitude are also due to technicians Ms G. Mchotika, Mr H. Mafazy, of the Department of Veterinary Pathology (SUA) and Mr L. Msalilwa, Mr E. Rugaimukamu, Mr M. Shoo and Mr T. Mwanjala of the Department of Microbiology and Parasitology for their kind cooperation on technical aspects of this work.

I further wish to acknowledge the favourable academic environment I enjoyed during my stay in Denmark. For this I wish to thank the Staff of Danida Fellowship Centre and Dr. Kaj Bruhn the Director of Centre for Tropical Agriculture and Environment for his

effective facilitation on all academic, administrative and logistical matters.

I further wish to express my thanks to the administration of Kingolwira Prisons farm (KPF), Mlali farm, Azam Estate Dairy farm, SUA farm and Magadu Dairy Unit for allowing me to conduct my study in their farms.

I also wish to acknowledge the kind support of my employer the Sokoine University of Agriculture (SUA) who provided me with a study leave to carry out this study.

This study was sponsored by the Danish International Development Agency (DANIDA) to which I am most thankful.

Last but not least I thank my family for patience and understanding during my engagement in this endeavour.

**DEDICATION**

This work is dedicated to my wife Natujwa and our beloved children Hope Nina, Gratias  
Sipira and little Glory Namugole.

**TABLE OF CONTENTS**

|   |             |
|---|-------------|
| <b>ABSTRACT</b> . . . . .                                 | <b>ii</b>   |
| <b>DECLARATION</b> . . . . .                              | <b>iv</b>   |
| <b>COPYRIGHT</b> . . . . .                                | <b>v</b>    |
| <b>ACKNOWLEDGEMENT</b> . . . . .                          | <b>vi</b>   |
| <b>DEDICATION</b> . . . . .                               | <b>viii</b> |
| <b>LIST OF TABLES</b> . . . . .                           | <b>xiii</b> |
| <b>LIST OF FIGURES</b> . . . . .                          | <b>xv</b>   |
| <b>ABBREVIATIONS AND SYMBOLS</b> . . . . .                | <b>xvii</b> |
| <br>  |             |
| <b>1.0 INTRODUCTION</b> . . . . .                         | <b>1</b>    |
| <br>  |             |
| <b>2.0 LITERATURE REVIEW</b> . . . . .                    | <b>3</b>    |
| <br>  |             |
| <b>2.1 General aspects</b> . . . . .                      | <b>3</b>    |
| <b>2.2 Influence of environment on diseases</b> . . . . . | <b>7</b>    |
| <b>2.3 Diseases of calves</b> . . . . .                   | <b>9</b>    |
| <b>2.3.1 Neonatal calf Diarrhoea</b> . . . . .            | <b>9</b>    |
| <b>2.3.2 Calf Pneumonia</b> . . . . .                     | <b>20</b>   |
| <b>2.3.3 Parasitic gastroenteritis</b> . . . . .          | <b>26</b>   |

|  |           |
|--|-----------|
| 2.3.4 Coccidiosis . . . . .                                  | 32        |
| 2.3.5 Indigestion . . . . .                                  | 39        |
| 2.3.6 Tickborne diseases . . . . .                           | 40        |
| 2.3.7 Bovine Parasitic otitis . . . . .                      | 41        |
| 2.3.8 Dermatophytosis . . . . .                              | 42        |
| <br>   |           |
| <b>3.0 MATERIALS AND METHODS . . . . .</b>                   | <b>43</b> |
| <br>   |           |
| 3.1 Study design . . . . .                                   | 43        |
| 3.2 Farms, breeds and management . . . . .                   | 44        |
| 3.2.1 Kingolwira Prisons Farm (KPF) . . . . .                | 44        |
| 3.2.2 Mlali Farm . . . . .                                   | 45        |
| 3.2.3 Azam estate . . . . .                                  | 46        |
| 3.2.4 Magadu Dairy Unit (MDU) . . . . .                      | 47        |
| 3.2.5 Sokoine University of Agriculture (SUA) Farm . . . . . | 48        |
| <br>   |           |
| 3.3 Methodology . . . . .                                    | 48        |
| 3.3.1 Sampling procedure . . . . .                           | 50        |
| 3.3.1.1 Blood Samples . . . . .                              | 50        |
| 3.3.1.2 Faecal samples . . . . .                             | 50        |
| 3.3.1.3 Blood smears . . . . .                               | 51        |

|  |           |
|--|-----------|
| 3.3.1.4 Skin scrapings . . . . .   | 51        |
| 3.3.1.5 Aural swabs . . . . .  | 51        |
| 3.3.1.6 Nasal swabs . . . . .  | 52        |
| <br>   |           |
| 3.4 Analysis . . . . .   | 52        |
| 3.4.1 Determination of Serum Total Protein . . . . .                           | 52        |
| 3.4.2 Determination of serum albumin . . . . .                                 | 55        |
| 3.4.3 Determination of globulin fraction . . . . .                             | 55        |
| 3.4.4 Determination of PCV . . . . .   | 56        |
| 3.4.5 Estimation of haemoglobin . . . . .                                      | 56        |
| 3.4.6 Faecal analysis . . . . .  | 57        |
| 3.4.7 Identification of fungi . . . . .  | 58        |
| 3.4.8 Bovine coronavirus, rotavirus and <i>E. coli</i> K 99 antigens . . . . . | 58        |
| 3.5 Autopsy and histopathology . . . . .                                       | 59        |
| 3.5.1 Autopsy . . . . .  | 60        |
| 3.5.2 Histopathology . . . . .   | 61        |
| 3.6 Statistical Analysis . . . . .   | 61        |
| <br>   |           |
| <b>4.0 RESULTS . . . . .</b>   | <b>62</b> |
| <br>   |           |
| 4.1 Influence of management . . . . .  | 62        |

|   |            |
|---|------------|
| 4.2 Disease control schemes . . . . .               | 73         |
| 4.3 Influence of season . . . . .                   | 76         |
| 4.4 Disease conditions observed . . . . .           | 76         |
| <br>  |            |
| <b>5.0 DISCUSSION . . . . .</b>                     | <b>93</b>  |
| <br>  |            |
| <b>6.0 CONCLUSION AND RECOMMENDATIONS . . . . .</b> | <b>106</b> |
| <br>  |            |
| <b>7.0 REFERENCES . . . . .</b>                     | <b>107</b> |

**LIST OF TABLES**

|         |   |    |
|---------|---|----|
| Table 1 | Methods and duration of feeding colostrum and milk to calves at Azam estate, KPF, Mlali, SUA and Magadu dairy farms . . . . . | 63 |
| Table 2 | Prevalence of diarrhoea among calves less than 28 days of age at Azam estate, KPF, Mlali, Magadu and SUA farms. . . . .       | 65 |
| Table 3 | Calf mortality rates (MR) at Azam estate, Mlali, KPF, Magadu and SUA farms. . . . .   | 67 |
| Table 4 | Pasture and feed supplement given to calves at Magadu dairy unit, KPF, Azam Estate, SUA farm and Mlali farm. . . . .          | 68 |
| Table 5 | Rainfall and point prevalence of unthriftiness in weaned calves at KPF, Azam estate and Mlali farm. . . . .                   | 70 |

|          |  |    |
|----------|--|----|
| Figure 8 | Bacterial otitis in calf from Mlali farm. Whitish creamy pus occluding the ear canal. . . . .                    | 89 |
| Figure 9 | A case of bovine parasitic otitis showing curling of the pinna and complete occlusion of the aural canal.. . . . | 91 |

## LIST OF FIGURES

|           |  |    |
|-----------|--|----|
| Figure 1: | Clinical refractometer used for determination of serum protein concentration. . . . .            | 54 |
| Figure 2  | Gross muscular wasting in a calf 4 weeks after weaning at Kingolwira Prisons farm (KPF). . . . . | 71 |
| Figure 3  | A healthy calf 2 days after weaning at KPF. . . . .  | 72 |
| Figure 4  | Atrophy of villi and dilated lacteals in jejunum of a diarrhoeic calf from SUA farm.. . . .      | 81 |
| Figure 5  | Hyperkeratosis, vacuolation, hydropic degeneration and microabscess in the rumen... . . . .      | 82 |
| Figure 6  | <i>Trichophyton spp</i> on Sabouraud's dextrose agar containing cycloheximide. . . . .           | 84 |
| Figure 7  | Alopecia on the back of the calf from Mlali farm. . . . .  | 87 |

|          |  |    |
|----------|--|----|
| Figure 8 | Bacterial otitis in calf from Mlali farm. Whitish<br>creamy pus occluding the ear canal. . . . .                       | 89 |
| Figure 9 | A case of bovine parasitic otitis showing curling<br>of the pinna and complete occlusion of the<br>aural canal.. . . . | 91 |

**ABBREVIATIONS AND SYMBOLS**

**BAV = Bovine adenovirus**

**BCV = Bovine coronavirus**

**BPO = Bovine parasitic otitis**

**BRSV = Bovine respiratory syncytial virus**

**°C = Degrees Centigrade**

**CBPP = Contagious bovine pleuropneumonia**

**ECF = East coast fever**

**EPG = Eggs per gram of faeces**

**ETEC = Enterotoxigenic *Escherichia coli***

**F1 = First Filial generation**

**FMD = Foot and mouth disease**

**FPT = Failure of passive transfer**

**Ig = Immunoglobulin**

**IMViC = Indole, Methylene blue, Voges proskauer, Citrate**

**KPF = Kingolwira prisons farm**

**MALD = Ministry of Agriculture and Livestock Development**

**mg/kg = Milligrams per kilogram**

**OR = Odds ratio**

**PCV = Packed cell volume**

PI3 = Parainfluenza 3 virus

SDA = Sabouraud's dextrose agar

SE = Standard error

TBD = Tick borne diseases

TP = Total protein

TSZ = Tanzania Shorthorn Zebu

X<sup>2</sup> = Chi Square

## 1.0 INTRODUCTION

The National Livestock Policy of the Ministry of Agriculture and Livestock Development and Cooperatives indicates that, diseases are the single most important constraint limiting livestock production in the country (MALD, 1984). The impact of diseases on livestock productivity is aggravated by the inadequacy of investigative and supportive veterinary services (Mpelumbe, 1984). Diseases alone, have been estimated to account for as much as 20-25% in mortality losses alone (Mchechu, 1983).

The bulk of the 13.1 million head of cattle is made of the local Tanzania Shorthorn Zebu (TSZ) which constitutes 98% of the national herd (Komba, 1992). This herd produces 74% of bovine milk. The dairy herd constitutes only 2% of the national herd (MALD, 1993). On the other hand, the dairy herd small as it is produce the remaining 26% of bovine milk (MALD, 1993). In a situation where demand for milk is far above supply and hence prices are high, Tanzanian farmers are increasingly moving towards the keeping of dairy cattle (Shoo *et al.* 1990). This has accounted for the rise in dairy cattle population from 1% of the national herd in 1984 to 2% in 1988 (MALD, 1987; MALD, 1993).

However, calf mortality has continued to pose a serious limitation to the expansion of especially the dairy herds (Das *et al.*, 1988), thereby reducing the source of replacement

stock and eroding the genetic improvement of the national herd.

Although the factors associated with diseases and mortality of calves in Tanzania may not differ much from those studies in other parts of the world (Lema and Banda, 1991), climate and differences in management practices is bound to exercise influence on various agents known to be associated with diseases and mortality of calves.

Further, a number of diseases have been associated with mortality of calves in Tanzania (Kifaro and Temba, 1990; Shoo *et al.*, 1990), but no studies have been conducted on the verification of the relationship between the disease conditions and susceptibility of calves in various age groups in different management systems. It was the objective of this study therefore to:

(a) investigate the nature and relative significance of conditions associated with diseases and mortality of calves under different management systems in Morogoro; (b) investigate on the causes of disease and mortality of calves of up to 6 months of age in Morogoro; (c) investigate on the impact of different management practices on the susceptibility of various age groups to diseases affecting calves in Morogoro.

It is hoped that, the output of this work will have an impact towards better management of calves and thereby enhance productivity by cutting down morbidity and mortality rates.

## 2.0 LITERATURE REVIEW

### 2.1 General aspects

Health problems in calves may appear in the form of high disease incidence or general unthriftiness without clinical signs specific of any disease (Simensen and Norheim, 1983). Simensen and Norheim (1993) indicated that, health problems or reduced growth are related to unfavourable environmental conditions, low standard of management and poor nutrition. Where unfavourable environmental conditions are predisposing factors enteric and respiratory disorders are the two main hazards to health (Roy, 1980). In both cases growth of the calf may be retarded resulting into serious economic losses of calves from birth to six months of age with mortality and sub optimal performance caused by diseases or inadequacies in production management (Blood *et al.*, 1990). Calf mortality is a serious problem in all countries where cattle are bred (Simensen, 1982).

The majority of deaths occur at or around birth and during the first weeks of life (Simensen, 1982). Infections acquired under housing conditions are considered to be the major cause of mortality in young calves. Diarrhoea has been singled out to be the most important cause of death (Oxender *et al.*, 1973). However, Blom and Thyssen (1980) found that, more calves died of pneumonia than other causes. Post mortem examinations have in most cases confirmed that gastrointestinal diseases contribute the most important cause of death (Ploger *et al.*, 1980).

These gastroenteric diseases include scours, bloat, helminthoses and coccidiosis (Roy, 1983). Other causes of deaths have been reported to be tick borne diseases (TBD), pneumonia, trypanosomiasis and nutritional deficiencies particularly in calves up to 6 months of age (Kifaro and Temba, 1990; Shoo *et al.*, 1990). The occurrence of calf diseases differ in magnitude and severity from one area to another. This variation is due to varied environmental, managemental, nutritional and physiological factors pertinent to the calf itself (Snodgrass *et al.*, 1986).

In Tanzania, infectious diseases are serious health problems in all domestic animals but gastroenteritis and pneumonia are the most serious problems in calves (Lema and Banda, 1991; Masanja and Matovelo, 1993). Scours, pneumonia and tick borne diseases were the major calf killers in Iringa dairy farms, however, post weaning stress and helminthoses were also reported to be important (Kifaro and Temba, 1990). Shoo *et al.* (1990) reported that, diarrhoea, pneumonia and weak calf syndrome were the main causes of deaths on dairy farms in Eastern zone of Tanzania. At Livestock Production Research Institute (LPRI) Mpwapwa diarrhoea and pneumonia were the main causes of calf mortality (Mpiri *et al.*, 1986). In a survey conducted by Lema and Banda (1991), rotavirus, salmonella, mannose resistant hemagglutinating *Escherichia coli* and Cryptosporidia were reported to be the major causes of neonatal calf diarrhoea in selected farms in Tanzania.

While infection rate and the aetiological agents of calf diarrhoea increases with age of the calves (Lema and Banda, 1991), mortality rates of calves due to various causes decreases with increasing age of calves (Sharma and Jain, 1976). Sharma and Jain (1976) concluded that, losses due to mortality appear to account for only a small proportion of causes of total economic losses in the herd.

Calf losses due to mortality can be divided into deaths occurring during perinatal period, during neonatal period and when calves are beyond a month in age (Roy, 1983). Roy (1983) defined perinatal mortality as calf death occurring between 24 hours before and after parturition. Neonatal mortality meant calves born alive that die between 24 hours and 28 days postpartum, whereas, older calf mortality was calves dying between 29 and 180 days of age (Roy, 1983).

It has been shown that, neonatal mortality is caused by *Escherichia coli* with or without viral or Cryptosporidium involvement (Roy, 1983). Older calf mortality is due to respiratory infections, salmonellosis, bloat, helminthiasis and coccidiosis (Roy, 1983). Superimposed on these are effects of management and environment (Roy, 1980) and genetic attributes of both mother and offspring (Spooner *et al.*, 1975). In the tropics calf mortality rates have been reported to be as high as 50% (Williamson and Payne, 1978). The reason for this high mortality include the hostility of tropical environment to *Bos taurus* dairy breeds in particular, severity of enzootic diseases and to some extent

unsatisfactory management standards (Shaka, 1977). Measures that constitute bad management include poor calf husbandry, poor disease control program and malnutrition (Das *et al.*, 1988). Shoo *et al.* (1990) reported mortality rate of between 20-27% in selected farms in Morogoro. Mortality rates of 41.2% and 9.5 % were reported in a study of morbid states of calves of Sokoine University of Agriculture farms (Masanja and Matovelo, 1993).

According to Withers (1952), the mortality rate and incidence of disease varies under different management practices. Withers (1952) noted that, twice as much mortality rate was found in heifer calves up to six months of age in dairy herds as compared to calf mortality in beef and dual purpose herds. Withers (1952) associated the lower death rate in beef herds with the more natural conditions of rearing which include allowing the calves to suckle colostrum for 4 days postpartum. Suckled calves are used for premilking stimulation of the dam (milk let down) and to empty the mammary gland after each milking.

Sharma and Jain (1976) reported that, the mortality rate of calves up to one year old were fairly similar for Brown Swiss and Zebu hybrids, Sahiwal Zebu and Red Sindhi Zebu. Zebu calves had higher preweaning survival rate than crossbred calves whereas, crossbred calves had higher viability than zebu calves up to one year of age (Das *et al.*, 1988). The lower viability of Zebu calves was attributed to high mortality due to their

low birth weight and lack of vigour (Das *et al.*, 1988).

Hartmann *et al.* (1974) stated that, mortality of calves depended on the type of housing, calving facilities, the person caring for the calves and attendance of calving thus, he reported that, calf mortality was lower when the owner manages the calves rather than when employees performed those duties. This suggests that, owner-managers may be sufficiently motivated to provide the care necessary to ensure a high survival rate in calves. McBeath *et al.* (1971) found that, calves deficient in plasma immunoglobulin are more susceptible to neonatal infections but good husbandry and treatment are likely to ameliorate the mortality rate of such calves. The successful rearing of calves therefore, depend on a well managed combination of early feeding of colostrum, adequate housing and adequate nutrition following the colostrum feeding period (Roy, 1983).

## **2.2 Influence of environment on diseases**

Climatic and environmental conditions are important to newborn calves particularly where enteric and pulmonary infections are causes of calf losses (Simensen, 1982). Infectious diseases of the respiratory tract are a major cause of economic losses in dairy calves, particularly during the winter in the temperate climates (Roe, 1982). The economic losses are due to mortality and unthriftiness caused by acute and chronic enzootic calf pneumonia (Simensen, 1982). Epidemiologic determinants of diseases in neonates are crowded calf pens, inadequate ventilation, high relative humidity and

dampness and generally poor feeding of the calves (Radostits and Blood, 1985).

Dampness has been shown to favour a lot of microbes causing infections in domestic animals and man and in the calf house the quality of air is critical in the control of respiratory disease (Pritchard *et al.*, 1981). Ventilation of the house serves to remove from the environment certain products of the animals' metabolism such as heat, moisture, carbon dioxide, dusty noxious gases and also the microbes (Wathes *et al.*, 1983). Ventilation replaces such products with fresh air. The maximum ventilation rate is usually chosen to limit the temperature difference to 2°C or 3°C above ambient temperature (Randall, 1981). Marked changes occur in the concentration of airborne bacteria in the calf house following changes in the outside temperature and humidity (Jones and Webster, 1981). Side effects of excessive ventilation include predisposing the animals to more severe respiratory problems through organic gases within the animal house. Among the gases, hydrogen sulphide and ammonia are common causes of livestock poisoning (Wathes *et al.*, 1983). Hydrogen sulphide is an irritant at sublethal concentrations causing inflammation of exposed surface tissues (Wathes *et al.*, 1983). Ammonia at 50 ppm was incriminated in the damage of the mucosa lining of the respiratory tract in chicken (Anderson *et al.*, 1964). Draughts resulting from excessive ventilation can be prevented by the correct design of air inlets for example placing them well above animals' height (Randall, 1981).

### 2.3 Diseases of calves

Enteric and respiratory disorders are the two main hazards to calf health but they occur in calves of different ages (Roy, 1990). Mortality from enteric disorders are limited largely to the first 14 days of life and respiratory problems are important from 6-8 weeks of age onwards (Roy, 1980). Neonatal enteric diseases depend on the level of passive immunity that the calf has received from colostrum, its innate resistance to infection, burden of the infection in the environment, and the nutrition of the calf (Logan and Penhale, 1972; Roy, 1983). Calves are born with low gammaglobulins and are more susceptible to infection unless they ingest colostrum and absorb sufficient quantities of lactoglobulin from colostrum (Gay *et al.*, 1965; Logan and Penhale, 1972; McBeath *et al.*, 1971; McEwan *et al.*, 1970).

#### 2.3.1 Neonatal calf Diarrhoea

Neonatal calf diarrhea is caused by different aetiological agents either singly or in combination, both in the tropics and in temperate countries (Saif and Smith, 1985; Snodgrass *et al.*, 1986). Rotavirus, coronavirus, *E. coli*, *Salmonella spp* and *Cryptosporidium spp* are the most important causes of calf diarrhoea in temperate countries (McNulty *et al.*, 1976; Reynolds *et al.*, 1986; Roy, 1983; Snodgrass *et al.*, 1986; Tzipori, 1981). The importance of an organism is usually based on the proportion of diarrhoeic calves from which a particular organism is isolated (Roy, 1990). Bovine coronavirus, rotavirus, *Escherichia coli* K 99, *Cryptosporidium spp* and *Salmonella spp*

were reported to be the main causes of neonatal calf diarrhoea in Tanzania dairy farms (Lema and Banda, 1991). Rotavirus and rotavirus and coronavirus have been detected from healthy calves with no clinical illness (Lema and Banda, 1991; Snodgrass *et al.*, 1986). Rotavirus was found to be more important than others in causing diarrhoea in young calves of around one week of age (Snodgrass *et al.*, 1986). Diarrhoea caused by enterotoxigenic *Escherichia coli* begins during the first few days of life, although peak mortality occurs about 6-7 days of age (Selim *et al.*, 1991; Tzipori, 1981). The importance of predisposing factors in the aetiology of calf diarrhoea has been demonstrated (Reynolds *et al.*, 1986; Snodgrass *et al.*, 1986). These factors include, the build up of infection in the calf house, the system of rearing, the physical environment and animal breed. The rate of build up of infection is related to the immune status of the calf, post colostrum diet of the calf, and the air space in terms of capacity per calf and the ventilation rate in the calf house (Snodgrass *et al.*, 1986).

Regarding the immune status of the calf, the time of colostrum ingestion particularly the first meal after birth is of major importance (Logan and Penhale, 1972).

Highest rates of absorption based on the concentration of immunoglobulin in serum occurs when colostrum is fed during the first 4 hours after birth (Kruse, 1970; Stott *et al.*, 1979). Bacteria that are established in the small intestine before first colostrum feed can be absorbed by pinocytosis in the same way as for immunoglobulin (Corley *et al.*, 1977). The delay in colostrum feed will therefore allow bacteria to multiply in the gut

of the non immune calf (Corley *et al.*, 1977). Non immune calves are immediately exposed to viruses and bacteria that may be shed by carrier older animals. Isolation of the calf from the adult herd, periodic depopulation of the calf barn and sanitizing the calf nursing area are the measures that can reduce the prevalence of neonatal diseases (Turnbull, 1980).

Moreover, the presence and multiplication of bacteria may reduce absorption by accelerating cell migration along the villi and reduce the length of time from birth that intact Ig can be transferred into the blood (James and Polan, 1978). To prevent bacterial adherence, colostrum must be fed within 6 hours postpartum before establishment of the microflora in the digestive tract (Logan *et al.*, 1977). In the bovine three classes of immunoglobulin (Ig) have been identified, including immunoglobulin G (IgG), immunoglobulin M (IgM) and immunoglobulin A (IgA) (Tizard, 1987). Quantitatively, IgG and IgM are the principle immunoglobulin in bovine serum whereas IgA is the major one in many external secretions and IgG accounts for 85-90% of the total immunoglobulin in both serum and colostrum (Tizard, 1987). It is clear that, the calf that has obtained enough colostrum would absorb IgG in large quantities. Successful absorption of immunoglobulins from colostrum provides protection against enteritis, septicaemia and pneumonia (Penhale *et al.*, 1973). Feeding large volumes of colostrum, has been shown to increase the amount of immunoglobulin in calf's sera (Kruse, 1970). However, if the Ig concentration of colostrum is low, inadequate Ig absorption may

occur regardless of the volume fed (Stott and Fellah, 1983). Calves ingesting 2-3 litres of colostrum during the first meal were found to have low serum immunoglobulins if colostrum concentration of Ig was below 20 mg/ml (Penhale *et al.*, 1973). Colostrum immunoglobulin concentration influences absorption and hence the subsequent serum Ig concentration in the calf.

Following milking there is a rapid fall in the Ig content of the mammary secretions (Porter, 1972). This rapid fall in Ig levels is of important in relation to the feeding of colostrum to neonates. This implies that, neonates are be given the first meal of colostrum immediately after birth because immunoglobulin concentration in colostrum is reduced with subsequent milking (Roy, 1980). Penhale *et al.* (1973) stated that, there is differential absorption between individual classes of immunoglobulins, thus 90% of IgG, 59% IgM and 48% IgA are absorbed.

Penhale *et al.* (1973) reported that, a defect in the absorption mechanism occurs in 10-16% of calves and hence higher Ig concentration of serum in suckled calves was attributable to three factors (Selman *et al.*, 1970). First, in most cases suckling occurs within the first 6 hours postpartum. Secondly, during suckling the amounts of colostrum consumed is usually larger when compared to bucket feeding. Thirdly, calves are rendered far more efficient in absorption of immune lactoglobulin if left with the dam possibly as a result of the prolonged grooming they usually receive. Selman *et al.*

(1970) further described the effect of parity on concentration of immunoglobulin in serum and colostrum of the dam. Concentrations of Ig in colostrum were similar for the first three parities of holstein cows, but older animal had a higher IgG concentration in colostrum (Kruse, 1970). However, calves from dams beyond their eighth parity tended to have lower Ig concentration in their serum (Logan and Gibson, 1975).

The quality of immunoglobulin may be more important than the quantity (Logan and Penhale, 1972). Compared to whole milk, good quality colostrum has high quantities of micronutrient although their concentration might differ from cow to cow. Good quality colostrum therefore should have at least 336 g/kg dry matter, that is, 56 g/kg casein protein, 145 g/kg immunoglobulin fraction, 65 g/kg fat, 21 g/kg lactose, 14 g/kg minerals, 116 mg/kg vitamin A, and 6 mg/kg vitamin B<sub>2</sub> (Roy, 1980). Except for lactose which tends to be high in whole milk, all other ingredients are much higher in colostrum than in whole milk (Fallon and Harte, 1987). It is important therefore that calves are fed colostrum instead of normal milk. Logan and Penhale (1972) demonstrated that, the colostral immunoglobulin apart from conferring humoral immunity also had a local protective action within the small intestine. Thus for the calf's maximum protection, both serum and intestinal immunoglobulin must be present in adequate quantities.

When calves are grouped by immunoglobulin status, the highest mortality rate occurred among those with lower levels (Harold and Hassan, 1984). However, and in some cases, even when the calf is given adequate colostrum, serum immunoglobulin level of any individual calf may not be a reliable guide to its future viability because, there have been incidence where higher mortality was found in calves with high immunoglobulin concentration than immunoglobulin deficient calves (Harold and Hassan, 1984). It has been shown that, calves left with their dams may have lower Immunoglobulin concentrations than those bucket fed (Mitchell *et al.*, 1974). Logan *et al.* (1981) also reported that, calves fed colostrum artificially had substantially higher immune status than their naturally fed counterparts. A defect in absorption of immunoglobulin in some calves and the breed difference in efficiency of colostral immunoglobulin absorption could explain the low immunoglobulin concentration in suckled calves (Kruse, 1970; Logan and Penhale, 1972). Friesian and Jersey calves have been shown to have a higher efficiency of absorption of Ig than have Red Danish calves. Reasons for the difference in colostral Ig absorption is not clear. However, breed difference in efficiency of digestion of post colostral diets may be due to differences of breeds in the amounts of digestive enzymes (Roy, 1980; Ternouth *et al.*, 1976). Ayrshire and Jersey calves appear to be more susceptible to enteric disorder than Friesian (Kruse, 1970). Blue-grey cows had higher concentrates of IgG1 and IgM in their colostrum than did Hereford and Friesian crosses and this was reflected in the serum Ig concentration in their calves (Halliday, 1978). High frequency of hypoglobulinemia is usually interpreted as the result

of late feeding of colostrum, inadequate amount of colostrum given, or the occurrence of an early loss of absorption capability (Kruse, 1970). Reduced mothering ability, difficult calving, weak or malformed calves and badly shaped udders may also affect colostrum ingestion by the calf (Barber, 1976; Logan and Gibson, 1975). Pendulous udders of the dairy cows had a significant delaying effect on the time taken to first suckling and 25% of calves suckled 8 hours postpartum (Selman *et al.*, 1970). Selman *et al.* (1970) also noted that, suckler beef cows possessed a much better mothering instinct than dairy cows and heifers. Selman *et al.* (1970) stated that suckling beef cows pay more attention to their offspring. They would lick their calf for a much longer period. Their calves were able to stand up sooner after birth and most important the cows suckle their calves within 1½ hours after birth, compared to dairy calves which are separated from the dam and given colostrum by bucket 4 hours later. Calves whose serum immunoglobulin concentration is not sufficient to protect the calf against enteric and respiratory diseases during neonatal and postnatal period are therefore likely to suffer from these diseases. Clinical manifestation pertinent to a disease and pathological changes associated with it would depend on the organism involved and whether the calf suckled or fed colostrum by bucket.

### **Pathology**

In diarrhoea caused by enterotoxigenic *Escherichia coli* (ETEC) the tail become soiled with faeces. The colour of faeces seems to vary in different outbreaks of the disease

(Roy, 1990). In some it is yellow and watery, whereas in others it is white and pasty. In succeeding days appetite becomes variable and the calf becomes dehydrated with sinking of eyes being the most obvious symptom. If no treatment is given the calf becomes prostrated and hypothermic (Tennant *et al.*, 1978).

Coronavirus infect both the small and large intestine but infection in the small intestine is in the ileum rather than in the anterior small intestine (Mebus, 1976)). In this case diarrhoea is acute and faeces may be soft in consistency and may contain coagulated milk or mucus. Diarrhoea associated with rotavirus infection varies from inapparent infection and mild diarrhoea to severe profuse watery diarrhoea with yellow coloured faeces and severe dehydration (Mebus *et al.*, 1971). In both cases diarrhoea is a result of villous atrophy in small intestinal mucosa and consequent malabsorption and loss of intestinal enzymatic activity (Mebus *et al.*, 1975).

The pathogenesis of diarrhoea varies with the causative agent involved (Mebus, 1976). Secretory diarrhoea results from derangement of normal secretory and absorptive mechanism in the small intestine, thus, an excess of secretion over absorption of fluid in the small intestine (Jubb *et al.*, 1992). In surface enterocytes the heat labile toxin produced by *Escherichia coli* K 99 act through the mediation of cyclic adenosine monophosphate (cAMP). This enterotoxin shuts down sodium chloride co-transport at the luminal cell membrane, thus reducing passive water absorption. Concomitantly, in the crypt epithelium, cAMP stimulated chloride secretion is promoted, and water follows

(Jubb *et al.*, 1992). This hypersecretion is a net intestinal flux of fluid and electrolytes occurring independent of changes in permeability, absorptive capacity or exogenously generated osmotic gradients (Moon *et al.*, 1982).

Kennedy *et al.* (1984) could not find discernible morphological changes to the enterocytes although the mechanism of diarrhoea due to *Escherichia coli* K 99 enterotoxin was by altering the normal secretory mechanism of the surface enterocyte (Rose *et al.*, 1987). However, Whipp *et al.* (1986) confirmed villous atrophy resulting from the heat stable enterotoxin b of *Escherichia coli* infection in pigs. Malabsorptive diarrhoea commonly result from villous atrophy of any cause. Rotavirus cause the villi of the small intestine to be shortened and thickened with tall columnar epithelium replaced by a short cuboidal form (Mebus, 1976). Stunting and fusion of the villi, exfoliations, disarrangement and vacuolation of enterocytes was observed in rotavirus infected calves (Reynolds *et al.*, 1986). Coronavirus seems to affect the villi in a manner similar to rotavirus (Mebus, 1976). However, coronavirus produces more severe lesions than rotavirus with lesions extending from the small intestine to the colon (Mebus *et al.*, 1969). Electrolyte and nutrient solutes malabsorbed as the result of reduced villous and microvillous surface area are retained in the lumen of the bowel, then water follows osmotically.

As in ostertagiasis effusive diarrhoea has been associated with lymphangiectasia,

inflamed lamina propria, increased vascular permeability, propria oedema and enteric plasma protein loss (Jubb *et al.*, 1992; Nielsen, 1976). Increased permeability of the mucosa permits increased retrograde movement of solute and fluid from the lateral intercellular space to the lumen or by facilitating transportation of the tissue fluid. Increased exfoliation of epithelium and transient micro erosions may provide further potential sites for effusions of interstitial fluid (Symons, 1976).

#### **Prophylaxis and treatment**

Antibiotics are frequently used in the treatment of the diarrhoeic calf. Antibiotic resistance is very high in *E. coli* (Hinton and Linton, 1983). To enhance the value of medication with antibiotics, bacterial sensitivity tests of the organism isolated against various antibiotics may be necessary to ascertain the best antibiotic to use. Indiscriminate use of antibiotics only lead to selection in favour of resistant strains and complicates future therapeutic efforts (Hinton and Linton, 1983). Most other causes of diarrhoea in calves are viral or parasitic and are therefore not sensitive to antibiotics. Systemic antimicrobial therapy might have some benefit in diarrhoeic calves and survival is enhanced partly by controlling intercurrent infections. Certain sulphur drugs and antibiotics have been used to good effects (Hinton and Linton, 1983). It has been indicated that colostrum is important in preventing morbidity and mortality from calf scours and that, local enteric immunity is important (Boyd *et al.*, 1974; Woode *et al.* 1975).

Hyperimmunization of pregnant cows with *Escherichia coli* K 99 antigen has been used to produce colostrum with augmented protection against *E. coli* infections (Nagy, 1980). Since most enteric infections occur in calves less than 3 weeks of age passive lactogenic immunity within the gut plays an important role in protection of susceptible intestinal cells (Saif and Smith, 1985). New born calves inoculated orally with attenuated bovine rotavirus vaccine responded with local IgA production (Saif and Smith, 1985). Hyperimmune colostrum whey from pregnant cows vaccinated with rotavirus have been used to protect calves from rotavirus infection (Castrucci *et al.*, 1988). It has however been shown that, colostrum rotavirus antibody titre decrease dramatically in the transition to milk (Castrucci *et al.*, 1984; Saif and Smith, 1985). The beneficial effect in calves would therefore depend on the continued feeding of immune colostrum to calves throughout the period of greatest risk to neonatal diseases (Castrucci *et al.*, 1984). Acidified milk (pH 4.3-5.5) and fermented colostrum reduce the incidence of diarrhoea and that fermentation of mastitic milk is a method of utilizing this milk for calf feeding without causing health problems and thus the reduced incidence of diarrhoea is related to reduction in bacterial challenge on the calf digestive system, the improved clotting time and improved activity of pepsinogen in the acid environment (Fallon and Harte, 1987). The intestinal loss of fluid and electrolytes may involve hypersecretion, malabsorption or both. Oral rehydration is particularly used under practical conditions and is effective whether diarrhoea is due to bacteria or viruses (Saif and Smith, 1985). The principle of rehydration is that if salt and water are presented slowly enough to the

intestine then absorption should be complete (Bywater, 1977). Rotavirus infected calves often have a severe metabolic acidosis which persist even after intravenous infusion of sodium bicarbonate solutions thus, oral fluid therapy must be given to a diarrhoeic calf even after signs of acidosis have disappeared (Fallon and Harte, 1987).

Roy (1990) suggests that, a calf rearer in the farm can take either of the following two measures:

1. At the first onset of diarrhoea, warm water only or warm water containing 9 gram sodium chloride /litre should be fed for 24 hours at the rate of 1.1 kg/feed, 3 times a day. During this period no other dry or liquid feed should be given. For the following 24 hours 1.7 kg milk with 2.6 kg water should be given and for the next 24 hours feed 2.6 kg milk with 1.7 kg water ought to be given in each case in 3 feeds a day. If the calf recovers, undiluted milk may then be offered.

2. Starve the calf for one feed and then reduce the daily ration of milk to 1.8 kg/feed. This amount is considered enough for maintenance of body weight in a 25 kg calf.

### **2.3.2 Calf Pneumonia**

This is an infectious pulmonary disease of calves or young cattle housed together in groups either indoors or in yards. It is primarily a problem in calves 2-6 months of age although pneumonia may occur in groups of young cattle up to one year old. The morbidity and mortality vary considerably depending on the conditions of housing, the quality of management and the kind and number of viruses and bacteria which

predominate at any one time but severity of mortality may be associated with the degree of secondary bacterial invasion (Blood *et al.*, 1990; Roy, 1990).

Initial infections in the respiratory tract occur at specific sites resulting from interaction between a complex and effective mechanism and the characteristic of the organism involved. For Pasteurella, Mycoplasma, Parainfluenza 3 and infectious bovine rhinotracheitis viruses, the main site of infection is the upper respiratory tract whereas for bovine respiratory syncytial virus and adenovirus it is the lower respiratory tract (Kahrs, 1981).

#### **Predisposing factors**

The role of antibody in the protection of calves from enzootic pneumonia has been described by Corbeil *et al.* (1984). He associated the peak incidence of enzootic pneumonia with the time when serum immunoglobulin levels are at their lowest. This is between 2-4 weeks of age. Early outbreaks of pneumonia do not affect calves younger than one month of age (Corbeil *et al.*, 1984). Calves are probably protected during this time by the passive immunity received from their dams. Whether the protection afforded is to the secondary invaders or to the virus or both, is not clear. Pneumonia frequently follows on attacks of scours (Curtis and Drummond, 1982). Poor hygiene, poor housing, muddy wet conditions, faulty feeding, deficiency of vitamin A and inadequate early supply of colostrum to the calf are all potent predisposing factors (Selman *et al.*, 1970).

Crowding results into close contact and promote spread of infection. Crowding also result in excess moisture which in the presence of inadequate ventilation and heat causes a high relative humidity and chilling of calves (Pritchard *et al.*, 1981). Increased relative humidity, condensation of moisture on walls and on calves lead to wet conditions. Reduced ventilation results in an increase in the concentration of droplet infection (Pritchard *et al.*, 1981). Sudden changes in climate may affect the colonization of the respiratory tract by microorganisms partly as a result of increased pathogen survival and partly as a result of reduced host resistance (Webster, 1981).

Air borne dust in animal houses predisposes the animals to respiratory infections. Dust arises from three main sources including the animals themselves, in the form of skin squame, bedding or litter and feed (Wathes *et al.*, 1983). Exposure to dust has been associated with pneumoconiosis, allergies and toxic effects (Wathes *et al.*, 1983). Chronic exposure to carbon dioxide affects dietary functions, clearance of bacteria by alveolar macrophage and bactericidal properties of the lungs and has been shown to stimulate or depress the human immune system (Wright, 1966). Curtis and Drummond (1982) stated that, even minor structural damage to the epithelium of the upper respiratory tract may lead to severe functional impairment and thus increased incidence and severity of lung diseases. Calf pneumonia has been shown to be caused by different aetiological agents. Some mycoplasmas and viruses are thought to act as primary agents, with bacteria as secondary invaders exacerbating the initial infection. These bacteria are

principally *Escherichia coli*, *Pasteurella* spp, *Staphylococcus* spp, *Corynebacteria* spp and *Pseudomonas* spp (Baker and Frey, 1985). Mycoplasmas isolated from pneumonic lungs of fatal cases are at times considered as secondary invaders opportunistically infecting tissues previously injured by viral infections, parasites, allergic reactions or inhalation of foreign material.

The role of viral infections in calf pneumonia is uncertain (Dawson *et al.*, 1964). However, Lopez *et al.* (1988) associated Parainfluenza 3 (PI3) with interference of normal pulmonary clearance mechanism thereby allowing invasion of bacteria and mycoplasmas. Curtis and Drummond (1982) described enzootic pneumonia of calves and listed Chlamydia, Parainfluenza 3 virus, reoviruses, rhinoviruses, adenoviruses and mycoplasmas as potential causes. Curtis and Drummond (1982) stated that, predisposing factors particularly overcrowding and inadequate ventilation in respect to the temperature and humidity are even more important than the aetiological agents. The disease spreads from calf to calf with greatest of ease when they are kept under unsuitable conditions. Frequently infection occurs through the umbilicus at birth, leading to septicaemia and later to pneumonia (Curtis and Drummond, 1982).

Acute primary pneumonia has been associated with adenoviruses, respiratory syncytial viruses, Parainfluenza-3 virus and other viruses (Kahrs, 1981). Bovine adenoviruses (BAV) infections are frequently inapparent, but in calves they can cause pneumonia,

enteritis, and conjunctivitis and weak calf syndrome (Cole, 1970). He pointed out stress factor to be associated with some but not all of outbreaks. The virus is present in nasal and lacrimal excretions and is transmitted by direct calf to calf contact. Raising calves in close proximity to adult cattle may result in constant exposure to infectious agents to which the mature cattle are immune. Age at weaning appear to affect susceptibility of calves to respiratory infections. Calves weaned early at the age of 5 weeks were more affected by pneumonia than those weaned at 8-12 weeks of age (Roy, 1990).

Clinical symptoms of respiratory disease are variable, but there is usually a nasal discharge. The discharge is sometimes thin and watery, but may be thick and purulent at times. A slight discharge may also occur from the eyes. In most outbreaks, there is a dry cough which often persist after the calf has recovered and is particularly noticeable on exercise. Lesions of variable extend usually occur in the lungs especially in the apical lobes (Kahrs, 1981).

Pneumonia associated with bovine adenovirus (BAV) infection is more common in neonatal than adult cows (Mattson, 1973). Bovine respiratory syncytial virus (BRSV) infections can be inapparent but are associated frequently with acute, rapidly spreading respiratory disease characterized by fever, nasal discharge, cough and sometimes pneumonia, which may be the result of secondary infections (Inaba *et al.*, 1972). Marshall and Frank (1975) summarized the gross respiratory lesions reported after experimental infections of (PI3) virus as congestion of respiratory mucosa, consolidation

of ventral portions of the lungs and enlargement of the bronchial and retropharyngeal lymphnodes.

### **Treatment and prevention**

Management of the calves is major means of preventing the disease in calves. It has been shown that, the problem of pneumonia in young housed calves was overcome by ensuring an adequate intake of colostrum and avoidance of nutritional stress, the mixing of groups of calves, poor ventilation, dust and infection with lung worm (Roy, 1980). In view of the variety of organisms involved in respiratory disease, vaccination should be considered only where specific agents have been identified.

The use of antibiotics for prophylaxis is more effective than their use for therapy of a clinical disease. Treatment is largely aimed at reducing the effect of secondary bacterial invasion. Mass medication of purchased calves against shipping fever-bovine respiratory disease complex by intramuscular injection of oxytetracycline for three successive days followed by oral sulphadimethoxine resulted in 81% percent reduction in "treatment days" per calf purchased (Roy, 1980).

Long acting oxytetracycline preparation given intramuscularly to beef bulls at performance testing at about 6 months of age tended to reduce the risk of respiratory infection, but the effect was not significant (Peters, 1986). Since dyspnoea and

hyperpnoea rapidly lead to dehydration and the calf may not take water voluntarily administration of 2 liters fluid intravenously can help (Roy, 1980).

Selenium has been found to have a preventive effect against diarrhoea and pneumonia (Phillippo *et al.*, 1987). However, Hall *et al.* (1988) disputed the effect of selenium treatment on pneumonia in housed calves. Nevertheless, when selenium as barium selenite was given to suckler cows, no antibiotic treatment was needed to the calves (Hall *et al.*, 1988).

### 2.3.3 Parasitic gastroenteritis

Gastrointestinal helminthosis is an important disease affecting small ruminants production in Tanzania (Njau, 1987). These parasites are also important in cattle in many tropical and subtropical environments (Hansen and Perry, 1994). *Haemonchus contortus*, *Oesophagostomum columbianum*, *Cooperia spp*, *Trichostrongylus spp*, and *Trichuris spp* were the predominant species isolated in an autopsy survey conducted at Sokoine University of Agriculture farms (Ngomuo *et al.*, 1994).

The same parasites are predominant helminths of cattle in Oregon (Rickard and Zimmermann, 1992), Argentina (Suarez *et al.*, 1992) and Nigeria (Fakae, 1990). *Haemonchus spp* and *Trichostrongylus axei* infests the abomasum, *Cooperia spp*, *Bunostomum*, *Strongyloides*, *Nematodirus*, *Trichostrongylus* infests the small intestine. *Paramphistomum* larvae reside in the small intestine, whereas *Trichuris* and

Oesophagostomum infests the large intestine of domestic ruminants (Campos *et al.*, 1990). *Toxocara vitulorum* was the commonest parasite in the age group of 0-60 days (Pradhan *et al.*, 1991; Taira and Fujita, 1991). Pradhan *et al.* (1991) associated the high incidence in the younger group with prenatal infection due to poor hygienic condition. *Toxocara vitulorum* eggs were found in calves faeces of as early as two days old and the maximum patency occurred after two months (Agyei, 1991; Chandrawathani and Sani, 1989). *T. vitulorum* is an important cause of mortality in buffalo calves in India and are acquired by the calf by transfer of larvae in colostrum and buffalo calves may become a source of infection to dairy or beef calves (Chandrawathani and Sani, 1989).

### **Epidemiology**

Incidence of parasitic diseases vary greatly between areas depending on the relative nutritional states and specific nutritional deficiencies, pasture management, barn management, climate and host immunity (Blood *et al.*, 1990). A dietary deficiency of specific nutrients such as copper, cobalt, phosphorus or protein can lead to a reduction of animal's body resistance similar to general malnutrition. On the contrary, *Haemonchus contortus* develops better in sheep fed cobalt supplement than in sheep on cobalt deficient diet (Blood *et al.*, 1990). This observation suggests that, there may be some advantages related to dietary requirements of the helminths themselves.

Heavy stocking rate in areas of improved pastures result into faecal contamination especially when animals of different ages are grazed together. Greater length and bulk

of pasture protect helminth eggs and larvae from sunlight and desiccation. Heavy stocking rate therefore, forces animals to graze on contaminated pastures that they normally avoid. On extensive range, especially in dry season, pasture contamination occurs around water sources.

In intensively kept animals faecal contamination of feed can occur when animals are fed on the floor or in very low troughs or the animals are overcrowded (Blood *et al.*, 1990). Inadequate nutrition is a constant hazard in animals housed for long periods. Young animals are more susceptible to the effects of helminths infestation.

Development and survival of infective larvae in the environment depends upon warm adequate moisture (Soulsby, 1982). In tropical and subtropical countries, temperatures are permanently favourable for larval development in the environment. The ideal temperature for larval development of many species in the microclimate of the tuft of grass or vegetation is between 22 - 26°C, and ideal humidity of 85% to 100% (Hansen and Perry, 1994).

### **Pathology**

The ways in which the parasite exert their effect upon the host are manifold (Nielsen, 1976). These include blood sucking, tissue destruction during host migration or feeding, mechanical or chemical irritation of contact surfaces, liberation of toxic metabolites, obstruction of excretory ducts, air passages and blood vessels (Jubb *et al.*, 1992). Structural alterations caused by the parasites depend not only upon the mass of

infestation but also environmental and managemental factors including the level of nutrition, climate and grazing procedures (Nielsen, 1976). In heavy infestation severe damage of the internal lining of the gut occur (Sykes, 1982). Plasma proteins may therefore leak back into the gut and get lost either in faeces or urine.

Pathological changes due to parasitic gastroenteritis depend on the predilection site of the parasite. There are five groups of parasites depending on their localization in the digestive tract (Jubb *et al.*, 1992), these groups include:

1. A group of helminths residing freely in the intestinal lumen of the intestine for example *Moniezia spp* whose members are generally of low pathogenicity except in massive infestation when they cause obstruction.
2. A group of helminths known for causing physical trauma to the intestinal wall by burrowing or inciting inflammatory foci in the submucosa or deeper layers e.g *Oesophagostomum spp*. Protein loss may occur from ulcerated areas or when larvae emerge from submucosa.
3. A group which includes *Haemonchus spp*, *Bunostomum* cause blood loss by feeding on mucosa, causing bleeding or actively sucking blood. Anaemia and hypoproteinemia and their sequelae cause production loss, clinical disease and death.
4. A group exemplified by *Ostertagia spp* and *Trichostrongylus axei* causing protein losing gastroenteropathy usually associated with inappetence and diarrhoea. In addition mucus metaplasia and hyperplasia of gastric glands occur in the abomasum.

5. A group of helminths including *Cooperia spp*, *Nematodirus spp*, *Trichostrongylus spp* and paramphistomum larvae causing immune mediated villous atrophy (Jubb *et al.*, 1992). Nodular worm of sheep *Oesophagostomum columbianum* cause an immune mediated colonic function which result into mucus secretion and diarrhoea whereas, *Trichuris spp* cause mucosal typhlocolitis which may be related to immune mediated inflammatory responses to the worm (Jubb *et al.*, 1992).

Malabsorption of nutrients, electrolytes and water occur with the associated loss of endogenous protein into the gut. Erosions as a result of heavy infestations cause loss of absorptive function and effusion of tissue fluids or hemorrhagic exudate. Animals infected with such parasites may die but reduction of intake and inefficient utilization of food lead to poor performance and poor carcass quality in survivors (Sykes, 1982); Holmes, 1985). Sykes (1982) revealed that, gastrointestinal nematodes can cause reduction in feed intake, nitrogen and mineral retention. However, in many infections of the gastrointestinal tract the lesions particularly of the mucosa are obvious (Symons, 1976). Malabsorption occurs when lesions are preponderant in the duodenum or jejunum where a large part of digestion and absorption of important constituents of the diet take place. The degree of malabsorption is related to the severity of the infestation and that malabsorption in early infection was associated with a loss of absorptive area as a result of flattening and fusion of villi caused by *Trichinella* infection in man (Symons, 1976). Symons (1976) further suggests that, derangement of enzymatic activity might account

for malabsorption. Although enzyme deficiency may affect digestion and absorption at the site of infection, there is no unequivocal evidence that they are a major cause of malabsorption (Symons, 1976).

Gastrointestinal parasitism is associated with a range of clinical signs (Holmes, 1985). Infections with gastrointestinal nematodes usually involves several different species of parasites which may have an additive pathogenic effect on the host. In subclinical infections, obvious signs like diarrhoea may be absent (Holmes, 1985).

Clinical signs in mixed infections of *Haemonchus*, *Ostertagia*, *Cooperia*, *Bunostomum*, *Oesophagostomum*, *Trichuris*, *Trichostrongylus* are weight loss, reduced feed intake, diarrhoea, mortality and reduced carcass quality (Hansen and Perry, 1994). Severe blood and protein loss into the abomasum and intestine due to damage caused by *Haemonchus*, *Bunostomum* and *Oesophagostomum* often result into bottle jaw (Blood *et al.*, 1990) and young animals are most susceptible. Pain, discomfort and alterations in the rate of passage of ingesta could be associated with changes in voluntary feed intake and hence loss of weight (Sykes, 1982). Infected calves had significant decrease in packed cell volume, haemoglobin, total serum protein, globulins and albumin (Pradhan *et al.*, 1991).

### Treatment and control

Overstocking is a major problem in larger part of the world particularly in Africa outside the tsetse infested areas (Murray and Gray, 1984), where soil erosion and pasture degradation forces the animal to graze closer to faecal material which inevitably result in the uptake of higher numbers of infective larvae (Donald *et al.*, 1978). Improving grazing management and introducing the safe pasture concept can reduce the use of anthelmintic resistance (Donald *et al.*, 1978). Suppressive treatment is meant to reduce markedly the parasite numbers by removing more susceptible species of the parasites in the gut, thereby reducing the total parasite population to a size below what is necessary to produce clinical parasitism (Anderson *et al.*, 1976). Due to susceptibility of young stock to parasitism, strategic treatments should be instituted such that they provide maximum protection until weaning and at weaning when calves suffer their greatest nutritional stress (Herd *et al.*, 1984). Strategic treatments are usually administered two to four times a year depending on climate and management procedures (Herd *et al.*, 1984).

#### 2.3.4 Coccidiosis

Coccidiosis is caused by intestinal infection with *Eimeria spp* and *Isospora spp*. The two protozoan parasites differ mainly in the details of development within the oocyst and the free living stages of the parasites (Levine, 1973). The disease has been reported from

both vertebrates and invertebrates, including domestic animals, game and birds ((Fitzgerald, 1972; Fox, 1978; Reid, 1985; Vassiliades, 1969). It is primarily a disease of young non immune animals crowded together in unsanitary housing lots particularly in larger concentrated operations (Fox, 1978). Under these conditions the environment is highly contaminated with oocysts and immunity may be compromised by stress related factors. Often outbreaks are associated with the stress of shipping, weaning, and dietary changes. Although coccidiosis is considered a disease of confinement, outbreaks have been reported in range animals (Radostits and Stockdale, 1980). Nonetheless, the disease is prevalent in lambs or beef feedlots and in goats raised on dry lot (Fitzgerald, 1972)). *Coccidia* causes serious economic losses in a variety of animal species than any other protozoa (Fitzgerald, 1972; Fox, 1978; Reid, 1985). Losses due to coccidiosis include mortality, reduced weight gain, unthriftiness, weakness and costs involved in treatment and control of the diseases (Vassiliades, 1969).

The prevalence of 35% of coccidiosis was observed in calves in selected farms in Morogoro (Chibunda, 1995). *Eimeria bovis* and *E. zuernii* had higher prevalence rates of 68% and 57% respectively than other species. Schrag (1968) and Ruiz (1973b) reported 72% prevalence in South and East Africa. Ruiz (1973a) reported a prevalence of 67% in South Dakota, USA. In Mexican cattle Skandar (1973) reported a prevalence rate of 81%. It seems therefore that, the prevalence of coccidiosis is influenced by geographical and climatic variation and management.

Occasionally, adult animals immune to their own endemic species of coccidia develop disease when they are moved to a location with different species of parasites (Radostits and Stockdale, 1980). High infection rates were found in the rainy season than in the dry season ( Mwasomola, 1983; Waruiru and Mbutia, 1991). Severe coccidia infection has been associated with mixed infection with helminthiasis and Clostridial infections (Hateren *et al.*, 1985; Mwasomola, 1983). Although most animals are exposed and infected with coccidia, usually the infection is asymptomatic and self limiting. Cattle of all age groups can be infected (Marquardt, 1962) but often, young animals exhibit severe clinical signs (Levine, 1973). Older animals are important symptomless carriers which continue to excrete oocysts, thereby becoming sources of infection to the young stock (Levine, 1973).

### **Epidemiology**

The two major pathogenic species in cattle are *Eimeria bovis* and *Eimeria zuernii* (Mage *et al.*, 1990), but *Eimeria auburnensis* may at times contribute to general clinical picture (Soulsby, 1982). In Malaysia the most common parasite in calves was *Eimeria* which was present in 100% of the calves especially during the first six months of age (Chandrawathani and Sani, 1989). *Eimeria ellipsoidalis* was found in only two calves in the group whereas, *Eimeria bovis* and *Eimeria zuernii* was identified in 88% of the cases (Chandrawathani and Sani, 1989).

Mwasomola (1983) reported that 8 species of *Eimeria* prevalent in calves at Morogoro. *Eimeria bovis* and *Eimeria zuernii* were frequent isolates in diarrhoeic and non diarrhoeic calves (Chibunda, 1995; Matovelo, personal communication, 1996; Mwasomola, 1983). Other species isolated in Morogoro include, *E. cylindrica*, *E. subspherica*, *E. canadensis*, *E. ellipsoidalis*, *E. pellita*, *E. bukidnodensis* and *E. wyomingensis* and *E. alabamensis* and *E. auburnensis* (Chibunda, 1995; Mwasomola, 1983). Diarrhoeic calves had more counts of coccidia oocyst than apparently healthy ones. Mixed infections involving 2 or 3 species was common (Chibunda, 1995; Mwasomola, 1983).

### Pathogenesis

Following ingestion of the infective stage, oocyst are liberated and penetrate the epithelial cells of the intestine where they undergo a rather complicated cycle of rapid growth and reproduction (Levine, 1980). The amount of damage done to the intestinal epithelium is directly related to the number of oocyst ingested and the host immunity to the parasite (Jensen and Miner, 1976). A low level of oocyst ingestion may result in subclinical infection that would not be diagnosed as coccidiosis but could have subtle adverse effect on the animal (Marquardt, 1962). If the number of ingested oocyst is low, non immune healthy animals may tolerate infection and demonstrate little or inapparent clinical signs (Lipova, 1985). If a non immune animal is exposed to many oocyst, widespread rupture and exfoliation of intestinal cells alters gut function and allows

secondary bacterial invasion. Intestinal cells are destroyed but since they are normally replaced at high rate, the damage done to the gut is minimal (Fitzgerald, 1975).

Two weeks after a heavy infection with oocyst most epithelial cells at the base of the intestinal glands are occupied by gametocyte. As these rupture damage is severe and causes loss of blood, fluid and electrolytes into the gut. Fitzgerald (1975) reported that, the infection by *E. bovis*, caused a decrease in serum albumin and total protein three weeks after infection in calves. In mild infections calves may show signs of diarrhoea, weakness and loss of appetite (Svensson, 1993).

Clinical signs of coccidiosis caused by various species are similar in all animal species (Levine, 1973), but clinical and pathological sign in *E. zuernii* infection are severe because it is a more pathogenic Eimeria specie (Soulsby, 1982). In cattle the clinical signs of coccidiosis often appear after 3-8 weeks post infection (Levine, 1973). Marked diarrhoea with stringy masses of mucus and clotted blood occur (Levine, 1973). This is often accompanied by loss of appetite, general weakness, dehydration and loss of vigour (Fox, 1978). In the weakened state, the animal become subjected to secondary diseases such as pneumonia, bacterial enteritis and viral infections.

The destruction of epithelial cells and subsequent loss of albumin, fluid and electrolyte typically cause a profuse diarrhoea containing mucus and blood. Despite the blood loss, anaemia is not usually clear in acute cases. Animals typically strain to defecate and

rectal prolapse may occur. Animals become anorectic and feed consumption may be decreased for extended periods of time. In the young, coccidiosis is associated with poor weight gain, weakness and unthriftiness. After recovery from the disease, the gut does not return to normal function for several weeks and appetite may be suppressed (Schillhorn, 1986). Severely affected calves which survive the acute phase of the disease do not regain body weight unless they are fed properly (Fitzgerald and Mansfield, 1972). This suggest that, bovine coccidia have a marked effect on growth performance. In calves and weaned beef cattle, neurological signs have been reported during outbreaks of coccidiosis (Fanelli, 1983). A heat labile neurotoxin in the serum of calves with nervous coccidiosis has been identified but its significance is unknown (Blood *et al.*, 1990). The pathology of coccidiosis depend on the specie of the parasite (Jubb *et al.*, 1992). The colon, ileum and caecum are areas mostly affected in *Eimeria bovis* infection (Levine, 1973). Initially the mucosa becomes congested and edematous. Large amount of blood occur in the lumen of the intestine after destruction of the mucosa and submucosa. In *Eimeria zuernii* infections, the carcass is anaemic, dehydrated and emaciated (Lapage, 1968). According to Levine (1980), the small intestine, caecum and colon may be filled with semi fluid bloody material. The mucosa is thickened and thrown into irregular ridges in the large intestine. Diffuse haemorrhages occur in acute cases but later, desquamation and ulceration occur (Jubb *et al.*, 1992).

### Treatment and control

Coccidiosis is a self limiting disease and clinical signs subside spontaneously when the multiplication stage of the parasite has passed (Blood *et al.*, 1990). In an outbreak, the clinically affected animals should be isolated and treated. Overcrowding is a common feature in epidemics of coccidiosis. All feed and water troughs should be high enough off the ground to avoid faecal contamination (Gregory *et al.*, 1982). Mass medication of feed and water may be indicated in an attempt to abort an outbreak and to minimize the rate of development of new cases (Schillhorn, 1986). Cattle affected with coccidiosis associated with nervous signs should be brought indoors, kept well bedded and warm (Blood *et al.*, 1990).

A variety of drugs have been used to prevent or treat coccidiosis (Baker and Frey, 1985). Sulphamethazine (140 mg/kg) is used widely for the treatment of the acute clinical coccidiosis in calves (Schillhorn, 1982). Amprolium is also used for treatment with an added advantage of weight gain in animals (Gregory *et al.*, 1982). Other anticoccidials include Nitrofurazone (15 mg/Kg) in feed or water, Monensin (33 g/tonne of feed) and Lasalocid (Gregory *et al.*, 1982). These drugs are most often administered in water or feed. However the efficacy of treatment of a clinical disease is limited by several factors. First, treatment of animals maintained in a contaminated environment may be insufficient to prevent subsequent outbreaks. Secondly, most products have only partial efficacy against ruminant coccidia (Gregory *et al.*, 1982). Thirdly, food or water

may not be ingested by the sick animal especially when anorexia is a feature. Finally many drugs are effective against later developmental stages of the parasite and are less effective in preventing the pathologic changes that lead to disease. The best strategies in preventing coccidiosis are therefore managerial rather than treatment (Gregory *et al.*, 1982). These strategic preventive measures are directed at decreasing exposure to oocyst. This is accomplished by decreasing stocking rates, minimizing stress and providing clean housing and feed.

#### **2.3.5 Indigestion**

Simple indigestion is clinically characterized by inappetence, decreased ruminal movements and abnormal faeces (Blood *et al.*, 1990). Faeces may be scanty or voluminous and associated with diarrhoea. The disease is usually associated with some dietary abnormality particularly in intensive management of dairy cattle and stall fed beef cattle because of variability in quality and the large amounts of food consumed. Indigestion in calves has been reported recently in Tanzania and rumenitis is interpreted as a sequel to rumen acidosis especially in animals exposed to concentrates ad libitum (Matovelo, personal communication, 1996). Affected calves may develop severe forestomach lesions associated with chronic rumen acidosis (Mgassa, 1991). The most common lesions in acidotic cases are extensive branching with clamping of papillae, hyperkeratosis, acanthosis, necrosis and ulceration of epithelium (Mgassa, 1991).

Indigestion was reported in calves fed ground barley (Landsverk, 1978). Hyperplasia of secondary papillae and epithelia layers and microabscess associated with penetrating hairs were frequent lesions (Landsverk, 1978).

### 2.3.6 Tickborne diseases

Tickborne diseases (TBDs) including theileriosis, babesiosis, anaplasmosis and cowdriosis are recognized as major constraints to livestock production on the African continent (Mukhebi *et al.*, 1992). East Coast fever (ECF) or bovine theileriosis caused by *Theileria parva parva* (Uilenberg *et al.*, 1987) continues to be of significant economic importance in endemic areas (Mukhebi *et al.*, 1992), especially because of the high mortality, productivity losses on recovery, costs of control, and exclusion of Taurine (*Bos taurus*) and Taurine cross cattle from endemic regions due to their high susceptibility (Young *et al.*, 1973). Despite being a virulent pathogen, there are situations where *T. parva parva* infections cause minimal losses which are usually confined to calves due to endemic stability ((Moll *et al.*, 1986).

Factors associated with endemic stability include, innate resistance of zebu cattle to the effects of *T. parva* in endemic areas and possible protection afforded in calves by passively acquired antibodies from colostrum (Mbassa, 1994). However, innate resistance to ECF has been questionable (Mbassa, personal communication, 1996). Maternally acquired antibody levels decline exponentially and the duration depends on

the age of the calf (Mbassa, 1994). This was a function of vector-calf interaction, antibodies conferred by the dam which in turn are based on the parasite dose and genetic breed resistance (Mbassa, 1994).

Other variables contributing to endemic stability include seasonality and abundance of ticks, host breed and age susceptibility to tick and pathogen, acquired immunity to tick and pathogen which is further related to level of parasitemia (Yeoman, 1966).

Animals which recover spontaneously from the disease are solidly immune to homologous challenge and it has been shown that this protection lasts for up to 3½ years in the absence of challenge (Burrige *et al.*, 1975). Losses due to ECF are low in areas where animals were intensively dipped weekly using acaricide (Mbassa and Silayo, 1995). Mbassa and Silayo (1995) observed that, in areas where no acaricide were used, there was a vector and parasite population build up resulting in massive infections which overcome immunity.

### **2.3.7 Bovine Parasitic otitis**

This is a chronic or occasionally acute ear infection of cattle clinically manifested by foul smelling aural discharges, swelling o the base of the affected ear and its regional lymphnodes, constant ear shaking in early stages and droopy ears in later stages (Msolla

*et al.*, 1986). Chronically affected animals becomes deaf due to occlusion of the auditory meatus and the involvement of the brain cause circling, stiff neck, recumbency and death (Msolla *et al.*, 1986). The causative agent is a saprophytic nematode known as *Rhabditis bovis* (Kreiss, 1964, Cited by Msolla *et al.*, 1985). All ages and breeds of cattle are affected but severe cases are seen in immature zebu cattle due to a tuft of hair in the ear which allow growth and multiplication of the nematode (Msolla *et al.*, 1986). *Musca domestica* and dip tanks have been shown to play a part in the transmission of the disease (Jibbo, 1966; Msolla *et al.*, 1986). The habit of cattle scratching their ears using hooves may contribute to the transfer of the nematode from soil and manure to the ears (Jibbo, 1966).

### **2.3.8 Dermatophytosis**

In ruminants dermatophytosis refer to infection of the skin and hair by *Microsporum* and *Trichophyton* species (Scott, 1988). Transmission of the disease is usually by direct contact between animals or indirectly through fomites such as grooming instruments, housing, fence and feed bunks (Fadok, 1984). Young animals are susceptible to infection due to lack of prior exposure which ususally confer immunity (Fadok, 1984). Poor nutrition, concurrent diseases, crowding and hot humid climate predispose animals to infection (Scott, 1988).

### **3.0 MATERIALS AND METHODS**

#### **3.1 Study design**

The study was divided into two parts;

(a) A study on disease prevalence and mortality rates of calves in different management systems. In this study a total of 323 calves from Kingolwira Prisons Farm, Mlali Farm and Azam Estate were covered. The Kingolwira Prisons farm practises semintensive system, Azam estate practises an intensive system, where as, Mlali farm practises free range management system. These farms were selected on the basis of differing management systems, number of cattle in the farm and access to the farm by road.

(b) A study on calf management and its association with diarrhoea. In this study 46 calves were registered at Magadu dairy unit and SUA farms. Eighteen calves were born at Magadu Dairy Unit (MDU) in the dry season (June through December 1995). The Magadu dairy farm practises an intensive management system. Also during the dry season 24 calves were born at the Sokoine University of Agriculture (SUA) farm. The SUA farm practises semi intensive management system. Four more calves were born at Magadu dairy Unit in the rainy season (February to April 1996) but no calf was born at SUA farm during the same rainy season. A total of 28 faecal samples from diarrhoeic calves were obtained. Five samples were from Magadu dairy unit, 6 from SUA farm, 6 from KPF, 9 from Azam estate and 3 from Mlali farm.

## **3.2 Farms, breeds and management**

### **3.2.1 Kingolwira Prisons Farm (KPF)**

KPF is 22 km East of Morogoro off Morogoro-Dar-es-salaam road. During the study the farm had 460 cattle. The herd was of crosses of Ayrshire and Friesian breeds with Tanzania shorthorn zebu (TSZ). Adult cattle and weaners were taken to pasture during the day. At night adult cattle were kept in a paddock, while weaner calves were kept in a communal pen. The floor of the weaners' house was made of earth. Young calves were allowed to suckle colostrum from the dam for 5-7 days postpartum. After colostrum period, calves were given 4 liters of milk per day by bucket until weaning at 3 months of age. Young preweaned calves were kept in individual pens with concrete floors. Dry grass was used as bedding. More bedding was usually added on top of the wet bedding every morning. Dirty bedding was removed only when necessary. During the day, these calves were allowed to rest and feed in a paddock near the calf house. No hay or silage was provided as supplementary feed. The same paddock was used at times to confine weaned calves before taking them to pasture.

Ectoparasitic diseases were controlled by dipping all animals in a plunge dip containing acaricide. Dipping was done twice a week. The acaricide in use was Chlorfenviphos 110% WV (Super dip<sup>®</sup>, SA Cyanamid Pty Ltd, South Africa). Gastrointestinal helminths

control in calves was done using available anthelmintics namely, Albendazole (Valbazen<sup>®</sup>, Smith Kline, Beecham) or Levamisole (Milsan<sup>®</sup>, Interchem Pharm). Prophylactic treatment of animals against Bovine trypanosomiasis was done after every 3 months by administration of Isometamidium chloride (Samorin<sup>®</sup> RM, France) at 0.25 mg/kg body weight.

### **3.2.2 Mlali Farm**

Mlali Farm is located 28 km South of Morogoro off Morogoro-Iringa road. During the study there was a total of 380 heads of cattle in this farm and a total of 18 water buffaloes. Tanzania Shorthorn Zebu (TSZ) constitute 50% of the herd, whereas, crosses of TSZ with Friesian or Ayrshire constituted the other 50%.

Every morning adult cattle and buffaloes were taken out for grazing. There was no specific weaning period. The animals were herded in separate groups divided according to age. These groups were brought back during the afternoon and during the night each group stayed in a separate kraal. The kraal for adult cattle and that of calves except colostrum feeding ones had neither roof nor floors. The calf shelter had roof which is supported by iron pillars. The walls of the calf shelter are of angle iron which doesn't protect calves from draughts. Young calves suckled colostrum from the dam for up to 7 days.

Ectoparasitic diseases were controlled by immersing animals in a plunge dip twice a week. Acaricide in use was Chlorovenfiphos 110% WV (SuperDip<sup>®</sup>). Deworming of calves was done only when the health of animal appeared to deteriorate. Prophylactic treatment against bovine trypanosomiasis was done by administration of Isometamidium chloride (Samorin<sup>®</sup>) every after 3 months at the dosage rate of 0.25 mg/kg body weight.

### 3.2.3 Azam estate

This Farm is situated 48 kilometers North-West of morogoro along Morogoro-Dodoma road. During the study the farm had 128 heads of cattle. The herd was made of Ayrshire and Friesian breeds. Adult cattle and weaners grazed on pastures in paddocks on rotational basis. Unweaned calves were kept in individual pens with concrete floors. The floors had no bedding. Calf pens were cleaned every morning and allowed dry. During the day, unweaned calves rest and fed on lush pastures in a water logged paddock within the vicinity of the calf house. Young calves were each given 4 liters of colostrum in bucket. Colostrum feeding period was 5-7 days. Calves were weaned when they were 3 months old.

Routine deworming, prophylaxis against endemic bovine trypanosomiasis and ectoparasite control was practised. Ectoparasite were controlled by dipping animals in a plunge dip containing acaricide. The Acaricide in use was Chlorfenviphos EC 300

(Steladone<sup>®</sup>). Routine deworming against gastrointestinal helminths control was strictly done every 3 months using Milsan (Levemisole) or Valbazen (Albendazole).

#### **3.2.4 Magadu Dairy Unit (MDU)**

The Magadu dairy Unit is within the University premises. Ayrshire and Friesian breeds constituted 95% of the herd. The herd size was 116 cattle during the study. The dry herd grazed on pastures in the paddocks during the day, while the milk herd was stall fed during the day and at nights. In the dry season all animals were given hay, but the milk herd and the calves were also provided with hay and silage.

Neonates were allowed to suckle colostrum for up to 7 days postpartum. After colostrum period, were kept in individual pens on concrete floors with bedding until when they were weaned. Wet straw was changed and replaced with dry straw every morning. Post colostrum feed in calves was mainly whole milk and hay in a rack outside the calf house. Calves were given 4 liters of whole milk per day by bucket feeding and were supplemented with hay from 2 weeks of age.

Diseases control included spraying animals with Chlorfenviphos 110% WV (Superdip<sup>®</sup>) against tick borne diseases. Deworming of calves depended on body condition of calves.

### **3.2.5 Sokoine University of Agriculture (SUA) Farm**

The SUA Farm is also within the University premises. The herd was of 80 cattle. Boran cattle constituted 80% of the herd and crosses of Friesian and Ayrshire with Boran constitute 20% of the herd. Weaned calves were not separated from adult cattle and they were grazed on pasture grass in paddocks during the day. At night adult cattle and weaners were sheltered in a yard where milking cows rested before milking. Neonates suckled colostrum for up to 7 days postpartum and were kept in a group pen within the calf house. The floor of the calf house had no bedding. Unweaned calves were each given 4 liters of milk daily in bucket. Hay or silage was not provided. Milk feeding calves were allowed to graze on pastures in the paddock near the farm buildings. Calves were weaned at the age of 4 months or more when they were in poor body condition. There was no programme for gastrointestinal helminths control or prophylaxis against endemic diseases in the farm. All animals were dipped in a plunge dip containing acaricide once weekly, however, there were times when the acaricide was not available when animals could go without dipping for as long as three weeks.

### **3.3 Methodology**

In this study the target population was unthrifty calves aged 1 day to 6 months. The calf was considered unthrifty when the body condition was poor and showed general signs

of ill health. The signs included, general body weakness, the rough coat, visible bone prominence (pin bones, hock bones, vertebral column and ribs), and gross signs of muscular wasting. Samples collection and health examination was done for 7 months representing both the rainy season and the dry season. During monthly health checks, unthrifty calves were separated from healthy ones. An elaborate examination was performed on them. Natural orifices (ears, eyes, vagina, mouth and nostrils) were closely examined. Externally accessible lymphnodes were palpated and mucus membranes were checked for pathological changes. Healthy calves were only screened for gastrointestinal helminths and coccidia. Whenever available dead calves from the farms of study were subjected to post mortem examination. Questionnaires were used to get information on management practice. Further information was obtained through actual observations made during sample collection.

Blood samples were collected from the jugular vein and analyzed for packed cell volume (PCV), hemoglobin concentration (Hb), serum total protein (TP), Albumin and globulin fraction. Thin and thick blood smears were made and used to screen unthrifty calves for hemoparasites. Faeces were collected per rectum and screened for gastrointestinal helminths and coccidia oocysts.

Faecal samples from diarrhoeic calves were screened for coronavirus, rotavirus and *Escherichia coli* K 99 using antigen detection ELISA kits supplied by Pathasure<sup>®</sup>,

Cambridge Laboratories, UK). Faecal samples in which *E. coli* K 99 antigen was detected by the ELISA kits were cultured on blood agar and McConkey agar in order to isolate the bacterium. The isolate was later subcultured in the slant agar and stocked in bijoux bottles at 4°C.

Blood samples from neonates were collected on the first week of life during which serum immunoglobulin concentration would be high.

### **3.3.1 Sampling procedure**

#### **3.3.1.1 Blood Samples**

A pair of blood samples was collected. One sample was collected in plain vacutainer tubes containing clot activator. Another sample was collected in tubes containing heparin. Serum was harvested from the clotted blood by centrifuging at 3000 rpm for 10 minutes. Sera obtained were stored at -20°C until they were analyzed.

#### **3.3.1.2 Faecal samples**

Faecal samples were collected directly from rectum using disposable gloves. The samples were put into labelled plastic containers. Samples were stored in a cool box during transportation to the laboratory.

#### **3.3.1.3 Blood smears**

Thin and thick blood smears were made at the time of faecal collection. Sometimes the smears were made on arrival to the laboratory using heparinated blood sample. Thin smears were dried and fixed immediately in absolute methyl alcohol. Thick blood smears were not fixed.

#### **3.3.1.4 Skin scrapings**

Skin scrapping around hairless areas were taken using sterile surgical blade. The samples were put into clean dry test tubes and taken to the laboratory for detection of aetiological agents.

#### **3.3.1.5 Aural swabs**

Aural swabs were collected from calves which showed signs of otitis. The signs included purulent or brownish foul smelling aural discharges, alopecia on the base of the infected ear and tilting of the head. A sterile cotton swab was gently introduced into the infected ear canal and a mixture of pus and aural debris was taken by rolling the cotton swab gently within the ear canal. The swab was put into a test tube. The lid of the test tube was tightened gently. During transportation the test tubes were stored in the cool box.

### **3.3.1.6 Nasal swabs**

Nasal swabs were collected from calves with purulent nasal discharges. The sterile cotton swab was inserted into the nostrils of the affected calf. The swab containing the nasal discharge was put into a sterile test tube. The lid of the test tube was tightened and stored in a cool box during transportation to the laboratory.

## **3.4 Analysis**

### **3.4.1 Determination of Serum Total Protein**

Refractometer method was adopted and the clinical refractometer (ATAGO, JAPAN) was used in this case (Fig.1). This test assumes that, the increase in serum protein over that of presuckled newborn ruminants is caused by absorption of immunoglobulin. In the absence of dehydration, a serum protein over 6 g/dl is considered to be associated with a successful passive transfer. Values less than 5 g/dl are consistent with failure of passive transfer (FPT). Refractometer utilizes the principle that the refractive index of a solution is determined by its concentration and that, that of blood serum or plasma depends principally upon the protein concentration as these are the major components. The immunoglobulin fraction makes up approximately one-third of the total serum protein. Since immunoglobulin deficient calves will consequently have less total protein, this will be indicated by lower refractometer reading. Prior to protein measurement, the readings were checked by placing a few drops of distilled water on the face of the

prism, closing the cover plate and focusing the scale. If the scale was correct the boundary line would fall on the dark line. If not the boundary line was adjusted to coincide with the dark line by turning the screw. The cover plate and the prism face were then wiped with soft tissue. A drop of serum was applied to the face of the prism. Refractive index of the serum which is proportional to the concentration of total protein (g/dl) was read through the eye piece against direct natural light.

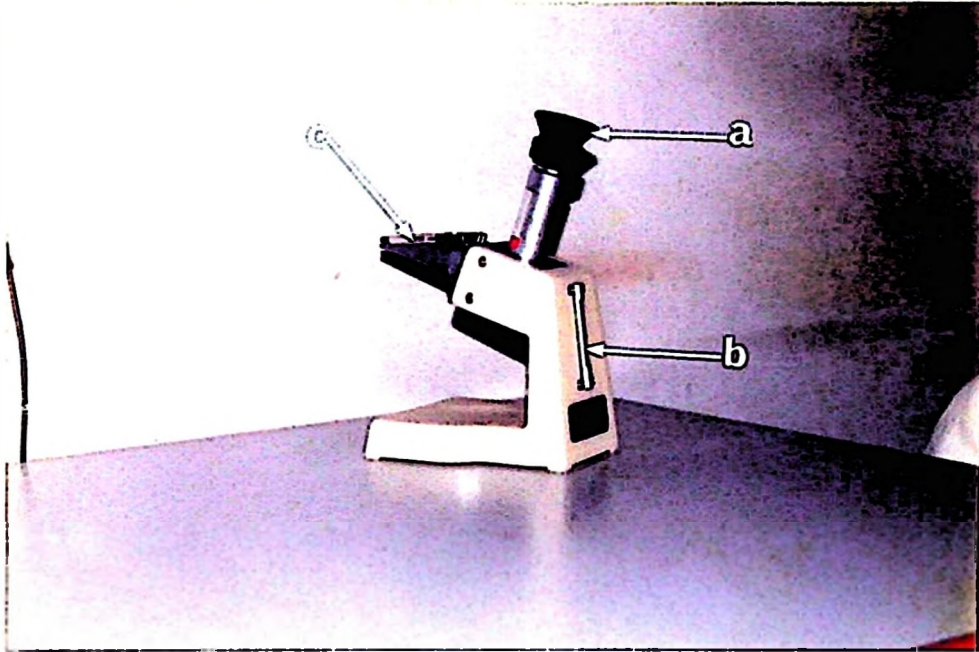


Figure 1: Clinical refractometer used for determination of serum protein concentration.

a: eye piece b: thermometer c: glass prism

### 3.4.2 Determination of serum albumin

The method of Dumas *et al.* (1971) was adopted. This method utilizes the principle that, bromocresol green binds qualitatively with serum albumin to give a bright blue green complex. The intensity of the colours produced is compared with that of a standard albumin similarly treated. The addition of albumin to a solution of bromocresol green results in an increase in absorbance at 628nm wave length. Five milliliters of bromocresol green working solution was added into three test tubes labelled blank, test sample and standard. To each tube 25 $\mu$ l of distilled water, working and standard solution were added respectively. All samples in the tubes were well mixed and allowed to stand for 10 minutes at 25°C. The absorbance of standard and test samples were read against blank at 628nm. The concentration of albumin in the sample was calculated as:

$$\text{Albumin (g/dl)} = \frac{\text{Absorbance of sample} \times \text{Conc. of Standard}}{\text{Absorbance of Standard}}$$

### 3.4.3 Determination of globulin fraction

Globulin fraction concentration was obtained by the difference between total protein and albumin concentration as determined by the method of Dumas *et al.* (1971) and expressed in g/dl.

#### **3.4.4 Determination of PCV**

Packed cell volume (PCV) is the volume of red cells expressed as proportion (L/L) of the given volume of the whole blood.

The stabilized blood was mixed by repeatedly turning the test tube upside down several times. Whole blood was drawn to three quarters of the microhematocrit capillary tube. The end of the capillary tube was sealed with plasticine. The capillary tube was placed in the microhematocrit centrifuge, the sealed end outermost. The safety cover of the centrifuge was screwed on. The lid of the centrifuge was closed. Spinning was done for 5 minutes. The PCV was read on the microhematocrit reader.

#### **3.4.5 Estimation of haemoglobin**

The cyanmethemoglobin method of ICSH (1967) was adopted. Five milliliters of Drabkin's solution was dispensed into a clean test tube. 20  $\mu$ l of blood was added into the tube containing Drabkin's solution. The pipette was rinsed with Drabkin's solution. The mixture was shaken and allowed to stand for 3-5 minutes. Absorbance of the mixture was read at 540 nm wavelength against Drabkin's solution. The absorbance of the Cyanmethemoglobin standard solution was read against water. Cyanmethemoglobin was calculated as:

$$\text{Cyanmethemoglobin (g/dl)} = \frac{\text{Absorbance of sample} \times \text{Conc. Std}}{\text{Absorbance of Std}}$$

Where:        Conc. Std        = concentration of standard  
                   D                = The dilution factor of 0.251

### 3.4.6 Faecal analysis

Four grams of faeces were weighed and placed into cellophane cup containing 56 ml of 40% glucose monohydrate solution. The contents were mixed using a wooden spatula. The filtered sample was stirred, pipetted and filled in both chambers of the McMaster counting chamber. The counting chamber was allowed to stand for 5 minutes before examining the filtrate under a microscope. The 10 times objective was used.

The number of eggs per gram of faeces (epg) was calculated as:

EPG = Number of eggs in both chambers multiplied by 50. Basing on mixed infections of parasites, eggs were classified according to (Hansen and Perry, 1994) as: Low (50-200) epg, Medium (250-750) epg and High (800 and above) epg. Eimeria oocyst were counted for each sample and recorded as; Negative = 0 Oocyst; (+) = 10-50 Oocyst; (++) = 51-100 Oocyst; (+++) = Over 101 Oocyst.

### 3.4.7 Identification of fungi

Crust material was cultured in Sabouraud's dextrose agar containing cycloheximide. The medium was incubated at room temperature (25°C) for six days.

### 3.4.8 Bovine coronavirus, rotavirus and *E. coli* K 99 antigens

The Pathasure<sup>®</sup> Bovine enteritis kit (Cambridge Veterinary Laboratories, UK) were used for faecal ELISA. A 10% suspension of the faecal sample was made by adding 0.25 grams of faeces into 2.5 ml of sample diluent provided. Washing solution was made by adding 10 milliliter of the wash concentrate provided into the plastic wash bottle and made up to 500 milliliter with cold distilled water. Two drops of the positive control solution was added into appropriate microwell for bovine coronavirus (BCV), rotavirus (ROTA) and *Escherichia coli* K 99 (K99). Two drops of the negative control were added into appropriate microwells for BCV, rotavirus and *Escherichia coli* K 99. Using a fresh plastic pipette for each previously diluted faecal sample, two drops of the sample were carefully added to the appropriate microwell for BCV, rotavirus and *Escherichia coli* K 99. The microwell plate was covered with the lid and left for 30 minutes at room temperature (18°C to 28°C). Microwells were washed five times with a washing solution. Two drops (blue, bovine coronavirus conjugate, red, rotavirus conjugate and green, *E. coli* K 99 conjugate) were added into each microwell located over a blue line,

red line and green line on the colour guide respectively. The microwell plate was covered and left for 30 minutes at room temperature. Two drops of substrate part A were first added to every microwell then two drops of substrate part B were added to the same wells. The wells were carefully mixed by agitating the whole plate in a swirling motion with care not to spill the contents of the wells. The microwells were covered and left at room temperature for 15 minutes while avoiding direct sunlight. The contents of the microwell were mixed again and the holder was placed on plain white paper.

Interpretation of results was based on colour reaction. The negative control microwells were faint blue and the positive control microwells were distinctly blue indicating that the assay was functioning well. Any blue colour microwell which was distinctly darker than that of its respective negative control was scored as a positive result, otherwise the result was considered as negative.

### **3.5 Autopsy and histopathology**

Three dead calves were submitted for necropsy and one calf was brought to be sacrificed. The first calf was one from SUA farm, with a history of coughing, grunting and producing purulent nasal discharge. The second was from KPF and had a history of salivation, lameness, emaciation and diarrhoea. The third was a 2 weeks old calf

from Azam Estate with a history of diarrhoea and enlarged umbilicus. The one which was sacrificed was from SUA farm was severely emaciated and dehydrated.

### 3.5.1 Autopsy

The dead calves were examined closely before any incision was made. Body orifices were closely checked. Thereafter each carcass was prosected, eviscerated and examined as per routine postmortem examination and sampling techniques. The sacrificed calf was anaesthetized with pentobarbitone sodium prior to total autopsy. This was to enable sampling of intestine before they undergo putrefaction. Five pieces of small intestine (SI 1, SI 2, SI 3, SI 4, and SI 5) approximately 10 cm in length were located. These pieces were 15cm, 72 cm, 162 cm, 432 cm, 667 cm and 767 cm proximal to the ileal caecal junction. A double ligature was put cranial and aboral to each 10 cm piece. In each piece 20 ml of buffered formalin was infused. Each piece was removed in situ by cutting the cranial and aboral ligature. Another 10 cm piece of colon was ligated, infused with 20 ml of buffered formalin and transected. One piece 2x3 cm of dorsal and another of the ventral rumenal sac were removed and fixed in buffered formalin.

### **3.5.2 Histopathology**

Formalin fixed intestine samples were trimmed and processed for sectioning into 5  $\mu\text{m}$  thick paraffin sections by standard techniques as outlined by Bancroft and Stevens (1990). The sections were stained with hematoxylin and Eosin (H&E) or by selected special stains namely periodic acid schiff (PAS) and Van Giesson as the cases required. Selected sections were recorded photomicrographically under an Olympus BH 2 Microscope with Agfapan 25 (ASA25) film.

### **3.6 Statistical Analysis**

The Graph Pad Statistical Package "Instat" 1991 was used. Causal effect relationship was determined by 2X2 contingency tables using Yates corrected Chi square and Fisher's exact test for 2X2 contingency tables wherever necessary. T test was used to test the difference between two means among parameters measured. The difference was considered significant only when  $P < 0.05$  or less.

## **4.0 RESULTS**

### **4.1 Influence of management**

#### **(a) Bucket feeding versus suckling**

At Azam estate two litres of colostrum were provided to the calf per meal twice daily (Table 1). After days of colostrum feeding, calves were given 2 liters of skim milk in the morning and 2 liters in the afternoon. At Magadu Dairy, KPF and SUA farm calves suckled colostrum for 5-7 days. After 5 days of colostrum, calves were fed 2 litres of whole milk in bucket in the morning and 2 litres in the afternoon. Calves were given this amount until weaning. At Mlali farm calves suckled both colostrum and milk. They continued to suckle for up to 6-8 months.

**Table 1: Methods and duration of feeding colostrum and milk to calves at Azam estate, KPF, Mlali, SUA and Magadu dairy farms**

| <b>Farm</b> | <b>Colostrum feeding</b> | <b>Duration (days)</b> | <b>Milk feeding</b> | <b>Duration (months)</b> |
|-------------|--------------------------|------------------------|---------------------|--------------------------|
| Azam        | bucket                   | 5                      | bucket              | 3                        |
| KPF         | suckling                 | 5-7                    | bucket              | 3                        |
| Mlali       | suckling                 | 7                      | suckling            | 6-8                      |
| SUA         | suckling                 | 5-7                    | bucket              | 3                        |
| Magadu      | suckling                 | 5-7                    | bucket              | 3                        |

#### **Feeding method and its relation to diarrhoea**

The only disease which was observed in calves of less than 4 weeks of age was diarrhoea. Diarrhoea was characterized by fever in 95% of cases. In all cases faeces were whitish grey in colour, porridge like in consistence and with a foetid smell. Prevalence of diarrhoea in different management systems practised by individual farms is shown (Table 2). In the intensive system which was practised in Azam Estate, the

prevalence of diarrhoea was significantly higher than semi intensive and free range management systems  $P < 0.01$  and  $P < 0.001$  respectively (Table 2). It was also observed that, prevalence of diarrhoea was significantly higher in Azam Estate than in Magadu dairy unit although both farms practised intensive management system.

Table 2: Prevalence of diarrhoea among calves less than 28 days of age (Azam estate, KPF, Mlali, Magadu and SUA farms).

| Farm   | Management system | Diarrhoeic | Non diarrhoeic | Sample size | Prevalence (%) |
|--------|-------------------|------------|----------------|-------------|----------------|
| Azam   | I                 | 9          | 5              | 14          | 64.3           |
| KPF    | SI                | 6          | 11             | 17          | 35.3           |
| Mlali  | FR                | 3          | 20             | 23          | 15             |
| Magadu | I                 | 5          | 17             | 22          | 29.4           |
| SUA    | SI                | 6          | 18             | 24          | 33.3           |

NB: I=Intensive, SI=Semi intensive, FR=Free range

Comparison between management systems using Fisher's exact Test:

Azam Vs KPF:  $P > 0.05$ , Odds ratio = 3.3

Azam Vs Mlali:  $**P < 0.001$ , Odds ratio = 12.00

Azam Vs Magadu:  $*P < 0.01$ , Odds ratio = 6.12

Azam Vs SUA farm:  $*P < 0.01$ , odds ratio = 5.4

KPF Vs Mlali:  $P > 0.05$ , Odds ratio = 3.636

KPF Vs Magadu:  $P > 0.05$ , odds ratio = 1.85

KPF Vs SUA farm:  $p > 0.05$ , odds ratio = 1.636

Mlali Vs Magadu:  $P > 0.05$ , Odds ratio = 0.51

Mlali Vs SUA farm:  $P > 0.05$ , Odds ratio = 0.45

#### **Microorganisms isolated in faeces of diarrhoeic calves**

The aetiology of diarrhoea in calves was based on 28 faecal samples collected from diarrhoeic calves in the 5 farms. Rotavirus antigen was detected in 46.4%, *Escherichia coli* K 99 antigen in 35.7% and coronavirus antigen in 10.7% of the faecal samples collected. While mixed infection of rotavirus and *E.coli* was observed in 21.4% of the samples, mixed infection of coronavirus and *E.coli* was detected in 3.6% and infection of coronavirus and rotavirus was detected in 3.6% of the samples. No attempt was made to detect other common causes of diarrhoea like *Cryptosporidium spp.*

#### **Calf mortality**

Calf pneumonia, diarrhoea and Foot and Mouth Disease (FMD) were causes of death among unweaned calves of 0-4 months of age and accounted for 39.1%, 21.7%, and 17.1% respectively. Cause of death in 21.8% of the cases could not be determined. In calves aged 4-6 months, causes of death were helminthosis 60%, bloat 4%, anaemia 22%. The cause of death in 14% of the cases could not be determined. Mortality of calves aged 0-4 months accounted for 47.9%, whereas mortality of calves aged 4-6 months accounted for 52.1% (Table 3).

Table 3: Calf mortality rates (MR) at Azam estate, Mlali, KPF, Magadu and SUA farms.

| Farm  | Born | died      |           | Total | MR    |
|-------|------|-----------|-----------|-------|-------|
|       |      | 0-4 month | 4-6 month |       |       |
| Azam  | 57   | 5         | 0         | 5     | 8.7%  |
| Mlali | 127  | 5         | 5         | 10    | 7.8%  |
| KPF   | 138  | 7         | 17        | 24    | 17.4% |
| MDU   | 22   | 3         | 0         | 3     | 13.6% |
| SUA   | 24   | 3         | 3         | 6     | 25%   |
| Total | 368  | 23        | 25        | 48    | 13%   |

**(b) Post colostrum feeds and supplements**

At Azam estate, post colostrum feed consisted of natural pasture grass in a paddock in which the calf house is located. Hominy feed locally known as "Pumba" was also provided at the rate of 0.25 kg per calf per day. At Magadu Dairy Unit hay and silage was provided during the dry season, while at KPF and Mlali farms calves depended solely on local pasture grass. At SUA farm hay was provided to calves in addition to pasture grass (Table 4).

Table 4: Pasture and feed supplement given to calves at Magadu dairy unit, KPF, Azam Estate, SUA farm and Mlali farm.

| Farm            | Pasture<br>grass | Hay | Silage | Pumba |
|-----------------|------------------|-----|--------|-------|
| Magadu<br>dairy | +                | +   | +      | -     |
| Azam Estate     | +                | +   | +      | +     |
| KPF             | +                | -   | -      | -     |
| Mlali farm      | +                | -   | -      | -     |
| SUA farm        | +                | +   | -      | -     |

#### Body condition of calves

Point prevalence of unthriftiness in calves and its relationship to the amount of rainfall in Morogoro is shown in Table 5. Point prevalence of unthriftiness was 54%, 63.8% and 70.2% at KPF in June, July and September respectively. The amount of rainfall in these months was 0 mm, 1.3 mm and 13.1 mm respectively. At Mlali farm the highest recorded point prevalence was 42% in December, whereas at Azam Estate calves did not face a period of unthriftiness (table 5).

Unthriftiness in weaned calves occurred as a syndrome of weight loss with apparently normal appetite. The syndrome was apparent 4-6 weeks after weaning. The coat of affected calves was rough and muscles were grossly wasted (Figure 2) compared to healthy calves immediately after weaning (Figure 3). Affected calves became weak and could not walk long distances. This syndrome occurred irrespective of the season of the year. However, calves weaned in the dry season were more affected than calves weaned in the rainy season. Monthly prevalence of unthriftiness in weaned calves was high at KPF compared to Mlali farm (table 5). Calves at Azam Estate did not suffer from unthriftiness during the whole study period. A total of 35 (72.9%) calves that died during the study period in all farms occurred in the period covering the months of June to October, where point prevalence of unthriftiness was higher (Table 5).

Table 5: Amount of rainfall (mm) in Morogoro and point prevalence of unthriftiness and deaths of calves at KPF, Azam Estate and Mlali farm.

| Month | Rainfall (mm) | Point prevalence (%) |            |      | Deaths |
|-------|---------------|----------------------|------------|------|--------|
|       |               | KPF                  | Mlali farm | Azam |        |
| May   | 132           | 31.8                 | n.r        | n.r  | 0      |
| June  | 0             | 54                   | 39.1       | 0    | 7      |
| July  | 1.3           | 63.8                 | 23.9       | 0    | 6      |
| Aug   | 28.8          | n.r                  | n.r        | n.r  | 15     |
| Sept  | 13.1          | 70.2                 | 34.9       | 0    | 4      |
| Oct   | 64.2          | 48                   | n.r        | 0    | 1      |
| Nov   | 0.6           | n.r                  | n.r        | n.r  | 0      |
| Dec   | 73.4          | 32.8                 | 42.6       | 0    | 2      |
| Jan   | 105           | n.r                  | n.r        | n.r  | 0      |
| Feb   | 151.5         | 27.5                 | 31.9       | 0    | 8      |
| March | 127.9         | 22.6                 | 24.3       | 0    | 4      |
| April | 208.5         | 46.9                 | 34.5       | 0    | 1      |

n.r = not recorded; Source of rainfall data: Morogoro Metereological station SUA.



**Figure 2: Gross muscular wasting indicative of unthriftiness in a calf four weeks after weaning at KPF. The cause was helminthosis and underfeeding in the dry season.**



**Figure 3: A healthy calf two days after weaning at KPF. The calf suckled colostrum and was fed milk by bucket.**

### **(c) Housing of calves**

At Azam estate, KPF and Magadu Dairy unweaned calves were kept in individual pens with concrete floors. Bedding was provided at Magadu dairy and KPF but not Azam Estate. Unlike Magadu dairy where bedding was changed every morning, bedding was changed only when it became wet at KPF. Calf pens were therefore littered with damp bedding at KPF. At SUA farm each 6 unweaned calves were kept in a pen measuring 3 x 4 meters. In those pens the floor was slant but usually without bedding. At Mlali farm calves aged 1 week up to 6 months old were mixed together and confined in a kraal. The kraal had neither walls nor concrete floor. At Azam estate and SUA farm weaned calves were housed in barn during the night. At KPF and Magadu dairy a group of 10 to 15 weaned calves were confined in group pens at night.

## **4.2 Disease control schemes**

### **Control of helminthosis**

Helminths control at Azam estate was achieved by deworming of calves just before weaning and later, on a three monthly basis in weaned calves. Anthelmintic used were Albendazole (Valbazen<sup>®</sup>, Smith Kline Beecham, or Levamisole (Milsan<sup>®</sup>, Interchem Pharm). At Mlali farm and KPF helminthosis control was done only when weight loss in calves became severe. No data on helminthosis was collected from Magadu dairy unit and SUA farm to avoid interfering another research which had already started.

### Control of tick borne diseases

Endemic tick borne diseases in Morogoro include East Coast Fever, Anaplasmosis, Babesiosis and Heart water. Control of these diseases was achieved by dipping/spraying animals with available acaricide (Table 6).

Table 6: Method of tick control, acaricide used and days/week of dipping/spraying in selected farms in Morogoro.

| Farm         | Method   | Days/week | Acaricide              |
|--------------|----------|-----------|------------------------|
| Azam Estate  | Dipping  | 2         | Chlorfenviphos EC 300  |
| KPF          | Dipping  | 2         | Chlorfenviphos 110% wv |
| Mlali farm   | Dipping  | 2         | Chlorfenviphos 110% wv |
| SUA farm     | Dipping  | 2*        | Chlorfenviphos 110% wv |
| Magadu dairy | Spraying | 2         | Chlorfenviphos 110% wv |

\* Dipping depended on availability of acaricide.

Chlorfenviphos EC 300 is Steladone<sup>®</sup> (Ciba) and Chlorfenviphos 110% wv is Super Dip<sup>®</sup> (SA Cyanamid, South Africa)

### **Control of trypanosomiasis**

Prophylactic treatment against trypanosomiasis was done at Azam estate, Mlali farm and KPF by injection of animals with Isometamidium chloride (Samorin<sup>®</sup>) at the dosage rate of 0.25 mg/kg body weight on a 3 monthly basis. Impact of control of tick borne and tsetse transmitted diseases was reflected by few positive cases of these diseases in Morogoro. Thus, a total of 339 blood samples from unthrifty calves were screened for hemoparasites. Among the samples only 4 (1.1%) were found with hemoparasites. *Theileria* piroplasms were present in 2 samples and *Trypanosoma congolense* was found in 1 sample, the 3 calves were from KPF. *Babesia bovis* was observed in a blood smear collected from a dying unthrifty calf from SUA farm. The calf died of severe anaemia resulting from gastrointestinal parasitism and diarrhoea. Blood samples from Mlali farm, Magadu dairy farm, and Azam Estate did not reveal any hemoparasite.

### **Vaccination against endemic contagious diseases**

Vaccination against Foot and Mouth disease (FMD) was done at Azam estate using a vaccine imported from Kenya. Other farms did not vaccinate their animals against FMD because the vaccine was not available in the country and they could not import. In 1996 there was a national campaign against contagious bovine pleuropneumonia (CBPP). Animals were vaccinated in many parts of the country including Morogoro. No vaccination was done against any other disease in Morogoro during the study period.

### 4.3 Influence of season

There are four seasons in Morogoro. These seasons included, short rainy season (November to December), long rainy season (February to May), cold windy season (June and July) and the dry season (August to October). A total of 35 (72.9%) calves died in the cold season and the dry season which covered June to October, whereas, 13 (27.1%) calves died in the rainy season which covered February to May 1996, nonetheless, monthly mean deaths in the dry season did not differ significantly from that of the rainy season ( $P > 0.05$ ).

### 4.4 Disease conditions observed

#### Helminthosis

Helminthosis featured to be a serious disease affecting both weaned and unweaned calves. Affected calves were unthrifty and diarrhoeic. Affected calves were characterized by soiling of the perineum with brownish soft faeces, had normal appetite and normal rectal temperature. Calves of 0-4 months old were equally infested as those aged 4-6 months ( $P > 0.05$ ) (Table 8).

Although this was the case, calves whose age was less than one month were less likely infested compared to those aged 2-3 months of age  $P < 0.001$  (table 9). As the calf

grew older, chances of being infested with gastrointestinal nematodes becomes higher. Common gastrointestinal nematodes frequently isolated from faeces of infested calves were *Haemonchus spp*, *Trichostrongylus axei*, *Oesophagostomum spp*, *Strongyloides papillosus*, and *Cooperia oncophora* (Table 7). *Haemonchus spp* and *Oesophagostomum spp* formed the great majority of nematodes infesting calves in Morogoro and *Trichostrongylus spp* and *Strongyloides spp* followed. *Toxocara vitulorum* was identified in faeces of calves of less than 4 weeks of age at Mlali farm.

Mean counts and standard deviation of worm eggs per gram of faeces were  $1600 \pm 1400$  and  $1045 \pm 1500$  in July and September respectively at KPF. At Mlali farm highest counts of  $257 \pm 212$  and  $500 \pm 226$  were observed in September and December respectively, whereas, counts of  $227 \pm 436$  and  $116 \pm 90$  were observed in June and February respectively at Azam Estate. Corresponding serum globulin concentration in g/dl are shown (Table 10).

Helminthosis was also associated with pathological changes occurring in the small intestine. These lesions included atrophy of villi in the jejunum, dilated lacteals, flattening of villi surfaces and joining of adjacent jejunal villi (Figure 4) and there was dyskeratosis of the rumen manifested by hyperkeratosis, vacuolation, hydropic degeneration and microabscess formation (Figure 5).

Table 7: Helminths species identified after culturing faeces of calves from KPF, Mlali and Azam Estate farms.

| Nematode spp                | Farm |       |      | Total (%)  |
|-----------------------------|------|-------|------|------------|
|                             | KPF  | Mlali | Azam |            |
| <i>Haemonchus spp</i>       | 5    | 2     | 9    | 16 (35.5%) |
| <i>Trichostrongylus spp</i> | 0    | 1     | 6    | 7 (15.5%)  |
| <i>Strongyloides spp</i>    | 3    | 2     | 2    | 7 (15.5%)  |
| <i>Oesophagostomum spp</i>  | 11   | 0     | 3    | 14 (31.1%) |
| <i>Cooperia spp</i>         | 1    | 0     | 0    | 1 (2.2%)   |
| Larvae counted              | 20   | 5     | 20   | 45         |

NB: SUA farm and Magadu dairy unit not screened.

Table 8: Helminths infestation among calves aged 0-4 month old and those aged 4-6 months at KPF in July 1995.

|            | Infested | Not infested | Total |
|------------|----------|--------------|-------|
| 0-4 month  | 7        | 15           | 22    |
| 4-6 months | 7        | 20           | 27    |
| Total      | 14       | 35           | 49    |

$X^2 = 0.0186$ ;  $P > 0.05$

Odds ratio = 1.33

Table 9: Helminths infestation among calves aged less than 28 days and those aged 2-3 months at Azam Estate in September 1995.

|                       | Infested | Not infested | Total |
|-----------------------|----------|--------------|-------|
| Less than 1 month old | 0        | 5            | 5     |
| Aged 2-3 months       | 9        | 3            | 12    |
| Total                 | 9        | 8            | 17    |

Fisher's exact test P value = 0.05 - 0.001.

Table 10: Mean worm egg per gram of faeces and mean globulin concentration (g/dl) in calves at KPF, Mlali farm and Azam Estate.

| Farm  | KPF         |             | Mlali     |             | Azam      |             |
|-------|-------------|-------------|-----------|-------------|-----------|-------------|
| Month | EPG         | Globulin    | EPG       | Globulin    | EPG       | Globulin    |
| May   | 493 ± 605   | 2.84 ± 1.29 | n.r       | n.r         | n.r       | n.r         |
| June  | 595 ± 1138  | 1.63 ± 1.6  | 114 ± 213 | 1.75 ± 0.8  | 221 ± 436 | 2.44 ± 0.85 |
| July  | 1045 ± 1500 | 2.17 ± 0.96 | 24 ± 39   | 2.89 ± 0.52 | 95 ± 193  | 1.68 ± 0.85 |
| Aug   | n.r         | n.r         | n.r       | n.r         | n.r       | n.r         |
| Sep   | 1600 ± 1400 | 0.99 ± 0.74 | 257 ± 212 | 1.24 ± 0.8  | 47 ± 98   | 3.0 ± 1.56  |
| Oct   | 817 ± 738   | 1.8 ± 0.68  | n.r       | n.r         | 51 ± 68   | 2.6 ± 1.24  |
| Nov   | n.r         | n.r         | n.r       | n.r         | n.r       | n.r         |
| Dec   | 595 ± 470   | 1.52 ± 1.2  | 500 ± 226 | 1.89 ± 0.65 | 78 ± 103  | 1.89 ± 0.9  |
| Jan   | n.r         | n.r         | n.r       | n.r         | n.r       | n.r         |
| Feb   | 181 ± 205   | 1.75 ± 0.36 | 200 ± 400 | 1.87 ± 0.37 | 116 ± 90  | 2.01 ± 0.4  |

Note: n.r = not recorded



Figure 4: Histological section of the wall of the jejunum showing atrophy of villi and dilated lacteals in a diarrhoeic calf from SUA farm. The calf was sacrificed and sections were removed under general anaesthesia. The calf was later euthanized.

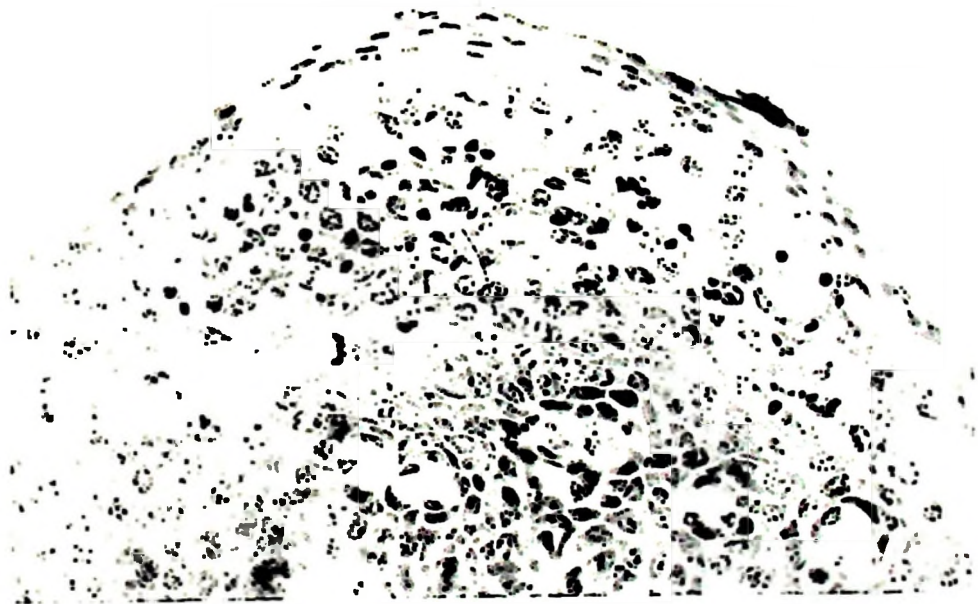


Figure 5: Histological section of the wall of the rumen showing hyperkeratosis, vacuolation, hydropic degeneration and microabscess. The problem was indigestion and the calf was from SUA farm.

**Cutaneous mycosis**

Cutaneous mycosis was observed at KPF involving 18% of the weaned calves. At Magadu dairy unit both weaned and unweaned calves suffered from the diseases at the prevalence rate of 82%. Thick crusts were distributed around eyes and on the neck. Microscopic examination of material from the crust contained many branching filaments with few but large spores. On the hair shaft the spores were characteristically ectothrix (arthrospore) suggesting *Trichophyton spp.* On Sabouraud's dextrose agar containing cycloheximide *Trichophyton spp* grew after 6 days of incubation at room temperature of 25-28°C (Figure 6).

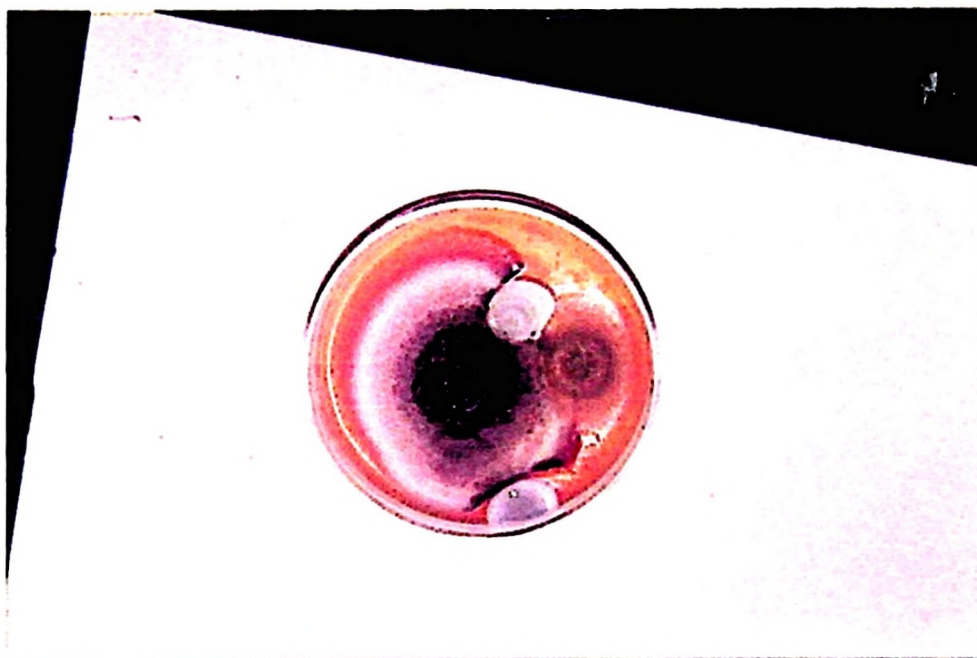


Figure 6: *Trichophyton spp* on Sabouraud's dextrose agar containing cycloheximide.

Skin scrapping of a calf from Magadu Dairy unit. Calves were kept in group pens.

### **Foot and Mouth Disease (FMD)**

An outbreak of FMD occurred in the herd of KPF in June 1995. During the outbreak, 4 out of 26 calves aged less than 2 weeks and 1 weaned calf aged 4 months died from the disease. The dams of the neonatal calves suffered from clinical FMD a week earlier. Since other farm did not suffer FMD epidemic, no calf losses were associated with the disease in those farms. Pathological specimens to confirm the disease were not submitted to the laboratory. *Post mortem* examination done to a calf which died on the day and had history of salivation, lameness and diarrhoea, revealed severe emaciation, gelatinous subcutaneous, coronary and renal fat. There were erosions on the tongue, gums and inter digital cleft. The bone marrow was also hyperplastic. Further more, a mixture of larvae and adult stages of *Haemonchus spp* were recovered in the abomasum. Starvation due to Foot and Mouth Disease and anaemia resulted from chronic haemonchosis was suspected to be responsible for the death of the calf.

### **Thelaziasis**

A total of 6 (0.04%) calves succumbed to thelaziasis at KPF. Worms of *Thelazia spp* were seen under the cornea of infected calves. At Azam estate and Mlali farms only ocular conjunctivitis and lacrimation was observed infrequently.

### **Alopecia**

An alopecia was observed in 14 (11%) calves at Mlali farm. Affected calves were those less than two month old. Materials from the affected area revealed neither mange, dermatophytes nor *Dermatophylous congolence*. Lesion were either symmetrical or asymmetrical along the back line (Figure 7). Areas of alopecia in some cases extended to the head of the tail caudally. Anteriorly the alopecic area extended to the base of the ears and the entire back side of ears. Alopecia persisted in 8 out 14 calves for up to the age of 8 months. The same condition has been observed at Dakawa ranch and no aetiological agent was associated with the disease but photosensitization was suspected.



**Figure 7: Alopecic lesions on the back of the calf. The aetiology was not determined. The calf was from Mlali farm practising free range management system.**

### **Bacterial otitis**

An otitis caused by *Escherichia coli* infection was observed in 2 out of 368 calves. One calf belonged to SUA farm and another belonged to Mlali farm. Pus from the affected ears was creamy to whitish yellow in colour (Figure 8). The infection was bilateral in calf from Mlali farm and unilateral in calf from SUA farm. Both calves were less than 1 month of age. Affected calves bent the head towards the infected ear. Affected animals resented touching of the affected ears and were reluctant to eat perhaps due to pain. Beta hemolytic *Escherichia coli* was isolated in pure culture from cultures of the swabs collected from the calves. The colonies were shiny, raised and round. The bacterium was a Gram negative coccoid rod. The bacterium fermented lactose in both brilliant green agar and McConkey agar. Biochemically the bacterium was IMViC (+ + - -). The bacterium was resistant to penicillin, tetracycline and neomycin but was sensitive to gentamycin. The calf from Mlali farm died 3 weeks after the onset of the disease and that from SUA farm recovered after treatment with gentamycin injection intramuscularly for 5 days.



**Figure 8: Bacterial otitis in calf from Mlali farm. Note the whitish creamy pus occluding the ear canal.**

**Bovine parasitic otitis**

An aural disease characterized by foul smelling dark brown aural discharge, lymphadenitis, alopecia on the ear base, curling of the pinna and occlusion of the ear canal was observed in calves at Mlali farm (Figure 9). The disease was not observed in calves of other farms. In early infections only frank blood was discharged from the infected ear. Calves over 3 month of age were the only ones affected. Under the microscope, myriads of worms were seen in aural debris mixed with a drop of normal saline on a microscopic glass slide. The nematodes *Rhabditis bovis* and *Rhabditis blumi* were identified from the aural swabs of infected ears. There were more *Rhabditis bovis* than *Rhabditis blumi* in the infected ears. The two parasites have been isolated from soil and manure collected the same day from the calf pens. The proportion of *Rhabditis blumi* in soil sample was higher than *Rhabditis bovis*. The higher prevalence of 13% was observed in April (rainy season) compared to prevalence of 4.5% which was observed in the dry season.



Figure 9: A case of Bovine parasitic otitis showing curling of the pinna and complete occlusion of the aural canal. A calf from Mlali farm.

### **Calf pneumonia**

Respiratory disease characterized by purulent nasal discharges and coughing was observed in 12% of weaned calves at KPF and 62% of weaned calves at Mlali farm. On the other hand, 3 out of 57 calves died of pneumonia at Azam estate. The dead calves were less than 28 days old and had a history of failure to get colostrum due to problems pertinent to their dams. Post mortem examination in one calf from Magadu dairy unit revealed froth in the trachea extending down to the entire bronchial tree. Tonsils were congested and acute bronchopneumonia was evident. None fatal cases were observed at KPF and Mlali farms. Purulent naso-ocular discharge associated with low frequency of coughing was observed in the affected calves. Weaned calves were more affected than preweaned calves. *Pasteurella spp* was isolated in pure culture from nasal swabs obtained from affected calves. Following incubation for 48 hours on Blood Agar, the bacterium was Gram negative and bipolarly stained. Morphologically it was a coccoid rod. There was no growth in McConkey Agar. On biochemical tests the organism was oxidase negative, catalase positive, urease negative, sorbitol negative, citrate negative, arabinose negative and non lactose fermenter.

## **5.0 DISCUSSION**

The intensive, semi intensive and free range management systems of production whose influence on the nature and rate of calf morbidity and mortality was the focus of this study were the major dairy production systems in the country. Therefore, the farms of KPF, Azam Estate, Mlali, Magadu dairy unit and SUA which were selected for this study each represent one of the widely practised systems of dairy production in Tanzania. Intensive system was practised at Azam estate and Magadu dairy unit, semi intensive system at SUA farm and KPF, whereas, the free range system was practised at Mlali farm.

The choice of the management system adopted by each farm appears to have been influenced by the farm's financial situation, breed of cattle kept, the balance between the thrust for pure dairy breeds and dual purpose production, as well as the location of the farm. Cattle breeds at Azam estate and Magadu dairy unit were of Friesian and Ayrshire breeds, where as, at KPF crosses of Zebu and Ayrshire or Friesian constituted over 90% of the herd. Eighty percent of the SUA herd was Boran and crosses of Boran with Friesian or Ayrshire constituted the remaining 20%. The 50% of the herd of Mlali was Zebu breed, whereas, crosses of Zebu and Friesian or Zebu and Ayrshire made the remaining 50%.

Beside the type of management in force, other practises that seemed to vary between farms with significant influence on the subsequent performance of calf health were the methods of feeding colostrum and milk to calves (Table 1), provision of post colostrum feed and supplements (Table 2), nature of housing and disease control schemes. Moreover, other factors unrelated to management practise were season of the year and breed of cattle kept. These have also been demonstrated to exert notable influence on calf health with regard to the following diseases encountered during the study:

#### **Calf diarrhoea**

In this study, the prevalence of diarrhoea in calves of less than 28 days old was 64.3% and 29.4% at Azam Estate and Magadu dairy unit both practising intensive system. The prevalence of 35.3% and 33.3% was observed in farms practising semi intensive system including KPF and SUA farms, and prevalence of 15% was observed at Mlali farm which practised free range management system (Table 2). Prevalence of diarrhoea differed within farms practising the same management system due to variations in hygiene and routine calf management practised in the farm. The highest prevalence of diarrhoea at Azam Estate could have resulted from bucket feeding of colostrum and skim milk to calves. Bucket feeding increases chances of contamination and possibility of feeding of cold milk to calves. Skim milk and milk replacers have been associated with outbreaks of diarrhoea, as a resulted of cold milk and poor hygienic condition during feeding (Roy, 1980; Withers, 1952). Compared to suckled calves prevalence of

diarrhoea was high in bucket fed calves because, suckled calves have high serum immunoglobulin concentration than bucket fed calves (Selman *et al.*, 1970; Stott *et al.*, 1979). The system of allowing calves to suckle colostrum may have helped to reduce the incidence and prevalence of diarrhoea in SUA farm, Mlali farm, KPF and Magadu dairy unit compared to Azam Estate practising bucket feeding of colostrum and milk to calves.

In this study, the correlation between prevalence of diarrhoea and serum immunoglobulin concentration could not be ascertained because immunoglobulin concentration in colostrum and that in calves' sera was not determined. These antibodies could protect the calves from enteric and respiratory diseases (Penhale *et al.*, 1973). Reynolds *et al.* (1986) suggested that, infection in the calf house, the rearing system and physical environment have been responsible for variable prevalence rate of diarrhoea in different management systems. Snodgrass *et al.* (1986) further indicated that, the difference in severity and magnitude of calf diarrhoea depend among other things on environmental and physiological factors including failure in absorption of immunoglobulin, in the small intestine of the calf and the causal agents in different localities. It has been found in this study that, causes of calf diarrhoea in Morogoro farms were coronavirus, rotavirus and *Escherichia coli* K 99. Mixed infection of rotavirus and *E. coli* K 99 was detected in 21.4% of the samples, whereas mixed infection of coronavirus and rotavirus was detected in 3.6%, and in 3.6% of the

samples, mixed infection of coronavirus and rotavirus was detected. Nonetheless rotavirus was frequently detected followed by *E. coli* and rarely coronavirus, where they accounted for 46.4%, 35.7% and 10.7% respectively. Mixed infection of these agents, with cryptosporidia and *Salmonella spp* have also been reported for causing calf diarrhoea in different parts of Tanzania including Morogoro and other parts of the world (Lema and Banda, 1991; Snodgrass *et al.*, 1986).

### **Dermatophytosis**

Prevalence of dermatophytosis was 82% in the intensive system (Magadu dairy unit) compared to the prevalence rate of 18% which was observed in the semi intensive system (KPF). Transmission of cutaneous mycosis occurs by directly by contact between animals and indirectly through fomites such as grooming instruments, fences and feed bunks (Fadok, 1984). Wooden feed troughs used at Magadu dairy unit and group pens used to confine calves are thought to have have contributed to such high prevalence of dermatophytosis. The disease was not observed in other farms, but what ever the reason, animals becomes immune to subsequent fungal infections and after primary infection (Blood *et al.*, 1990).

### **Haemoparasitic diseases**

The prevalence of haemoparasitic diseases including East coast fever and trypanosomiasis was 1.1%. This prevalence was low to be considered significant in well

managed farms in Morogoro. It seemed that, the five farms succeeded in controlling calf losses due to haemoparasitic diseases perhaps due awareness of high mortality usually associated with these diseases. In contrary, tickborne diseases were among causes of calf deaths in some farms in Tanzania (Kifaro and Temba, 1990). In Morogoro however, calf deaths due to TBDs and trypanosomiasis may be high in pastoral herds of Maasai and Barbaighs where the practise of dipping/spraying animals with acaricide is not common perhaps due to collapse of the communal based dip tanks previously operated by government (Komba, 1992). A study on diseases and causes of mortality of calves in traditional pastoral herds is suggested.

#### **Alopecia of undetermined cause**

Alopecia of unknown cause have been observed at the prevalence rate of 11% in calves kept under free range management system. This type of alopecia was also observed at Dakawa ranch situated 40 kilometers East of Mlali farm off Morogoro-Dodoma road (Kassuku, personal communication, 1996). The disease involved calves of less than 4 weeks of age. No organism was incriminated for causing the disease although photosensitization was suspected. A study on aetiology and epidemiology of this disease is suggested.

#### **Bovine parasitic otitis**

Bovine parasitic otitis (BPO) was observed at a prevalence rate of 13% and 4.5% in the

rainy season and the dry season respectively at Mlali farm practising free range management system. The disease was caused by the nematode *Rhabditis bovis* (Kassuku, personal communication, 1996). BPO is common in the coastal belt and some up country regions of Tanzania and Zebu cattle being more affected than dairy breeds (Msolla *et al.*, 1986). A tuft of hair in ears of Zebu cattle provide moist environment and warmth conducive for the nematode to multiply (Msolla *et al.*, 1986).

An act of pawing the ears by hooves is thought to introduce the nematode from soil to the ears especially in calf sheds in which the floors were of earth such as that of Mlali farm. Prevalence of 13% in calves in the rain season was attributed to muddy condition which allowed easy transfer of the nematode from soil to the clean ears. The disease at Mlali farm could also be related to the dominance of Zebu breed in the herd and perhaps unchecked concentration of the acaricide and continuous accumulation of manure in the kraal particularly in the rainy season. The disease was not observed in calves at Azam estate, Magadu dairy unit, SUA farm and Kingolwira Prisons farm.

#### **Bovine coccidiosis**

Coccidiosis caused by *Eimeria spp* was confined to young preweaned calves at Azam Estate although oocyst concentration was 50-100 per gram of faeces. These counts were too low compared to 80,000 to 100, 000 oocysts per gram of faeces which are required to cause clinical disease in calves (Blood *et al.*, 1990). The paddock within which the

calf house was located at Azam estate was water logged, thus, continuous wetting in the paddock provides favourable environment for multiplication of coccidia organisms (Fox, 1978). Roy (1983) indicated coccidiosis as one of the causes of mortality in weaned calves particularly in the intensive systems. It can be concluded from these findings that, clinical coccidiosis may not be a serious disease of calves in Morogoro, because even in samples from other farms where there was no clinical illness, oocyst counts per gram of faeces were between 50-100. Roy (1983) however, reported that coccidiosis and helminthosis were major causes of death among weaned calves in temperate countries due to confinement especially during winter. Climatic conditions in Morogoro and the management practises perhaps do not favour multiplication of coccidia in calves in the studied farms, resulting into low counts of *Eimeria* oocysts.

### **Bacterial otitis**

An otitis caused by beta-hemolytic *Escherichia coli* was observed in 2 calves. One calf was from SUA farm and another was from Mlali farm. These affected calves rotated their heads towards the infected ears. Besides the presence of this disease one calf concurrently had an omphalophlebitis and another had diarrhoea. Beta hemolytic *E. coli* was also isolated in pure culture from aspirated pus of the inflamed umbilicus. *Proteus spp* which is known to be a contaminant in the laboratory was isolated from faeces of the calf which had diarrhoea. An association of bacterial otitis and the observed intercurrent diseases could not be established.

However, infection of the middle ear occurs in young animals of all species especially pigs and to a less extent calves and lambs (Olson, 1982). Infectious agents gain entrance from the external ear but the spread is chiefly hematogenous especially in calves of 1-4 weeks of age (Blood *et al.*, 1990). In ruminants however, otitis media and interna is common and usually occur as a sequel to severe respiratory infection by *Pasteurella spp.* Purulent omphaloplebitis in the calf may have resulted in dissemination of infection into the ear canal through hematogenous route.

#### **Calf pneumonia**

None fatal cases of pneumonia characterized by purulent nasal discharges and infrequent cough were observed in 12% of weaned calves at KPF and 62% of weaned calves at Mlali farm during the months of June to August. During this period, 3 calves died of pneumonia at Azam estate and 1 died also of pneumonia at Magadu dairy. At Azam estate dead calves were less than 28 days old and had a history of failure to get colostrum due to problems pertinent to their dams.

Colostrum usually confer immunity to neonates against enteric and respiratory infections early in life (Corbeil *et al.*, 1984). Shoo *et al.* (1990) observed that, higher calf mortality in Morogoro farms occurred between June and August and were due to pneumonia. Shoo *et al.* (1990) further associated these mortalities with low environmental temperatures and draughts during the same period. The two factors

predisposes young animals to respiratory diseases.

At Mlali farm, calves were kept in a barn with neither wall nor concrete floor. This type of housing allows draughts and dust which predisposes calves to respiratory infections (Wathes *et al.*, 1983). *Post mortem* examination of the calf from Magadu dairy unit which was previously treated of pneumonia, revealed froth in the trachea extending down to the entire bronchial tree and tonsils were congested. Acute bronchopneumonia was the cause of death. *Pasteurella spp* was isolated in pure culture from nasal swabs of 4 calves from KPF and 13 from Mlali farm. The role of this bacterium in development of pneumonia in these calves could not be established. However, *Mycoplasma* and bacteria including *Pasteurella spp* are secondary invaders which exacerbate the initial pulmonary infection caused by viruses (Baker and Frey, 1985).

### **Helminthosis**

Helminthosis was characterized by soiling of the perineum with brownish soft faeces, normal appetite and normal rectal temperature. Calves aged 0-4 months were equally infested as those aged 4-6 months because of pasture contamination and uncontrolled grazing (Table 8). In semi intensive system (KPF), unweaned calves were grazed in paddocks also used to confine young calves during the day. Indoor calves of up to 1 month old particularly in intensive system (Azam Estate) were less likely infested compared to those aged 2-3 months grazing in paddocks also used by adult cattle

$P < 0.001$  (table 9). Therefore, as the calf grew older, chances of being infested with gastrointestinal nematodes becomes higher.

Common gastrointestinal nematodes frequently isolated from faeces of infested calves were shown in (Table 7). Sera of infected calves showed a decrease in serum globulin concentration, indicating that gastrointestinal worms causes a reduction of serum protein components, and calf deaths which were associated with helminthosis were observed in the dry season covering July to September (Table 10). *Toxocara vitulorum* was identified in faeces of calves of less than 4 weeks of age at Mlali farm where water buffaloes were grazed together with cattle. In Malaysia, water buffaloes grazing with cattle, were thought to be sources of infestation to cattle (Chandrawathani and Sani, 1989).

### **Calf Mortality**

Highest mortality of 25% and 17.4% were observed in farms practising semi intensive system. Lowest mortality of 7.8% was observed in free range system. Mortality rate of calves in the intensive system practised at Azam Estate (13.6%) and Magadu dairy unit (8.7%) could be explained by well planned disease control programmes, which included deworming before weaning, regular dipping and provision of supplemental feeds in the dry season. Low mortality rate observed in the free range management system could be explained by pasture grass reserves in the dairy season as a result of a vast area of grazing in this farm, continuous suckling of calves a system which was thought to boost

nutritional requirement of calves and possibly dominance of Zebu which are fairly resistant to tropical conditions.

Williamson and Payne (1978) associated low mortality of Zebu calves in the tropics with fair resistance of Zebu breeds to helminthoses and tick borne diseases compared to their exotic counterparts. In the tropics calf mortality was said to be as high as 50% (Williamson and Payne, 1978). Masanja and Matovelo (1993) reported mortality rates of 41.5% and 9.5% in calves at SUA farm and Magadu dairy unit respectively which differed slightly from values of 25% and 13.6% respectively obtained in this study. It seem therefore that, mortality rates in individual farms may vary from time to time depending on management, predisposing factors and severity of draught.

In this study, mortality of calves aged 0-4 months and that of calves aged between 4-6 months contributed equally to total calf mortality (Table 3).

The age of calves during which mortality occurred varied with the type of management system. In intensively managed farms (Azam Estate and Magadu dairy unit), mortality involved calves of up to 4 months of age. In semi intensively managed calves, for example at KPF which also accounted for 50% calf deaths in total, about 71% of calves that died were weaners aged 4-6 months. These finding implies that, while problems in rearing are confined to unweaned calves in the intensive system, problems inherent to calf rearing in semi intensive system affect weaned calves more than unweaned ones.

Weaned calf mortality particularly at KPF followed severe unthriftiness.

Three points were thought to have been associated with these mortalities. First, supplemental feeds have not been provided to unweaned calves in the dry season. Calves were therefore weaned with low body weights and hence affected by weaning stress and helminthosis. Secondly, no deworming regime before weaning at KPF and SUA farm as had been at Azam estate. Questionnaires revealed that, deworming depended upon financial situation. When calves were said to have been dewormed, drug administration and dosage rates were not as per manufacturers instructions as long as the operation was performed by labourers. The link between farmers and extension workers should therefore be improved to address such problem of drug administration, dose formulation and their outcomes. It appeared further that, the level of undernutrition in the dry season could be responsible for variable mortality rate in weaned calves.

Free range calves which also suckled for more than 6 months were nutritionally favoured compared to dairy calves which were given shares of milk twice a day before weaning at 3 month of age. Although the amount taken by the calf may not be known exactly especially in suckled calves, small amounts of milk boosts the calf nutritional requirement in the dry season. Free range management system is nonetheless recommended for beef cattle in tropical range lands than for dairy purposes. This is because, such a management system is too hostile to dairy breeds and perhaps due to

natural susceptibility of dairy breeds and their crosses to unsatisfactory management standards (Shaka, 1977).

It is therefore imperative that, acceptable level of calf rearing and disease control schemes should be directed to both preweaned and weaned calves alike in both intensive and semi intensive system for, failure to provide supplemental feed to calves during the dry season led into weaning of calves with low body weight as compared to those in the intensive system which were provided with hominy feed and hay and/or silage in the dry season and therefore did not face a period of weight loss. These findings concur with those of Williamson and Payne (1978) who observed that, the seriousness of parasitism in the tropics is the association between parasitic diseases and malnutrition. Parasitic gastroenteritis causes death or ill health when nutritional levels decline at the end of the rains and during the subsequent drought periods. Notwithstanding its seriousness, the relatively insidious nature of parasitic gastroenteritis, helminthosis lends itself amenable to second rate consideration with respect to allocation of resources for control measures especially in financially constrained institutional and state farms, while in fact, parasitic gastroenteritis is the main cause of unthriftiness and calf mortality.

## 6.0 CONCLUSION AND RECOMMENDATIONS

- ▶ Helminthosis coupled with weaning stress is a major cause of unthriftiness and mortality of weaned calves in semi intensively managed farms especially in the dry season.
- ▶ Calves weaned in the dry season are relatively at higher risk of death than calves weaned in the rain season. Supplemental feeds must be generously provided to calves weaned in the dry season.
- ▶ The association of calf diarrhoea and immunoglobulin concentration in calf serum and dam's colostrum is suggested.
- ▶ Mixed infections of coronavirus, rotavirus and *Escherichia coli* K 99 were common causes of calves diarrhoea in Morogoro farms, but rotavirus followed by *Escherichia coli* K 99 were frequent isolates.
- ▶ Coccidiosis was not a serious disease in weaned calves as it was in preweaned milk feeding calves in the intensive system.
- ▶ Epidemiology of calf diseases in pastoral herds at Morogoro need to be conducted.

**7.0 REFERENCES**

- Agyei, A.D. (1991) Epidemiological observation on helminth infections of calves in Southern Ghana. *Tropical Animal Health and Production* **23**, 134-140.
- Anderson, F.L.; Hammond, P. and Mincer, M.L. (1964) Response of immunized and non immunized calves to caecal inoculations of first generation merozoite of *Eimeria bovis*. *Journal of Parasitology* **50**, 209-213.
- Anderson, N.; Morris, R.S. and McTaggart, I.K. (1976) An economic analysis of two schemes for the anthelmintic control of helminthiasis in weaned lambs. *Australian Veterinary Journal* **52**, 174-178.
- Baker, J.C. and Frey, M.L. (1985) Bovine respiratory syncytial virus. *Veterinary clinics of North America, Food animal Practice* **1**, 259-275.
- Bancroft, J.D. and Stevens, A. (1990) *Theory and practice of histological techniques*. Ed 3. Churchill Livingstone Publishers, London. pp 726.
- Barber, D.M.L. (1976) Assessment of immune globulin status. *Veterinary Record* **98**, 121.

- Blom, J.Y. and Thyssen, I. (1980) Klimates indflydelse pa kalves sundhed. In: *Berning for statens husdyrbrugsforsog* (Edited by Ostergaard, B. and Hindhede J. Kobenhavn. pp 138-153. (Abstract)
- Blood, D.C.; Radostits, O.M. and Henderson, J.A. (1990) *A text book of the diseases of cattle, sheep, pigs, goats, and horses*. Edition 6, Bailliere Tindall, London. pp 215-224.
- Boyd, J.W.; Baker, J.R. and Leyland, A. (1974) Neonatal diarrhoea in calves. *Veterinary Record* 95, 310-313.
- Burridge, M.T.; Morzaria, S.P.; Cunningham, M.P. and Brown, C.B.G. (1975) Duration of immunity to East Coast Fever (*Theileria parva*) infections in cattle. *Parasitology* 25, 213-226.
- Bywater, R.J. (1977) Evaluation of an oral glucose-glycine electrolyte formulation and Amoxicillin for treatment of diarrhoea in calves. *American Journal of Veterinary Research* 38, 1983-1987.
- Campos, R.R.; Liebano, H.E.; Herrera, R.D. and Godinez, G.A. (1990) Identification of gastrointestinal nematode larvae in bovine faeces in the cattle rearing area of

Morelos state. *Veterinaria Mexico* 21, 415-417.

Castrucci, G.; Frigeri, F.; Ferrari, M.; Cilli.; Caleffi, F.; Aldrovandi, V. and Nigrelli, A. (1984) The efficacy of colostrum from cows vaccinated with rotavirus in protecting calves to experimentally induced rotavirus infection. *Comparative Immunology, Microbiology and Infectious Diseases* 7, 11-18.

Castrucci, G.; Frigeri F.; Ferrari, M.; Aldrovandi, V.; Tassim, F. and Gatti, R. (1988) Protection of newborn calves against experimental rotavirus infection by feeding mammary secretions from vaccinated cows. *Microbiologica II*, 379-385.

Chandrawathani, P. and Sani, R.A. (1989) Incidence of gastrointestinal parasites in Kedah-Kelantan calves. *Jurnal Veterinar Malaysia* 1, 27-31.

Chibunda, R.T. (1985) An epidemiological study of Eimeriosis in selected cattle farms in Morogoro Municipality. A special project submitted in fulfilment for the award of the degree of Bachelor of Veterinary Medicine, Sokoine University of Agriculture.

Cole, A.M. (1970) The aetiology of calf pneumonia in Queensland. *Australian Veterinary Journal* 46, 569-575.

- Corbeil, L.B.; Watt, B.; Corbeil R.R.; Betzen, T.; Brownson, R.K. and Morrill, J.L. (1984) Immunoglobulin concentration in serum and nasal secretions of calves at the onset of pneumonia. *American Journal of Veterinary Research* **45**, 773-778.
- Corley, L.D.; Staley T.E.; Bush, L.J. and Jones, E.W. (1977) Influence of colostrum on transepithelial movement of E.Coli 055. *Journal of Dairy Science* **60**, 1416.
- Curtis, S.E. and Drummond, J.G. (1982) *Handbook of Agricultural productivity II. Animal productivity*. Edited by Reicheigh, M. CRC Press, Florida. pp 107.
- Das, S.M.; Mgheni, M.; Kitanyi, J.I. and Mkonyi, J.I. (1988) Genetic and environmental factors affecting viability of improved *Bos indicus* calves in central Tanzania. *Proceedings of the 6th Tanzania Veterinary Association Scientific Conference*. **6**, 87-92.
- Dawson, P.S.; Darbyshire, J.H.; Lamont P.; P.H. and Patterson, A.B. (1964) Pneumonia in calves caused by parainfluenza-3 virus. *Veterinary Record* **76**, 434-435.
- Donald, A.D.; Southcott, W.H. and Dineen, J.K. (1978) The epidemiology and control of gastrointestinal parasites in sheep in Australia. *Commonwealth Scientific and*

Industrial Organization Publications, Melbourne. pp 132.

Dumas, B.T.; Watson, W. and Biggs, G.H. (1971) Albumins standards and the measurements of serum albumin with Bromocresol green. *Clinica Chimica Acta* 31, 87-96.

Fadok, Y.A. (1984) Parasitic diseases of large animals. *Veterinary Clinics of North America*. Large animal Practise 6, 3-262.

Fakae, B.B. (1990) The epidemiology of helminthosis in small ruminants under traditional husbandry system in Nigeria. *Veterinary Research Communication* 14, 381-392.

Fallon, R.J. and Harte, F.J. (1987) *Calf feeding and Management*. Beef Series No 1. An Foras Taluntais Publishers, Ireland. pp 34.

Fanelli, H.H. (1983) Observation on nervous coccidiosis in calves. *Bovine Practitioner* 18, 50-53.

Fitzgerald, P.R. (1972) "*Economics of bovine coccidiosis*". *Feedstuffs*, Minneapolis 44, 28-30.

- Fitzgerald, P.R. (1975) The significance of bovine coccidiosis as a disease in United States of America. *Bovine Practitioner* 10, 28-30, 32-33.
- Fitzgerald, P.R. and Mansfield, M.E. (1972) The economic impact of coccidiosis in domestic animals. *American Journal of Veterinary Research* 33, 1931.
- Fox, J.E. (1978) Bovine coccidiosis review including field safety studies with decoquinoate for prevention. *Modern Veterinary Practice* 59, 599-603.
- Gay, C.C.; Anderson, N.; Fisher, E.W. and McEwan, A.D. (1965) Gammaglobulin levels and neonatal mortality in market calves. *Veterinary Record* 77, 148-149.
- Gregory, M.W.; Joyner, L.P. and Catchpole, J. (1982) Medication against ovine coccidiosis. A review. *Veterinary Research Communication* 5, 307-325.
- Hall, G.A.; Reynolds, D.J.; Parssons, R.K.; Bland, A.P. and Morgan J.A. (1988) Pathology of calves with diarrhea in Southern Britain. *Research in Veterinary Science* 45, 240-250.
- Halliday, R. (1978) Immunity and health in young lambs. *Veterinary Record* 103, 489.

- Hansen, J. and Perry, B. (1994) *A Handbook of the Epidemiology Diagnosis and Control of Helminth and Parasites of Ruminants*. ILRAD, Nairobi. pp 236.
- Harrold, J.G. and Hassan, A.B. (1984) A study of the aetiology, epidemiology and control of calf diarrhoea in Ireland. *Irish veterinary Journal* 38, 63-67.
- Hartmann, D.A.; Everett, R.W.; Salck, S.T. and Werner, R.G. (1974) Calf mortality. *Journal of dairy Science* 57, 576-578.
- Hateren, A.D.; Cuperns, T. and Walgemoed, J. (1985) Is coccidiosis in adult cattle an exception. *Tidshrift-voor-Diergeneeskunde* 110 (4), 558-559.
- Herd, R.H.; Parker, C.F.; McClure, K.E. (1984) Epidemiologic approach to the control of sheep nematodes. *Journal of American Veterinary Medical Association* 184 (6), 680-687.
- Hinton, M. and Linton, A.H. (1983) Antibacterial drug resistance among *Escherichia coli* isolated from calves fed on a milk substrate diet. *Veterinary Record* 112, 567-568.
- Holmes, P.H. (1985) Pathophysiology of parasite infections. *Parasitology* 94, 829-551.

Inaba, Y.; Tanaka, Y.; Sato, K.; Omori, T. and Matumoto, M. (1972) Bovine respiratory syncytial virus, studies on an outbreak in Japan. *Japanese Journal of Microbiology* 16, 373-383.

International Committee for Standardisation in Haematology (1967) Recommendations and requirements for haemoglobinometry in human blood. *British Journal of haematology* 13, Supplement 71.

James, R.E. and Polan, C.E. (1978) Effect of orally administered duodenal fluid on serum proteins in neonatal calve. *Journal of Dairy Science* 61, 1444.

Jenssen, J.B. and Miner, M.L. (1976) Decoquinoate in the control of experimentally induced coccidiosis in calves. *American Journal of Veterinary Research* 37, 1043-1045.

Jibbo, J.M.C. (1966) Bovine parasitic otitis. *Bulletin of Epizootic diseases in Africa* 14, 56-63.

Jones, C.R. and Webster, A.J.F. (1981) Weather induced changes in air borne bacteria within a calf house. *Veterinary Record* 109, 493-494.

- Jubb, K.V.F.; Kennedy, P.C. and Palmer, N. (1992). *Pathology of domestic animals*. Ed 4, vol. 2. Academic press Inc. New York. pp 747.
- Kahrs, R.F. (1981) *Viral Diseases of cattle*. Iowa State University Press, Ames, Iowa. pp 299.
- Kennedy, D.J.; Greenberg, R.N.; Dunn, J.A.; Abernathy, R.; Ryerse, J.S.; Guerrant, R.L. (1984) Effects of *E.coli* Heat stable enterotoxin b on intestines of mice, rats, rabbits and piglets. *Infectious immunology* 46, 639.
- Kifaro, G.C. and Temba, E.A. (1990) Calf mortality and culling rates in two dairy farms in Iringa region, Tanzania. *Proceeding of the Scientific conference of the Tanzania Society of Animal Production vol 17*: 138-148.
- Komba, G.L. (1992) Problems associated with cost sharing in the provision of Veterinary services in Tanzania. In: *The role of veterinary Profession of Animal health and production*. ED Mbassa G.K, SUA, Morogoro. pp 25-27.
- Kreiss, H.A. (1964). In: Epidemiology of Bovine parasitic otitis. Eds Msolla, P.; Matafu, E.P.M. and Monrad, J. (1986) *Tropical Animal Health and Production Journal* 18, 51-52.

- Kruse, V. (1970) Absorption of immunoglobulin from colostrum in newborn calves. *Animal Production* 12, 627-638.
- Landsverk, T. (1978) Indigestion in calves.IV. Lesions of ruminal papillae in young calves fed barley and barley plus hay. *Acta Veterinaria Scandinavica* 19, 377-391.
- Lapage, G. (1968) *Veterinary Parasitology*. Ed " Oliver, J. and Boyd, G. London. pp 935-978.
- Lema, B.E. and Banda, G. (1991) Infectious causes of calf diarrhoea in selected regions of Tanzania. *Proceedings of the Ninth Tanzania Veterinary Association Scientific Conference, held at Arusha in dec.1991.* pp 216-221.
- Levine, D.M. (1973) *Protozoan parasites of domestic animals and man*. Edition 2. Burgess Publishing Company. Minneapolis, Minnesota. pp 156-157, 179-181.
- Levine, D.M. (1980) Protozoan of Veterinary importance. *Journal of Protozoology* 27, 37-58.
- Lipova, E. (1985) Coccidia and coccidiosis of calves in large scale calf barns.

*Veterinastvi* 35 (7), 310-311.

Logan, E.F. and Gibson, T. (1975) Serum immunoglobulin levels in suckled beef calves. *Veterinary Record* 97, 229.

Logan, E.F.; Muskett, B.D. and Herron, R.J. (1981) Colostrum feeding of dairy calves. *Veterinary Record* 108, 283-284.

Logan, E.F.; Pearson, G.R. and McNulty, M.S. (1977) Studies on the immunity of the calf to colibacillosis. 7. The experimental reproduction of colibacillosis in colostrum fed calves. *Veterinary Record* 101, 433.

Logan, E.F. and Penhale, W.J. (1972) Study on the immunity of the calf to colibacillosis. III. The local protective activity of colostrum within the gastrointestinal tract. *Veterinary Record* 89, 628-632.

Lopez, J.W.; Allen, S.D.; Mitchell, J. and Quinn, M. (1988) Rotavirus and *Cryptosporidium* shedding in dairy calf faeces and its relationship to colostrum immune transfer. *Journal of Dairy Science* 71, 1288-1294.

Mage, C.; Faeces, P. and Chasteloux, C. (1990) Coccidiosis in suckled limousine

calves. *Revue de Médecine-Vétérinaire* 141 (8-9), 671-676.

MALD, (1984) Ministry of Agriculture and Livestock Development, National Livestock Census, Dar es salaam.

MALD, (1987) Ministry of Agriculture and Livestock Development. Review of ruminant livestock industry. Dar es salaam, Tanzania.

MALD, (1993) Ministry of Agriculture and Livestock Development. Basic data. Agriculture and Livestock sector 1986/87- 1991/92. URT Mainland, Dar es salaam p 134-202.

Masanja, S.L. and Matovelo, J.A. (1993). Pathological and aetiological observations associated with calf mortality in herds at Sokoine University of Agriculture, Morogoro, Tanzania. *Tanzania Veterinary Journal* 13 (2), 27-35.

Mattson, D.E. (1973) Adenovirus infection in calves. *American Journal of Veterinary Medical Association* 163, 894-896.

Marquardt, W.C. (1962) Subclinical infections with coccidia in cattle and their

transmission to susceptible calves. *Journal of Parasitology* 48, 270-275.

Marshall, R.G. and Frank, G.H. (1975) Clinical and immunological responses of calves with colostral acquired maternal antibody against parainfluenza 3 virus. *American Journal of Veterinary Research* 32, 1699-1706.

Mbassa, G.K. (1994). Driving and state variables of immunity to Theileriosis in Ankole Zebu calves in Lake Victoria basin. *The Kenyan Veterinarian* 18 (2), 367.

Mbassa, G.K. and Silayo, R.S. (1995) The effect of massive natural *Theileria parva* infections on the trivalent sporozoite Vaccine in Epidemiologically unstable areas. *Tanzania Veterinary Journal* 15 (2), 60-71.

McBeath, D.G.; Penhale, W.J. and Logan, E.F. (1971) An examination on the influence of husbandry on the plasma immunoglobulin level of the newborn calf using a rapid refractometer test for assessing immunoglobulin content. *Veterinary Record* 88, 266-270.

McEwan, A.D.; Fisher, E.W.; Selman, I.E. and Penhale W.J. (1970) A turbidity test for the estimation of immune globulin levels in neonatal calf serum. *Clinica Chimica Acta* 27, 155-163.

Mchechu, J.E.U. (1983) Present status of Livestock Industry in Tanzania. *Proceedings of the Tanzania Society of Animal Production* **10**, 8-43.

McNulty, M.S.; McFerran, J.B.; Bryson, D.G.; Logan, E.F. and Curran, W.L. (1976) Studies on Rotavirus infection and diarrhoea in young calves. *Veterinary Record* **99**, 229.

Mebus, C.A. (1976) Viral enteritis of calves. *Journal of Dairy Science* **59**, 1175.

Mebus C.A.; Newman, L.E. and Stair, E.L. (1975) Scanning electron light and immunofluorescent microscopy of intestine of a gnotobiotic calf infected with calf diarrheal coronavirus. *American Journal of veterinary Research* **36**, 1719-1729.

Mebus, C.A.; Underdahl, N.R.; Rhodes, M.B.; and Twiehaus, M.J. (1969) Calf diarrhea (Scours): Reproduced with a virus from a field outbreak. *University of Nebraska Research Bulletin* **233**, 1-16.

Mebus, C.A.; Stair, E.L.; Underhal, N.R.; and Twiehaus, M.J. (1971) Pathology of neonatal calf diarrhea induced by reo-like virus. *Veterinary Pathology* **8**, 490-505.

- Mgassa, M.N. (1991) Orthopaedic diseases related to nutrition and forestomach pathology in ruminant. PhD Thesis. p 14-32.
- Mitchell, C.D.; McCoy, G.C. and Olson, H.H. (1974) Influence of colostrum feeding on serum protein constituents of neonatal calves. *Journal of Dairy Science* **57**, 642.
- Moll, G.; Lohding, A.; Young, A.S. and Leitch, B.L. (1986) Epidemiology of theileriosis in calves in an endemic area of Kenya. *Veterinary Parasitology* **19**, 255-273.
- Moon, H.W.; Woode, G.N. and Ahrens, F.A. (1982) Attempted chemoprophylaxis of Cryptosporidiosis in calves. *Veterinary Record* **110**, 181.
- Mpelumbe, I.S. (1984) Current animal diseases situation in Tanzania. *Proceeding of the Second Tanzania Veterinary Association Scientific Conference* **2**, 1-11.
- Mpiri, D.B.; Yongolo, M.G.S. and Wella, E.B. (1986) An analysis of bovine mortality at LPRI Mpwapwa. Paper presented at 13th Scientific Conference of Tanzania Society of Animal Production Vol 13, December 1987, Arusha, Tanzania.

- Msolla, P.; Matafu, E.P.M. and Monrad, J. (1986) Epidemiology of Bovine Parasitic Otitis. *Tropical Animal Health and Production Journal* 18, 51-52.
- Mukhebi, A.W.; Perry, B. and Kurska, R. (1992) Estimate economics of theileriosis control in Africa. *Preventive Veterinary Medicine* 12, 73-85.
- Murray, M. and Gray, A.R. (1984) The current situation on animal trypanosomiasis in Africa. *Preventive Veterinary Medicine* 2, 23-30.
- Mwasomola, A.T.M. (1983) The study of coccidia in calves. A special project submitted in fulfilment for the award of the degree of Bachelor of Veterinary Science, University of Dar es salaam.
- Nagy, B. (1980) Vaccination of cows with K 99 extract to protect newborn calves against experimental enterotoxin colibacillosis. *Infection and Immunity* 27 (1), 21-24.
- Ngomuo, A.J.; Kassuku, A.A. and Boa, M.E. (1994) The prevalence of gastrointestinal nematodes at Sokoine University of Agriculture and their susceptibility to Levamisole preparations. *Tanzania Veterinary Journal* 14 (3-4), 170-180.

- Nielsen, K. (1976) Pathophysiology of Parasitic infections. Plasma protein metabolism. In: *Pathophysiology of parasitic infection*, Edited by Soulsby, E.J.L.; Academic Press, New York. pp 23-40.
- Njau, B.C. (1987) Gastrointestinal nematodes of small ruminants at King'ori in Northern Tanzania. *Bulletin of Animal Health and production in Africa* 35, 298-303.
- Olson, L.D. (1982) Gross and microscopic lesions of the middle and inner ear infections in swine. *American Journal of Veterinary Research* 42, 1433-1440.
- Oxender, W.D.; Newman, L.E. and Morrow D.A. (1973) Factors influencing dairy calf mortality in Michigan. *Journal of American Veterinary Medical Association* 162, 458-460.
- Penhale, W.J.; Logan, E.F.; Selman, I.E.; Fisher, F.W. and McEwan, A.D. (1973) Observation on the absorption of colostral immunoglobulin by the neonatal calf and their significance in colibacillosis. *Annals Recherche Veterinaire* 4, 223.
- Peters, A.R. (1986) Some husbandry factors affecting mortality and morbidity on a calf rearing unit. *Veterinary Record* 119, 355-357.

- Phillippo, M.; Arthur, J.R.; Price, J.; and Halliday, G.J. (1987) The effects of selenium, housing and management on the incidence of pneumonia in housed calves. *Veterinary Record* 121, 509-512.
- Ploger, W.; Buitkamp, J.; Newmann, W.; Bechmann, G. and Reuss U. (1980) Untersuchungen uiber die ursachen der kalersteblichkeit im nordwest deustchen kurstengebiet. *Tierarztl umschau* 35, 659-671 (Abstract).
- Porter, P. (1972) Quantitative changes in early lactation and absorption by the neonatal calf. *Immunology* 23, 225.
- Pradhan, K.B.; Thakur, D.K. and Sudhan, N.A. (1991) Hemato-biological changes in calves with natural helminthic infection in Ranchi. *Journal of Research. Birsa Agricultural University* 3, 119-121.
- Pritchard, D.G.; Carpenter, C.A. and Morzaria, S.P. (1981) Effect of air filtration on respiratory diseases in intensively housed veal calves. *Veterinary Record* 109, 5.
- Radostits, O.M. and Blood, D.C. (1985) Aspects of Dairy Cattle Nutrition and Housing. *Herd Health. A text book of Health and Production Management of Agricultural Animals*. Edited by Radostits O.M and Blood, W.B. Saunders

Company. Toronto. p 173-188.

Radostits, O.M. and Stockdale, P.H.G. (1980) A brief review of coccidiosis in western Canada. *Canadian Veterinary Journal* 21, 227-230.

Randall, J.M. (1981) *Environmental Aspects of Housing for animal production*. Edited by J.A Clark. London. pp 351.

Reid, W.M. (1975). Progress in the control of coccidiosis with anticoccidials and planned immunization. *American Journal of Veterinary Research* 36, 593-596.

Reynolds, D.J.; Morgan, J.H.; Chanter, N.; Jones, P.W.; Bridger, J.C.; Debney, T.G. and Bunch, K.J. (1986) Microbiology of calf diarrhoea in Southern Britain. *Veterinary Record* 119, 34-39.

Rickard, L.G. and Zimmerman, G.L. (1992) Epizootiology of gastrointestinal nematodes of cattle in selected areas of Oregon. *Veterinary Parasitology* 43, 271-291.

Roe, C.P. (1982) A review of the environmental factors influencing calf respiratory disease. *Agricultural Meteorology* 26, 127-144.

- Rose, R.; Whipp, S.C. and Moon, H.W. (1987) Effects of *Escherichia coli* heat stable enterotoxin b on small intestine villi in pigs, rabbits and lambs. *Veterinary Pathology* 24, 71-79.
- Roy, J.H.B. (1980) Factors affecting susceptibility of calves to disease. *Journal of dairy Science* 63, 650-664.
- Roy, J.H.B. (1983) Problems of calf rearing in connection with their mortality and optimal growth: A review. *Livestock Production Science* 10, 339-349.
- Roy, J.H.B. (1990) *The Calf. Management of health*. Edition 5 Vol. 1, Philadelphia Publishers, Philadelphia. pp 258.
- Ruiz, A.V. (1973a) The seasonal incidence of coccidial infections in cattle of different ages in South Dakota- USA. *Veterinary Bulletin* 44, 93.
- Ruiz, A.V. (1973b) On the natural history of the coccidial infections in range and feeder cattle in West Germany. *Veterinary Medicine* 20, 594-602.
- Saif, L.J. and Smith, K.L. (1985) Enteric infections of calves and passive immunity. *Journal of Dairy Science* 68, 206-228.

- Schillhorn van Veen T.W. (1986) Coccidiosis in ruminants. *Compendium of Continuing Education* 8 (16), F52-F58.
- Schrag, C.H. (1968) Incidence of coccidia in cattle, sheep and goats in some regions of South and East africa. *Veterinary Bulletin* 39, 331.
- Scott, D.W. (1988) *Large animal dermatology*. W B Saunders Co. Philadelphia. pp 172-182.
- Selim, S.A.; Aziz, K.M.S.; Sarker, A.J. and Rahman, H. (1991). Rotavirus infection in calves in Bangladesh. *Veterinary Research communications* 15 (4), 327-333.
- Selman, I.E.; McEwan, A.D. and Fisher, E.W. (1970) Serum immune globulin concentrations of calves left with their dams for the first two days of life. *Journal of Comparative Pathology* 80, 419-427.
- Shaka, S. (1977) Disease control in dairy cattle with particular reference to production diseases. *Proceedings of the Tanzania Society of Animal Production Scientific conference Vol 4*, 90-113.
- Sharma, K.N.S. and Jain, D.K. (1976) Mortality in cross bred calves vis-a-vis zebu

calves. *Indian Journal of Dairy Science* 29 (1), 53-58.

Shoo, M.K.; Semvua, R.H.; Kazwala, R.R. and Msolla, P. (1990) Dairy calf mortality rates, causes of deaths and associated factors on farms in Eastern zone of Tanzania. *Proceedings of the 8th Tanzania Veterinary Association Scientific Conference held at Arusha, Tanzania* 8 158-163.

Simensen, E. (1982) An epidemiological study of calf health and performance in Norwegian dairy herds. 1. Mortality: Literature review, rates and characteristics. *Acta Agriculture Scandinavica* 32, 411-419.

Simensen, E. and Norheim, K. (1983) An epidemiological study of calf health and performance in Norwegian dairy herds IV. Factors affecting morbidity and performance. *Acta Agriculture Scandinavica* 33, 65-74.

Skandar, Q.F (1973) Frequency of coccidiosis in cattle and identification of *Eimeria* species in Mexico. *Veterinary Bulletin* 44, 406.

Snodgrass, D.R.; Terzolo, H.R.; Sherwood, D.; Campbell, I.; Menzies, J.D and Syngé, A.D (1986) Aetiology of diarrhoea in young calves. *Veterinary Records* 119, 31-34.

- Soulsby, E.J.L. (1982) *Helminths Arthropods and Protozoa of Domesticated Animals*. Edition 7, Bailliere Tindall, London. pp 809.
- Spooner, R.L.; Bradley, J.S. and Young, G.B. (1975) Genetics and diseases in domestic animals with particular reference to dairy cattle. *Veterinary Record* **97**, 125.
- Stott, G.H. and Fellah, A. (1983) Colostral immunoglobulin absorption linearly related to concentration of calves. *Journal of Dairy Science* **66**, 1319-1328.
- Stott, G.H.; Marx, D.B.; Meneffe, B.E. and Nightingale, G.T. (1979) Colostral immunoglobulin transfer in calves 1V. Effect of Suckling. *Journal of Dairy Science* **62**, 1908-1913.
- Suarez, V.H.; Busetti, M.R. and Fort, M.C. (1992) Epidemiology and effects of nematodes infections on beef cow-calf systems of Argentina western Pampas. *Veterinary Parasitology* **42**, 73-81.
- Svensson, C. (1993) Peripartal excretion of *Eimeria* oocyst by cows on Swedish dairy farms and the age of the calves at first excretion. *Acta Veterinaria Scandinavica* **34** (1), 77-81.

- Sykes, A.R. (1982) Nutritional and Physiological Aspects of helminthiasis in sheep. In: *Biology and control of Endoparasite*. Edited by Symmons L.E.A.; Donald A.D and Dineen, J.K. p 217-232.
- Symons, L.E.A. (1976) Malabsorption. In: *Pathophysiology of parasitic infections*. Edited by Soulsby E.J.L.; Academic Press, New York pp 11-21.
- Taira, N. and Fujita, J. (1991) Morphological observation of *Taxocara vitulorum*, new record found in Japanese calves. *Journal of Veterinary Medical Science* 53, 409-414.
- Tennant, B.; Ward, D.E.; Braun, R.K.; Hunt, E.L. and Baldwin, B.H. (1978) Clinical management and control of neonatal enteric infections in calves. *American Journal of Veterinary Medical Association* 173, 654-661.
- Ternourth, J.H.; Roy, J.H.B. and Shotton, S.M. (1976) Concurrent studies of the flow of digesta in the duodenum and exocrine pancreatic secretion of calves. 4. The effects of age. *British Journal of Nutrition* 36, 523.
- Tizard, I.R. (1987) *An Introduction to Veterinary Immunology*. Edition 3. W.B Saunders Company. Philadelphia. 365-366.

- Turnbull, J.E. (1980) Housing and Environment for Dairy Calves. *Canadian Veterinary Journal* **21**, 85-90.
- Tzipori, S. (1981) The aetiology and diagnosis of calf diarrhoea. *Veterinary Record* **108**, 510-514.
- Uilenberg, G. (1987) Theileria species of domestic animals. In: *Advances in the control of Theileriosis*. Edited by A.D. Irvin, M.P. Cunningham and A.S. Young. Martinus Nijhoff, The Hague. p 4-37.
- Vassiliades, G. (1969) Intestinal coccidiosis of domestic ruminants in Senegal. Epidemiology, geographical distribution and economic importance. *Veterinary Bulletin* **39**, 832.
- Waruiru, R.M. and Mbutia, C.O. (1991) Prevalence of gastrointestinal parasites and liver flukes in calves in Mathira division of Nyeri district Kenya. *Bulletin of Animal health and Production in Africa*, XL 1 (4), 70-73.
- Wathes, C.M.; Jones, C.D.R. and Webster, A.J.F. (1983) Farm animal Housing. Ventilation, air hygiene and Animal Health. *Veterinary Record* **113** 554-559.

- Webster, A.J.F. (1981) *Environmental Aspects of Housing for Animal Production*. Edited by J.A.Clark. London Philadelphia. p 217.
- Whipp, S.C.; Moseby, S.L. and Moon, H.W. (1986) Microscopic alterations in jejunal epithelium of 3 weeks old pigs induced specific mouse negative heat stable *Escherichia coli* enterotoxin. *American Journal of Veterinary Research* 47, 615.
- Williamson, G. and Payne, W.J.A. (1978) *An introduction to animal Husbandry in the tropics*. Edition 2. Longmans, London. p 31, 171.
- Withers, F.W. (1952) Mortality rates and disease incidence in calves in relation to feeding, management and other environmental factors. *British Veterinary Journal* 108, 472.
- Woode, G.N.; Jones, J. and Bridger, J. (1975) Levels of colostral antibodies against neonatal calf diarrhoea virus. *Veterinary Record* 97, 148-149.
- Wright, G.W. (1966) *American Review of Respiratory Diseases* 93, Supplement 103.
- Yeoman, G.H. (1966) Field vector studies of epizootic East Coast Fever 1. A quantitative relationship between *Rhipicephalus appendicularis* and the

epizooticity of East Coast Fever. *Bulletin of Epizootic Diseases of Africa* 14, 5-27.

Young, A.S.; Brown C.G.D.; Burrige, M.J.; Cunningham, M.P.; Kiriimi, I.M. and Irvin, A.D. (1973). Observations on the cross immunity between *Theileria lawrencei* (Serengeti) and *Theileria parva* (Muguga) in cattle. *International Journal of Parasitology* 3, 723-728.