FREIE UNIVERSITÄT BERLIN - ADDIS ABABA UNIVERSITY

STUDIES ON PREVALENCE AND ECONOMIC IMPACT OF POULTRY COCCIDIOSIS IN DIFFERENT PRODUCTION SYSTEMS IN DEBRE ZEIT AND ADDIS ABABA, ETHIOPIA

A thesis submitted in partial fulfilment for the degree of Master of Science in Tropical Veterinary Epidemiology at the Freie Universität Berlin and Addis Ababa University

by

Safari Methusela Kinung'hi

December, 2001

STUDIES ON PREVALENCE AND ECONOMIC IMIPACT OF POULTRY COCCIDIOSIS IN DIFFERENT PRODUCTION SYSTEMS IN DEBRE ZEIT AND ADDIS ABABA, ETHIOPIA

by

Safari Kinung'hi Methusela

Board of Examiners

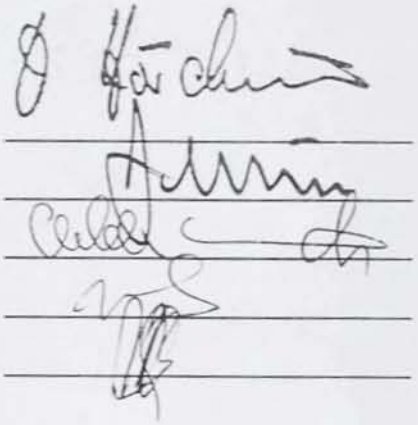
Professor Dr. F. Hörchner

Professor Dr. K.-H. Zessin

Professor Dr. G. Hildebrandt

Dr. Getachew Tilahun

Dr. Bayleyegn Molla

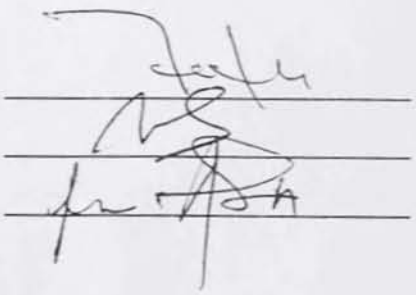


Academic Advisors

Prof. Hafez

Dr. Getachew Tilahun

Dr. Moges Woldemeskel



Academic Advisors:

Prof. Dr. H. M. Hafez

Freie Universität Berlin
Fachbereich Veterinärmedizin
Institut für Geflügelkrankheiten
Germany

Dr. Getachew Tilahun

Faculty of Veterinary Medicine
Addis Ababa University
Debre Zeit
Ethiopia

Dr. Moges Woldemeskel

Faculty of Veterinary Medicine
Addis Ababa University
Debre Zeit
Ethiopia

DEDICATION

This thesis work is dedicated to:

My family; my father, the late Methusela Kinung'hi and my mother Minza Mhameji, and my brothers and sisters.

My colleagues in the veterinary profession, especially parasitologists and epidemiologists whose devotion and commitment relieve disease burden and suffering from animals and ensure their optimal health, productivity and welfare.

Almighty God who gives us life, good health, love and energy to perform our daily duties.

TABLE OF CONTENTS page

TABLE OF CONTENTS i
LIST OF TABLES iii
LIST OF FIGURES iv
LIST OF APPENDICES v
LIST OF ABBREVIATIONS vi
ACKNOWLEDGEMENTS vii
ABSTRACT viii

1. INTRODUCTION AND OBJECTIVES 1

2. LITERATURE REVIEW 3
2.1. General considerations 3
2.1.1. Definition and aetiology 3
2.1.2. Epidemiology 3
2.1.3. General life cycle 4
2.1.4. Immunity to coccidia infections 6
2.2. Coccidiosis in chicken 7
2.2.1. Species of coccidia in chicken 7
2.2.2. Distribution and occurrence of the agent and the disease 13
2.2.3. Risk Factors for Coccidia Infection in Chicken 13
2.2.3.1. Agent related factors 13
2.2.3.2. Host related factors 15
2.2.3.3. Environmental related factors 16
2.2.3.4. Management related factors 17
2.2.4. Transmission and Susceptibility 19
2.2.5. Pathogenicity, pathogenic effect and host-parasite relationship 20
2.2.6. Diagnosis of chicken coccidiosis 21
2.2.7. Treatment, Prevention and Control of Chicken Coccidiosis 23
2.2.8. The Economic Impact of Coccidiosis on the Poultry Industry 25

3. MATERIALS AND METHODS 27
3.1. Description of study area and study population 27
3.1.1. The study area 27
3.1.2. The study population 27
3.2. Study design 28
3.2.1. Cross sectional study 28
3.2.1.1. Parasitological examination of faecal samples 28
3.2.1.2. Antemortem and postmortem examination of birds 29
3.2.1.3. Identification of species of coccidia 30
3.2.1.4. Questionnaire survey and assessment for potential risk factors for coccidiosis 31
3.2.2. Retrospective study 31
3.2.2.1. Qualitative assessment 31
3.2.2.2. Quantitative assessment 32
3.3. Sample size determination for cross sectional study 32

3.4. Sampling procedures 33
3.5. Data management and analysis 33

4. RESULTS 34
4.1. Cross sectional study 34
4.1.1. Parasitological examination of faecal samples 34
4.1.2. Antemortem and postmortem examination of birds 35
4.1.3. Identification of species of coccidia. 40
4.1.4. Gross and histopathological changes associated with coccidiosis in chicken 42
4.1.5. Questionnaire survey and assessment for potential risk factors for coccidiosis ... 46
4.1.5.1. Farm management practices 46
4.1.5.2. Potential risk factors for coccidiosis 46
4.2. Retrospective study 48
4.2.1. Assessment of the economic impact of coccidiosis 48

5. DISCUSSION AND CONCLUSIONS 53
5.1. Discussion 53
5.2. Conclusion 57
5.3. Recommendations 58

6. REFERENCES 60

7. APPENDICES 69
Appendix 7.1. Questionnaires 67
Appendix 7.2. Summery of responses from questionnaire survey 73
Appendix 7.3. Gross margin analysis for quantification of economic losses 75
Appendix 7.4. Summary of poultry farms and and samples 76
Appendix 7.5. Curriculum vitae 78
Appendix 7.6. Signed declaration sheet 80

LIST OF TABLES page

Table 1. Frequency of detection of coccidia oocysts in faecal samples from different production and rearing systems 34

Table 2. Frequency of detection of coccidiosis lesions in intestinal tracts of necropsied birds from different production systems 35

Table 3. Proportions of clinical and subclinical coccidiosis in small scale and large scale poultry production systems by age groups 38

Table 4. Proportions of severe and mild/moderate lesions compared to the occurence of clinical and subclinical coccidiosis in different production systems.....40

Table 5. Prevalent species of chicken coccidia identified at necropsy examination of birds from different production systems 41

Table 6. Distribution of coccidia species in chicken in small scale and large scale poultry farms 42

Table 7. Cross-tabulation for potential risk factors against occurence of coccidiosis in small scale and large scale farms.....47

Table 8. Quantification of economic losses resulting from coccidiosis in small scale and large scale poultry farms 48

Table 9. Estimation of farmers incorne from poultry keeping enterprises in small scale and large scale poultry farms 49

LIST OF FIGURES page

Figure 1. Comparison between proportions of clinical and subclinical coccidiosis among different poultry production systems.....36

Figure 2. Comparison between frequency of detection of coccidia oocysts in faecal samples and occurence of coccidiosis lesions in the gastro intestinal tracts of birds necropsied from different production systems 37

Figure 3. Comparison between proportions of clinical and subclinical coccidiosis among different age groups of chicken in small scale and large scale poultry farms.....39

Figure 4. Comparison of farmers income (profit/chicken) against total loss due to coccidiosis (loss/chicken) between small scale and large scale poultry farms 50

Figure 5a. Average costs of major inputs used in small scale poultry farms 51

Figure 5b. Average costs of major inputs used in large scale poultry farms 52

LIST OF APPENDICES

page

Appendix 1. Questionnaires 69

Appendix 2. Summary of responses from questionnaire survey on farm disease history, management practices, and potential risk factors 73

Appendix 3. Gross Margin Analysis for quantification of economic losses due to coccidiosis 75

Appendix 4. Summary of poultry farms and samples examined in each farm 76

Appendix 5. Curriculum vitae.....78

Appendix 6. Signed declaration sheet.....80

LIST OF ABBREVIATIONS

AAU.....Addis Ababa University
CSA.....Central Statistics Authority
Coc .....Coccidiosis
CI .....Confidence Interval
CTA.....Centre for Tropical Agriculture
DVM.....Doctor of Veterinary Medicine
DZARC.....Debre Zeit Agricultural Research Centre
EARO.....Ethiopian Agricultural Research Organization
ESR.....Erythrocyte Sedmentation Rate
FAO.....Food and Agricultural Organization
FUB.....Free University of Berlin
FVM.....Faculty of Veterinary Medicine
GDP.....Gross Domestick Product
GM .....Gross Margin
IAR.....Insitute of Agricultural Research
ILCA.....International Livestock Centre for Africa
ILRI.....International Livestock Research Institute
LMA.....Livestock Marketing Authority
LS-BR.....Large Scale Broiler Farms
LS-LY.....Large Scale Layer Farms
LS.....Large Scale poultry farms
MAFF.....Ministry of Agriculture Food and Fisheries
Mil.....Millions
MS.....Microsoft
MSc.....Masters of Science
NMSA .....National Meteorological Services Agency
No.....Number
OPG.....Oocysts Per Gram of faeces
PCV.....Packed Cell Volume
PMR .....Proportional Mortality Rates
rpm.....revolutions per minute
SD .....Standard Deviation
SS-BR/PL.....Small Scale Broiler or Pullet Farms
SS.....Small Scale poultry farms
SVA .....Statens Veterinär Medicinska Anstalt
Tfc .....Total fixed costs
Tvc .....Total variable costs
UK.....United Kingdom
USA.....United States of America
WB.....World Bank
Umext .....University of Maine Cooperative Extension

ACKNOWLEDGEMENTS

I am highly endebted to my supervisors, Professor Dr. H. M. Hafez, Head of the Institute for Poultry Diseases, FUB, Associate Professors Getachew Tilahun and Moges Woldemeskel both of AAU for their technical input in the form of comments, guidance and provision of reference materials necessary for my thesis work. I also acknowlege the inputs of Drs Moses Kyule and Matthias Greiner in their capacity as Epidemiology advisors and Ato Girmaye Medhin for his advice in statistics.

I would like to express my sincere gratitude to the Germany Government for funding my studies through Germany Academic Exchange Service (DAAD) and Germany Technical Cooperation (GTZ). I also convey my special thanks to the management and staff of the Department for International Animal Health of the Freie Universität Berlin (FUB) and the Department for Pathology and Parasitology of the Addis Ababa University (AAU) for their assistance and cooperation in various ways during the course and research work. In this regard I wish to acknowledge the efforts and commitments of Dr. Maximillian Baumann Course Coordinator FUB and Drs. Bayelegn Molla and Getachew Abebe Course Coordinators AAU for running the course succesfully. My sincere thanks also go to Ato Alemayehu Meshesha for his technical assistance during laboratory work and W/t Eskedar Haillegebral for assisting me to proceess tissue samples for histopathology.

My thanks also go to the farm authorities and individual poultry farmers of all poultry farms which were included in this study for allowing me to collect samples from their farms, answering my questionnaires and attending my various requests. Their cooperation during data collection is highly appreciated.

I sincerely acknowledge the contribution of the management and staff of EARO Debre Zeit and ILRI Addis Ababa for allowing me acces to their library facilities and services.

Thanks to my family members particularly my young brothers Peter and Jacob and my wife to be Esther for their moral support and continuous communication with me, keeping me in touch with my family as well as my country.

ABSTRACT

Coccidia infections in poultry are prevalent and causes significant economic losses worldwide. In Ethiopia, previous studies did not attempt to substantially assessment of economic losses due to coccidiosis. This study was designed to determine the prevalence of poultry coccidiosis, identify species of coccidia involved, examine pathological changes associated with coccidia infection and assess the economic impact of the disease in small scale and large scale poultry farms in urban and peri-urban areas of Ethiopia. The study population consisted of broiler and layer chicken in 26 poultry farms. They included 5 large scale broiler farms, 2 large scale layer farms with deep litter rearing system, 4 large scale layer farms with cage rearing system, 12 small scale broiler farms and 3 small scale pullet rearing farms both with deep litter rearing systems.

Faecal samples were randomly collected from all poultry farms as follows: 25-30 samples from every small scale farm and 35-75 samples from every large scale farm. Overall 1028 faecal samples were collected. Qualitative and quantitative faecal examinations were carried out using simple salt floatation and modified McMaster techniques, respectively. Sporulation of coccidia oocysts was done followed by microscopic examination for shape and size.

Further, a sample of 420 birds was randomly selected. The selected birds were distributed as follows: 15-20 birds from every farm in small scale farms and 30-35 birds from every farm in large scale farms (except in one very large farm in which 62 birds were sampled). Antemortem and postmortem examinations were conducted to detect lesions due to coccidiosis in the gastrointestinal tracts, and to identify the species of coccidia involved. A questionnaire survey was conducted to assess farm performance, farm disease history, management practices and potential risk factors for coccidiosis.

Assessment of the economic impact of coccidiosis was conducted in 8 selected farms using historical data.

Coccidia oocysts were detected in 220 (80.3%), 8 (6.8%), 11 (4.8%), 231 (72.9%) and 73 (81.1%) faecal samples which were collected from large scale broiler farms, large scale layer farms with deep litter rearing system, large scale layer farms with cage rearing system, small scale broiler and small scale pullet rearing farms, respectively. There were significant (p-value =

0.000) differences in the proportions of coccidia oocysts detected in faecal samples among the different poultry production systems. The means of OPG counts were significantly (p-value = 0.024) higher in broiler and pullet rearing farms than those of layer farms.

Lesions due to coccidiosis in the gastro-intestinal tracts were detected in 92 (48.2%), 94 (47.2%) and 12 (40%) birds which were examined in large scale broiler, small scale broiler and small scale pullet rearing farms, respectively. Proportions of subclinical coccidiosis were higher (82.6%, 76.6%, and 66.7%) than those of clinical coccidiosis (17.4%, 23.4% and 33.3%) in large scale broiler, small scale broiler and small scale pullet rearing farms, respectively. However, there was no significant (p-value = 0.731) difference in the detection rates of coccidia lesions in the gastro intestinal tracts among the different production systems or among the different age groups (p-value = 0.905).

Four species of coccidia were identified namely *Eimeria acervulina* (34.4%), *Eimeria tenella* (32.8%), *Eimeria maxima* (8.6%) and *Eimeria necatrix* (4%). Over 20% were cases of mixed infections. Most observed lesions due to these species were mild to moderate (Lesion scores 1-2). Lesions due to *Eimeria tenella* and *Eimeria necatrix* were characterised by haemorrhagic typhilitis and enteritis, respectively, whereas those due to *Eimeria acervulina* and *Eimeria maxima* were characterised by mucoid enteritis.

Use of coccidiostats in the feed was the major method of managing coccidiosis in most farms. The method has been effective in controlling clinical coccidiosis despite occurence of major risk factors for the disease especially in small scale poultry farms.

Economic losses due to coccidiosis were high especially in small scale poultry farms. They occured in the form of mortalities, reduced market value of the affected birds and sometimes culling or delayed offtake. Improved management practices, biosecurity measures and drug sensitivity testing are recommended.

1. INTRODUCTION AND OBJECTIVES

Ethiopia, with a human population of about 60 million owns the largest number of livestock in the continent but produces and consumes less livestock products by international standards. The current mean daily caloric intake from livestock products is estimated between 1600 and 1800 Kcal/person which is about 70% of minimum recommended level (FAO/ILRI, 1995; FAO/WB, 1993; EARO, 2000b). The country's economy depends mainly on agriculture of which the livestock subsector contributes 40 % of Gross Domestic Product and 20 % of foreign export earnings (CSA 1998).

Poultry is among the important species of livestock kept in Ethiopia. Alemu and Tadelle (1997) identified three poultry production systems. These are backyard poultry production system, small scale and large scale intensive poultry production systems. The total poultry population in the country is estimated to be 56.5 million. The subsector is concerned with egg and meat production for income generation and home consumption (ILCA, 1993; EARO, 2000a).

To be able to feed her people and ensure food security, Ethiopia needs to intensify livestock production, particularly poultry production systems. Poultry production has the potential to create both rural and urban employment, improve the nutritional status of the people and can be easily sold in time of economic difficulty to generate income (EARO, 2000b). However, poultry production in Ethiopia has been hindered by diseases among other factors. Mortalities due to diseases are estimated between 20 % to 50% but can go as high as 80 % during times of epidemics (Alamargot, 1987; Alemu, 1995).

Among parasitic diseases, economic losses are caused by coccidiosis (Nasser, 1998; Alamargot, 1987). Coccidiosis has been refered to as a disease of intensification worldwide. This makes coccidiosis an important disease deserving attention especially with regard to its prevention and control (Reid, 1990; David, 1979).

In Ethiopia, coccidiosis is endemic, causing great economic losses particularly in young growing birds in all production systems. In the past years coccidiosis used to be the most important cause of mortalities in all farms. Incidences of the disease were as higher as 80% usually occuring in the form of ourbreaks (Alamargot, 1987). The disease continued to be a problem as reported by

Fessesse-work (1990) who recorded prevalence rates of 50.8% and 11% in deep litter intensive systems and backyard poultry production systems, respectively and Kalifa (1997) who reported prevalence of coccidia lesions in dead birds of 44.8% in Lemlem and 21.5% in Tsedey farms both of which are large scale commercial poultry farms in Debre Zeit. Likewise Hagos (2000) reported prevalence of 20% clinical and subclinical coccidiosis in local chickens in Central Ethiopia agro-climatic zones.

Despite the economic significance of the disease, no substantial research work has been done to determine its prevalence and particularly to assess its economic impact. This study is, therefore, intended to contribute on these aspects.

General objectives

1. To establish baseline data on prevalence and economic impact of poultry coccidiosis in different production systems in the study area.

2. To recommend appropriate measures for controling the disease and alleviating its associated economic losses.

Specific objectives

1. To determine the prevalence of poultry coccidiosis in small scale and large scale poultry farms in the study area.

2. To identify the prevalent species of poultry coccidia in the study area.

3. To examine gross and histopathological changes associated with coccidiosis in poultry.

4. To assess the economic impact of the disease.

5. To recommend appropriate control measures.

2. LITERATURE REVIEW

2.1. General considerations

2.1.1. Definition and aetiology

Coccidiosis is an infectious disease of vertebrate animals caused by intracellular protozoan parasites of the phylum Apicomplexa, subclass Coccidia (Urquhart *et al*, 1987). Coccidia of economic importance in domestic animals belong to the genera Eimeria and Isospora and hence the term coccidosis is usually reserved for disease resulting from infection caused by these species (Elmer and Glenn 1982; Urquhart *at al* 1987).

Coccidia of the genus Eimeria cause severe intestinal infections in poultry and ruminants whereas Isospora is the predominant genus in pigs and carnivores (SVA, 2000). The majority of infections in domestic animals results from genus Eimeria (Soulsby, 1982).

2.1.2. Epidemiology

Coccidiosis has worldwide distribution. It is a disease of universal importance in livestock production. All domestic and wild animals are susceptible. Young and growing animals are mostly affected resulting in high morbidity and mortality rates up to 10% or more (Gordon and Jordan, 1982). The disease occurs in all farms where large numbers of animals are kept in confinement in an environment contaminated with sporulated coccidia oocysts (McDougald and Reid, 1997; Radostits *et al*, 2000).

Transmission is by faecal-oral route, through ingestion of sporulated oocysts shed by infected animals via contaminated drinking water or feed (Radostits *et al* 2000). Dissemination of oocysts is often by feet, beaks, wings of birds, boots of attendants, equipment, birds and rodents. These can convey oocysts mechanically from one place to another in the same house or from one house to another (Backer, 1969). The maintanance of infection depends on repeated infections and contamination of the environment with oocysts. Clinically sick or recovered animals serve as carriers for a long time (Backer, 1969).

Excreted oocysts are not infective unless they undergo a maturation process called sporulation which takes place under favourable conditions of temperature about 27°C and high humidity and oxygen (Urquhart, *et al* 1987). Sporulated oocysts are resistant to harsh environmental conditions and commonly used disinfectants and can survive in the environment for up to 18 months (Backer, 1969; McDougald and Reid, 1997).

A large number of animals can be infected and shed oocysts in faeces but few develop clinical disease. The degree of reproduction within the susceptible host and hence the severity of the disease depends on the number of oocysts ingested, the frequency of ingestion of oocysts, the species of coccidia involved and the immune status of the host. This is because the rate of development is fixed by the number of schizont generations which occur in any given coccidia specie (Gordon and Jordan, 1982).

Infections associated with ingestion of few oocysts are mild and subclinical but acute and severe disease may occur following ingestion of large numbers of sporulated oocysts (Reid, 1990). Immunity develops immediately following first exposure making coccidiosis to be a self limiting disease not common in immune competent animals (Radostits *et al* 2000).

2.1.3. General life cycle

The general life history of the coccidian parasites is common to all members of the phylum Apicomplexa and is represented by the life cycle of the genus Eimeria (Dwight, 1995). The life cycle of the genus Eimeria is direct and characterised by sexual and asexual multiplicative phases which except sporogony or sporulation, takes place in the cytoplasm of parasitized host cells (Schmidt, 1990). For the purpose of description, the life cycle is devided into three phases namely sporogony, schizogony or merogony and gametogony (Urquhart, 1987).

a). Sporogony: is an asexual form of multiplicative development of the unsporulated oocyst. It includes development of the unsporulated oocysts to form sporulated oocysts containing the infective forms or sporozoites. It takes place in the ground outside the host (Schmidt, 1990).

This phase starts immediately after the unsporulated oocysts consisting of a mass of protoplasm are dischaged out from the infected host. Upon finding favourable conditions of high humidity,

temperature about 27°c and oxygen, sporulation takes place within 2-4 days. The single cell in the unsporulated oocyst (sporont) devides into four sporoblasts each of which develops into a sporocyst containing two haploid banana shaped sporozoites. The oocyst now consists of an outer wall enclosing 4 sporocysts each containing two sporozoites. The oocyst becomes an infective sporulated oocyst (Urquhart, *et al* 1987; Dwight, 1995).

b). Schizogony or merogony: is an asexual multiple reproductive divisions of the parasite which takes place in host cells and results in numerous organisms called merozoites (Schmidt, 1990). Schizogony follows ingestion of sporulated oocysts containing sporozoites by a susceptible host (Dwight, 1995). When the sporulated oocyt reaches its specific site in the host, the oocyst wall disolves. The sporocysts are set free, and activated by trypsin or bile the sporozoites leave the sporocysts and penetrate host epithelial cells (Urquhart *et al*, 1987). Parasitized cells swell as sporozoites round up to form trophozoites which grow large to become first generation schizonts. This first generation schizonts produce several first generation merozoites which cause the parasitized cells to burst. The free first generation merozoites invade new cells and develop into second generation schizonts. Though there may be many generations of schizonts and merozoites, 2 or 3 generations are usually a limit for many Eimeria species (Dwight, 1995). Schizogony terminates when merozoites of the final generation of schizonts differentiate into male and female sex cells called micro and macrogametocytes respectively (Urquhart *et al*, 1987).

The salient features of schizogony thus include exponential increase in the number of parasites which results from one sporozoite, destruction of host cells in proportion to the number of sporulated oocysts ingested and automatic termination of the asexual phase and beginning of the sexual phase of development. The number of asexual generations, the type and location of host cells parasitized and the number of merozoites formed at each asexual generation depends on the specie of coccidia involved (Dwight, 1995).

c). Gametogony: The sexual developmental phase, gametogony, is the formation from schizogony of male and female sex cells called micro and macrogametocytes respectively, and their subsequent fusion to form zygote which develops into oocysts. It takes place inside the host cells (Schmidt, 1990). During this phase, merozoites produced by the final stage of schizonts penetrate new host cells where they differentiate into micro and macrogametocytes (Dwight,

1995). The microgametocytes undergo repeated divisions to form large numbers of motile flagellated uninucleate microgametes which are freed by the rupture of the host cells. The macrogametocytes increase in size to fill the parasitized host cells and develop into macrogametes (Urquhart *et al.* 1987). One microgamete fertilizes a macrogamete forming a zygote. A resistant wall forms around the zygote to form an unsporulated oocyst which is released by rupture of the host cell. No further development takes place untill the unsporulated oocyst passes out with faeces. The prepatent period varies with specie from about 5 days in poultry to about 3-4 weeks in ruminants (Urquhart *et al* , 1987; Dwight, 1995).

2.1.4. Immunity to coccidia infections

Exposure to coccidia infection confers immunity to subsequent infections. The evidence accumulated by scientific research supports the hypothesis that by using an initial subclinical dose of Eimeria oocysts, the high susceptibility of chicken to coccidiosis is reduced and subsequent infections confer the host with strong immunity (Jiffar, 1982). Edgar *et al.* (1968) observed that birds become resistant to a specie of coccidia 6-8 weeks of age. A significant degree of resistance persists for at least one year. He also observed that birds which developed Avian Leukosis and Marek's Disease lost the acquired immunity to coccidiosis.

Immunity develops immediatelly following first exposure and is species specific. It gives protection against subsequent infections with the same specie but doesn't give protection againt infection with other species. This species specificity of the immunity developed against Eimeria have been used for identification of species (McDougald and Reid, 1997; Gordon and Jordan, 1982). The immunogenic stages of coccidia vary according to specie, but generally are sporozoites, second stage schizonts and free second stage merozoites (Kouwenhoven and Kuil, 1976). The mechanisms of immune response is a combination of both cellular and humoral factors (Elmer and Glenn, 1982; Reid, 1990). However the main mechanism of defence in the gut wall is cell mediated (Carmichael, 1998). Development of immunity and antibody titres are not correlated with initial number of oocysts ingested (Graat *et al*, 1996). Acquired immunity is not life long, it has to be maintained through repeated exposure with small numbers of oocysts. Reid (1990) observed that repeated innoculation with single or few oocysts of Eimeria maxima in chicken for 20 consecutive days produced stronger immunity than much larger number of oocysts given in a single innoculation. This phenomena termed as trickle infection results in premunity or

premmunization and accounts for the naturally acquared immunity in flocks that have never shown signs of clinical coccidiosis. Small numbers of oocysts in litter are therefore necessary to reinforce waning immunity initiated by earlier infections (Elmer and Glenn, 1982, Reid, 1990).

2.2. Coccidiosis in chicken

2.2.1. Species of coccidia in chicken: Description, major clinical signs and postmortem findings

In chicken, coccidiosis is caused by nine species of Eimeria which have been described based on nature and location of the lesion in the intestine, morphology of oocysts, location of the parasites in the host tissue, timing of parasite's patent and prepatent periods, minimum sporulation time and immunological specificity (Long *et al* 1976). These are *Eimeria tenella*, *Eimeria necatrix*, *Eimeria maxima*, *Eimeria acervulina*, *Eimeria brunetti*, *Eimeria mitis*, *Eimeria mivati*, *Eimeria praecox* and *Eimeria hagani* (MAFF, 1982; McDougald and Reid 1997). The first six species are important pathogens of chicken while the last three are mildly pathogenic (Long et al 1976). The following account on species of coccidia in chicken is due to Long *et al* (1976) Soulsby (1982), MAFF (1982), and McDougald and Reid (1997):

Eimeria tenella: This is the most pathogenic and widespread species causing severe disease and economic losses particularly in the commercial broilers production. It inhabits the caeca and adjacent tissues causing caecal coccidiosis characterised by bleeding from the caeca walls, high morbidity and mortality rates, reduced weight gain, reduced feed convention efficiency and reduced egg production in layers. Oocysts and schizonts are larger about 22x19 and 54mμ, respectively. Following infection, sporozoites round up and mature to form first generation schizonts. Each of the first generation schizonts undergo multiple divisions to produce about 900 merozoites which cause the parasitized cells to bulge and burst setting the merozoites free to parasitize other cells and form second generation schizonts. Colonies of second generation schizonts are apparent 72-96 hours post-infection and each produce 200-350 second generation merozoites (Soulsby, 1982). These initiate another cycle of asexual development to form third generation of schizonts. *Eimeria tenella* has 3 generations of schizonts.

Infection with 1000-3000 sporulated oocysts of *Eimeria tenella* can cause sudden onset of disease with high mortality rates of about 20% or more in the first 3-6 days post infection. The most pathogenic stages are the second generation schizonts.

Gross lesions include distension of the caecal pouches due to presence of clotted blood and pieces of caecal mucosa in the lumen. By the 4th day post-infection the second generation schizonts are mature causing haemorrhages and necrosis. The caeca wall are thickened due to oedema and infiltration with inflammatory cells. Dark spotted necrotic foci and petechiae appear on the caecal serosa which become coalesced in more severe infections. Hard caecal cores composed of necrotic debris, gametocytes and oocysts are passed in faeces and can be found at recovery.

Microscopically first generation schizonts are found in colonies or clusters. As the second generation schizonts develop in the lamina propria, there is heterophil infiltration in the submucosa. Oocysts, micro and macrogametocytes are seen.

Histopathological changes include denuded epithelia and necrosis of underlying connective tissues. There eosinophillia, lymphocytosis monocytosis and infiltration with plasma cells.

Eimeria necatrix: This is the second pathogenic species after *Eimeria tenella*. It parasitises the mid gut near the yolk sac (Meckel's) diverticulum. In severe infections it may extends the entire length of small intestine. Development of sporozoites and their associated pathogenic effects is similar to *Eimeria tenella*. Oocysts and schizonts are large, 20.4x17.2 and 66mμ, respectively. First generation schizonts develop proximal to the host cell nucleus. Merozoites appear two to three days post-infection. The haemorrhages and associated lesions observed during *Eimeria necatrix* infection are due to large, deeply seated second generation schizonts which are seen as white opaque foci surrounded by a zone of haemorrhage (Soulsby, 1982).

The infection is characterised by ballooning of the middle intestinal tract to about twice its normal size. There is congestion, bleeding and necrosis of the mucosa with passage of clotted blood, mucous, fibrin and necrotic intestinal epithelial tissue. Infection with 75,000-100,000 oocysts of *Eimeria necatrix* causes higher morbidity and mortality rates and severe weight loss than any specie except *Eimeria tenella*. *Eimeria necatrix* can cause mortaliy in excess of 25% in

commercial flocks. Signs of morbidity continue for a weak or more leading to chronic coccidiosis, a term used to describe coccidiosis due to *Eimeria necatrix* infection.

Gross lesions appear on the 3rd day post-infection and are associated with first generation schizogony. Serosal surfaces are bright red with petechiations. The intestine is ballooned, mucosa thickened and the lumen filled with blood, fluid and tissue debris. Pathognomonic small whitish-yellow plaques or red petechiae containing schizonts appear. Necrosis and bleeding of parasitized epithelial cells occur.

Smears examined microscopically may contain clusters of large (66mμ) schizonts often containing hundreds of merozoites which are characteristic for this species and distinguishes it from others that parasitize the same area.

Histopathological changes include infiltration with inflammatory cells, development of focal parasitic granulommas in the lamina propria and sloughing of epithelial cells being replaced by fibrin containing mononuclear cells. Developmental stages of the parasite are seen superficial to the nucleus of the epithelial cells.

Eimeria acervulina: This species is less pathogenic compared to *Eimeria tenella* and *Eimeria necatrix*. Sporozoites develop proximal to the host cell nucleus in the epithelial cells of the duodenum and jejunum, though in heavy infections light development in the caecum may occur (Long and Millard, 1976). Mature schizonts produce 16-32 merozoites, which initiate another asexual circle of development. Infections produce characteristic discrete localised whitish lesions which are oriented in a ladder-like arrangement visible from the serosal and mucosal surfaces. These may coalesce in heavy infections. Intestinal walls are thickened and congested.

Oocysts are ovoid and show thinning of the shell at the small end with an average size of 18.3 x 14.6μm. Schizonts are small with an average size of 8-10.3μm. Minimum sporulation time is 17 hours. Prepatent period is 97 hours.

Infections are mild to severe depending on the number of oocysts ingested and the immune status of the bird. In experimental infections, ingestion of 1000, 30,000, 100,000 or 1,000,000 results in mild to severe coccidiosis with morbidity and mortaliy rates varying from 0-70%, pronounced

reduction in weight gain and feed conversion efficiency. In layers this is accompanied by decline in egg production, weight loss and depigmentation of the skin (Fitz-coy and Edgar, 1992).

The lesions are of little pathogenic significance and are associated with sexual forms, gametocytes and oocysts (Gordon and Jordan, 1982). They are usually limited to the duodenal loop with only few plagues but may overlap and coalesce to cover some distance along the small intestine. The intestine is pale and contain watery fluid. The lesions contain schizonts, gametocytes and developing oocysts.

Histopathological changes are characterised by ovoid gametocytes lining epithelial cells on the villi. In moderate to heavy infections, infected villi are sloughed off leading to truncation and fusion of villi and thickening of the mucosa. Several parasites may invade one cell and are located superficial to the epithelial cells without extension to the lamina propria.

Eimeria maxima: Parasitizes middle small intestine from below the duodenal loop past the Meckel's diverticulum. In heavy infections it may extend throughout the small intestine. Oocysts are the largest of all chicken coccidia and diagnostic about 30.5 x 20.7µm (21.5-42.5 x 16.5-29.8) with a distinctive golden colour but schizonts are small about 9.4 µm. Only two generations of schizonts, are formed with the second generation of schizonts capable of producing 8-16 merozoites.

The infection is characterised by mucoid to haemorrhagic enteritis, thickening of intestinal walls and ballooning. Intestinal contents are brown, orange, pink to red and contain mucoid exudates. In heavy infections, there is haemorrhages, emaciation and anaemia. Light infections induce depigmentation. Mortality rates are moderate. Tissue damage is minimal except where infection involves deeper tissues by developing gametocytes. There is congestion and oedema marked by sub epithelial thickening and rapid growth in size and number of parasites. Invading gametocytes develop beneath the host cell nucleus.

Eimeria maxima is less pathogenic with morbidity and mortality rates of about 25%. Infection with 200,000 oocysts is sufficient to cause morbidity with poor weight gain, diarrhoea, anaemia and sometimes mortaliy. *Eimeria maxima* can be differentiated from *Eimeria necatrix* by lack of

large schizonts associated with the lesion and from *Eimeria brunetti* by the larger oocysts and the location and appearance of the lesions.

Gross lesions involve minimal tissue damage due to first two sexual cycles which develop superficially in the epithelial cells of the mucosa. The intestine is filled with fluid and the lumen contains yellow or orange mucous and blood, a condition described as "ballooning". Less gross lesions are seen from the serosa except slight swelling, petechiae and congestion.

Histopathologically there is oedema, cellular infiltration, thickening and considerable disruption of the mucosa. Developing schizonts, micro and macrogametes are seen in deeper tissues. Microscopic haemorrhages can be seen near the tips of villi. Infected host cells become enlarged, pushing into the subepithelial zone.

Eimeria brunetti: This species is less to moderate pathogenic due to rapid development of immunity. Effects on mortality rates, weight gain and feed conversion efficiencies are low. Pathogenicity is due to both sexual and asexual stages (Gordon and Jordan, 1982; Soulsby, 1982). The specie produces lesions in the posterior parts of small intestines usually from the Meckel's diverticulum to near the caecal junction.

Oocysts are large about 24.6 x 18.8µm. Schizonts are 30µm can easily be confused with E. tenella. Minimum sporulation time is 18 hours and prepatent period is 120 hours. Second and first generation schizonts and gametocytes occupy a zone above the nucleus in the epithelial cells. In light infections the mucosa show tiny petechiae, thickening and loss of colour. Heavy infections are characterised by damage of mucosa and haemorrhages with blood stained exudates. Yellow-white caseous materials are found in the lumen of lower intestines due to coagulative necrosis. There is oedema and thickening of the mucosa. Sloughing and coagulation of epithelial cells and bloody exudates may cause complete blockage of the intestines.

Histopathology on day 4-5 post-infection reveal schizonts, cellular infiltration and damage of mucosa. In severe cases there is denudation of the villi.

Eimeria mitis: This species parasitises the anterior half of the small intestine from the yolk sac diverticulum to the caecal necks. It have been mentioned as rare, mild to non-pathogenic species

in most of the literature, though Fitz-coy and Edgar (1992) reported pathogenicity due to this specie in laying hens. Layer chicken experimentally infected had watery diarrhoea, as early as day 5 postifection. Egg production decreased and some hens temporarily ceased laying for about 14 days. Weight gain, egg quality and pigmentation was also affected. Infections causing mortalities have also been reported. Infections are subclinical with no immunity on repeated infection.

There is no gross lesions produced because the developing parasites do not localize in colonies and schizonts and gametocytes are superficial in the mucosa.

Microscopic examination of the smears from the mucosa may reveal numerous tiny oocysts (15.6 x 14.2µm).

Eimeria praecox: Is a rare and non pathogenic species found in the duodenum. Heavy infections result in depressed weigh gain, loss of pigmentation, loss of fluid and poor feed conversion. Oocysts are rounded and larger (21.3 x 17.1µm) than all species found in the duodenum.

Infections are confined to the duodenal loop. Experimental infections of other parts of the intestines such as caecum failed (Long and Millard, 1976). Epithelial cells of the sides of the villi, and not the tip are often infected with several parasites found in one cell.

Gross lesions consist of watery intestinal contents, mucus and mucoid casts. Small pin point haemorrhages may be seen on mucosa.

Eimeria mivati: This species is rare, first identified as a strain of *Eimeria acervulina*, parasitizes the duodenal loop to the caeca. Light infections produce lesions similar to those of *Eimeria acervulina* but more circular in shape. Heavy infections of about 1,000,000 oocysts cause morbidity and reduced weight gain. Mortality occurs only in experimental infections.

Eimeria hagani: Is a rare and moderate to non pathogenic specie parasitizing the duodenum. Infections are characterised by haemorrhagic spots, catarrhal enteritis with fluid contents

2.2.2. Distribution and occurrence of the agent and the disease

Chicken coccidia are found everywhere chicken are kept. All known species of Eimeria in chicken have been reported throughout the world. This worldwide distribution of species indicate that the distribution of the species of coccidia in chicken is limited only by the distribution of the susceptible host species (McDougald and Reid, 1997; Kaufmann, 1996). However the occurrence and frequency of the disease can be determined by management factors. In chicken, deep litter intensive rearing systems offers optimal conditions of temperature and humidity favourable for accumulation and sporulation of viable oocysts and hence infection. Thus intensive methods of rearing birds favour the reproduction of Eimeria species causing the usually well balanced host-parasite relationship which commonly occurs in birds kept under natural conditions to break down causing acute and normally fatal disease outbreak (Gordon and Jordan, 1982).

2.2.3. Risk Factors for Coccidia Infection in Chicken

2.2.3.1. Agent related factors

a). **Species and pathogenicity of the Coccidia**: Coccidiosis occurs when birds are exposed to large numbers of sporulated oocysts and that the severity of the infection is proportional to the numbers of the organisms ingested (McDougald and Reid, 1997; Soulsby, 1982). The number of generations of schizonts and the number of merozoites formed at each generation of schizonts which in turn determine the the number of organisms formed at each cycle, the rate of host tissue damage and hence the severity of the infection depends on the species of coccidia involved (Dwight, 1995). Most pathogenic species are those with more generations of schizonts and which produce large numbers of merozoites at each generation of schizonts. *Eimeria tenella*, the most pathogenic of all chicken coccidia species for example has 3 generations of schizonts producing 900 and 200 to 350 first and second generation merozoites, respectively compared to *Eimeria maxima*, a moderately pathogenic specie which has 2 generations of schizonts each producing about 8-16 merozoites. The risk for coccidiosis, therefore depends on the specie of cocidia involved. It is high when birds are infected by a more pathogenic specie than with a less pathogenic one (Soulsby, 1982).

b). **High biotic potential**: Chicken coccidia have high capacity to reproduce within the host. This leads to a rapid increase and success of the parasite within the susceptible host and subsequent high level contamination of the environment (McDougald and Reid, 1997; Urquhart *et al* 1987). Due to short direct life cycle, a single oocyst of *Eimeria tenella* for example is capable of producing eight sporozoites each of which can produce 900x350 second generation merozoites within 4-5 days only (Gordon and Jordan 1982). This high reproductive potential of chicken coccidia ensures survival of the parasite in commercial poultry flocks, and increases the potential risk for heavy infection and severe outbreaks of the disease (McDougald and Reid, 1997).

c). **Lack of cross immunity between species:** Protective immunity acquired following coccidia infection is species specific and not long lasting. This predisposes otherwise immune birds to infection and disease outbreaks caused by different species. Under natural field conditions immunity is reliable and of long duration due to mixed infections and continuous reinfection and hence reinforcement of immunity. However if reinfection is prevented, new litter added or birds are moved to new location, natural reinforcement of immunity fails and birds becomes susceptible. These factors complicates vaccination programmes and act as risk factors for coccidiosis (McDougald and Reid, 1997).

d). **Resistance of Coccidian Oocysts:** Resistance of sporulated coccidia oocysts to harsh environmental conditions and hence their viability outside the host is an important factor regarding the course of the disease. Though sporulated oocysts may be desroyed by freezing, they survive cool tempreratures below 10°C and high temperatures up to 45°C. They survive drying conditions and most commonly used chemical disinfectants which kill viruses and bacteria. They are capable of surviving the cold winter conditions for up to two years (Gordon and Jordan, 1982). Cleaning and disinfection measures are recommended for general hygiene so as to reduce the number of coccidia parasites in the environment to acceptable levels (Fred *et al*, 2001). Thus resistant coccidia oocysts shed in the environment remain viable and infective for a long period of time and can be mechanically transmitted over long distances and from one poultry house to another by such things as dirty boots, birds, rodents and equipments (McDougald and Reid, 1997; Bailey, 2001). This constitutes a potential risk for susceptible birds (Gordon and Jordan, 1982).

2.2.3.2. Host related factors

a). Age of the birds: Though coccidiosis in chicken is generally known to be a disease of young birds, slight differences occur among coccidia species regarding age susceptibility of birds. Caecal coccidiosis due to *Eimeria tenella* infection for example is common in young chicken aged about 4 weeks whereas intestinal Coccidiosis due to *Eimeria necatrix, Eimeria acervulina, Eimeria maxima* and other species occurs in slightly older chicken about 6 weeks to maturity (MAFF, 1982; Soulsby, 1982). The reported peak of infection and coccidiosis is at 4-5 weeks of age though subclinical coccidiosis increases with age (Razmi and Kalideri, 1999). However age resistance to coccidia infection in poultry becomes less important if chicken are kept free of re-infection in which case susceptibility increases with age (Gordon and Jordan, 1982).

b). Immune status of the birds: The role of immunity in protecting against coccidiosis outbreaks have been noted as early as 1926, when Johnson showed that species specific immunity develops following repeated infections with small numbers of oocysts (Reid, 1990). The immunity developed depends on the specie of coccidia involved. Some species induce rapid solid immunity whereas in other species several infections are needed before complete immunity is developed (Gordon and Jordan 1982). Research have shown that immune compromised birds fail to mount effective immunity against coccidiosis. Braunius (1987) observed that broiler farms with higher viral and bacterial infection pressures had higher incidences of mixed coccidia infections compared with farms in which such infections were controlled.

c). Health status of the birds: Coccidiosis is known to be a self limiting disease following development of immunity if re-infection is prevented. Proper management particularly in terms of housing and nutrition plays a big role to ensure good health status of the birds (Gordon and Jordan, 1982). However concurrent infection with certain immunosuppressive bacteria and viruses as in the case of Infectious Bursal Disease (IBD) Avian Leucosis and Marek's Disease where development of immunity is affected, the host defence mechanisms fails to retard or stop reproduction of the parasite and hence multiplication of the agent continues until the host succumbs to the disease. Thus the presence of immunosuppresive diseases in the flock increases the severity of coccidiosis and acts as risk factors for the disease (Reid, 1990).

2.2.3.3. Environmental related factors

a). Temperature in the poultry house: Proper temperature in the poultry house not only reduces the probability of diseases but also determines the growth perfomance of the birds. During the rearing period, a range of 24-30°C is associated with the best performance. Below and above this range feed and water intake, feed convertion efficiencies and weight gains are affected and consequently the likelyhood of disease incresease. The temperature is too low if it is below 18°C and too high if it is above 35°C (Sainsbury, 1992). In the case of coccidiosis a temperature of asbout 27°C plus favourable humidity and oxygen levels causes accumulation and sporulation of viable oocysts in the poultry house thus posing a risk of infection and disease outbreak (Soulsby, 1982; Urquhart *et al* , 1987).

b). Ventilation system: Ventilation, the movement of air in and out of the poultry house is concerned with control of the climatic environment in the poultry house. It brings in fresh air and takes out stale air. It eliminates byproducts of respiration and excreation of birds and evaporations from litter and droppings. It also controls temperature and humidity in the house. Ventilation is facilitated if the house is built in such a way that air enters at the sides and extracted at the roof. In most tropical countries like Ethiopia naturally ventilated poultry houses with controllable windows at the sides of the poultry house is used (Sainsbury, 1992). If the ventilation system used is not effective, control of the climatic environment in the poultry house with respect to temperature and relative humidity is not efficient which will give the ideal conditions for sporulation and accumulation of viable coccidia oocysts and disease outbreak may occur (Sainsbury, 1992; Urquhart *et al*, 1987).

c). Relative humidity: Relative humidity, the percentage of moisture content in the air or litter have already been cited as one of the key factors determining sporulation of coccidia oocysts. Under normal conditions of poultry management, the optimum level required for sporulation of oocysts ranges from 50% to a maximum of about 75% (Sainsbury, 1992; Urquhart *et al*, 1987). Thus if relative humidity in the poultry house and litter is kept below this range, sporulation of oocysts is prevented and the risk of infection and coccidiosis outbreak is reduced (Soulsby, 1982).

d). Season of the year: The effect of season of the year on incidence of coccidiosis is generally emphasised in the literature. Coccidiosis outbreaks are known to be common during warm spring

and summer than cooler fall and winter conditions (McDougald and Reid 1997). Braunius (1987) recorded lowest incidence of coccidiosis in March and April and highest incidence in the months of November and December in the Netherlands. In another experiment he found higher incidences in autumn and winter than in other seasons of the year. Razmi and Kalideri (1999) working with broiler farms in the municipality of Mashdad, Iran observed that higher infection risk was associated with winter or spring than with summer or autumn conditions. In another study conducted by Maungyai *et al* (1990) it was shown that the incidence of subclinical coccidiosis was higher during the rainy season than other seasons. In Ethiopia, Fessesse-work (1990) observed higher incidences of coccidiosis in wet and humid seasons of the year than in dry seasons.

2.2.3.4. Management related factors

a). Poultry house design and system of management: Chicken may be reared on litter or in cages. These have a role on transmission, resistance and control of parasitic diseases like Coccidiosis (Sainsbury, 1992). If birds are reared on litter which is not properly managed in respect to temperature, relative humidity and oxygen, coccidia oocysts will accumulate and sporulate and coccidiosis is likely to break out. While coccidiosis is known to be transmitted through the oral route, cage rearing systems prevents both accumulation and sporulation of coccidia oocysts and their ingestion by susceptible birds and hence the risk of coccidiosis is reduced (Reid, 1990).

Proper poultry house design in terms of floor, walls and roof types, size and location of windows will facilitate biosecurity measures and control of climatic environment in the poultry house, which in turn will reduce the risk of infection and disease outbreak (Smith, 1995).

b). Flock size and stocking density: Clinical coccidiosis has been recognised as a disease associated with growing larger numbers of birds in a limited area (Reid, 1990). Surveys have shown that prevalence of Coccidiosis increases with flock size and stocking density. The explanation given is that large farms with large number of birds require more water, feed, litter and generate greater volumes of faeces thus presenting higher risk of infection (Braunius, 1987; Razmi and Kalideri, 1999).

c). **Feeding and watering system**: A feeding and watering system is efficient if feeding and watering equipments are placed in such a way that they can be easily reached by all birds and feeding and watering space is adequate. There should be no feed or water wastage, nor contamination of feed or water with droppings from birds. Equipments should be easy to clean and disinfect (Sainsbury, 1992). If the feeding and watering system is inefficient, 20% of feed and water is wasted and the rest is contaminated with litter or droppings from birds. This will result into less feed available for growth and humidity problem in the poultry house. Such an inefficient system increases the possibility of chicken ingesting feed or water contaminated with coccidia oocysts from chicken droppings and hence increases the risk of infection and disease (Smith, 1995).

d). **Inedequate biosecurity practices:** Biosecutity practices in poultry facilities include all measures which when followed will limit the spread of disease causing parasites and their vectors within the poultry facility or from one facility to another. They are aimed at reducing or controlling disease causing organisms to acceptable non-infectious levels (Fred *et al*, 2001). Biosecurity becomes a risk factor if it is inadequate hence contributing to disease outbreaks. For control of coccidiosis and other disease causing parasites in poultry facilities, biosecurity practices include hygiene, disinfection and litter management. Adequate amounts of potable water and approved methods for timely treatment and disposal of manure and wastes are necessary (Fred *et al*, 2001).

Although it is difficult to control coccidiosis by biosecurity measures alone, it is still an important component of good management practices which will control coccidiosis and other diseases and their associated economic losses (Fred *et al* 2001; Kabay, 2001). Litter should be managed in such a way that moisture and temperature does not favour accumulation and sporulation of oocysts. Litter should be kept dry at moisture content of 25-30% (Umext, 2001). When broiler chicken are emptied for new batches old litter should be replaced by dry and clean litter and the house disinfected. Altenatively, litter should be heaped for 12 hours or more to generate a temperature of 51°C sufficient to destroy coccidia oocysts (Soulsby, 1982).

e). **Continuous use of the same coccidiostat(s):** Proper coccidiostat(s) use in terms of duration and dosage is an important aspect regarding control of coccidiosis. Research have shown that continuous use of an anticoccidial drug leads to increased incidence of mixed coccidial

infections, whereas rotation of chemical products as in the case of shuttle programmes results in reduced incidence of coccidiosis. This is due to development of drug resistance by the parasite, which results in reduced activity of the drug against the parasite (Graat *et al*, 1996). In the study which was conducted by Razmi and Kalideri (1999) it was found that no significant difference in the prevalence of subclinical coccidiosis existed between medicated and unmedicated flocks and this was attributed to declining efficacy of the coccidiostats used against Eimeria species due to the same reason. Braunius (1987) observed the same phenomenon.

2.2.4. Transmission and Susceptibility

The only natural method of transmission is through ingestion of viable sporulated oocysts. Infected chickens shed oocysts in faeces for several days or weeks that sporulate and become infective (McDougald and Reid, 1997). Though there is experimental evidence supporting the existence of genetic resistance of different breeds of chickens to coccidiosis in the literature, this requires selective breeding to be effective. The most important factors determining the susceptibility of the host to coccidiosis are therefore age and immune status of the host, specie of coccidia involved and the number of oocysts ingested. Susceptible birds pick up the oocysts from contaminated environment and ingest them with feed or water or through litter pecking activities common to all chicken (Jiffar, 1982; McDougald and Reid, 1997).

There are no intermediate hosts to Eimeria species, though oocysts can be spread mechanically by animals, insects, rodents, contaminated equipment, wild birds, wind and dust. Oocysts are resistant to extreme environmental conditions and disinfectants and can survive in the environment for weeks, months, or years under favourable conditions of temperature and moisture. The optimal temperature and humidity are 24-30°c and 50%-75%, respectively (Sainsbury, 1992; Urquhart *et al*, 1987). Oocysts can easily be killed by exposure to 55°C, extreme dryness or freezing (Gordon and Jordan, 1982).

Movement of personnel or equipment can easily facilitate transmission from one house to another or from one farm to another. New farms can remain free of coccidia for a long time until infection is introduced from outside in which case severe outbreaks occur.

Coccidiosis becomes an important disease when birds are reared under conditions that act as stress factors and permit build up of infective sporulated oocysts. The intensive systems of management provide such conditions (Kennedy, 2001).

2.2.5. Pathogenicity, pathogenic effect and host-parasite relationship

Based on the degree of pathogenicity and the severity of lesions, species of coccidia in chicken can be divided into two major groups. Group one includes *Eimeria tenella* and *Eimeria necatrix*, which are the most pathogenic species, associated with severe lesions and marked haemorrhages. Group two includes the rest of Eimeria species in chicken which are either non-pathogenic or mild to moderately pathogenic associated with less severe non-haemorrhagic lesions (Urquhart, 1982; MAFF, 1982).

The pathogenic effect and damage caused by the coccidian parasite to the host is related to destruction of intestinal epithelial cells caused by the multiplying and migrating sexual and asexual stages of the parasite. The magnitude of tissue damage and severity of the disease depends on the number of oocysts ingested, virulence of the coccidia specie involved, number of host cells destroyed age and the immune status of the bird (Elmer and Glenn, 1982; Kaufmann, 1996). Oocyst numbers in the thousands produce signs of clinical coccidiosis with intestinal lesions. Less than 1000 oocysts normally produce mild disease or subclinical infections that can be detected only by laboratory investigations (Reid, 1990).

The effects resulting from that damage includes direct cellular and tissue damage caused by the developing parasite, loss of water and electrolytes leading to severe dehydration and loss of serum protein into the gut lumen. There is impaired feed and water intake and disturbance of digestive enzymes and gut p^H causing impaired digestion, absorption and utilisation of nutrients (Soulsby, 1982; Braunius, 1987).

In a controlled experiment, Jiffar (1982) observed a marked decrease in carbohydrates, protein, lipid and mineral absorption and utilisation during the acute phase of coccidia infection in chicken. The author also observed that coccidia infection caused severe alteration in blood parameters such as PCV, plasma proteins, plasma lipids, alkaline phosphatases and serum pigments (carotenoids) which decreased by about 50%. ESR increased on the 6-10 days post-

infection. It was concluded that intestinal coccidiosis interferes with digestion absorption and utilisation of nutrients due to severe enteritis and destruction of intestinal mucosa associated with development of the parasite. The reduced weight gain and weight loss is due to a combination of both reduced feed intake, impairment of digestion and absorption and subsequent utilisation of absorbed nutrients.

The most pathogenic developmental stages are schizonts. These negative effects therefore occurs during the first and second generations of schizogony during which there is maximum interaction between the host and the parasite and they are expressed as higher mortalities, growth retardation, poor feed conversion efficiency and poor carcase quality. An indirect effect is that birds suffering from coccidiosis are more susceptible to bacterial and viral diseases (Soulsby, 1982; Jiffar, 1982; MAFF, 1982). These negative effects explain the cause of economic losses in poultry flocks (Braunius, 1987).

Under natural conditions of management, this host-parasite relationship is such that birds are always exposed to small numbers of oocysts and quickly develop immunity often without clinical signs of disease. However under modern management conditions, where large numbers of birds are kept together in confinement, the usually well balanced host-parasite relationship breaks down. The management practices provides favourable conditions for the parasite resulting into large numbers of sporulated oocysts being available to the birds and hence overt disease occurs.

2.2.6. Diagnosis of chicken coccidiosis

Coccidiosis should be suspected when clinical signs such as diarrhoea occurs in the presence of coccidiosis promoting factors. Tentative diagnosis can be made based on history and gross lesions (Long *et al*, 1976). The infection is diagnosed from the nature and location of the lesion and the appearance of developmental stages in tissue sections (MAFF, 1982). The type and location of the lesion provide a guide to the specie involved. The diagnosis is confirmed by direct detection of oocysts in faeces, schizonts, gametocytes or oocysts in the scrapings of the gut or stained tissue sections taken from intestines (Urquhart *et al*, 1987). Finding of oocysts in faecal samples indicates infection but not a diagnosis of clinical coccidiosis because major pathogenic effect usually occurs prior to oocyst production and/or presence of larger number of oocysts may

not be correlated with severe pathogenic changes in the gut (Urquhart *et al*, 1987). The methods commonly used for diagnosis of coccidiosis includes:

a). Detection of oocysts in faeces: Oocysts in faeces of infected birds can be detected using floatation methods with saturated salt or sugar solution. While this method is not reliable for diagnosis of coccidiosis, it can be a useful indicator of subclinical infection. High numbers of oocyst counts in faeces indicate a high infection risk to which the birds are exposed (MAFF, 1982; Braunius, 1987).

b). Gross examination of necropsied birds: The entire intestinal tract should be examined. The characteristics of the observed lesion such as its location on the intestinal tract, its appearance and severity, the nature of intestinal contents and other associated gross changes can be useful in establishing a diagnosis (Long and Reid, 1982).

c). Microscopic Examination: Microscopic Examination is useful for confirmation of diagnosis after observing gross lesions at necropsy. Developmental stages of coccidia can be seen in smears taken from suspected lesions. Small amount of mucosal scrapings should be diluted with one or two drops of physiological saline on a slide and covered with a coverslip. Oocysts, schizonts or gametocytes are easily seen and can be pathognomonic for certain species of coccidia. The information from microscopic examination should be combined with other observations such as gross lesions and identification of individual species (McDougald and Reid, 1997).

d). Histopathological examination: Examination of infected tissue sections can be satisfactorily performed using routine histopathological procedures. The tissue is fixed in 10% neutral formalin and stained with Haematoxylin and Eosin (H&E). Using this technique, developmental stages of schizogony and gametocytes can be demonstrated upon examination under light microscope at high magnification. Histological methods are useful in establishing the type and location of the lesion within the tissue and to determine whether schizonts or gametocytes are the main cause of tissue damage (MAFF, 1982).

2.2.7. Treatment, Prevention and Control of Chicken Coccidiosis

a). Treatment: Treatment of coccidiosis in chicken is recommended if an outbreak occurs in a flock. Commonly used drugs are sulfonamides, amprolium, ionophorous antibiotics such as lasalocid and triazines such as toltrazuril (Baycox). These drugs are used at dosage rates according to manufacturers recommendations (Kabay, 2001). However as most of coccidial infections are subclinical, and that most of the damage due to coccidia infection occurs before clinical signs are seen, the most prefered method of control is preventive medication (McDougald and Reid, 1997).

b). Prevention and Control: This is based on a combination of good management practices and chemoprophylaxis (Graat *et al.* 1996). Methods used to control coccidiosis include:

1. Disinfection and sanitation: This method is not effective due to resistance of oocysts against commonly used disinfectants. Complete house sterilization is not possible, and an oocyst sterile environment could prevent build up of immunity. Disinfection of poultry houses after mechanical cleaning such as application of hot water under high pressure and avoidance of coccidiosis promoting factors can be convenient and effective (McDougald and Reid, 1997).

2. Chemoprophylaxis: This is based on the concept of preventive medication administered in feed or water. Drugs with different chemical groups and different modes of actions have been used. All drugs are classified as coccidiostatic if they only arrest the development of the parasite or coccidiocidal if they kill the parasites. Arrested parasites continue growing and coccidiosis relapses if coccidiostatic medication is withdrawn (McDougald and Reid, 1997).

A major limitation to chemoprophylaxis is the development of drug resistance by the parasites when one product is used continuously for a long period. This problem can be reduced through rotation of products or use of shuttle programmes. In a rotation programme an anticoccidial drug is used over a defined period of time normally six months after which it is replaced by another drug of different chemical group. In a shuttle programme two drugs of different chemical groups are used, one in the starter feed and another in the grower or finisher feed. In both programmes there is a booster effect on productivity and improvement in coccidiosis control through defence against drug resistance (McDougald and Reid, 1997).

In layers or breeders reared on litter, a programme designed to stimulate immunity is used. In this programme two products are administered in feed in a decreasing level over the first 16 or 18 weeks of life. The procedure can involve a two or three stage reduction between 0-8 and 8-16 or 0-6, 6-12 and 12-18. Using this technique, it is possible to protect young growing birds, and the reduced drug rate in the older birds allows limited exposure to the parasite so that acquired immunity can develop (Urquhart *et al*, 1987).

3. Vaccination: Use of anticoccidial drugs to control coccidiosis have limitations due to development of drug resistance by coccidia parasites. With coccidiosis remaining the most important disease of intensively raised poultry, and the declared ban on rising poultry in cages in most parts of the world, altenative ways of controlling coccidiosis are necessary (SVA, 2000). From this point of view, there have been recent developments and interests in developing vaccines against poultry coccidiosis (Vermeulen *et al*, 2001; Graat *et al* 1996). Different modes of vaccination have been tried based on the characteristics of the vaccine including:

i). *Use of live virulent strains:* These comprises of a number of wild type strains (Vermeulen, *et al*, 2001). The most promising option available is "strategic or planned immunization" (Carmichael, 1998; Reid, 1990). By this method, controlled application of small numbers of sporulated oocysts of several species of chicken coccidia are given in drinking water or feed. This results in mild infection sufficient to trigger immune response which can be maintained by tricke infection (Reid, 1990). This method have been used in layers and breeders stock with success. Oocysts of eight species of Eimeria are commercially prepared under the name **Coccivac** in the USA or **Immucox** in Canada and administered in water or feed to young chicks about 10 days of age, and 10 days latter a coccidiostat is introduced in the feed for about 3-4 weeks. (Urquhart *et al*, 1987; Reid, 1990).

ii). *Use of live attenuated strains:* Attenuated strains are prepared by repeated selection for early maturation (Pre-cociousness) or by serial passage in chorioallantoic membranes of chicken embryos or by irradiation. The important feature of these lines is their reduced proliferative capacity resulting in less damage to intestinal tissue of vaccinated birds after one passage through the gut (Reid, 1990; Vermeulen *et al*, 2001).

Precocious strains of chicken coccidia are prepared by repeated selection (isolation and serial passages) of oocysts with the shortest prepatent period until the shortened prepatency becomes genetically stabilised. They are characterised by short prepatent period, low reproduction rate, smaller schizonts and even loss one generation of schizonts. They are less pathogenic but immunogenic enough to stimulate immunity (Reid, 1990; Vermeulen, *et al* 2001). Examples are **Paracox** in UK and **Livacox** in the Czech Republic.

iii). Use of live strains tolerant to ionophores: Example of these vaccines is **Nobilis COX ATM** in USA, which comprises of three Eimeria species namely *Eimeria acervulina*, *Eimeria tenella* and *Eimeria maxima*. These vaccines can be used together with ionophores (example salinomycin) during the first 3-4 weeks of age when immunity is not complete and thus reducing the risk of coccidiosis (Vermeulen *et al* ,2001).

iv). Use of monoclonal antibody technology: Involves identification and use of coccidia proteins to protect against coccidia infections by inoculating young chicks (McDougald and Reid, 1997).

Development of an effective vaccine against chicken coccidia is slow. Problems include poor growth performance of vaccinated birds, possibility of disseminating new species of coccidia in areas where they never existed, large scale production of sufficient vaccine for practical field use and its application. Furthermore, complete success of any vaccination technique depends on subsequent exposure to sporulated oocysts under natural infection to boost immunity developed through vaccination (Urquhart *et al*, 1987; Graat *et al*, 1996).

2.2.8. The Economic Impact of Coccidiosis on Poultry Industry

In livestock health and production economics, disease in the herd or flock has been refered to as a negative input. It reduces productivity by reducing the efficiency into which inputs are converted to useful products and necessitates use of additional inputs not required under normal health condition, thus rising the cost of production (Thrusfield, 1995). Disease causes direct loses in the form of mortalities, stunting, reduced productivity and reduced market value. It also causes indirect loses in the form of drug, vaccine and other veterinary costs incurred for disease treatment and control (Rushton *et al*, 1999).

Coccidiosis remains one of the most prevalent and most expensive diseases of poultry production worldwide. The disease continues to cause significant economic losses, despite advances in chemotherapy, management, nutrition and genetics (Graat *et al*, 1996; McDougald and Reid, 1997). It is one of the top five chicken diseases most frequently diagnosed in the field and laboratory and is involved in 5-15% of all mortalities (Gordon, and Jordan, 1982; Danforth, 2000; Mopate and Lony, 2000). Each year the disease costs the poultry industry 600 million USD in treatment and low carcase weights (Danforth, 2000). Williams (1999) estimated that losses due to coccidiosis in broiler industry in the UK were approximately 40 million pounds sterling. The total cost due to coccidiosis is estimated to be USD 1500 million worldwide (Lister, 2000). Subclinical coccidiosis is more common than clinical coccidiosis. Losses due to this form of the disease are heavy and cannot be estimated (Gordon and Jordan 1982; Reid, 1990).

In Ethiopia, quantitative losses due to coccidiosis are not well documented but previous studies reported high prevalences of the disease in all production systems (Alamargot, 1987; Fessesework, 1990; Kalifa, 1997; Hagos, 2000).

3. MATERIALS AND METHODS

3.1. Description of study area and study population

3.1.1. The study area

The study was conducted between February and August, 2001. Study areas included urban and peri-urban areas of Addis Ababa and Debre Zeit, Ethiopia. Addis Ababa is the capital city and administration centre for the Federal Democratic Republic of Ethiopia. It lies in the Central Highlands of Ethiopia at an altitude of 2500 meters above sea level and has an estimated human population of about 3 million. The average annual temperature and rainfall are 21°C, and 1800 mm and respectively. Addis Ababa have relative humidity varying between 70% to 80% during the rainy season and between 40% to 50% during the dry season (CSA, 1998; NMSA, 1999).

Debre Zeit with a human population of about 95,000 people is located 45 kilometers south east of Addis-Ababa at an altitude of 1850 meters above sea level. It is an important small town where most government institutions, national and international agricultural research centres are located. The average annual temperature, rainfall and humidity are 18.7°C, 866 mm and 50.9%, respectivelly (CSA, 1998; NMSA, 1999).

3.1.2. The study population

The study population consisted of broiler and layer chicken in 26 small scale and large scale poultry farms. These included 5 large scale broiler farms, 6 large scale layer farms, 12 small scale broiler farms and 3 small scale pullet rearing farms. Large scale poultry farms had flock sizes that ranged from 4000 to over 80,000 per farm. They are owned by both the public and private sectors. Small scale poultry farms had flock sizes that ranged from 300 to 3000 birds per farm. These farms are owned mainly by individuals such as civil workers, ex-soldiers, house wives and other unemployed people. Breeds of poultry kept in these farms were exotic mainly, white leghorns, hybro, mobals and bovans.

3.2. Study design

The study comprised of a cross sectional study to determine the prevalence of poultry coccidiosis and the prevalent species of coccidia in the study areas. A retrospective study to assess the economic impact of the disease was also carried out.

3.2.1. Cross sectional study

This study was conducted through parasitological examination of faecal samples collected from selected poultry houses. The samples were collected in small plastic bottles. This was followed by antemortem and postmortem examination of representative birds selected from the same poultry houses and identification of species of coccidia involved.

3.2.1.1. Parasitological examination of faecal samples

Faecal examination was carried out to demonstrate the presence and level of coccidia infection in the poultry farms. The samples were collected in plastic bottles and brought to the parasitology laboratory where they were examined. Where the samples were not immediately examined they were stored at refregeration temperature (about 4 °C) until examination.

a). Qualitative faecal examination: This involved separation of the coccidia oocysts from the faecal samples, followed by direct microscopic detection. It was carried out by salt floatation using saturated sodium chloride solution according to the technique described by Hansen and Perry (1993).

b). Quantitative faecal examination: Determination of coccidia oocysts per gram of faeces (OPG) was performed for samples in which oocysts were detected in 3.2.1.1. (a). The Modified McMaster Technique as described in MAFF (1982) was followed. In cases of very high counts, dilution of the faecal suspension to 10 times its original volume was done to simplify counting.

c). Sporulation of coccidia oocysts: To assist in species identification, sporulation of coccidia oocysts was carried out. Coccidia oocysts were separated from faecal debris and subjected to salt floatation as described in 3.2.1.1 (a). The oocysts were removed from the surface of the fluid,

washed with water to remove excess salt, were suspended in 2.5% (w/v) potassium dichromate and incubated at 26°C. At intervals, air was bubbled through the suspension. The oocysts were observed for sporulation for 2-4 days. Sporulation time was recorded when at least 90% of oocysts have sporulated (Conway and McKenzie, 1991).

d). Morphology and measurement of sizes of sporulated coccidia oocysts: A suspension containing sporulated oocysts was drawn from the petri dish using a pasteur pipette. One or two drops of the suspension was placed on a microscopic slide and covered with a cover slip. The morphology and sizes of sporulated oocysts were microscopically determined using a calibrated ocular micrometer at 40X magnification. The sizes for a given specie (length and width) were determined by measuring at least 50 oocysts and calculating the average acording to Long and Reid (1982).

3.2.1.2. Antemortem and postmortem examination of birds

Birds for antemortem and postmortem examination were randomly selected from poultry houses in all the study farms in which coccidiosis was a problem. The procedure followed was as described by Long and Reid (1982) and Conway and McKenzie (1991).

General body condition of the birds was assessed. The birds were then examined for clinical signs suggestive of coccidiosis for example diarrhoea, dysentry, soiling of the vent as well as change in colour, composition and consistency of faeces.

a) Gross examination. After antemortem examination, the birds were killed by cervical dislocation and opened to expose abdominal cavity and viscera. The abdominal cavity and viscera were examined for gross pathological changes. The unopened intestines were thereafter freed from the mesentries along the entire length and rinsed in running tap water to remove traces of blood. The intestines were examined for gross changes of the serosal surfaces. Using a scissor, the intestines were opened at different positions, ie, duodenum, mid intestine before and after the Meckel's diverticulum, the lower intestine and the caeca. The intestinal walls and the mucosa were examined for gross lesions related to coccidiosis. Lesions were scored on a scale ranging from 1-4 whereby 1 = mild lesion, 2 = moderate lesion, 3 = severe lesion and 4 = very severe lesion. A score of 0 meant no lesion (Conway and McKenzie, 1991).

b) **Microscopic examination:** Mucosal scrapings for microscopic examination of developmental stages of coccidia were taken from segments of intestines with lesions. They were placed on microscopic slide, diluted with one or two drops of tape water, mixed thoroughly and covered with coverslip. They were examined under light microscope.

A bird was defined as a clinical case if it showed suggestive signs of coccidiosis and was confirmed at postmortem (gross and microscopic) examination. A bird was defined as a subclinical case if no clinical signs suggestive of coccidiosis were detected at antemortem examination but coccidiosis was diagnosed following postmortem examination.

c) **Histopathological examination**: Segments of intestines of about 1-2 cm for histopathology were taken from parts of the intestine with lesions and fixed in 10% (v/v) neutral buffered formalin. The segments were trimmed, dehydrated in alcohol, cleared in xylene and embedded in paraffin wax. Sections of about 2-3μm thickness were made and stained with Haematoxylin and Eosine (H & E) as described in Luna (1968). They were examined for histopathological changes under light microscope.

3.2.1.3. Identification of species of coccidia

This was based on the findings in subsections 3.2.1.1 (c), 3.2.1.1 (d) and 3.2.1.2 regarding sporulation time, shape and size of sporulated oocysts, and on the nature/type and location of the observed gross, microscopic and histopathological changes. The information from all these findings were combined and compared with the identification key given by Long and Reid (1982) for confirmation of the species identified.

3.2.1.4. Questionnaire survey and assessment for potential risk factors for coccidiosis

At least one set of questionnaires was administered to every farm which was included in the study. Targeted respondents were farm managers or farm veterinarians for large scale poultry farms. For small scale poultry farms respondents were farm owners.

The questionnaires focused on general management practises, farm disease history, disease control measures and on major factors which could act as potential risk factors for coccidiosis.

Potential risk factors included rearing systems used in the farm, stocking densities, ventilation systems, biosecurity practices, use of coccidiostats in the feed or water, availability and quality of feed, watering and feeding equipments, litter management and relative humidity in the poultry houses. Chi-Square test and Odds Ratios was used to determine existence of association between the potential risk factors and occurence of disease. Both Chi-square test and Odds ratios showed that no significant (p-values > 0.05 and odds ratios ≤ 1) association existed between any of the potential risk factors and occurence of the disease.

3.2.2. Retrospective study

A retrospective study was carried out in a total of 8 poultry farms (4 large scale and 4 small scale) to assess the economic impact of the diseases by examining farm records. Only farms in which coccidiosis was a problem were included.

Records were collected on major inputs and outputs involved in the poultry enterprises. The economic impact of the disease was assessed in the context of how and how much the disease affected the level of inputs and outputs and hence the level of income.
Structured questionnaires were used to obtain additional information on the disease and its economic impact that could not be obtained from farm records. They were also used to assess the farmers awareness of the disease and its impact on the performance of their enterprises.

The information from farm records and questionnaires were later combined to enable qualitative and quantitative assessment of the economic impact of the disease (DijKhuizen and Morris, 1996).

3.2.2.1. Qualitative assessment

This involved identifying and describing the various forms in which economic loses due to coccidiosis occured and the ways in which they affected the farmers income and expenditure.

3.2.2.2. Quantitative assessment

Enterprise Gross Margin Analysis (Martin *et al*, 1987; Rushton *et al*, 1999) was used to quantify the effects identified under 3.2.2.1. An attempt was made to compare the economic impact of the disease between production systems.

3.3. Sample size determination for cross sectional study

a). Faecal samples: The sample size (n) was estimated depending on the expected prevalence (p) of coccidiosis in the study area and the desired absolute precision (d) according to Thrusfield (1995) as follows:

$$n = \frac{Z^2 \times p(1 - p)}{d^2}$$

Where Z (a multiplier for 95% confidence interval based on the normal distribution) = 1.96, p = 50% (Fessesse-work, 1990; Kalifa 1997) and d = 5%. The required sample size for each production system was estimated to be 384. Thus, total faecal samples for two production systems were estimated to be 384 x 2 = 768. However, taking into account farm variabilities such as farm size (number of houses per farm and number of birds per house) and rearing systems used in different farms a total of 1028 faecal samples were collected as follows: 621 samples from 11 large scale farms and 407 samples from 15 small scale farms.

b). Number of live birds: A modification of minimum sample size suggested by Braunius (1987) and Razmi and Kalideri (1999) of 10 birds/10, 000 birds was taken. On average, 15 to 20 birds per farm were selected from every small scale poultry farm depending on flock size. For large scale poultry farms 30 to 35 birds per farm were selected, except in one very large farm from which 62 birds were selected. A total of 420 birds were selected of which 191 were from large scale poultry farms and 229 from small scale poultry farms.

3.4. Sampling procedures

The production systems in which the study was conducted were purposively selected. For each production system, all poultry farms which could be identified were included in the study.

Except in large farms with more than four poultry houses, faecal and necropsy samples were randomly selected from all poultry houses. Where the farm had more than four poultry houses, a two stage simple random sampling procedure was adopted by selecting the poultry houses first and then the faecal or necropsy samples in the selected poultry houses (Putt *et al*, 1988; Thrusfield, 1995). Feacal samples were picked by moving in a zigzag way around the entire poultry house. In farms where birds were kept in cages, faecal sampling was done in such a way that all places were represented. Birds for necropsy were picked in a similar mannar.

3.5. Data management and analysis

The data collected were managed as databases in MS-Access and MS-Excel.

Descriptive statistics were performed using MS-Excel and Statgraphics. OPG counts were logarithmically transformed prior analysis. Means of OPG counts and their respective standard deviations were recorded using their original scale (Berriatua *et al*, 1994). The mean OPG counts among different production systems were compared using Kruskal Wallis test. Testing for significant difference in frequency of detection of coccidia oocysts in faecal samples and prevalence of coccidiosis among different poultry production systems and age groups of chicken was performed using Chi-Square test. Assessment for economic impact of coccidiosis was performed using gross margin analysis (Rushton *et al*, 1999). All graphs were drawn in MS-Excel. Advanced statistical analysis such as testing for association between occurence of coccidiosis and factors such as age of birds, production system, management practices, ventilation system, amount of moisture in litter, flock size, farm age, level of biosecurity practices and water and feeding systems was performed using Chi-Square test and Odds ratios in Epi info 2000.

4. RESULTS

4.1. Cross sectional study

4.1.1. Parasitological examination of faecal samples

Coccidia oocysts were detected in 220 (80.3%), 8 (6.8%), 11 (4.8%), 231 (72.9%), and 73 (81.1%) faecal samples examined from large scale broiler farms, large scale layer farms with deep litter rearing system, large scale layer farms with cage rearing system, small scale broiler and small scale pullet rearing farms, respectively (Table 1).

Table 1. **Frequency of detection of coccidia oocysts in faecal samples from different production and rearing systems in Debre Zeit and Addis Ababa, Ethiopia. (n=1028).**

Production system	Rearing system	Number of farms	Samples examined	Positive samples	Percentage positive (CI)	Mean OPG ± SD (CI)
Large scale broilers	Deep litter	5	274	220	80.3% (75-84.8)	3687 ± 6 (2925-4645)
Large scale layers	Deep litter	2	118	8	6.8% (3-12.9)	193 ± 2 (148-251)
Large scale layers	Cage system	4	229	11	4.8% (2.4-8.4)	305 ± 2 (243-386)
Small scale broilers	Deep litter	12	317	231	72.9% (67.6-77.7)	2553 ± 7 (1981-3289)
Small scale pullets	Deep litter	3	90	73	81.1% (71.5-88.6)	1450 ± 4 (1070-1965)

There was a significant (p-value = 0.000) difference in frequency of detection of coccidia oocysts in faecal samples among the different poultry production systems. The frequency was highest in small scale pullet farms (81.1%) followed by large scale broiler farms (80.3%) and small scale broiler farms (72.9%). It was lowest in large scale layer farms with cage system (4.8%) followed by large scale layer farms with deep litter rearing system (6.8%).

The means of OPG counts were significantly (p-value = 0.024) higher in broilers and pullet rearing farms than those from layer farms. There was no significant (p-value = 0.296) difference between means of OPG counts in large scale broiler and small scale broiler/pullet farms. Similarly no significant (p-value = 0.563) difference of means of OPG counts between small scale broiler farms and small scale pullet farms, and between large scale layer farms with different rearing systems (p-value = 0.064).

4.1.2. Antemortem and postmortem examination of birds

Clinical signs suggestive of coccidiosis observed at antemortem examination included unthriftness, rough feathers, soft to watery faeces with mucus or blood. Specific lesions were observed following opening the abdominal cavity and systematic examination of the gastrointestinal tracts (Table 2).

Table 2. **Frequency of detection of coccidiosis lesions in intestinal tracts of necropsied birds from different production systems in Debre Zeit, Ethiopia. (n=420).**

Production system	Number of farms	Birds examined	Birds positive	Clinical coccidiosis	Subclinical coccidiosis	Percentage positive (CI)
Large scale broiler	5	191	92	16/92 (17.4%)	76/92 (82.6%)	(48.2%) (40.9-55.5)
Small scale broilers	12	199	94	22/94 (23.4%)	72/94 (76.6%)	47.2% (40.1-54.4)
Small scale pullets	2	30	12	4/12 (33.3%)	8/12 (66.7%)	40% (22.7-59.4)

Infection with coccidia was diagnosed in 92 (48.2%), 94 (47.2%) and 12 (40%) birds from large scale broiler, small scale broiler and small scale pullet rearing farms, respectively. There was no significant (p-value = 0.731) difference in overall prevalence of coccidiosis among large scale and small scale poultry farms. Most cases were subclinical with proportions of 82.6%, 76.6% and 66.7% of all positive cases coming from large scale broiler, small scale broiler and small scale pullets rearing farms, respectively. These were characterised by mild to moderate lesions. Only few cases 17.4%, 23.4% and 33.3% of all positive cases from large scale broiler, small scale broiler and small scale pullet rearing farms, respectively, showed clinical signs of coccidiosis. Figure 1 compares the proportions of clinical and subclinical coccidiosis among the different production systems. There was no significant (p-value = 0.761) difference in cases of clinical coccidiosis among the different production systems.

Figure 1. **Comparison between proportions of clinical and subclinical coccidiosis among different poultry production systems in Debre Zeit, Ethiopia. (n=420).**

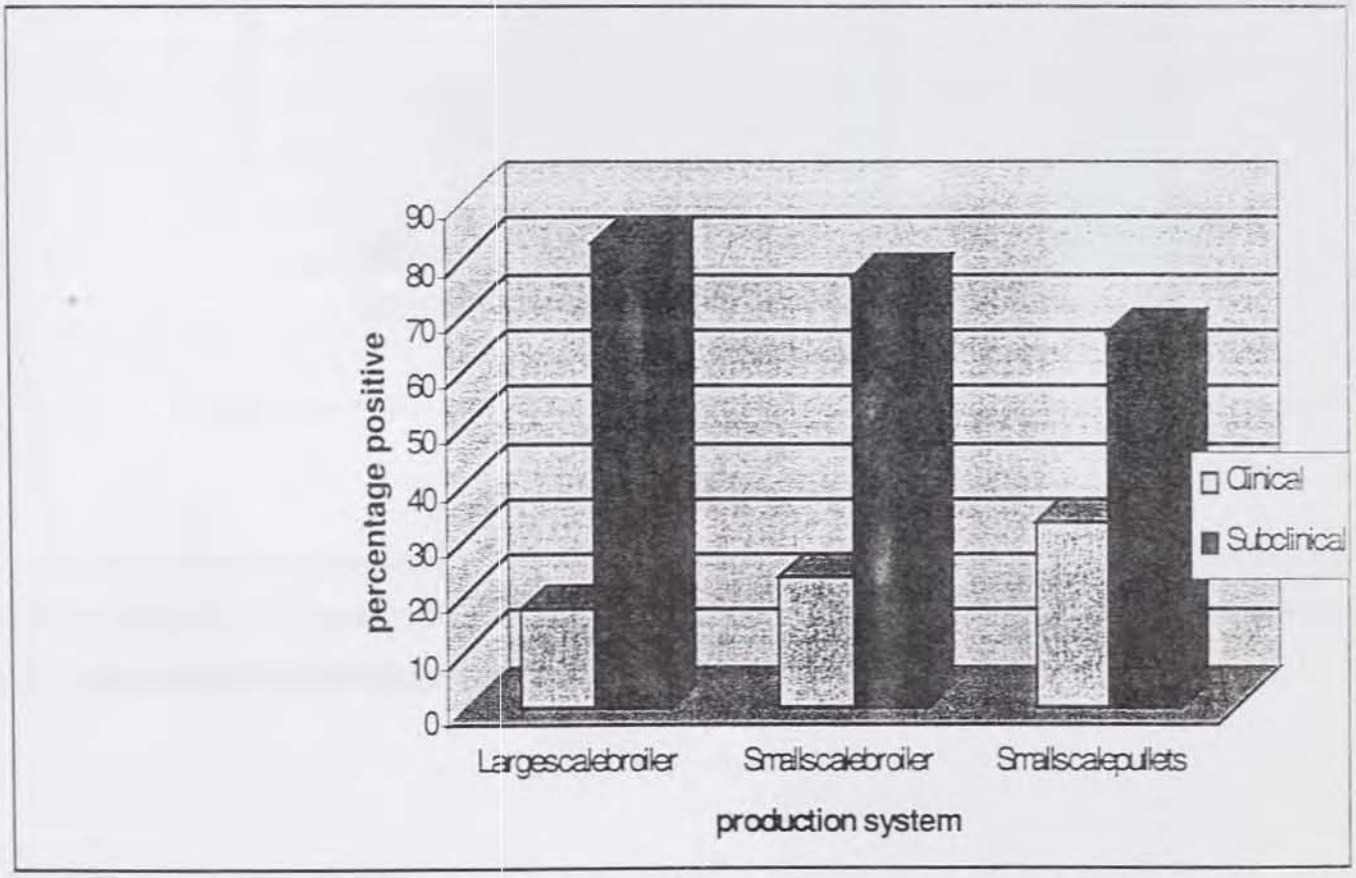
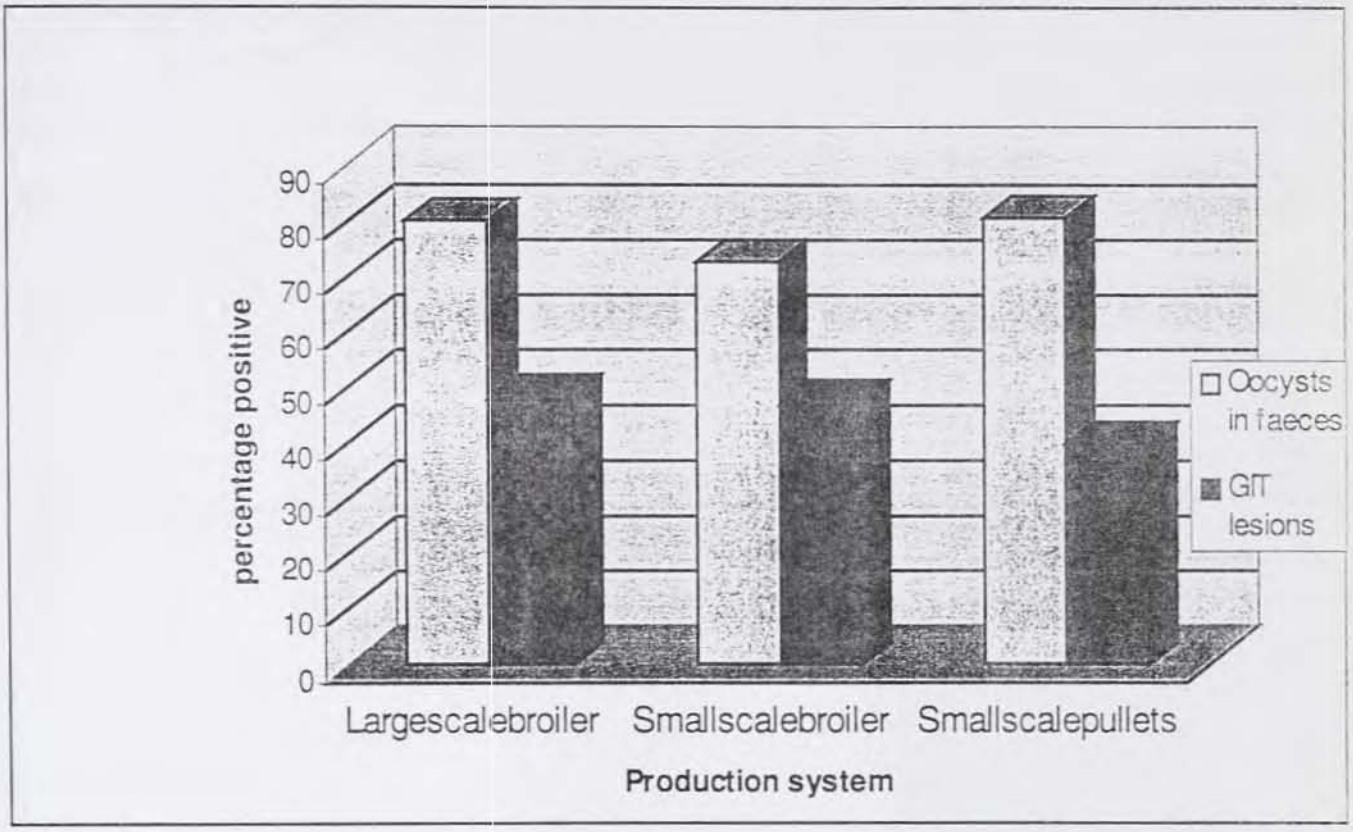


Figure 2 shows a comparison between frequency of detection of coccidia oocysts in faecal samples and coccidiosis lesions in the intestinal tracts of necropsied birds from the same poultry houses. The frequency of detection of coccidia oocysts was significantly (p-value = 0.048) as higher compared to the frequency of detection of coccidia lesions.

Figure 2. **Comparison between frequency of detection of coccidia oocysts in faecal samples and occurence of coccidiosis lesions in the gastro-intestinal tracts (GIT) of birds necropsied from different production systems in Debre Zeit, Ethiopia. (n=420).**



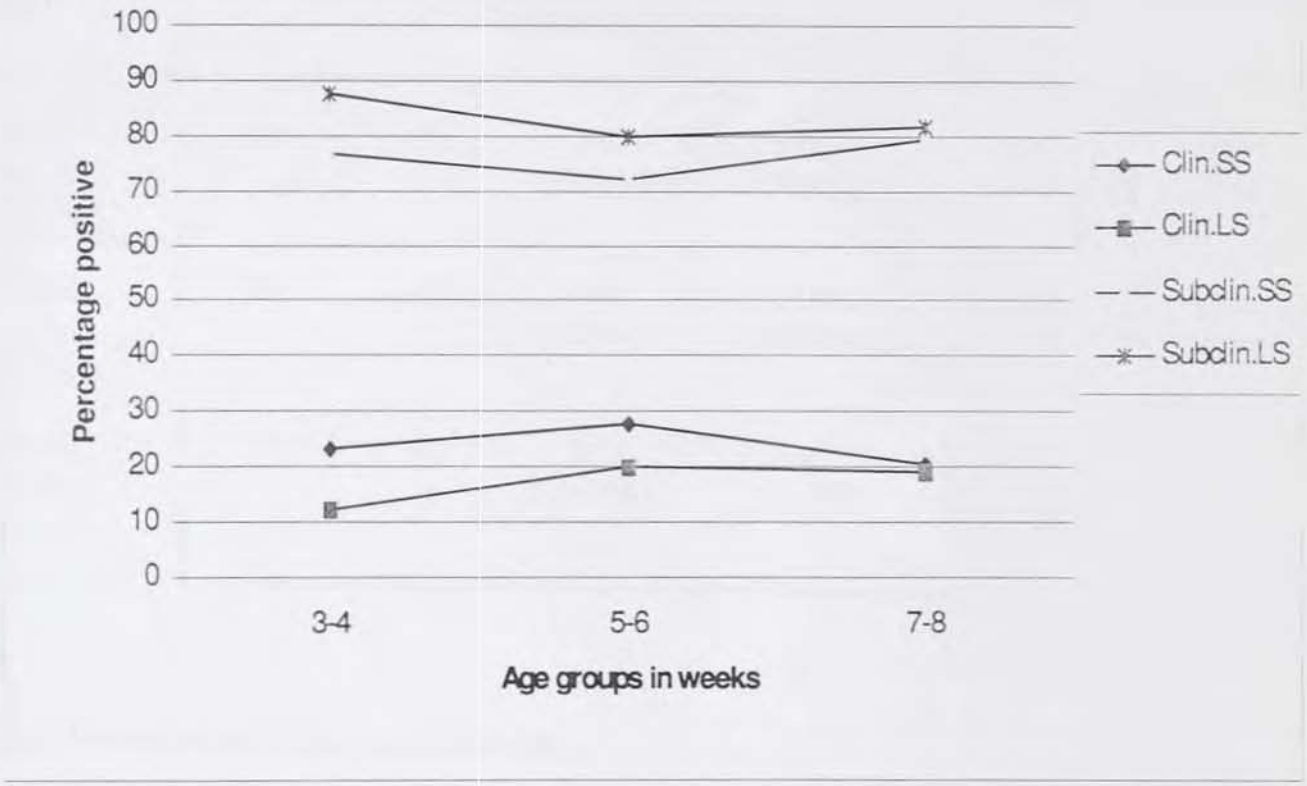
The proportions of clinical and subclinical coccidiosis among different age groups of chicken are shown in Table 3. Cases of clinical coccidiosis were higher, though not statistically significant (P-value = 0.905) in the age group of 5-6 weeks compared to other age groups.

Table 3. **Proportions of clinical and subclinical coccidiosis in large scale and small scale poultry production systems by age groups in Debre Zeit, Ethiopia. (n=420).**

Production system	Age group (weeks)	Number of farms	Samples examined	Positive samples	Clinical coccidiosis	Subclinical Coccidiosis
Large Scale	3-4	2	44	24 (54.5%)	3/24 (12.5%)	21/24 (87.5%)
	5-6	2	32	10 (31.3%)	2/10 (20%)	8/10 (80%)
	7-8	4	115	58 (50.4%)	11/58 (19%)	47/58 (81%)
Small Scale	3-4	3	47	17 (36.2%)	4/17 (23.5%)	13/17 (76.5%)
	5-6	7	115	50 (43.5%)	14/50 (28%)	36/50 (72%)
	7-8	5	67	39 (58.2%)	8/39 (20.5%)	31/39 (79.5%)

Figure 3 shows how cases of clinical and subclinical coccidiosis varied with age of chicken in small scale and large scale farms.

Figure 3. **Comparison between proportions of clinical (Clin) and subclinical (Sbclin) coccidiosis among different age groups of chicken in small scale (SS) and large scale (LS) farms in Debre Zeit, Ethiopia. (n=420).**



Clinical casese were relatively higher in the age group of 5-6 weeks as compared to other age groups in both small scale and large scale farms, whereas cases of subclinical coccidiosis were higher in the age groups of 3-4 and 7-8 weeks. At the age of 3-4 and 5-6 weeks, small scale farms had more clinical cases compared to large scale farms (23.5% and 28% compared to 12.5% and 20% respectively). However as the age increased to 7-8 weeks, the proportions of clinical cases became more or less the same in both production systems (19% and 20.5% respectively).

The proportions of observed severe and mild/moderate lesions compared to the occurence of clinical and subclinical coccidiosis are shown on table 4.

Table 4. **Proportions of severe and mild/moderate lesions compared to the occurence of clinical and subclinical coccidiosis in different production systems in Debre Zeit, Ethiopia. (n=420).**

Production system	Samples examined	Positive samples	Severe lesions (Score 3-4)	Mild/moderate lesions (Score 1-2)	Clinical coccidiosis	Subclinical coccidiosis
Large scale broilers	191	92	7/92 (7.6%)	85/92 (92.4%)	16/92 (17.4%)	76/92 (82.6%)
Small scale broilers	199	94	9/94 (9.6%)	85/94 (90.4%)	22/94 (23.4%)	72/94 (76.6%)
Small scale pullets	30	12	2/12 (16.7%)	10/12 (83.3%)	4/12 (33.3%)	8/12 (66.7%)

4.1.3. Identification of species of coccidia.

Four *Eimeria species* in chicken namely *Eimeria acervulina*, *Eimeria necatrix*, *Eimeria maxima* and *Eimeria tenalla* were identified in this study as shown in table 5. Mixed infections recorded were mainly due to *Eimeria acervulina* and *Eimeria tenella*.

Table 5. **Prevalent species of chicken coccidia identified at necropsy of birds from different production systems in Debre Zeit, Ethiopia. (n=420).**

Species	Region Parasitized	Shape of oocysts	Size of oocysts±SD	Sporulation time (Hrs±SD)	Number of samples	Percentage Positive
Eimeria acervulina	Doudenm/j ejunum	Ovoid	18.36 x 14.48 ±1.3 ±1.8	24 ± 2.2	68	34.4%
Eimeria necatrix	mid intestine	Ovoid	15.83 x 13.32 ±1.4 ± 0.96	48 ± 2.5	8	4%
Eimeria Maxima	mid intestine	Ovoid	30.4 x 22.6 ±1.8 ±2.9	48 ± 1.8	17	8.6%
Eimeria Tenella	Caeca	Ovoid	21.63 x 17.8 ±1.35 ±1.2	24 ± 2.3	65	32.8%
Mixed infections					40	20.2%

The proportions of the prevalent species in the study area indicate that *Eimeria acervulina* and *Eimeria tenella* were the most prevalent species (34.4% and 32.8%, respectively), whereas *Eimeria maxima* and *Eimeria necatrix* were less prevalent (8.6% and 4%, respectively).

The distribution of Eimeria species between the two production systems is shown in Table 6. The species were uniformly distributed between the two production systems except for *Eimeria necatrix* which occurred at a relatively higher frequency in small scale farms (75%) as compared to large scale farms (25%).

Table 6. **Distribution of coccidia species in chicken in small scale (n=14) and large scale (n=5) poultry farms in Debre Zeit, Ethiopia.**

Species	Number of samples	Species distribution	
		Small scale farms	Large scale farms
Eimeria acervulina	68	38/68 (55.9%)	30/68 (44.1%)
Eimeria necatrix	8	6/8 (75%)	2/8 (25%)
Eimeria maxima	17	10/17 (58.8%)	7/17 (41.2%)
Eimeria tenella	65	34/65 (52.3%)	31/65 (47.7%)
Mixed infections	40	19/40 (47.5%)	21/40 (52.5%)

4.1.4. Gross and histopathological changes associated with coccidiosis in chicken

The species specific lesions identified were as follows:

Eimeria acervulina: Affected parts of the duodenum were grossly enlarged except in mild to moderate infections where it appeared normal. Intestinal walls were thickened and congested. In most cases, intestines contained brown to yellowish watery fluid. In severe cases the lesions extended to the jejunum and were characterised by mucoid enteritis with pale to yellow or brownish mucoid exudate covering the duodenal mucosa. Small ovoid oocysts and gametocytes were observed in mucosal scrapings observed under light microscope.

Histopathology of affected tissues revealed schizonts in epithelial cells lining the borders of affected villi and numerous inflamrnatory cells. Affected villi lost their tips, were truncated and fused. Tissue damage caused by this species was minimal.

Eimeria necatrix: Lesions due to this species were found mainly in the jejunum to a small distance past the Meckel's diverticulum and were characterised by diffuse petechial haemorrhages seen from the serosal surface. Affected parts of the intestine were thickened and luminal contents were filled with blood and tissue debris. Mucosal scrapings contained large schizonts and merozoites.

Histopathology of affected segments revealed severe tissue destruction and sloughing off of epithelial cells into the lumen. Clusters of large schizonts were seen in epithelial cells. There was diffuse infiltration with inflammatory cells.

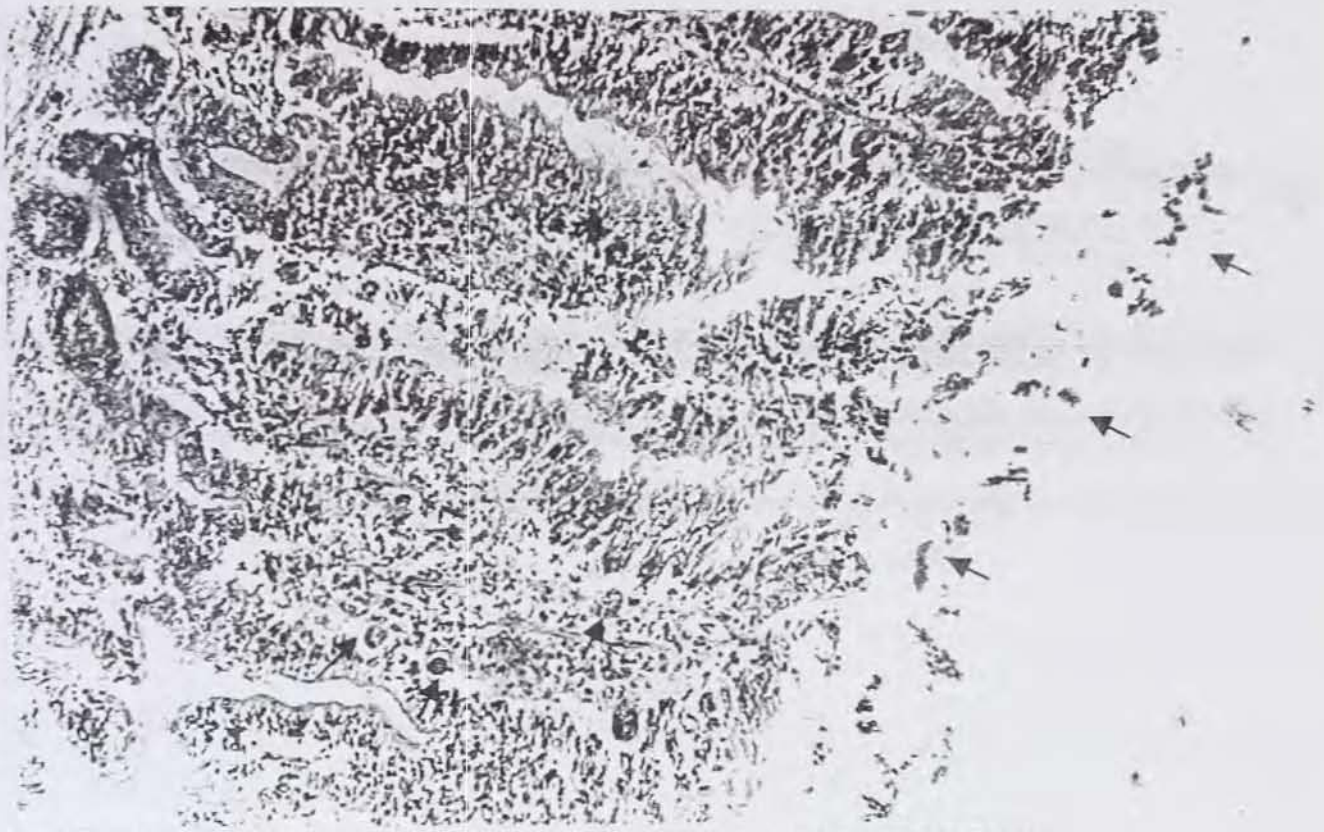
Eimeria maxima: Lesions due to this species were located in the mid intestine below duodenum. Grossly, affected parts appeared pale, flaccid and had no blood supply. Intestinal walls were thickened and contained thick brown to yellowish mucoid exudate. Characteristic large yellowish oocysts were seen in the mucosal scrapings examined under light microscope.

Histopathological findings included small schizonts and large gametocytes located proximal to host cell nucleus and muscularis mucosa, respectively. Tissue demage due to this species was minimal. There was evidence of infiltration with inflammatory cells.

Eimeria tenella: Lesions due to this species were located in the caeca, and were characterised by petechial haemorrhages clearly seen from the serosal surfaces. Except in mild infections, the caeca were distended, caecal walls thickened and the luminal contents were composed of blood and tissue debris. In few cases, hard caecal cores were observed. Microscopically, oocysts were seen in luminal contents and schizonts in mucosal scrapings.

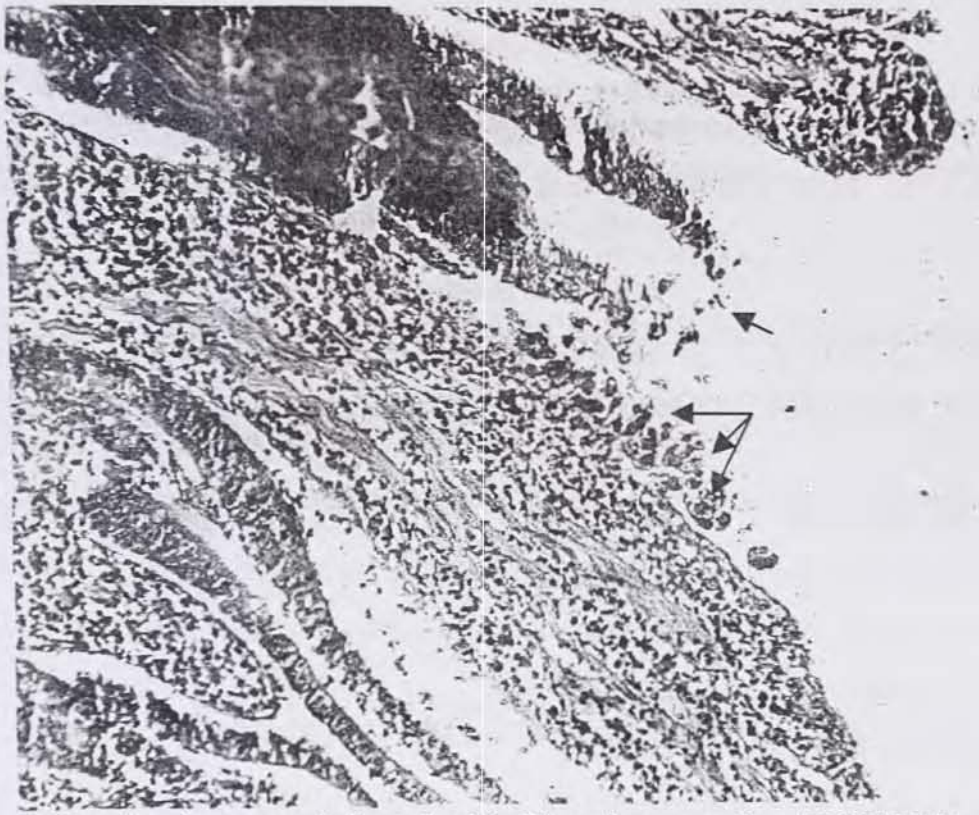
Histopathology of the affected tissues revealed severe destruction of the caecal mucosa with sloughing off of mucosal cells accompanied by cellular infiltration. Microscopic haemorrhages and congestion of blood vessels were observed in the submucosa and muscle layers. Clusters of large schizonts were seen in caecal glands.

In all species, cellular infiltration was a key feature. The cells which predominated were lymphocytes, monocytes, eocinophils, macrophages and plasma cells.

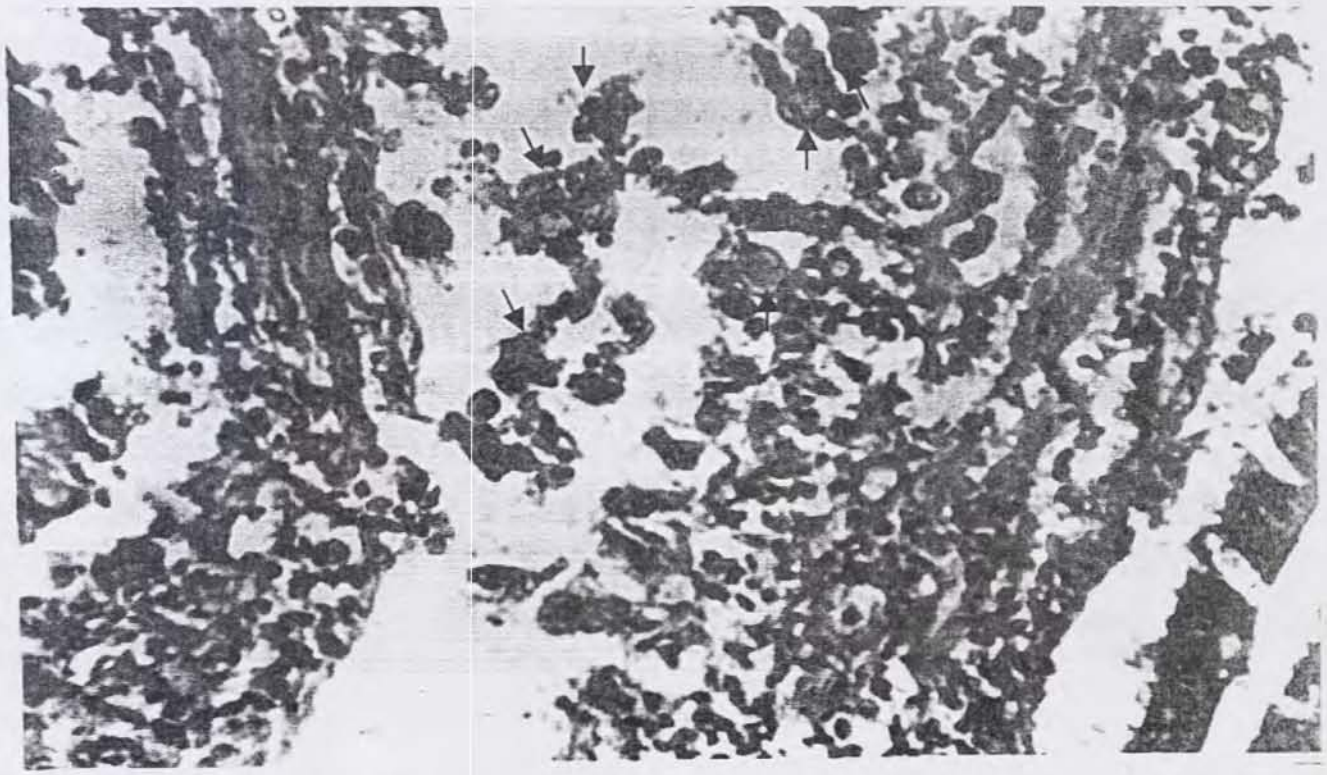


1. **Cells of mid-small intestine infected with *Eimeria maxima* (H&E X40)**
   Schizonts and gametocytes in epithelial and subepithelial cells of infected villi
   Evident tissue damage with some epithelial cells sloughed off
   Tissues underlying parasitized cells infiltrated with inflammatory cells

2. **Caecal mucosa infected with *Eimeria tenella* (H&E X40)**
   Destruction of caecal mucosa, sloughing off of epithelial cells
   Caecal glands dilated, and filled with clusters of large schizonts
   Microscopic haemorrhages
   Congested blood vessels



3. Duodenal mucosa infected with *Eimeria acervulina* (H&E X40)
Schizonts in epithelial cells lining the borders of parasitized villi
Affected villi fused, truncated and lost their tips
Tissues underlying parasitized cells infiltrated with inflammatory cells



4. Mucosa of the jejunum infected with *Eimeria necatrix* (H&E X40)
Severe tissue destruction and sloughing off of parasitized epithelial cells
Clusters of large schizonts in parasitized cells
Tissues undelying cells infiltrated with inflammatory cells

4.1.5. Questionnaire survey and assessment for potential risk factors for coccidiosis

Responses from questionnaire survey are summarised in Appendix 2. A description of farm management practices in the surveyed farms and potential risk factors for coccidiosis was as follows:

4.1.5.1. Farm management practices

All farms practised intensive system of management. Broiler and pullet farms had deep litter system of rearing, whereas layer farms had either cage rearing system or deep litter rearing system. General management practices with regard to housing, stocking density, ventilation system, quality of feeding and watering systems, biosecurity practices and disease control measures were done according to recommended standards in large scale farms. Feeders and drinkers were proportional to the number of birds in the poultry houses and were placed just at the right height from the ground. Water and feed was provided ad-libitum for broiler farms and was controlled acording to prescribed production standards in layer and pullet farms.

Some large scale farms had automated watering and feeding systems. Most large scale farms had computerised record keeping systems. Farms with deep litter systems of rearing used locally available teff straw which was replaced at the end of the rearing period for broiler and pullet farms, or at any time when necessary in layer farms. All broiler and pullet farms and some of layer farms used coccidiostats in the feed. Amprolium was used in the starter feed and Lasalosid in the finisher feed.

In small scale farms houses were simple with small or no windows. Spaces were small in proportion to the number of birds kept. Watering and feeding equipments allowed contamination of water and feed with bird droppings. Most farms did not keep records. Feed was provided ad-libitum and coccidiostats were used in the feed.

4.1.5.2. Potential risk factors for coccidiosis

Potential risk factors for coccidiosis in surveyed farms included age of birds, production system, flock size, stocking density, amount of moisture in the poultry houses, ventilation systems,

equipment quality, level of biosecurity practices and age of the farms. These were cross-tabulated against the frequency of occurence of coccidiosis and summarised in Table 7. No significant (p-values > 0.05) associations were obtained between coccidiosis and any of these factors.

Table 7. **Cross-tabulation for potential risk factors against occurence of coccidiosis in small scale and large scale farms in Debre Zeit, Ethiopia. (n=420).**

Risk factor	Level/category of risk factor	Degrees of freedom	Chi-Square	p-value	Inference
Age of the birds (weeks)	3-4 5-6 7-8	2	5.20	0.074	No association
Production system	Large scale broiler Small scale broiler Small scale pullets	2	0.62	0.731	No association
Flock size	300-1000 1001-4000 >4000	2	2.06	0.357	No association
Stocking density	Average High	1	0.55	0.457	No association
Litter/air moisture	Average High	1	0.04	0.851	No association
Ventilation	Good Satisfactory Poor	2	0.44	0.802	No association
Equipment quality	Good Satisfactory Poor	2	4.49	0.106	No association
Biosecurity practices	Good Poor	1	0.00	1.000	No association
Age of the farm	0-6 months 7-12 months 1-2 years >2 years	3	4.15	0.246	No association

4.2. Retrospective study

4.2.1. Assessment of the economic impact of coccidiosis

Coccidiosis was identified to be a major cause of both direct and indirect losses in all farms. Losses occured in the form of mortalities, coccidiostat costs, reduced weight gains, reduced market value of affected birds, culling and delayed offtake. In layers, losses occurred in the form of reduced egg production. Questionnaire survey combining expert opinion indicated that the disease ranked number 2 or 3 as a cause of morbidity and mortalities after broiler ascites, colibacillosis and diseases caused by miscellaneous agents.

Enterprise gross margin analysis (Rushton *et al*, 1999; Martin *et al*, 1987) was used to quantify economic losses (Tables 8 and 9).

Table 8. **Quantification of economic losses resulting from coccidiosis in small scale (n=4) and large scale (n=4) poultry farms in Debre Zeit, Ethiopia.**

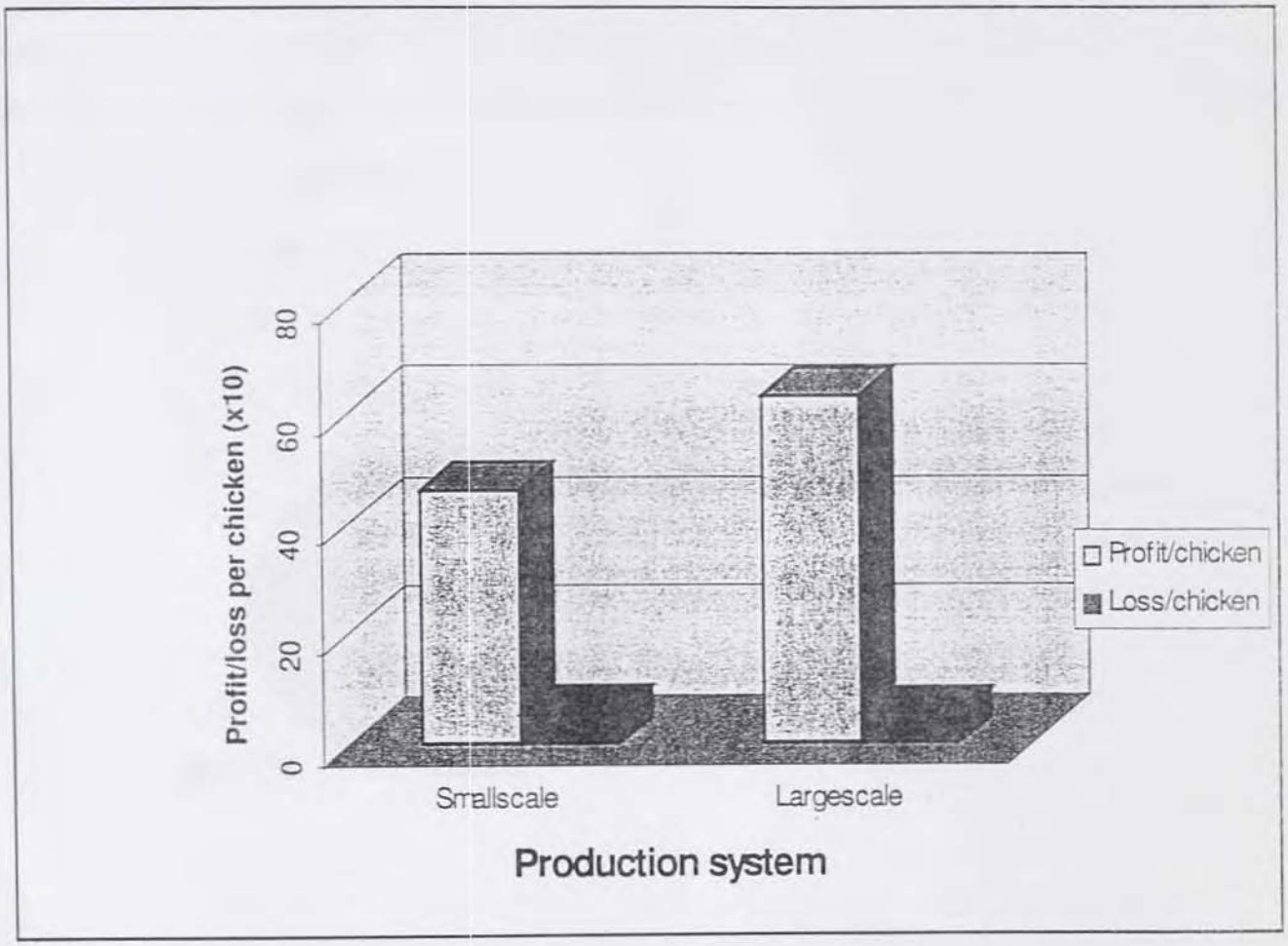
Loss factor		Loss (Ethiopian Birr)	
		Small scale farms	Large scale farms
Mortalities due to coccidiosis	(1)	429.24	3028.80
Coccidiostats cost	(2)	457.75	2273.00
Culling due to coccidiosis	(3)	11.78	0.00
Total loss	(1+2+3)	898.80	5301.80
Loss per chicken		0.55	0.53

Table 9. Estimation of farmers income from poultry keeping enterprises in small scale (n=4) and large scale (n=4) poultry farms in Debre Zeit, Ethiopia.

Income factor	Amount (Ethiopian Birr)	
	Small scale farms	Large scale farms
Output (1)	28883.05	156610.10
Variable costs (2)	20116.50	91479.53
Gross margin [3=(1-2)]	8766.55	65130.57
Fixed costs (4)	1187.00	2181.50
Profit (3-4)	7579.55	62949.07
Profit/chicken	4.64	6.33
Total loss due to coccidiosis as percentage of profit	11.86	8.42

Figure 4 shows a comparison of farmers income and losses due to coccidiosis between small scale and large scale poultry farms. Small scale farms incured more losses as compared to large scale farms, while large scale scale farms made more profits as compared to small scale farms.

Figure 4: **Comparison of farmers income (profit/chicken) against total loss due to coccidiosis (loss/chicken) between small scale (n=4) and large scale (n=4) poultry farms in Debre Zeit, Ethiopia.**

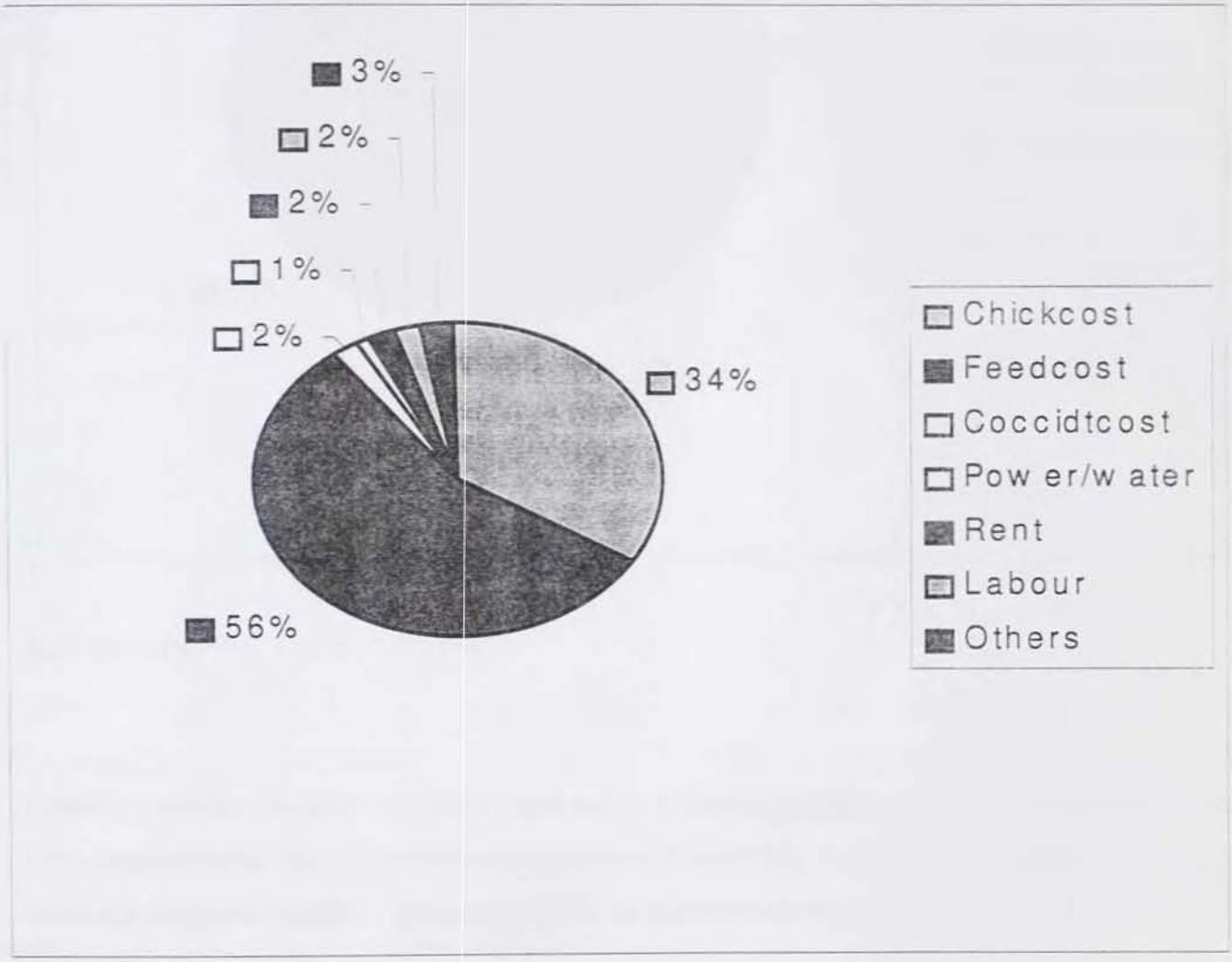


Total losses due to mortalities, culling and coccidiostat costs were estimated at Ethiopian Birr 898.80 and 5301.80/farm or 0.55 and 0.53 Ethiopian Birr/chicken in small scale and large scale poultry farms, respectively. This contributed to an average of 11.86% and 8.40% loss in enterprise profit/farm in the respective production systems. Average proportional mortality rates

due to coccidiosis were 14.5% and 13.3% in small scale and large scale poultry farms, respectively.

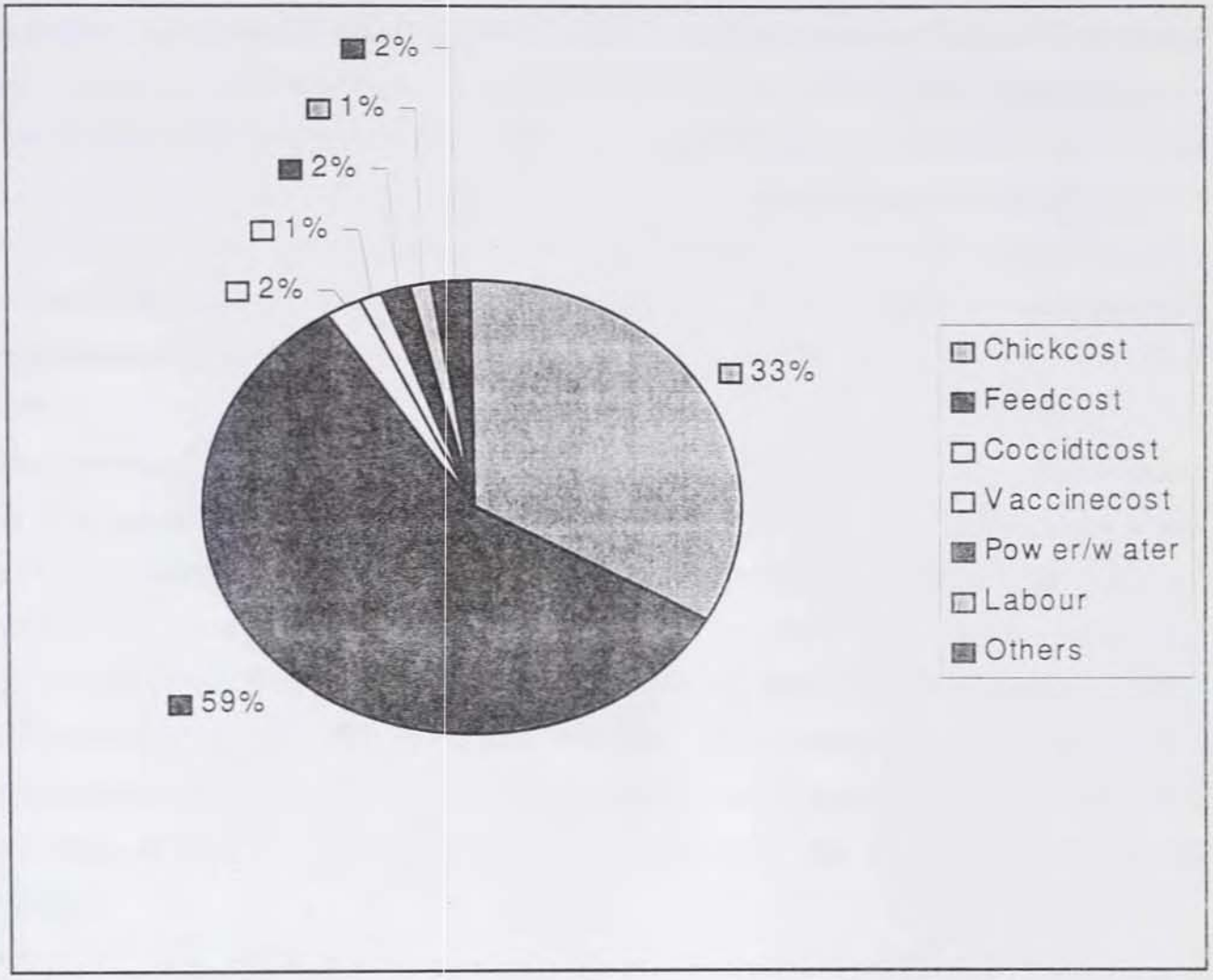
The average costs of major inputs used in both small scale and large scale poulty farms are shown in Figures 5a and 5b.

Figure 5a. **Average costs of major inputs used in small scale poultry farms in Debre Zeit, Ethiopia. (n=4).**



Key: Coccidtcost = Coccidiostat cost

Figure 5b. **Average costs of major inputs used in large scale poultry farms in Debre Zeit, Ethiopia. (n=4).**



Key: Coccidtcost = Coccidiostat cost

Chicken feed was the most expensive input for both small scale and large scale farmers (56% and 59%, respectively). This was followed by day old chicks (34% and 33%, respecively). Both small scale and large scale poultry farmers spent 2% of their overall expenditure for coccidiostats.

5. DISCUSSION AND CONCLUSIONS

5.1. Discussion

The present study showed that all tested flocks were positive for coccidia oocysts in faecal samples and coccidia lesions in the intestinal tracts of randomly selected birds. The percentages of faecal samples positive for coccidia oocysts were higher compared to the percentages of birds positive for coccidiosis lesions examined at postmortem examination from the same flocks. This provided further evidence that detection of coccidia oocysts in faeces does not mean diagnosis of coccidiosis (Urquhart, 1987; Reid, 1990; MAFF, 1982). In this case, there were birds shedding coccidia oocysts in faeces without having lesions due to coccidiosis.

Higher percentages of subclinical cases of coccidiosis were obtained compared to clinical cases. This is in agreement with published literature about the course of the disease, especially where control measures such as coccidiostats in feed were used. In such cases coccidia infections are known to take a subclinical course and only few cases manifest clinical signs of the disease. Similar findings have been reported by Voeten and Jansen (1983), McDougald and Reid (1997) and Braunius (1986). The results also showed that cases of severe lesions (lesion scores 3-4) did not correspond to the same number of cases of clinical coccidiosis and vice versa. This meant that some cases of mild to moderate lesions (lesion scores 1-2) also showed clinical signs of coccidiosis.

The study also recorded higher percentages of faecal samples positive for coccidia oocysts in broiler and pullet rearing farms compared to those that dealt with layers. This can be explained by the fact that layer farms kept older birds compared to broiler and pullet rearing farms. Coccidiosis in poultry is known to be a disease of young birds (Soulsby, 1982; McDougald and Reid, 1997). Therefore older birds kept in layer farms had built sufficient immunity. Furthermore, coccidiosis is transmited through the oral-faecal route. Most layer farms reared their birds in cages, hence birds had no access to coccidia oocysts since they had no contacts with faeces. This reduced the chance of infection. The low levels of coccidia oocysts detected in caged birds could be as a result of mechanical transmission by attendants or the birds might have been reared on litter prior to being caged.

Mean OPG counts recorded for each production system were not directly related to the levels of frequency and severity of the disease. One might expect a direct relation to exist due to the fact that coccidiosis results from ingestion of sporulated oocysts present in the environment where birds are kept. But in the current study results, this was not the case. Small scale pullet rearing farms had the lowest mean OPG counts as compared to large scale broiler and small scale broiler farms (Table 1). However they had the highest frequency of clinical cases (Table 2). This finding further supports the hypothesis that levels of frequency and severity of coccidiosis is not directly related to the level of shedding and hence contamination of the environment with coccidia oocysts (MAFF, 1982; Urquhart, 1987).

The results in the present study are in contrast with results from previous studies in Ethiopia that reported high percentages of clinical cases of coccidiosis up to 54.6 % (Fessesse-work, 1990) and 80% (Alamargot, 1987). This means that adopted control measures including improvements in management practices and use of coccidiostats in the feed have been effective.

When the overal prevalence of coccidia infections in the different production systems and among the different age groups of chicken were compared, there was no significant (p-values > 0.05) difference. This can be explained by the effect of management practices particularly with regard to the use of coccidiostats in the feeds. Questionnaire responses showed that all farms examined used coccidiostats in feed from the same manufacturer.

Assessment for potential risk factors for coccidiosis showed that all factors including those considered important such as age of the birds, flock size, stocking density, amount of moisture in litter, levels of biosecurity practices and equipment quality (McDougald and Reid, 1997; Braunius, 1987; Razmi and Kalideri, 1999; Graat *et al*, 1998) were not associated with the disease. This demonstrated the strong effect of coccidiostats in suppressing occurence of the disease despite the existence of coccidiosis promoting factors. Development of immunity might have contributed to this end.

Cases of clinical coccidiosis were higher in small scale farms as compared to large scale farms (Table 2 and figure 3), and higher percentages of severe lesions (lesion scores 3-4) were observed in small scale farms as compared to large scale farms (Table 4). On the contrary, the cases of subclinical coccidiosis and cases of mild to moderate lesions (lesion scores 1-2) occured at low

frequencies in small scale farms as compared to large scale farms. These differences however, were not justified following statistical analysis. There was no significant (P-values > 0.05) differences between prevalence rates of clinical coccidiosis or between severe lesions among the different production systems. Our observation and study however, indicated that management and environmental stress to which birds in small scale farms were exposed especially with regard to stocking densities, ventilation and high moisture levels in the poultry houses were important risk factors.

Higher percentages of cases of clinical coccidiosis in the 5-6 weeks age groups were observed compared to other age groups in all production systems (Table 3). This was because most coccidia infections occurs at the age of 3-4 weeks but clinical disease develop one or more weeks latter. As a result the peak of clinical disease appears at the age of 5-6 weeks. As age increases to 7-8 weeks, most birds develop immunity which suppresses development of clinical disease (Soulsby, 1982; McDougald and Reid, 1999). Because of the state of balance between immunity of the bird and tricke infection through continuous ingestion of small doses of oocysts, at the age of 7-8 weeks or more the most important form of the disease is subclinical. Braunius (1986), Reid (1990) and Razmi and Kalideri (1999) made similar observations.

In the present study four species of coccidia in chicken namely, *Eimeria acervulina, Eimeria necatrix, Eimeria maxima* and *Eimeria tenella* were identified. Although there were variations in the proportions of each species identified, the results are in agreement with previous studies in Ethiopia (Alamargot, 1987; Fessesse-work, 1990; Kalifa, 1997). Unlike in the study of Fessesse-work (1990), *Eimeria mivati* was not identified in this study. This can probably be due to management and environmental changes. Furthermore, there are close morphological and biological similarities between *Eimeria acervulina* and *Eimeria mivati* which might have made it difficult to differentiate the two species based on the methods of investigation applied in the current study.

The gross and microscopic changes observed for each species were quite similar to what was previously described in published literature (MAFF, 1982; Soulsby, 1982; McDougald and Reid, 1997). However, as most of the observed lesions were mild to moderate (lesion scaores 1-2), some of the features which are considered to be characteristic for a given species such as white

transverse (ladderlike) streaks in *Eimeria acervulina*, gross ballooning of the affected segments of intestines in *Eimeria necatrix* and *Eimeria maxima* were not be clearly observed in this study.

Questionnaire responses and our observation indicated that, except in one large scale farm and two small scale farms, coccidiosis was not highly ranked as the most important cause of losses as it used to be in the past (Alamargot, 1987; Kalifa, 1997). Most respondents ranked coccidiosis as second or third disease after broiler ascites, colibacillosis or cannibalism in layers. This was because the incidence of clinical coccidiosis have decreased due to implementation of effective control measures which include improvements in management and continuous administration of coccidiostats in the feed. Despite this advances in controling the disease, cases of subclinical coccidiosis are still significant. Therefore, taking into account the weight loss resulting from subclinical coccidiosis (Gordon and Jordan, 1982; McDougald and Reid, 1997;Vermeulen, *et al* 2001) and the high cost of medication (Table 8, Figures 5a and 5b), our opinion is that coccidiosis is still an important diseases that causes significant economic losses to the poultry industry in the study area.

Assessment of economic losses due to coccidiosis recorded average total losses of 898.8 and 5301.8 Ethiopian Birr/farm or 0.55 and 0.53 Ethiopian Birr/chicken in small scale and large scale farms, respectively. This contributed to an average of 11.86% and 8.4% loss in enterprise profit/farm in the respective production systems. This indicates that small scale poultry farms were losing more due to coccidiosis than large scale farms. However, when the amount spent on coccidiostats was compared (Figures 5a and 5b), it was observed that both farms spent 2% of their overall expenditure on inputs for coccidiostats. This observation meant that the excess financial losses that small scale poultry farms incurred were due to mortalities and (to a small extent) culling. This was supported by the cross sectional study (Table2) which showed that small scale poultry farms had more clinical cases of coccidiosis as compared to large scale farms. Proportional mortality rates due to coccidiosis were also high in small scale farms as compared to large scale farms (14.5% and 13.3%, respectively).

Small scale and large scale poultry farms spent 56% and 59%, respectively of their overal expenditure on chicken feed and 34% and 33%, respecively on day old chicks. This showed that chicken feed was the most expensive input on the producers budget. The reason could be that chicken as pigs compete with man for the same feed resources.

Large scale farms made more profit of 6.33 Ethiopian Birr/chicken as compared to 4.64 Ethiopian Birr/chicken for small scale farms. This reflected the market situation that is monopolised by large scale producers. They own almost 100% share of the market for poultry products (chicken meat and eggs) such that small scale farms have to sale their products through these large scale farms. At the same time large scale farms supply the major inputs for example day old chicks, feed and equipments to small scale farms. Thus large scale farms enjoys significant monopoly of the poultry bussiness in Ethiopia.

This study did not attempt to quantify economic losses due to losses in weight gains although it is known that they contribute the biggest portion of all losses due to coccidiosis (Braunius, 1980; Gordon and Jordan, 1982; McDougald and Reid, 1997; Vermeulen, 2001). The reason was that it was not easy to quantify losses in weight gains in the study area. Diseases other than coccidiosis, management and environmental factors such as nutrition, temperature, ventilation and higher stocking densities could also contribute to losses in weight gains. The three causes of losses which were quantified (mortalities, culling and costs of medication) showed significant economic losses.

5.2. Conclusion

In conclusion, coccidiosis is still a health problem of poultry production in urban and peri-urban areas of Ethiopia. The current study demonstrated high infection rates. The coccidia species identified were *Eimeria acervulina*, *Eimeria necatrix*, *Eimeria maxima* and *Eimeria tenella*, where *Eimeria acervulina* and *Eimeria tenella* were predorminant. Mixed infections of these species were detected. Infections due to *Eimeria tenalla* and *Eimeria necatrix* were characterised by haemorrhagic typhilitis and enteritis plus mortalities, whereas infections due to *Eimeria acervulina* and *Eimeria maxima* were characterised by catarrhal/mucoid enteritis with less mortalities. The disease caused high economic losses in the form of mortalities, costs of coccidiostats, reduced market value of affected birds, delayed offtake and in some cases led to culling of infected birds. According to this study, proportional mortality rates due to coccidiosis were 14.5% and 13.3% in small scale and large scale poultry farms, respectively. Total losses due to coccidiosis were estimated at an average of 898.80 and 5301.80 Ethiopian Birr/farm in small scale and large scale poultry farms, respectively which is equivalent to an average loss of 0.55

and 0.53 Ethiopian Birr/chicken, respectively. This contributed to an average loss in enterprise profits of 11.86%/farm in small scale farms and 8.4%/farm in large scale farms, respectively.

5.3. Recommendations

The results of the present study showed that clinical coccidiosis has been controled to a larger extent. In the past 10-15 years, incidences of clinical coccidiosis in the study area were higher and reached 80%. The disease used to be the most important cause of both morbidity and mortalities in all farms in Ethiopia (Alarmargot, 1987). However, the proportions of subclinical coccidia infections recorded by the present study are high, thus the current control measures should be strengthened. Other measures should be designed and implimented. Based on the results obtained, the following are recommended:

1. The high proportions of subclinical infections, and relatively high frequencies of occurence of species such as *Eimeria acervulina* and *Eimeria tenella* compared to such species as *Eimeria maxima* and *Eimeria necatrix* suggests existence of drug resistance. Drug sensitivity testing is therefore necessary to recommend the correct coccidiostats in the feed. Further programmes for using the coccidiostats like rotation or shuttle programmes should be applied.

2. Compliance with specified production standards in respect to stocking densities, ventilation systems, litter quality, equipment quality and number of birds in the poultry house in relation to available space and equipments is vital for control of coccidiosis.

3. Adherence to biosecurity measures and use of clean and dry litter will control not only coccidoisis but also bacterial and viral diseases.

4. Access to extension and veterinary services is very limited for small scale farms. An arrangement should be made at regional or national level to enable small scale farmers benefit from these inevitable services for the improvement and sustainability of this important production system. Enhancement of the small scale poultry production system will undoubtedly lead to poverty alleviation.

5. Establishment of small scale poultry farmers associations is recommended. This will facilitate access to credit facilities to meet the high initial investment in terms of animal, feed and equipment costs. It will also strengthen small scale poultry producer's market competitiveness.

6. REFERENCES

Alamargot, J (1987): Avian Pathology of Industrial Poultry Farms in Ethiopia. In: IAR (ed.) First National Livestock Improvement Conference. Addis Ababa: 11-13 February, 1987. Addis Ababa Agricultural Research Institute. pp 114-117.

Alemu, Y (1995): Poultry Production in Ethiopia. *World Poultry Science Journal* **51** (2), 197-201.

Alemu, Y., Tadelle, D (1997): The Status of Poultry Research and Development in Ethiopia. *Research Bulletin No.* 4. DZARC. 1997. pp. 40-46.

Backer, E. R (1969): Diseases of Poultry. 4th edition. Iowa, USA: The Iowa State University Press. pp 828-851.

Bailey, C. A (2001): Protozoan Diseases. A manual of Poultry Diseases. Texas A&M University. Texas Agricultural Extension Services. Internet information, August 26, 2000. www.GALLUS.TAMU.EDU/Diseases/AMOPD.html.

Berriatua, E., Green, L. E., Morgan, K. L (1994): A descriptive epidemioligical study of coccidiosis in early lambing housed flocks. *Veterinary Parasitology* **54**, 337-351.

Braunius, W. W (1980): Clinical aspects, detection methods and damage caused by coccidiosis in broilers. In: Some Aspects of Epidemiology and Control of Coccidiosis in Broilers. (Eds.): UTRECHT: Drukkerij Elinkwijik BV. pp 61-66.

Braunius, W. W (1986): Epidemiology of Eimeria in broilers in relation to anticoccidial drug. *Archive für geflugelkunde* **50** (3), 88-93.

Braunius, W. W. (1987): Epidemiology of Eimeria in broiler flocks and the effect of anticoccidial drugs on the economic performance. In: Some Aspects of Epidemiology and Control of Coccidiosis in Broilers. (Eds.): UTRECHT: Drukkerij Elinkwijik BV. pp 81-90.

Carmichael, I (1998): Coccidiosis. Proceedings of the General Meeting of the Marsupial Society of Australia Inc. Australia. Internet information, May. 21st, 1998. www.marsupialsociety.org.au/article001.htm.

Conway, D. P., McKenzie, M. E. (1991): Poultry coccidiosis. Diagnostic and Testing Procedures. 2nd edition. NETHERLANDS: Pfizer Inc. pp 7–14, 37-40.

CSA (1998): Ethiopian Livestock Estimates. Vol. I Bulletin 52. Addis Ababa, Ethiopia.

Danforth, H. D (2000): Making Coccidia Less Cocky. Agricultural Research Service, USA Department of Agriculture. Internet information, February 9, 1999. www.ars.usda.gov/is/AR/archive/jan99/cocc0199.html

David, W (1979): Poultry Production in Nigeria. In: Proceedings of the First National Seminar on Poultry Production. (Eds.): Zaria, Nigeria: Ahmadu Bello University.

DijKhuizen, A. A., Morris, R. S. (1997): Animal Health Economics. Postgraduate Foundations. (Eds.). Sydney: University of Sydney. Australia. pp 1-39.

Dwight, D. B (1995): Parasitology for Veterinarians. 7th edition. London: W. B. Saunders. pp 86-92.

EARO (2000a): Animal Science Research Strategy. Addis Ababa

EARO (2000b): Animal Science Research Directorate. Poultry Research Strategy. July 2000. Addis Ababa.

Edgar, S. A., Flangan, C., Tam, K. F., Bond, D (1968): Factors Related to Immunity of Chickens to Coccidiosis. *Poultry Science* **47**, 1668.

Elmer, R. N., Glenn, A. N (1982): Parasitology. The Biology of Animal Parasites. 5thedition. California: Lea & Febiger. pp 76-80.

FAO/ILRI (1995): Livestock Development Strategy for Low Income Countries. In: Proceedings of the Joint ILRI/FAO Round Table on Livestock Development Strategies for Low Income Countries, February 27-March 02, 1995. (Eds.): Addis Ababa: Ethiopia. pp 9-22.

FAO/WB (1993): Ethiopian Livestock Sector Development Project Report No. 24/93 CP-ETH 45 SR 18, Feb, 1993. Vol. I of II. Rome: FAO. 8-17 pp.

Fessesse-work (1990): Poultry Coccidiosis and Effect of Management System: An assessment trial in Debre Zeit and its Surroundings. Debre Zeit: FVM-AAU. DVM Thesis.

Fitz-coy, S. H., Edger, S. A (1992): The Effect of Eimeria mittis on Egg Production of Single Comb White Leghorn Hens. *Avian Disease* **36**, 719-721.

Fred, J. P., Carey, J. B., Jeffrey, J. S (2001): Biosecurity of Poultry Facilities. Prevention of Diseases Transmitted by Contaminated Facilities. Manual of Poultry Diseases. Texas A&M University. Internet information, August 26, 2000. www.gallus.tamu.edu/biofac.html

Gordon, R. F., Jordan, T. T. W (1982): Poultry Disease. 2nd edition. London: Bailliere Tindall. pp 166-181.

Graat, E. A. M., Ploeger, H. W., Henken, A. M., De Vries Reilingh, G., Noordhuizen, J. P. T. M., Van Beek, P. N. G. M (1996): Effects of Initial Litter Contamination Level with Eimeria acervulina on Population Dynamics and Production Characteristics in Broilers. *Veterinary Parasitology* **65**, 223-232.

Graat, E. A. M., Van der Kooj, E., Frankena, K., Hanken, A. M., Smeets, J. F. M., Hekerman, M. T. J (1998): Quantifying risk factors of coccidiosis in broilers using on farm data based on a veterinary practice. *Preventive Veterinary Medicine* **33**, 297-308.

Hagos, A (2000): Survey on Identification of Major Diseases of Local Chicken in Three Selected Agro-Climatic Zones in Central Ethiopia. Debre Zeit: FVM-AAU. DVM Thesis.

Hansen, J., Perry, B (1993): A handbook of Epidemiology, Diagnosis and Control of Helminth Parasites of Ruminants. 2nd edition. Nairobi: ILRAD Nairobi, Kenya. pp 57-62.

ILCA. (1993): Handbook of African Livestock Statistics. Addis Ababa, Ethiopia: International Livestock Centre for Africa. pp 15-52.

Jiffar, T (1982): Pathophysiological Studies on the Infectivity of the Coccidian parasite *Eimeria tenella* to the Woter Strain of Domestic Ethiopian Chicken and SC. White Leghon Breed of Chicken (*Gallus domesticus*). Addis Ababa: Addis Ababa University. MSC. Thesis.

Kabay, M (2001): Coccidiosis in Poultry. Disinfection. Agriculture Southern Australia. South Perth. Internet information, March 2nd, 2001. www.agric.wa.gov.au/agency/pubns/farmnote/1996/f02996.html

Kalifa, S. (1997): The Prevalence of Coccidiosis in Small and Large Scale Poultry Farms in Debre Zeit and Comparison of the Effect of Management Systems. Debre Zeit/Berlin: AAU/FUB. Postgraduate Diploma Thesis.

Kaufmann, J (1996): Parasitic Infections of Domestic Animals. A Diagnostic Manual. (Ed.) Berlin: Birkhäuser Verlag. pp 341-348.

Kennedy, M (2001): Coccidiosis in Chickens. Alberta Agriculture Food and Rural Development. Alberta. Internet information, March 2nd, 2001. www.agric.gov.ab.ca./agdex/600/63-65.html

Kouwenhoven, B., Kuil, H (1976): Demonstration of Circulating Antibodies to Eimeria tenella by the Indirect Immunofluorescent Antibody Test using Sporozoites and Second stage Schizonts as Antigens. *Veterinary Parasitology* **2**, 283-292.

Lister, S (2000): Coccodiosis, a continuing challenge? Antec International Limited. UK. Internet information, September 2nd, 2000. www.antecint.co.uk/main/coccidio.html

Long, P. L., Joyner, L. P., Millard, B. J., Norton, C. C (1976): A Guide to Laboratory Techniques used in the Study and Diasgnosis of Avian Coccidiosis. Folia Vet. Lat. 6, 201-217.

Long, P. L., Millard, B. J (1976): Studies on site finding and site specificity of *Eimeria praecox*, *Eimeria maxima* and *Eimeria acervulina* in chickens. *Parasitology* **73**, 327-336.

Long, P. L., Reid, W. M (1982): A Guide for Diagnosis of Coccidiosis in Chickens. Research Report 404. University of Georgia, USA: College of Agriculture Experimental Stations. pp 1–15.

Luna, L. G (1968): Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology. American Registry of Pathology. 3rd edition. New York: McGRAW-HILL BOOK COMPANY. pp 32-46.

MAFF (1982): Manual of Veterinary Parasitological Laboratory techniques. Technical Bulletin No. 18. 2nd edition. UK: HER Majesty's Stationary Office. pp 71-73.

Martin, S. W., Meek, A. H., Willeberg, P (1987): Veterinary Epidemiology. Principles and Methods. 1st edition. Ames, Iowa: Iowa State University Press. pp 225-230.

Maungyai, M., Sirichokchatchawan, S., Juranukul, U (1990): Efficacy of Toltrazuril and Maduramycin in the Control of Coccidiosis in Broilers. *Thailandian Journal of Veterinary Medicine* 20, 247-253.

McDougald, L. R., W. M. Reid (1997): Coccidiosis. In: B. W. Calnek., H. J. Barnes., C. W. Beard., L. R. McDougald and Y. M. Saif (Eds.): Diseases of Poultry. 10th edition. Ames. Iowa: Iowa State University Press. pp 865-883.

Mopate, L. Y., Lony, M (1998): Survey on family chicken farms in the rural areas of N'Djamena, Chad. In: Research Reports. (Eds.): International Network for Family Poultry Development (INFPD). INFPD Newsletter Vol. 8 No. 4, October-December, 1998.

Nasser, M. (1998): Oral New Castle Disease Vaccination Trials and Studies on New Castle Disease in Ethiopia. Debre Zeit/Berlin: AAU-FUB. MSc. Thesis.

NMSA (1999): Rainfall, humudity and temperature data. Addis Ababa: Ethiopia. 1999.

Putt, S. N. H., Shaw, A. P. M., Woods, A. J., Tyler, L., James, A. D (1988): Veterinary Epidemiolgy and Economics for Africa. 2nd edition. Addis Ababa: ILCA Addis Ababa. pp 30-32.

Radostits, O. M., Gay, C. C., Blood, D. C., Hinchcliff, K. W (2000): Veterinary Medicine. 9th edition. London: Harcout Publishers Ltd. pp 1296-1306.

Razmi, G. R., Kalideri, G. A (1999): Prevalence of Subclinical Coccidiosis in Broiler Chicken Farms in the Munucipality of Mashhad Khorasan, Iran. *Preventive Veterinary Medicine* 44, 247-253.

Reid, W. M. (1990): History of Avian Medicine in the United States. Control of Coccidiosis. *Avian Diseases* **34**, 509-525.

Rushton, J., Thornton, P. K., Otte, M. J (1999): Methods of Economic Impact Assessment. *Rev. Sci. Tech. Off. Int. Epiz.* **18** (2), 315-342.

Sainsbury, D (1992): Poultry Health and Management. 3rd edition. London: Blackwell Scientific Publications. pp 45-89.

Schmidt, G. D (1990): Essentials of Parasitology. 4th edition. New delhi: Universal Book Stall. p 20

Smith (1995): The Tropical Agriculturalist: Poultry. (Ed.) London: Macmillan Education Ltd. pp 62-64.

Soulsby, E. J. L (1982): Helminths, Arthropods and Protozoans of Domesticated Animals. 7th edition. London: Bailliere Tindall. pp 630-639

SVA (2000): Diagnosis and Control of Coccidial Infections. Statens Veterinär Medicinska Anstalt. Sweden. Internet information. September 2nd, 2000. www.sva.se/Efou/keyareas.htm. 22p

Thrusfield, M. (1995): Veterinary Epidemiology. 2nd edition. UK: Blackwell Science Ltd. pp183, 312-321.

Umext (2001): Control of Coccidiosis in Small Poultry Flocks: Litter management. University of Maine Coorperative Extension . Bulletin No. 2259. Maine. Internet Information, March 2nd, 2001. www.umext.maine.edu/onlinepubs/htmpubs/poultry/2259.html

Urquhart, G. M., Armour, J., Duncan, J. L., Dunn, A. M., Jennings, F. W (1987): Veterinary Parasitology. UK: Longman Group UK Ltd. pp 217-233.

Voeten, A. C., Jansen, B. A. P (1983): The monitoring and evaluation of coccidiostats in broilers. *Archive für Geflugekunde* **47**, 181-185.

Vermeulen, A. N., Schaap, D. C., Schetters, Th. P. M (2001): Control of coccidiosis in chicken by vaccination. *Veterinary Parasitology* **100**, 13-20.

Williams, R. B (1999): A compartimentalised model for the estimation of the cost of coccidiosis to the worlds chicken production industry. *International Journal of parasitology* **29**, 1209-1229.

7. APPENDICES

Appendix 7.1. Questionnaires

Please answer the following set of questionnaires by filling in the empty spaces or by marking with a cross (x) in the small box adjacent to the correct answer. You may skip questions which does not apply to your farm. Your participation will be highly appreciated and all information will be treated as confidential.

A. General information

Locality _____

Farm No _____House No _____

Owner's name and address _____

Production system _____

Breed _____. Production line: Broilers □; Layers □; Other (specify)_____

Farm size: No. of birds/farm _____, No. of bird/house _____

B. Information about farm history and causes of morbidity and mortality

1. Age of the farm:0-6 months □; 7-12 months □; 1-2years□; More tah 2 years □.

2. Purppose of keeping chicken: Family consumption □; Earning cash □; Other (specify)_____

3. Disease problem in the farm: Yes / No. If yes:

4. i) Major diseases in your farm: (In order of importance)

   a)_____b)_____ c)_____d)_____

   ii) Number of birds which get sick due to each of the above diseases:

   a) _____ b) _____ c) _____ d) _____ e) _____

5. Deaths of birds in your farm: Yes/No

6. i) Major causes of deaths (In order of importance):

   a) _____ b) _____ c) _____ d) _____

   ii) Number of birds which die due to each of the above causes:

   a) _____ b)_____ c) _____ d) _____

C. Information about disease control measures

7. Diseases control measures in your farm: Drugs □; Cleaning/Disinfection □: Vaccination □: Other (specify) _____

8. How long have you been using the above measures? 1 year □; 2 years □; More than 2 years □

10. Frequency of manure disposal : Once/week □; Once/month □; Once/batch □; Other (specify) _____

11. Drugs used in the farm starting with the most frequently used:

    a) _____ b) _____ c) _____ d) _____

11. Level of biosecurity measures in the farm: Good □; Poor □.

D. Information about production

12. . Source of day old chicks: Own □; Local supplier □; Imported □.

13. Age and weight at which birds are sold: Age _____ weight _____

14. Culling (removing) birds from the farm before sale age: Yes/No. If Yes:
Number culled _____ Reason for culling_____

15. Age at which birds start laying:_____

E. Information about feed and feeding

16. Source of feed:

□ Manufactured in the farm.

□ Bought from local suppliers.

□ Other (specify) _____

17. Feed management:

□ Ad-libitum

□ Controlled

□ Other(specify) _____

18. Quality and availability of feed:

□ Satisfactory

□ Poor

19. Source of feeding and watering equipment:

□ Made in the farm

□ Locally made

□ Imported

20. Quality of feeding and watering equipment in relation to feed and water spillage:

Good □; Satisfactoy □; Poor □.

21. . Type of feeding system: Automated □; Manual □.

22. Use coccidiostats:. Yes □; No □; Sometimes □.

23. Method of application: In feed □; In water □; In feed and water □.

F. Information about environmental/management factors:

24. Litter/house moisture: Average. □ ; High □

25 Stocking density: Average □; High □

26 Ventilation system: Good □; Satisfactory □; Poor □.

G. Information about coccidiosis

27. Coccidiosis in the farm: Yes/No. If Yes:

When did the problem start? _____

Effect on farm preformance _____

Number of birds showing signs _____

Number of birds which die per batch _____

Measures used to control the disease: a) _____ b) _____

c) _____ d) _____ e) _____

28. Have your control measures been continuous? Yes□ , No □

29. Use of production standards in the farm: Yes □, No □

Appendix 7.2. Summery of responses from questionnaire survey on farm disease history, management practices and potential risk factors

Question/Item	Level/Category of question/item	No. of farms which responded		
		LS-BR	SS-BR/PL	LS-LY
Farm age	0-6 months	1	3	0
	7-12 months	0	3	1
	1-2 years	0	4	0
	> 2 years	4	4	5
Purpose of keeping chicken	Income	5	14	6
	Subsistence	0	0	0
	Other	0	0	2
Disease in the farm	Yes	4	8	5
	No	1	6	1
Coccidiosis in the farm	Yes	4	4	2
	No	1	10	4
Important causes of morbidity	Coccidiosis	3	4	0
	Bacterial disease	0	2	1
	Viral disease	0	0	1
	Other/unknown	2	8	4
Important cause of mortalities	Coccidiosis	1	2	0
	Bacterial disease	0	1	2
	Viral disease	1	0	4
	Other/unknown	3	11	0
Important causes of culling	Disease	1	0	4
	Poor weight gain	2	1	0
	General weakness	1	0	0
	No culling	1	13	2
Disease control meassures used	Vaccination	0	0	0
	Disinf./hygiene	0	14	0
	Drugs	0	9	0
	All above	5	3	6
Biosecurity practices	Good	4	2	6
	Poor	1	12	0
Coccidiostats in feed	Yes	5	13	5
	No	0	1	1
Feed availability and quality	Satisfactory	5	14	6
	Poor	0	0	0
Source of day old	Own	1	0	2

chicks				
	Local supplier	3	14	4
	Imported	1	0	0
Source of feed and equipment	Locally made	3	14	2
	Imported	2	0	5
	Other	0	0	0
Method of feeding	Ad-libitum	5	14	0
	Controlled	0	0	6
Stocking density	Average	5	4	6
	High	0	10	0
Equipment quality	Good	5	0	5
	Satisfactory	0	3	1
	Poor	0	11	0
Feeding/watering system	Automated	2	0	1
	Manual	3	14	5
Ventilation system	Good	4	0	6
	Satisfactory	0	3	0
	Poor	1	11	0
Litter/house moisture	Average	4	2	6
	High	1	12	0
Manure disposal	Once/week	0	1	0
	Once/month	0	0	0
	Once/batch	5	13	2
	Other	0	0	4
Production standards	Yes	5	0	6
	No	0	14	0

Key:

LS-BR.....Large Scale Broiler Farms

SS-BR/PL....Small Scale Broiler or Pullet Farms

LS-LY.....Large Scale Layer Farms

Appendix 7.3. Gross margin analysis for quantification of economic losses due to coccidiosis

Item/Farm No.	Farm 1.	Farm 2	Farm 3	Farm 5	Farm 7	Farm 8	Farm 13	Farm.14.
Chicken sales	123628	278982	131904	86850.4	17524	15495.9	32490	48960
Home consumed	4500	576	0	0	0	329.7	0	288
Gifts	0	0	0	0	0	282.6	162	0
Output (1)	**128128**	**279558**	**131904**	**86850.4**	**17524**	**16108.2**	**32652**	**49248**
Chick Purchases	32000	41072	32000	20000	6000	2904	8000	12000
Feed Cost	55530	74460	52300	34500	6370	8540	18000	13800
Coccidiostat Cost	3450	3189	968	1485	290	258	453	830
Vaccine Cost	500	3792.6	608	500	60	75	0	450
Disinfectant Cost	414	1618	580	446	0	46	150	650
Drug Cost	0	1826	700	1149.5	0	0	250	300
Teff straw Cost	0	0	0	360	40	68	200	0
Veterinary Cost	450	0	480	400	0	0	0	0
Casual Labour	0	600	540	0	0	125	50	60
Marketing Cost	0	0	0	0	0	137	360	0
Tvc (2)	**92344**	**126557.6**	**88176**	**58840.5**	**12760**	**12153**	**27463**	**28090**
GM. (1-2)	**35784**	**153000.4**	**43728**	**28009.9**	**4764**	**3955.2**	**5189**	**21158**
Regular Labour	1200	1360	916	600	100	300	300	800
Power/water	2000	1800	450	400	240	138	80	530
Rent	0	0	0	0	160	0	600	1500
Tfc (3)	**3200**	**3160**	**1366**	**1000**	**500**	**438**	**980**	**2830**
Profit (GM-3)	**32584**	**149840.4**	**42362**	**27009.9**	**4264**	**3517.2**	**4209**	**18328**
Flocksize (Birds)	8000	18800	8000	5000	1500	726	2000	3000
No.offtaken (Birds)	7040	15520	7328	4772	1348	658	1814	2746
Total loss (mort)	17472	59040	12095	4150	1976	1554.3	3348	4752
Loss mort (coc)	1747.2	8154	1512	702	299	211.95	432	774
Loss culls (coc)	0	0	0	0	0	47.1	0	0
Feed price/100Kg	135	114	135	135	145	163	160	135
Chicken price/Kg	13	13	15	13	13	14	13	15
PMR (coc)	0.1	0.13811	0.12501	0.16916	0.15132	0.13636	0.12903	0.16288
Total loss (coc)	5197.2	11343	2480	2187	589	517.05	885	1604
Loss of profit (Coc)	0.159502	0.075701	0.05854	0.08097	0.13813	0.14701	0.21026	0.08752
Total loss/chicken	0.691117	0.661014	0.32359	0.44761	0.41362	0.74718	0.46408	0.5583

Key: **Tvc** .... Total variable costs. **Tfc** .... Total fixed costs. **GM** ... Gross Margin
PMR .... Proportional Mortality Rates. **Coc** .... Coccidiosis

Farm No. 1, 2, 3 and 5 were large scale farms.
Farm No. 7, 8, 13 and 14 were small scale farms.

Appendix 7.4. Summary of poultry farms and samples examined in each farm.

Farm Number	Production System	Faecal samples examined			Birds examined			
		Total	Positive samples	Mean OPG	Total	Positive samples	Clinical coccidiosis	Subclinical coccidiosis
1	LS-BR	68	59/68 (86.8%)	9953	32	19/32 (59.4%)	4/19 (21%)	15/19 (79%)
2	LS-BR	75	44/75 (58.7%)	5463	62	28/62 (45.2%)	7/28 (25%)	21/28 (75%)
3	LS-BR	60	60/60 (100%)	56897	30	14/30 (46.7%)	3/14 (21%)	11/14 (79%)
4	LS-BR	35	26/35 (74.3%)	562	32	15/32 (46.9%)	0/15 (0%)	15/15 (100%)
5	LS-BR	36	31/36 (86%)	3572	35	16/35 (45.7%)	2/16 (12.5%	14/16 (87.5%)
6	SS-PL	30	26/30 (86.6%)	6900	15	5/15 (33.3%)	2/5 (40%)	3/5 (60%)
7	SS-PL	30	23/30 (76.6%)	3122	15	7/15 (46.7%)	2/7 (28.6%)	5/7 (71.4%)
8	SS-BR	26	24/26 (92.3%)	10089	15	7/15 (46.7%)	2/7 (28.6%)	5/7 (28.6%)
9	SS-BR	26	15/26 (7.7%)	1267	15	6/15 (40%)	2/6 (33.3%)	4/6 (66.7%)
10	SS-BR	25	22/25 (88%)	57732	16	10/16 (62.5%)	3/10 (30%)	7/10 (70%)
11	SS-BR	25	12/25 (48%)	320	16	5/16 (31.25%)	1/5 (20%)	4/5 (80%)
12	SS-BR	26	14/26 (53.8%)	646	16	5/16 (31.25%)	1/5 (31.25%)	4/5 (31.25%)
13	SS-BR	30	19/30 (63%)	3034	20	9/20 (45%)	2/9 (22%)	7/9 (78%)
14	SS-BR	30	24/30 (80%)	5614	20	14/20 (70%)	3/14 (21.4%)	11/14 (79.6%)
15	SS-BR	25	19/25 (76%)	590	16	8/16 (50%)	1/8 (12.5%)	7/8 (87.5%)
16	SS-BR	25	19/25 (76%)	103908	16	7/16 (43.7%)	1/7 (14.3%)	6/7 (85.7%)
17	SS-BR	26	23/26 (88.5%)	31889	16	6/16 (37.5%)	1/6 (16.7)	5/6 (83.3%)
18	SS-BR	25	21/25 (84%)	15533	16	10/16 (62.5%)	2/10 (20%)	8/10 (80%)
19	SS-BR	28	19/28 (67.9%)	2200	17	7/17 (41%)	3/7 (42.8%)	4/7 (57.3%)
20	LY-LT	58	4/58 (7%)	200				
21	LY-CG	56	0/56 (0%)	0				

22	LY-CG	55	5/55 (9%)	450				
23	LY-LT	60	4/60 (6.7%)	186				
24	LY-CG	58	3/58 (5.2%)	300				
25	LY-CG	60	3/60 (5%)	433				
26	SS-PL	30	24/30 (80%)	471				

Key:

LS-BR .....Large scale broilers

SS-BR .....Small scale broilers

SS-PL .....Small scale pullets

LY-CG .....Large scale layers with cage rearing system

LYLT .....Large scale layers with deep litter rearing system

Appendix 7.5. Curriculum vitae

1. Personal particulars

Name: Safari Methusela Kinung'hi

Sex: Male

Date of birth: 19. 10. 1969

Place of birth: Mwanza, Tanzania

Nationality: Tanzanian

Profession: Veterinarian

Marital status: Single

Language proficiency: English-written and spoken; Swahili-written and spoken

Parmanent address: C/o James Kilaba, P. O. 7854, Dar-Es-Salaam, Tanzania.

2. Education background

1991-1996: University education: Sokoine University of Agriculture, Morogoro, Tanzania.
Award: Bachelor of Veterinary Medicine (BVM) degree.

1988-1990: Advanced level of secondary education, Shinyanga High School, Shinyanga, Tanzania.
Award: Advanced Certificate of Secondary Education

1984-1997: Ordinary level of secondary education, Mara Secondary School. Mara, Tanzania.
Award: Certificate of Secondary Education

1977-1983: Primary education: Mwabujose Primary School. Magu. Mwanza.

Award: Certificate of Primary Education

3. Working experience

1992/1994: Kinango Secondary School. Mwanza, Tanzania.

Responsibilities : Teaching secondary school students. Subjects: Chemistry, Biology and Agricultural Sciences.

1996-1998: Animal Health Care & Surgery (Veterinary Clinic)

Position: Clinician Incharge.

Responsibilities: Diagnosis, treatment and control of animal diseases.

Supervision of the clinic's day to day activities.

1998-1999: Tan Veterina Ltd (Pharmaceutical Company and Veterinary Consultancy Centre)

Position: Technical Manager

Responsibilities: Giving technical advise to individual customers and businessmen (Agro-Veterinary Enterprises).

Supervision of day to day activities of the centre.

4. Publications

Study on Natural Coccidia Infection in Domestic Rabbits in Morogoro Municipality, Tanzania. Tanzania Veterinary Journal, (1997).

5. Membership

Tanzania Veterinary Association (TVA).

Tanzania Society for Wildlife Conservation (TSWC).

Appendix 7.6. Signed declaration sheet

I, the undersigned, declare that the thesis is my original work and has not been presented for a degree in any university

Name: SAFARI METHUSELA KINUNG'HI

SIGNATURE .....

Date of submission: 23. 11. 2001.

This thesis has been submitted for examination with our approval as university advisors

1. Prof. Dr. H. M. Hafez .....

2. Dr. Getachew Tilahun .....

3. Dr. Moges Woldemeskel .....

Studies on prevalence and economic
Empact of Poultry coccidiosis in
Different production systems in Debre Zei
& Addis Ababa, Ethiopia.

Safari Methusela