# ON THE CONSTRUCTION OF MIXED POISSON DISTRIBUTIONS

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## Declaration

This is my original work and has never been presented for any academic award in any other learning institution.

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## Dedication

To my loving husband Rodgers Mutai and my beloved daughter Kimberly Jeptepkeny, thanks for your daily support and encouragements.

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## Abstract

Mixed Poisson distributions are very significant in modeling non - homogeneous populations, for instance in Actuarial applications for modeling total claims in insurance. In this work the concentration is mainly on the construction of these Mixed Poisson distributions. The methods of construction used are: Direct integration, obtaining Recursive relations for the Mixed Poisson distributions, using Laplace Transforms of the mixing distributions and use of Special Functions to express the Mixed Poisson distributions. A number of Mixed Poisson distributions are constructed using each of the mentioned methods of construction.

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# Chapter 1

## GENERAL INTRODUCTION

### 1.1 Statistical Distributions

One Major area of statistics is statistical distributions. Let $f(x)$ be a function of a random variable, $X$.

If,

$$f(x) > 0 \text{ and } \int_{-\infty}^{\infty} f(x)\,dx = 1 \tag{1.1}$$

then $f(x)$ is called a probability density function (pdf) of a continuous random variable $X$.

If,

$$0 \leq f(x) \leq 1 \text{ and } \sum_{-\infty}^{\infty} f(x) = 1 \tag{1.2}$$

then $f(x)$ is called a probability mass function (pmf) of a discrete random variable $X$.

Probability distributions have been classified according to methods used for the construction. Thus we have;

1. Power series based expansion
2. Transformation based, Jacobian and Cumulative Functions
3. Mixtures
4. Methods based on Recursive relations in probabilities
5. Lagrangian expansion

6. Distributions based on hazard functions of survival analysis
7. Distributions emerging from stochastic processes
8. Sums of independent random variables

## 1.2 Poisson Distribution

We can derive Poisson Distribution from the exponential power series, Binomial-Poisson mixture, a sum of iid random variables, pure birth process and recursive model.

A brief discussion follows:

### 1.2.1 Power series based expansion

$$e^{\lambda}=\sum_{x=0}^{\infty}\frac{\lambda^{x}}{x!}$$

Therefore,

$$1=\sum_{x=0}^{\infty}\frac{e^{-\lambda}\lambda^{x}}{x!}$$

hence,

$$f(x)=\frac{e^{-\lambda}\lambda^{x}}{x!};x=0,1,2,...$$

which is a Poisson distribution with parameter $\lambda$.

### 1.2.2 Binomial-Poisson Mixture

$$\begin{aligned} f(x) &= \sum_{n=0}^{\infty} f(x \mid n)\, g(n) \\ &= \sum_{n=0}^{\infty} \binom{n}{x} p^x q^{n-x} \frac{e^{-\lambda}\lambda^n}{n!} \\ &= e^{-\lambda} \sum_{n=0}^{\infty} \frac{n!}{x!\,(n-x)!} \frac{p^x q^{n-x} \lambda^x \lambda^{n-x}}{n!} \\ &= \frac{e^{-\lambda}}{x!} \sum_{n=0}^{(\infty)} \frac{(\lambda p)^x (\lambda q)^{n-x}}{(n-x)!} \\ &= \frac{e^{-\lambda}(\lambda p)^x}{x!} \sum_{n=0}^{\infty} \frac{(\lambda q)^{n-x}}{(n-x)!} \end{aligned}$$

Therefore,

$$\begin{aligned} f(x) &= \frac{e^{-\lambda}(\lambda p)^x}{x!} e^{\lambda q} \\ &= \frac{e^{-\lambda}(\lambda p)^x e^{\lambda(1-p)}}{x!} \\ &= \frac{e^{-\lambda}(\lambda p)^x e^{\lambda} e^{-\lambda p}}{x!} \end{aligned}$$

Hence,

$$f(x) = \frac{e^{-\lambda p}(\lambda p)^x}{x!}; x = 0,\ 1, 2, \ldots$$

which is a Poisson probability mass function with parameter $\lambda p$.

### 1.2.3 Sum of iid random variables

Case(i)

Let

$$S_N = X_1 + X_2 + \ldots + X_N$$

where, $X_i$'s are iid random variables and $N$ is also a random variable independent of $X_i$'s.

Further, let

$$G(s) = E(s^{x_i}) = \text{the pgf of } X_i$$

$$F(s) = E\left(s^{N}\right) = \text{the pgf of } N$$

$$H(s) = E(s^{s_N}) = \text{the pgf of } s_N$$

Then,

$$H(s) = F[G(s)]$$

If $X_i$ is Bernoulli with parameter $p$, and $N$ is Poisson with parameter $\lambda$, then

$$G(s) = q + ps, \text{ where } q = 1 - p$$

and

$$F(s) = e^{-\lambda(1-s)}$$

$$\begin{aligned} H(s) &= e^{-\lambda[1-G(s)]} \\ &= e^{-\lambda[1-(q+ps)]} \\ &= e^{-\lambda p(1-s)} \end{aligned}$$

which is the probability generating function of a Poisson distribution with parameter $\lambda p$; that is,

$$g(y) = \Pr[S_N = y] = \frac{e^{-\lambda p}(\lambda p)^y}{y!}; y = 0, 1, 2, \ldots$$

Case(ii)
Let

$$S_N = X_1 + X_2 + \ldots + X_N$$

where $X_i$'s are iid variables with fixed $N$.

If $X_i$ is Poisson $(\lambda)$, then

$$\begin{aligned} G_{s_N} &= H(s) = E(s^{s_N}) = E\left[s^{X_1+X_2+\ldots+X_N}\right] \\ &= \left[E\left(s^X\right)\right]^N = [G(s)]^N \\ &= \left[e^{-\lambda(1-s)}\right]^N = e^{-\lambda N(1-s)} \end{aligned}$$

Thus $S_N$ is Poisson $(\lambda N)$, that is,

$$g(y) = \Pr[s_N = y] = \frac{e^{-\lambda N}(\lambda N)^y}{y!}; y = 0, 1, 2, \ldots$$

### 1.2.4 Pure Birth Process

Let

$$X(t) = \text{the population size at time } t$$

and

$$p_n(t) = \Pr[X(t) = n]$$

Therefore,

$$\begin{aligned} p_n(t+\Delta t) &= \Pr[X(t+\Delta t) = n] \\ &= \Pr[X(t+\Delta t) = n, X(t) = n] + \Pr[X(t+\Delta t) = n, X(t) = n-1] \\ &= \Pr\{X(t+\Delta t) = n \mid X(t) = n\} \Pr[X(t) = n] \\ &\quad + \Pr\{X(t+\Delta t) = n \mid X(t) = n-1\} \Pr[X(t) = n-1] \\ &= [1 - \lambda_n \Delta t + o(\Delta t)] p_n(t) + [\lambda_{n-1}\Delta t + o(\Delta t)] p_{n-1}(t) \end{aligned}$$

where,

(i) $[\lambda_n \Delta t + o(\Delta t)]$ is the probability of a birth within an interval $\Delta t$ when the birth rate for population of size $n$ is $\lambda_n$.

(ii) $o(\Delta t)$ is order of $\Delta t$ which tends to zero as $\Delta t \to 0$

(iii) The probability of two or more births in the interval $\Delta t$ is $o(\Delta t)$.

(iv) The probability of no birth between time $t$ and $t+\Delta t$ when $X(t) = n-1$ is $1 - [\lambda_{n-1}\Delta t + o(\Delta t)]$

Therefore,

$$p_n'(t) = \lim_{\Delta t \to 0} \frac{p_n(t+\Delta t) - p_n(t)}{\Delta t} = -\lambda_n p_n(t) + \lambda_{n-1} p_{n-1}(t); n \geq 0$$

and

$$p_0'(t) = -\lambda_0 p_0(t)$$

When $\lambda_n = \lambda$ for all n we have a Poisson Process. Thus the basic difference differential equations are:

$$p_0'(t) = -\lambda p_0(t) \tag{i}$$

and

$$p_n'(t) = -\lambda p_n(t) + \lambda p_{n-1}(t); n \geq 1 \tag{ii}$$

Using the pgf technique to (ii), we have

$$\sum_{n=1}^{\infty} p_n'(t) s^n = -\lambda \sum_{n=1}^{\infty} p_n(t) s^n + \lambda \sum_{n=1}^{\infty} p_{n-1}(t) s^n$$

$$\Longrightarrow \frac{\partial G}{\partial t} - p_0'(t) = -\lambda [G(s,t) - p_0] + \lambda s G(s,t)$$

Applying (i) we get

$$\frac{\partial G(s,t)}{\partial t} = -\lambda (1-s) G(s,t)$$

where

$$G(s,t) = \sum_{n=0}^{\infty} p_n(t) s^n \text{ and } \frac{\partial G}{\partial t} = \sum_{n=0}^{\infty} p_n'(t) s^n$$

Therefore,

$$\int \frac{\partial G}{G} = -\lambda (1-s) \int dt$$

$$\Longrightarrow \ln G(s,t) = -\lambda (1-s) t + c$$

Therefore,

$$G(s,t) = e^{c}e^{-\lambda(1-s)t} = ke^{-\lambda t(1-s)}$$

$$X(0) = 0 \Longrightarrow p_0(0) = 1 \text{ and } p_n(0) = 0 \text{ for } n \neq 0$$

Therefore,

$$G(s,0) = p_0(0) + \sum_{n=1}^{\infty} p_n(0)\, s^n = 1 + 0 = 1$$

$$G(s,t) = e^{-\lambda t(1-s)}$$

which is the probability generating function of Poisson distribution with parameter $\lambda t$. That is,

$$p_n(t) = \frac{e^{-\lambda t}(\lambda t)^n}{n!}; n = 0, 1, 2, ...$$

### 1.2.5 Recursive Model

Let

$$f(x+1) = \left(\frac{\lambda + \beta x}{1+x}\right) f(x); x = 0, 1, 2, ...$$

When $\beta = 0$, we have

$$f(x+1) = \left(\frac{\lambda}{1+x}\right) f(x)$$

$$\Rightarrow \sum_{x=0}^{\infty} (1+x) f(x+1) s^x = \lambda \sum_{x=0}^{\infty} f(x) s^x$$

$$\Rightarrow \frac{d}{ds} G(s) = \lambda G(s)$$

$$\Rightarrow \int \frac{dG(s)}{G(s)} = \int \lambda ds \Rightarrow \ln G(s) = \lambda t + c$$

Therefore,

$$G(s) = e^{\lambda t + c} = ke^{\lambda s}$$

$$\Rightarrow G(1) = ke^{\lambda} \Rightarrow 1 = ke^{\lambda} \Rightarrow k = e^{-\lambda}$$

therefore,

$$G(s) = e^{-\lambda(1-s)}$$

which is the probability generating function of Poisson $(\lambda)$.

### 1.2.6 Summary

The following types of Poisson Distributions have emerged:

$$f(x) = \frac{e^{-\lambda}\lambda^{x}}{x!}; x = 0, 1, 2, ..., \lambda > 0$$

$$f(x) = \frac{e^{-\lambda p}(\lambda p)^{x}}{x!}; x = 0, 1, 2, ..., 0 < p < 1$$

$$f(x) = \frac{e^{-\lambda n}(\lambda n)^{x}}{x!}; x = 0, 1, 2, ...;$$

for fixed $n$, a positive integer.

$$f(x) = \frac{e^{-\lambda t}(\lambda t)^{x}}{x!}; x = 0, 1, 2, ..., t > 0 \text{ and } \lambda > 0$$

In this study, the mixed model will be based on;

$$f(x) = \frac{e^{-\lambda}\lambda^{x}}{x!}; x = 0, 1, 2, ...$$

Occasionally, we may look at

$$f(x) = \frac{e^{-\lambda p}(\lambda p)^{x}}{x!}; x = 0, 1, 2, ...$$

## 1.3 Distribution Mixtures

Let $f(x;\theta)$ be a probability distribution function (pdf) or a probability mass function (pmf) of a random variable $X$ with parameter $\theta$. If this parameter $\theta$ is varying, then it also becomes a random variable. Thus we have a conditional pdf or pmf $f(x \mid \theta)$; and the unconditional or marginal distribution becomes

$$f(x) = \int_{-\infty}^{\infty} f(x;\theta)\, d\theta = \int_{-\infty}^{\infty} f(x \mid \theta)\, g(\theta)\, d\theta$$

or

$$f(x) = \sum_{\theta} f(x \mid \theta)\, g(\theta)$$

where $g(\theta)$ is a pdf or pmf of $\Theta = \theta$ and is called a mixing or prior distribution.

For Mixed Poisson distribution, let $\theta = \lambda$. Thus

$$f(x \mid \lambda) = \frac{e^{-\lambda}\lambda^{x}}{x!}; x = 0, 1, 2, ..., \lambda > 0$$

and

$$f(x) = \int_{0}^{\infty} f(x \mid \lambda)\, g(\lambda)\, d\lambda$$

$$f(x) = \int_{0}^{\infty} \frac{e^{-\lambda}\lambda^{x}}{x!} g(\lambda)\, d\lambda \tag{1.5}$$

when $g(\lambda)$ is a continuous mixing distribution, otherwise

$$f(x) = \sum_{\lambda} \frac{e^{-\lambda}\lambda^{x}}{x!} g(\lambda) \tag{1.6}$$

for a discrete mixing distribution $g(\lambda)$. The term "Finite Mixture" is used when the mixing distribution is discrete.

A random variable $X$ with fixed parameter $\lambda$, has

$$E(X) = Var(X) = \lambda \tag{1.7}$$

If $\Lambda = \lambda$ is now varying then

$$E(X) = EE(X \mid \lambda) \tag{1.8}$$

and

$$\begin{aligned} Var(X) &= E[Var(X \mid \lambda)] + VarE(X \mid \lambda) \\ &= E[E(X \mid \lambda)] + VarE(X \mid \lambda) \end{aligned}$$

$$Var(X) = E(X) + VarE(X \mid \lambda) \tag{1.9}$$

## 1.4 Problem Statement

Consider the mixed Poisson distribution given by,

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda$$

where $g(\lambda)$ is a mixing distribution.

To obtain the mixed distribution, the evaluation of the above integrand explicitly is difficult with the exception of a few mixing distributions, (Albercht, 1984). The problem then is to find alternative ways of obtaining the mixed Poisson distribution for various probability density functions of $\Lambda = \lambda$, and also to identify the ones where explicit evaluation is possible.

The major problem in constructing or obtaining mixture distributions with continuous mixing distributions is the evaluation of the integrand as Albercht (1984) has stated. Only a few integrands can be evaluated explicitly, therefore, alternative methods had to be sought.

## 1.5 Objectives

The main objective of this work is to review some of these methods in determining Mixed Poisson distributions.

In this work, the specific objective will be to obtain the Mixed Poisson distributions through

(i) Direct integration where possible,

(ii) Recursive formulae,

(iii) Laplace Transform technique and

(iv) Use of special functions.

## 1.6 Significance of the study - Applications

Mixture models cover several distinct fields of statistical science. In recent years, the number of applications increased mainly because of the availability of high speed computer resources, which removed any obstacles to apply such methods.

"Thus, mixture models have found applications in fields as diverse as data modeling, discriminant analysis, cluster analysis, outlier-robustness studies, ANOVA models, kernel density estimation, latent structure models, empirical Bayes estimation, Bayesian statistics, random variable generation, approximation of the distribution of some statistic and others" (Karlis and Xekalaki, 2005).

The distribution of total claims payable by an insurer is considered when the frequency of claims is a Mixed Poisson random variable, (Willmot, 1986). Mixed Poisson distributions often have desirable properties for modelling claim frequencies. For instance, they often have thick tails which make them useful for long tailed data.

"Mixtures of distributions have been widely used for modeling observed situations whose various characteristics as reflected by the data differ from those that would be anticipated under the simple component distribution. In actuarial applications, for example, observed data on the number of claims often exhibit a variance that noticeably exceeds their mean. Hence, assuming a Poisson form (or any other form that would imply equality of the mean to the variance) for the claim frequency distribution is not appropriate in such cases, (Karlis and Xekalaki, 2005). To have overdispersion, then there is need to have models whose variance is greater than the mean. This is where mixture models such as Mixed Poisson distributions come in handy.

Mixed Poisson distributions have been used in a wide range of scientific fields for modeling non-homogeneous populations. (Antzoulakos, D.L. and S.Chadjiconstantinidis, 2004) quote the following example: "The use of the Poisson distribution as a model describing the number of claims caused by individual policy holders (e.g. in automobile insurance) during a certain period is usually rejected, since in practice the behavior of policy holders is heterogeneous. This means that the Poisson parameter, say $\lambda > 0$, varies

between the policy holders reflecting the different underlying risks and hence its value cannot be a constant for each policy holder. Therefore, it is natural to assume a model reflecting the uncertainty for claim frequencies. It may be reasonable to consider that the counting distribution of the number of claims or losses caused by each individual follows a Poisson distribution whose parameter $\lambda$ varies between the individuals, i.e. we consider that to each individual policy holder, there corresponds a personal Poisson distribution. Therefore, it seems natural to regard a personal $\lambda$ (characterizing each individual) as the outcome of a random variable $\Lambda$ (reflecting the risk level among the group of risks) with known pdf $g(\lambda)$, and thus given the risk level the number of claims follows a Poisson distribution with parameter the given value of the risk level. Hence, the number of claims or losses, $N$ (for a given reference period, say a year), caused by an individual chosen randomly from the portfolio, follows a mixed Poisson distribution." The paper also states that the class of Mixed Poisson distributions is one of the most important classes of counting distributions for modelling insurance claims.

A good example on how applicable Mixed Poisson distributions are in actuarial data is given by Klugman, et al (1998). The driving habits of some automobile drivers were studied in a class of automobile insurance by counting the number of accidents per driver in a one-year time period. Poisson and Negative Binomial distributions were then fitted to the data and the two models compared using likelihood ratio test. The model that was selected as the best fitting was that of the Negative Binomial distribution which is a Mixed Poisson distribution with Gamma as the mixing distribution.

## 1.7 Literature Review

Various Mixed Poisson distributions can be constructed depending on the choice of the mixing distribution using several ways such as the explicit evaluation, use of recursive relations, use of the Laplace transforms of the mixing distributions and representing the mixed Poisson distributions in terms of special functions.

Excellent work on the review of this subject has been done by Karlis and Xekalaki (2005).

### 1.7.1 Explicit Forms

The simplest choice of the distribution of $\Lambda$ is the Gamma density which results in the Negative Binomial Distribution, NBD (Greenwood & Yule, 1920). Johnson, et al, 1992, considered the Exponential distribution as the mixing density and this resulted in a Geometric distribution. Taking $\Lambda$ to have a Shifted Gamma (three parameter) distribution, the resulting mixed Poisson distribution is a convolution of a Negative Binomial distribution and a Poisson distribution, (Ruohonen, 1988). The Poisson distribution is mixed with Lindley distribution resulting in the Poisson - Lindley distribution, (Sankaran, 1970a). Further, Zakerzadeh and Dolati, (2010) generalized the Lindley distribution to obtain a Generalized Lindley distribution. Taking this distribution as the mixing density, Mahmoudi and Zakerzadeh, (2010) obtained a Generalized Poisson - Lindley distribution. Taking a mixture of the Poisson distribution with a normal distribution truncated at the left at zero, then we have a Poisson-Normal distribution, (Patil, 1964). The Poisson-Linear Exponential distribution is obtained by formally mixing the Poisson distribution with the linear exponential family of distributions, (Sankaran, 1970b).

### 1.7.2 Mixed Poisson Distributions in Recursive Forms

Willmot (1993) devised a method now known as Willmot Approach to determining Mixed Poisson distributions in recursive forms. He obtained recursive formulae for the following mixing distributions:

1. Gamma distribution to obtain NBD
2. Generalized Inverse Gaussian distribution to obtain the Sichel Distribution; Poisson - Inverse Gaussian distribution is a special case.
3. Beta Distribution to obtain Poisson - Beta
4. Generalized Pareto to obtain Poisson - Generalized Pareto. Poisson - Pareto is a special case.
5. Transformed or Generalized Gamma

6. Transformed Beta

7. Inverse Gamma

8. Mixing distributions based on hazard functions

9. Shifted and Truncated Mixing distributions; Shifted Gamma to obtain Delaporte's distribution, Shifted Pareto, Truncated Gamma, Truncated Normal.

Gupta and Ong (2005) obtained recursive forms of Poisson mixtures for the following Generalized Mixing Distributions:

- Generalized Gamma distribution. This is a different generalized Gamma from the one discussed by Willmot (1993). It is a four parameter Gamma considered by Armero and Bayan (1993) in the study of some queueing problems.

- Generalized Exponential distribution. Gupta and Ong (2005) did not use Willmot's Approach. Most likely they integrated by parts.

Sankaran (1968) obtained a recursive formula for Poisson - Inverse Gaussian using differential equation in pgf.

### 1.7.3 Use of Generating Functions and Laplace Transforms

Probability Generating function technique and Laplace Transforms have been handy in determining some mixed Poisson distributions.

Ruohonen (1988) obtained the Delaporte distribution in terms of a product of the pgf of NBD and Poisson distribution.

Gupta and Ong (2005) obtains pgfs for Poisson - Generalized Gamma, Poisson - Generalized Exponential distribution.

Power Variance Function (PVF) distribution is a three parameter family uniting Gamma and positive stable distributions. The distribution is denoted by PVF$(\alpha, \delta, \theta)$.

The Laplace transform is given by

$$L(s) = \exp\left\{-\frac{\delta}{\alpha}\left[(\theta+s)^{\alpha} - \theta^{\alpha}\right]\right\}$$

according to Hougaard et al (1997).

(i) For $\alpha = 0$, the Gamma distributions are obtained

(ii) For $\theta = 0$, the positive stable distributions are obtained

(iii) For $\alpha = \frac{1}{2}$, the Inverse Gaussian distributions are obtained

(iv) For $\alpha = -1$, the non-central Gamma distribution of shape parameter zero is obtained.

The mixed Poisson (Poisson - Power Variance) pmf can be obtained using the formula

$$f(x) = (-1)^x \frac{L^{(x)}(1)}{x!}$$

where $L^{(x)}(s)$ denotes the xth derivative of $L(s)$.

Willmot (1986) obtained the Poisson - Generalized Inverse Gaussian (Sichel) distribution by considering the Laplace Transform of GIG. He then converted the Laplace into pgf by the relation;

$$G_x(s) = L_\lambda(1 - s)$$

Hence the pmf as a coefficient of $s^k$. He also used the pgf to determine the recursive relation. Thus Hougaard et al (1997) used the relationship between $f(x)$ and $L_\lambda(s)$ to obtain $f(x)$.

Willmot (1986) used the relationship between $G_x(s)$ and $L_\lambda(s)$ to obtain $f(x)$.

Karlis and Xekalaki (2005) in their proposition 14 have given an alternative useful method which links the probability function of a mixed Poisson distribution to the moments of the mixing distribution.

$$f(x) = \frac{1}{x!} \sum_{r=0}^{\infty} \frac{(-1)^r}{r!} \mu_{x+r}(\lambda)$$

### 1.7.4 Mixtures in terms of Special Functions

Some integrands that cannot be evaluated explicitly can be expressed in terms of special functions.

Willmot (1993) did express the pgf of Poisson - Scaled Beta distribution in terms of a Confluent Hypergeometric distribution. This same result was

obtained by Gurland (1958) by mixing a Poisson with a parameter $\lambda p$ with the classical Beta distribution.

### 1.7.5 Other Cases

Brown and Holgate (1970) found that the Poisson - Lognormal distribution cannot be evaluated explicitly. Blumer (1974) also examined the Poisson - Lognormal as a model for species abundance. In the paper, it is confirmed that there appears to be no simple form. Thus Blumer evaluated the model by numerical integration.

# Chapter 2

## EXPLICIT MIXED POISSON DISTRIBUTIONS

### 2.1 Introduction

A random variable X follows a Mixed Poisson distribution with mixing distribution having probability density function $g$ if its probability function is given by;

$$f(x) = \int_{\lambda} \frac{e^{-\lambda}\lambda^{x}}{x!} g(\lambda)\, d\lambda \tag{2.1}$$

There are a few Mixed Poisson distributions which can be evaluated explicitly. This chapter looks at some of them, namely;

(i) Poisson - Exponential

(ii) Poisson - Gamma(with one parameter, two parameters and shifted)

(iii) Poisson - Lindley

(iv) Poisson - Zero Truncated Normal

(v) Poisson - Linear Exponential Family

Lately, the Poisson - Lindley distribution has been extended to Poisson - Generalized Lindley distribution. This distribution is also looked at in this chapter.

### 2.2 Mixing with Exponential Distribution

The pdf of Exponential distribution is

$$g(\lambda) = \mu e^{-\mu\lambda}; \lambda > 0 \tag{2.2}$$

The Mixed Poisson distribution is obtained as follows:

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\mu e^{-\mu\lambda}d\lambda \\ &= \frac{\mu}{x!}\int_0^\infty e^{-\lambda(1+\mu)}\lambda^x d\lambda \end{aligned}$$

Let

$$\begin{aligned} y &= \lambda(1+\mu) \Longrightarrow \lambda = \frac{y}{1+\mu} \\ dy &= (1+\mu)\,d\lambda \Longrightarrow d\lambda = \frac{dy}{1+\mu} \end{aligned}$$

Now

$$\begin{aligned} f(x) &= \frac{\mu}{x!}\int_0^\infty e^{-y}y^x\left(\frac{1}{1+\mu}\right)^x\frac{dy}{1+\mu} \\ &= \frac{\mu}{x!}\left(\frac{1}{1+\mu}\right)^{x+1}\int_0^\infty e^{-y}y^{(x+1)-1}dy \\ &= \frac{\mu}{x!}\left(\frac{1}{1+\mu}\right)^{x+1}\Gamma(x+1) \\ &= \mu\left(\frac{1}{1+\mu}\right)^{x+1}\frac{1}{x!}x! \\ &= \mu\left(\frac{1}{1+\mu}\right)^{x+1} \end{aligned}$$

Therefore,

$$f(x) = \left(\frac{\mu}{1+\mu}\right)\left(\frac{1}{1+\mu}\right)^x; x = 0, 1, 2, ... \qquad (2..3)$$

which is a Geometric Distribution (Johnson et al, 1992).

## 2.3 Mixing with Gamma Distribution with one parameter

The pdf of Gamma distribution with one parameter is,

$$g(\lambda) = \frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}; \lambda > 0, \alpha > 0 \qquad (2.4)$$

Therefore,

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}d\lambda \\ &= \frac{1}{x!\Gamma(\alpha)}\int_0^\infty e^{-2\lambda}\lambda^{x+\alpha-1}d\lambda \end{aligned}$$

Let

$$\begin{aligned} y &= 2\lambda \Longrightarrow \lambda = \frac{y}{2} \\ dy &= 2d\lambda \Longrightarrow d\lambda = \frac{dy}{2} \end{aligned}$$

Now,

$$\begin{aligned} f(x) &= \frac{1}{x!\Gamma(\alpha)}\int_0^\infty e^{-y}\left(\frac{y}{2}\right)^{x+\alpha-1}\frac{dy}{2} \\ &= \frac{1}{x!\Gamma(\alpha)}\left(\frac{1}{2}\right)^{x+\alpha}\int_0^\infty e^{-y}y^{x+\alpha-1}dy \\ &= \frac{1}{x!\Gamma(\alpha)}\left(\frac{1}{2}\right)^{x+\alpha}\Gamma(x+\alpha) \\ &= \frac{(x+\alpha-1)!}{x!\,(\alpha-1)!}\left(\frac{1}{2}\right)^{\alpha}\left(\frac{1}{2}\right)^{x} \end{aligned}$$

$$f(x) = \binom{x+\alpha-1}{x}\left(\frac{1}{2}\right)^{\alpha}\left(\frac{1}{2}\right)^{x}; x = 0,1,2,... \tag{2.5}$$

which is a Negative Binomial distribution with parameters $\alpha$ and $\frac{1}{2}$,(Greenwood and Yule, 1920).

## 2.4 Mixing with Gamma Distribution with two parameters

The pdf of Gamma distribution with two parameters is given by

$$g(\lambda) = \frac{\beta^\alpha}{\Gamma(\alpha)}e^{-\beta\lambda}\lambda^{\alpha-1}; \lambda > 0, \alpha > 0, \beta > 0 \tag{2.6}$$

The mixed Poisson distribution is thus

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\frac{\beta^\alpha}{\Gamma(\alpha)}e^{-\beta\lambda}\lambda^{\alpha-1}d\lambda \\ &= \frac{\beta^\alpha}{x!\Gamma(\alpha)}\int_0^\infty e^{-\lambda(1+\beta)}\lambda^{x+\alpha-1}d\lambda \end{aligned}$$

Let

$$\begin{aligned} y &= \lambda(1+\beta) \Longrightarrow \lambda = \frac{y}{1+\beta} \\ dy &= (1+\beta)\,d\lambda \Longrightarrow d\lambda = \frac{dy}{1+\beta} \end{aligned}$$

Now,

$$\begin{aligned} f(x) &= \frac{\beta^\alpha}{x!\Gamma(\alpha)}\int_0^\infty e^{-y}\left(\frac{y}{1+\beta}\right)^{x+\alpha-1}\frac{dy}{1+\beta} \\ &= \frac{\beta^\alpha}{x!\Gamma(\alpha)}\left(\frac{1}{1+\beta}\right)^{x+\alpha}\int_0^\infty e^{-y}y^{x+\alpha-1}dy \\ &= \frac{1}{x!\Gamma(\alpha)}\left(\frac{\beta}{1+\beta}\right)^{\alpha}\left(\frac{1}{1+\beta}\right)^{x}\Gamma(x+\alpha) \\ &= \frac{(x+\alpha-1)!}{x!\,(\alpha-1)!}\left(\frac{\beta}{1+\beta}\right)^{\alpha}\left(\frac{1}{1+\beta}\right)^{x} \end{aligned}$$

$$f(x) = \binom{x+\alpha-1}{x}\left(\frac{\beta}{1+\beta}\right)^{\alpha}\left(\frac{1}{1+\beta}\right)^{x}; x = 0, 1, 2, \ldots \qquad (2.7)$$

which is a Negative Binomial distribution with parameters $\alpha$ and $\frac{\beta}{1+\beta}$, (Greenwood and Yule, 1920).

## 2.5 Mixing with Shifted Gamma Distribution

For a two parameter Gamma distribution,

$$f(x) = \frac{\beta^\alpha}{\Gamma(\alpha)}e^{-\beta x}x^{\alpha-1}; x > 0, \alpha > 0, \beta > 0$$

Let

$$\begin{aligned} x &= y-\mu \Longrightarrow y = x+\mu \\ \frac{dy}{dx} &= 1 \end{aligned}$$

Using Jacobian of transformation,

$$\begin{aligned} g(y) &= f(x)\left|\frac{dy}{dx}\right| \\ &= \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta x} x^{\alpha-1} |1| \end{aligned}$$

$$g(y) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta(y-\mu)} (y-\mu)^{\alpha-1}; y>0, \alpha>0, \beta>0, \mu>0$$

Therefore replacing $y$ with $\lambda$,we have

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta(\lambda-\mu)} (\lambda-\mu)^{\alpha-1}; \lambda>0, \alpha>0, \beta>0, \mu>0 \qquad (2.8)$$

Now the mixed Poisson distribution becomes;

$$\begin{aligned} f(x) &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta(\lambda-\mu)} (\lambda-\mu)^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha}}{x!\Gamma(\alpha)} \int_0^{\infty} e^{-\lambda-\beta(\lambda-\mu)} \lambda^x (\lambda-\mu)^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha}}{x!\Gamma(\alpha)} e^{\beta\mu} \int_0^{\infty} e^{-\lambda(1+\beta)} \lambda^x (\lambda-\mu)^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha}}{x!\Gamma(\alpha)} e^{\beta\mu} \int_0^{\infty} e^{-(\lambda-\mu+\mu)(1+\beta)} \lambda^x (\lambda-\mu)^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha} e^{\beta\mu}}{x!\Gamma(\alpha)} \int_0^{\infty} e^{-(\lambda-\mu)(1+\beta)} e^{-\mu(1+\beta)} \lambda^x (\lambda-\mu)^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha} e^{\beta\mu} e^{-\beta\mu} e^{-\mu}}{x!\Gamma(\alpha)} \int_0^{\infty} e^{-(\lambda-\mu)(1+\beta)} \lambda^x (\lambda-\mu)^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha} e^{-\mu}}{x!\Gamma(\alpha)} \int_0^{\infty} e^{-(\lambda-\mu)(1+\beta)} \lambda^x (\lambda-\mu)^{\alpha-1} d\lambda \end{aligned}$$

Let

$$y = (\lambda-\mu)(1+\beta) \Longrightarrow (\lambda-\mu) = \frac{y}{1+\beta}; \lambda = \frac{y}{1+\beta}+\mu$$

$$dy = (1+\beta)\,d\lambda \Longrightarrow d\lambda = \frac{dy}{1+\beta}$$

$$\begin{aligned} f(x) &= \frac{\beta^{\alpha}e^{-\mu}}{x!\Gamma(\alpha)}\int_0^{\infty} e^{-y}\left(\frac{y+\mu(1+\beta)}{1+\beta}\right)^{x}\left(\frac{y}{1+\beta}\right)^{\alpha-1}\left(\frac{1}{1+\beta}\right)dy \\ &= \frac{\beta^{\alpha}e^{-\mu}}{x!\Gamma(\alpha)}\left(\frac{1}{1+\beta}\right)^{x+\alpha}\int_0^{\infty} e^{-y}\left[y+\mu(1+\beta)\right]^{x}y^{\alpha-1}dy \\ &= \frac{\beta^{\alpha}e^{-\mu}}{x!\Gamma(\alpha)}\left(\frac{1}{1+\beta}\right)^{x+\alpha}\int_0^{\infty} e^{-y}\left[\mu(1+\beta)\left(1+\frac{y}{\mu(1+\beta)}\right)\right]^{x}y^{\alpha-1}dy \\ &= \frac{\beta^{\alpha}e^{-\mu}\mu^{x}(1+\beta)^{x}}{x!\Gamma(\alpha)(1+\beta)^{x}(1+\beta)^{\alpha}}\int_0^{\infty} e^{-y}y^{\alpha-1}\left(1+\frac{y}{\mu(1+\beta)}\right)^{x}dy \end{aligned}$$

$$\begin{aligned} f(x) &= \frac{\beta^{\alpha}e^{-\mu}\mu^{x}}{x!\Gamma(\alpha)(1+\beta)^{\alpha}}\int_0^{\infty} e^{-y}y^{\alpha-1}\left[\sum_{k=0}^{x}\binom{x}{k}\left(\frac{y}{\mu(1+\beta)}\right)^{k}\right]dy \\ &= \frac{\beta^{\alpha}e^{-\mu}\mu^{x}}{x!\Gamma(\alpha)(1+\beta)^{\alpha}}\sum_{k=0}^{x}\left\{\binom{x}{k}\frac{1}{[\mu(1+\beta)]^{k}}\int_0^{\infty} e^{-y}y^{k+\alpha-1}dy\right\} \\ &= \frac{e^{-\mu}\mu^{x}}{x!\Gamma(\alpha)}\left(\frac{\beta}{1+\beta}\right)^{\alpha}\sum_{k=0}^{x}\left\{\binom{x}{k}\frac{1}{[\mu(1+\beta)]^{k}}\Gamma(k+\alpha)\right\} \\ &= \sum_{k=0}^{x}\binom{x}{k}\frac{e^{-\mu}\mu^{x}\Gamma(k+\alpha)}{x!\Gamma(\alpha)\mu^{k}}\left(\frac{\beta}{1+\beta}\right)^{\alpha}\left(\frac{1}{1+\beta}\right)^{k} \\ &= \sum_{k=0}^{x}\frac{x!}{k!(x-k)!}\frac{\Gamma(k+\alpha)}{x!\Gamma(\alpha)}\left(\frac{\beta}{1+\beta}\right)^{\alpha}\left(\frac{1}{1+\beta}\right)^{k}\mu^{x-k}e^{-\mu} \\ &= \sum_{k=0}^{x}\frac{\Gamma(k+\alpha)}{\Gamma(k+1)\Gamma(\alpha)(x-k)!}\left(\frac{1}{1+\beta}\right)^{k}\mu^{x-k}e^{-\mu} \end{aligned}$$

$$f(x) = \sum_{k=0}^{x} \frac{e^{-\mu}\mu^{x-k}}{(x-k)!} \frac{\Gamma(k+\alpha)}{\Gamma(k+1)\Gamma(\alpha)} \left(\frac{\beta}{1+\beta}\right)^{\alpha} \left(\frac{1}{1+\beta}\right)^{k} \tag{2.9}$$

which is a convolution of Poisson distribution and Negative Binomial distribution, known as Delaporte Distribution (Ruohonen, 1988).

## 2.6 Mixing with Lindley Distribution

The pdf for Lindley distribution is given by

$$g(\lambda) = \frac{\theta^2}{(\theta+1)} (\lambda+1) e^{-\lambda\theta}; \lambda > 0, \theta > 0 \tag{2.10}$$

The mixed Poisson distribution is

$$\begin{aligned} f(x) &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\theta^2}{(\theta+1)} (\lambda+1) e^{-\lambda\theta} d\lambda \\ &= \frac{\theta^2}{x!(\theta+1)} \int_0^{\infty} (\lambda+1) \lambda^x e^{-\lambda(1+\theta)} d\lambda \\ &= \frac{\theta^2}{(\theta+1)} \int_0^{\infty} \left[\frac{\lambda^{x+1}}{x!} e^{-\lambda(1+\theta)} + \frac{\lambda^x}{x!} e^{-\lambda(1+\theta)}\right] d\lambda \end{aligned}$$

Put

$$\phi = 1 + \theta \Longrightarrow \theta = \phi - 1$$

Therefore,

$$f(x) = \frac{(\phi-1)^2}{\phi} \int_0^{\infty} \left[\frac{\lambda^{x+1}}{x!} e^{-\phi\lambda} + \frac{\lambda^x}{x!} e^{-\phi\lambda}\right] d\lambda$$

Put

$$\begin{aligned} y &= \phi\lambda \Longrightarrow \lambda = \frac{y}{\phi} \\ d\lambda &= \frac{dy}{\phi} \end{aligned}$$

Therefore,

$$
\begin{aligned}
f(x) &= \frac{(\phi-1)^2}{\phi}\int_0^{\infty}\left[\frac{y^{x+1}}{\phi^{x+1}}\frac{e^{-y}}{x!}+\frac{y^x}{\phi^x}\frac{e^{-y}}{x!}\right]\frac{dy}{\phi} \\
&= \left(\frac{\phi-1}{\phi}\right)^2\left\{\frac{\Gamma(x+2)}{\phi^{x+1}x!}+\frac{\Gamma(x+1)}{\phi^x x!}\right\} \\
&= \left(\frac{\phi-1}{\phi}\right)^2\left\{\frac{(x+1)\Gamma(x+1)}{\phi^{x+1}x!}+\frac{\Gamma(x+1)}{\phi^x x!}\right\} \\
&= \left(\frac{\phi-1}{\phi}\right)^2\left[\frac{(x+1)}{\phi^{x+1}}+\frac{1}{\phi^x}\right]\frac{\Gamma(x+1)}{x!} \\
&= \left(\frac{\phi-1}{\phi}\right)^2\left[\frac{(x+1)}{\phi^{x+1}}+\frac{1}{\phi^x}\right] \\
&= \left(\frac{\phi-1}{\phi}\right)^2\left(\frac{x+1+\phi}{\phi^{x+1}}\right) \\
&= \frac{\theta^2}{(1+\theta)^2}\left[\frac{x+1+1+\theta}{(1+\theta)^{x+1}}\right]
\end{aligned}
$$

$$
f(x)=\frac{\theta^2(\theta+2+x)}{(1+\theta)^{x+3}};x=0,1,2,... \tag{2.11}
$$

which is the Poisson - Lindley distribution, (Sankaran, 1970).

## 2.7 Mixing with Generalized Lindley Distribution

### 2.7.1 Constructing Generalized Lindley Distribution

Let

$$v_1 \sim G(\alpha,\theta)$$

That is

$$g_1(v_1)=\frac{\theta^\alpha}{\Gamma(\alpha)}e^{-\theta v_1}v_1^{\alpha-1};\alpha>0,\theta>0,v_1>0$$

and

$$v_2 \sim G(\alpha+1, \theta)$$

That is

$$g_2(v_2) = \frac{\theta^{\alpha+1}}{\Gamma(\alpha+1)} e^{-\theta v_2} v_2^{\alpha}; \alpha > 0, \theta > 0, v_2 > 0$$

For a mixture distribution of $v_1$ and $v_2$, let

$$\begin{aligned} v_1 &= x \text{ with probability } p_1 \\ &\quad \text{and} \\ v_2 &= x \text{ with probability } p_2 \end{aligned}$$

Therefore,

$$f(x) = p_1 g_1(v_1) + p_2 g_2(v_2)$$

such that,

$$p_1 + p_2 = 1$$

Suppose

$$p_1 = \frac{\theta}{\theta+\gamma} \text{ and } p_2 = \frac{\gamma}{\theta+\gamma} \quad ; \theta > 0, \gamma > 0$$

Then

$$\begin{aligned} f(x) &= \frac{\theta}{\theta+\gamma} g_1(x) + \frac{\gamma}{\theta+\gamma} g_2(x) \\ &= \frac{\theta}{\theta+\gamma} \left[ \frac{\theta^{\alpha}}{\Gamma(\alpha)} e^{-\theta x} x^{\alpha-1} \right] + \frac{\gamma}{\theta+\gamma} \left[ \frac{\theta^{\alpha+1}}{\Gamma(\alpha+1)} e^{-\theta x} x^{\alpha} \right] \\ &= \frac{\theta^{\alpha+1} x^{\alpha-1} e^{-\theta x}}{(\theta+\gamma)\Gamma(\alpha)} \left[ 1 + \frac{\gamma x}{\alpha} \right] \\ &= \frac{\theta^{\alpha+1} x^{\alpha-1} e^{-\theta x}}{(\theta+\gamma)\Gamma(\alpha)} \left[ \frac{\alpha+\gamma x}{\alpha} \right] \\ &= \frac{\theta^{\alpha+1} x^{\alpha-1} (\alpha+\gamma x) e^{-\theta x}}{(\theta+\gamma)\Gamma(\alpha+1)} \end{aligned}$$

$$f(x) = \frac{\theta^2 (\theta x)^{\alpha-1} (\alpha+\gamma x) e^{-\theta x}}{(\theta+\gamma)\Gamma(\alpha+1)}; x > 0, \theta > 0, \alpha > 0, \gamma > 0$$

This is the probability density function of a 3-parameter Lindley distribution and it is denoted as $GL(\alpha, \theta, \gamma)$ ,(Zakerzadeh and Dolati, 2010).

Taking a special case when $\gamma = 1$, we have a 2-parameter Lindley pdf, i.e.,

$$f(x) = \frac{\theta^2 (\theta x)^{\alpha-1} (\alpha + x) e^{-\theta x}}{(\theta + 1) \Gamma(\alpha + 1)}; x > 0, \theta > 0, \alpha > 0$$

When $\alpha = 1$ and $\gamma = 1$, we have a one-parameter Lindley pdf, i.e.,

$$f(x) = \frac{\theta^2 e^{-\theta x} (1 + x)}{(\theta + 1)}$$

### 2.7.2 Poisson - Generalized Lindley Distribution

The mixed Poisson distribution is given by

$$f(x) = \int_0^\infty \frac{e^{-\lambda} \lambda^x}{x!} g(\lambda) \, d\lambda$$

Put

$$g(\lambda) = \frac{\theta^2 (\theta\lambda)^{\alpha-1} (\alpha + \lambda) e^{-\theta\lambda}}{(\theta + 1) \Gamma(\alpha + 1)}; \lambda > 0, \theta > 0, \alpha > 0 \tag{2.12}$$

$$\begin{aligned}
f(x) &= \int_0^\infty \frac{e^{-\lambda} \lambda^x}{x!} \frac{\theta^2 (\theta\lambda)^{\alpha-1} (\alpha + \lambda) e^{-\theta\lambda}}{(\theta + 1) \Gamma(\alpha + 1)} d\lambda \\
&= \frac{\theta^{\alpha+1}}{x! (\theta + 1) \Gamma(\alpha + 1)} \int_0^\infty e^{-\lambda(\theta+1)} \lambda^{x+\alpha-1} (\alpha + \lambda) \, d\lambda \\
&= \frac{\theta^{\alpha+1}}{x! (\theta + 1) \Gamma(\alpha + 1)} \left\{ \int_0^\infty \alpha e^{-\lambda(\theta+1)} \lambda^{x+\alpha-1} d\lambda + \int_0^\infty e^{-\lambda(\theta+1)} \lambda^{x+\alpha} d\lambda \right\} \\
&= \frac{\theta^{\alpha+1} \alpha}{x! (\theta + 1) \Gamma(\alpha + 1)} \int_0^\infty e^{-\lambda(\theta+1)} \lambda^{x+\alpha-1} d\lambda \\
&\quad + \frac{\theta^{\alpha+1}}{x! (\theta + 1) \Gamma(\alpha + 1)} \int_0^\infty e^{-\lambda(\theta+1)} \lambda^{x+\alpha} d\lambda
\end{aligned}$$

Let

$$y = \lambda(\theta + 1) \Longrightarrow \lambda = \frac{y}{\theta + 1}; d\lambda = \frac{dy}{\theta + 1}$$

therefore,

$$\begin{aligned} f(x) &= \frac{\theta^{\alpha+1}\alpha}{x!(\theta+1)\Gamma(\alpha+1)}\int_0^{\infty} e^{-y}\left(\frac{y}{\theta+1}\right)^{x+\alpha-1}\frac{dy}{(\theta+1)} \\ &\quad + \frac{\theta^{\alpha+1}}{x!(\theta+1)\Gamma(\alpha+1)}\int_0^{\infty} e^{-y}\left(\frac{y}{\theta+1}\right)^{x+\alpha}\frac{dy}{(\theta+1)} \\ &= \frac{\theta^{\alpha+1}\alpha}{x!(\theta+1)^{x+\alpha+1}\Gamma(\alpha+1)}\int_0^{\infty} e^{-y}y^{x+\alpha-1}dy \\ &\quad + \frac{\theta^{\alpha+1}}{x!(\theta+1)^{x+\alpha+2}\Gamma(\alpha+1)}\int_0^{\infty} e^{-y}y^{x+\alpha+1-1}dy \\ &= \frac{\theta^{\alpha+1}\alpha}{x!(\theta+1)^{x+\alpha+1}\Gamma(\alpha+1)}\Gamma(x+\alpha) \\ &\quad + \frac{\theta^{\alpha+1}}{x!(\theta+1)^{x+\alpha+2}\Gamma(\alpha+1)}\Gamma(x+\alpha+1) \end{aligned}$$

$$f(x) = \frac{\theta^{\alpha+1}\Gamma(x+\alpha)}{x!(\theta+1)^{x+\alpha+1}\Gamma(\alpha+1)}\left[\alpha + \frac{(x+\alpha)}{(\theta+1)}\right]; x = 0,1,2,...; \theta > 0, \alpha > 0 \tag{2.13}$$

Equation (2.13) is the Generalized Poisson - Lindley distribution and is denoted by $GPL(\alpha,\theta)$, (Mahmoudi & Zakerzadeh, 2010).

## 2.8 Mixing with Zero-Truncated Normal Distribution

If $x$ is a normally distributed random variable, then

$$\begin{aligned} f(x) &= \frac{1}{\sqrt{(2\pi\sigma^2)}}\exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right]; -\infty < x < \infty \\ -\infty &< \mu < \infty, \sigma^2 > 0 \end{aligned}$$

Since $f(x)$ is a probability density function, then

$$\int_{-\infty}^{\infty} f(x)\,dx = 1$$

$$\Longrightarrow \int_{-\infty}^{\infty} \frac{1}{\sqrt{(2\pi\sigma^2)}} \exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right] dx = 1$$

Now, let

$$I = \int_{0}^{\infty} \frac{1}{\sqrt{(2\pi\sigma^2)}} \exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right] dx$$

Let

$$z = \frac{x-\mu}{\sigma} \Longrightarrow dz = \frac{dx}{\sigma}; dx = \sigma dz$$

Therefore,

$$\begin{aligned} I &= \int_{-\frac{\mu}{\sigma}}^{\infty} \frac{1}{\sigma\sqrt{2\pi}} \exp\left[-\frac{z^2}{2}\right] \sigma dz \\ &= \int_{-\frac{\mu}{\sigma}}^{\infty} \frac{1}{\sqrt{2\pi}} \exp\left[-\frac{z^2}{2}\right] dz \\ &= \int_{-\infty}^{\frac{\mu}{\sigma}} \frac{1}{\sqrt{2\pi}} \exp\left[-\frac{z^2}{2}\right] dz \end{aligned}$$

Let

$$\phi(x) = \int_{x}^{\infty} f^*(x)\,dx$$

where $f^*(x)$ is the pdf of a standard normal distribution

Therefore,

$$I = \int_{-\infty}^{\frac{\mu}{\sigma}} \frac{1}{\sqrt{2\pi}} \exp\left[-\frac{z^2}{2}\right] dz = \phi\left(\frac{\mu}{\sigma}\right)$$

$$\Longrightarrow \int_{0}^{\infty} \frac{1}{\sqrt{(2\pi\sigma^2)}} \exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right] dx = \phi\left(\frac{\mu}{\sigma}\right)$$

Therefore,

$$\int_0^{\infty} \frac{1}{\phi\left(\frac{\mu}{\sigma}\right)\sqrt{(2\pi\sigma^2)}} \exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right] dx = 1$$

This implies that,

$$f(x) = \frac{1}{\phi\left(\frac{\mu}{\sigma}\right)\sqrt{(2\pi\sigma^2)}} \exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right]; 0 < x < \infty, -\infty < \mu < \infty, \sigma^2 > 0$$

is the probability density function of a Zero-Truncated Normal distribution.

Substituting $x$ with $\lambda$, we have;

$$g(\lambda) = \frac{1}{\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}} \exp\left[-\frac{(\lambda-\mu)^2}{2\sigma^2}\right]; 0 < \lambda < \infty, -\infty < \mu < \infty, \sigma^2 > 0 \tag{2.14}$$

On mixing with Poisson distribution, we have

$$\begin{aligned} f(x) &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda \\ &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} \frac{1}{\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}} e^{-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \\ &= \frac{1}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}} \int_0^{\infty} \lambda^x \exp\left[-\lambda - \frac{(\lambda-\mu)^2}{2\sigma^2}\right] d\lambda \\ &= \frac{1}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}} \int_0^{\infty} \lambda^x \exp\left[\frac{-2\lambda\sigma^2 - (\lambda-\mu)^2}{2\sigma^2}\right] d\lambda \end{aligned}$$

Now, lets consider the coefficient of the exponent, that is

$$\begin{aligned} \frac{2\lambda\sigma^2 + (\lambda-\mu)^2}{2\sigma^2} &= \frac{2\lambda\sigma^2 + \left(\lambda^2 - 2\mu\lambda + \mu^2\right)}{2\sigma^2} \\ &= \frac{1}{2\sigma^2}\left(\lambda^2 + 2\sigma^2\lambda - 2\mu\lambda + \mu^2\right) \\ &= \frac{1}{2\sigma^2}\left[\lambda^2 + 2\left(\sigma^2 - \mu\right)\lambda + \mu^2\right] \end{aligned}$$

By completing squares,

$$\begin{aligned}
\frac{2\lambda\sigma^2+(\lambda-\mu)^2}{2\sigma^2} &= \frac{1}{2\sigma^2}\left[\lambda^2+2\left(\sigma^2-\mu\right)\lambda+\left(\sigma^2-\mu\right)^2-\mu^2+\mu^2-\left(\sigma^2-\mu\right)^2+\mu^2\right] \\
&= \frac{1}{2\sigma^2}\left[\left(\lambda+\sigma^2-\mu\right)^2-\left(\sigma^2-\mu\right)^2+\mu^2\right] \\
&= \frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2}+\frac{\mu^2-\left(\sigma^2-\mu\right)^2}{2\sigma^2} \\
&= \frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2}+\frac{\mu^2-\sigma^4+2\mu\sigma^2-\mu^2}{2\sigma^2} \\
&= \frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2}+\frac{2\mu\sigma^2-\sigma^4}{2\sigma^2} \\
&= \frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2}+\left(\mu-\frac{\sigma^2}{2}\right)
\end{aligned}$$

Now,

$$\begin{aligned}
f(x) &= \frac{1}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}}\int_0^\infty \lambda^x e^{-\frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2}} e^{-\left(\mu-\frac{\sigma^2}{2}\right)} d\lambda \\
&= \frac{e^{\left(\frac{\sigma^2}{2}-\mu\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}}\int_0^\infty \lambda^x e^{-\frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2}} d\lambda
\end{aligned}$$

Let

$$\begin{aligned}
t &= \frac{\left(\lambda+\sigma^2-\mu\right)^2}{2\sigma^2} \Longrightarrow t^{\frac{1}{2}} = \frac{\left(\lambda+\sigma^2-\mu\right)}{\sqrt{2\sigma^2}} \\
&\Longrightarrow \sqrt{2\sigma^2 t} = \left(\lambda+\sigma^2-\mu\right) \\
&\Longrightarrow \lambda = \sqrt{2\sigma^2 t}-\sigma^2+\mu \\
&\Longrightarrow d\lambda = \frac{1}{2}\sqrt{\left(\frac{2\sigma^2}{t}\right)}dt
\end{aligned}$$

Now

$$f(x) = \frac{e^{\left(\frac{\sigma^2}{2}-\mu\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}} \int_{\frac{(\sigma^2-\mu)^2}{2\sigma^2}}^{\infty} e^{-t}\left[\mu-\sigma^2+\sqrt{2\sigma^2 t}\right]^x \frac{1}{2}\sqrt{\left(\frac{2\sigma^2}{t}\right)}dt$$

$$= \frac{e^{\left(\frac{\sigma^2}{2}-\mu\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi}} \int_{\frac{(\sigma^2-\mu)^2}{2\sigma^2}}^{\infty} \left[\left(\mu-\sigma^2\right)+\sqrt{2\sigma^2 t}\right]^x \frac{e^{-t}}{\sqrt{2t}}dt$$

$$= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi}} \int_{a}^{\infty} \left[\left(\mu-\sigma^2\right)+\left(2\sigma^2 t\right)^{\frac{1}{2}}\right]^x \frac{e^{-t}}{(2t)^{\frac{1}{2}}}dt$$

where

$$a = \frac{\left(\sigma^2-\mu\right)^2}{2\sigma^2}$$

$$
\begin{aligned}
f(x) &= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi}}\int_a^{\infty}\sum_{r=0}^{\infty}\binom{x}{r}\left(2\sigma^2 t\right)^{\frac{r}{2}}\left(\mu-\sigma^2\right)^{x-r}\frac{e^{-t}}{(2t)^{\frac{1}{2}}}dt \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi}}\int_a^{\infty}\sum_{r=0}^{\infty}\binom{x}{r}\frac{\left(\sqrt{2\sigma^2}\right)^r t^{\frac{r}{2}}\left(\mu-\sigma^2\right)^{x-r}e^{-t}}{\sqrt{2}t^{\frac{1}{2}}}dt \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\sum_{r=0}^{\infty}\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r\left(\mu-\sigma^2\right)^{x-r}\int_a^{\infty}t^{\frac{r}{2}-\frac{1}{2}}e^{-t}dt \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\sum_{r=0}^{\infty}\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r\left(\mu-\sigma^2\right)^{x-r}\int_a^{\infty}t^{\frac{r}{2}+\frac{1}{2}-1}e^{-t}dt \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\sum_{r=0}^{\infty}\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r\left(\mu-\sigma^2\right)^{x-r}\int_a^{\infty}t^{\frac{r+1}{2}-1}e^{-t}dt \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\sum_{r=0}^{\infty}\left\{\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r\left(\mu-\sigma^2\right)^{x-r}\left[\int_0^{\infty}t^{\frac{r+1}{2}-1}e^{-t}dt-\int_0^{a}t^{\frac{r+1}{2}-1}e^{-t}dt\right]\right\} \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\sum_{r=0}^{\infty}\left\{\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r\left(\mu-\sigma^2\right)^{x-r}\left[\Gamma\left(\frac{r+1}{2}\right)-\Gamma\left(\frac{r+1}{2}\right)\Gamma_a\left(\frac{r+1}{2}\right)\right]\right. \\
&= \frac{e^{-\left(\mu-\frac{\sigma^2}{2}\right)}}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\sum_{r=0}^{\infty}\left\{\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r\left(\mu-\sigma^2\right)^{x-r}\Gamma\left(\frac{r+1}{2}\right)\left[1-\Gamma_a\left(\frac{r+1}{2}\right)\right]\right\}
\end{aligned}
$$

$$f(x) = \frac{\left[e^{-\left(\mu-\frac{\sigma^2}{2}\right)}\right]\left(\mu-\sigma^2+\sqrt{2\sigma^2}\right)^x}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}$$

$$\sum_{r=0}^{\infty}\left\{\frac{\binom{x}{r}\left(\sqrt{2\sigma^2}\right)^r(\mu-\sigma^2)^{x-r}}{\left(\mu-\sigma^2+\sqrt{2\sigma^2}\right)^x}\Gamma\left(\frac{r+1}{2}\right)\left[1-\Gamma_a\left(\frac{r+1}{2}\right)\right]\right\}$$

$$= \left\{\frac{\left[e^{-\left(\mu-\frac{\sigma^2}{2}\right)}\right]\left(\mu-\sigma^2+\sqrt{2\sigma^2}\right)^x}{x!\phi\left(\frac{\mu}{\sigma}\right)2\sqrt{\pi}}\right\}$$

$$\left\{\sum_{r=0}^{\infty}\binom{x}{r}\frac{\left(\sqrt{2\sigma^2}\right)^r}{\left(\mu-\sigma^2+\sqrt{2\sigma^2}\right)^r}\frac{(\mu-\sigma^2)^{x-r}}{\left(\mu-\sigma^2+\sqrt{2\sigma^2}\right)^{x-r}}\Gamma\left(\frac{r+1}{2}\right)\left[1-\Gamma_a\left(\frac{r+1}{2}\right)\right]\right\}$$

Therefore, the mixed Poisson distribution is

$$f(x) = \frac{1}{x!2\sqrt{\pi}}\frac{1}{\phi\left(\frac{\mu}{\sigma}\right)}\exp\left[-\left(\mu-\frac{\sigma^2}{2}\right)\right]\left(\mu-\sigma^2+\sqrt{2\sigma^2}\right)^x$$

$$\sum_{r=0}^{x}\binom{x}{r}p^r(1-p)^{x-r}\left\{\Gamma\left(\frac{r+1}{2}\right)\left[1-\Gamma\left(\frac{r+1}{2}\right)\right]\right\} \quad (2.15)$$

where

$$p=\frac{\sqrt{2\sigma^2}}{\mu-\sigma^2+\sqrt{2\sigma^2}} \text{ and } 1-p=\frac{\mu-\sigma^2}{\mu-\sigma^2+\sqrt{2\sigma^2}}$$

Patil (1964).

## 2.9 Mixing with Linear Exponential Family

The pdf of Linear Exponential family is given as

$$g(\lambda)=\beta(\theta)e^{\lambda\theta}h(\lambda);h(\lambda)\geq 0,-\infty<\theta<\infty,\lambda>0 \quad (2.16)$$

Since $g(\lambda)$ is a pdf, then

$$\int_0^\infty \beta(\theta) e^{\lambda\theta} h(\lambda)\, d\lambda = 1$$

$$\Longrightarrow \frac{1}{\beta(\theta)} = \int_0^\infty e^{\lambda\theta} h(\lambda)\, d\lambda$$

Now the Mixed Poisson distribution becomes;

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda$$

$$= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \beta(\theta) e^{\lambda\theta} h(\lambda)\, d\lambda$$

$$= \frac{\beta(\theta)}{x!} \int_0^\infty e^{-\lambda(1-\theta)} \lambda^x h(\lambda)\, d\lambda$$

But

$$E(\lambda^x) = \int_0^\infty \lambda^x \beta(\theta) e^{\lambda\theta} h(\lambda)\, d\lambda$$

$$= \mu_x^{'}$$

Therefore,

$$\frac{\mu_x^{'}}{\beta(\theta)} = \int_0^\infty \lambda^x e^{\lambda\theta} h(\lambda)\, d\lambda$$

Now

$$f(x) = \frac{\beta(\theta)}{x!} \int_0^\infty e^{\lambda(\theta-1)} \lambda^x h(\lambda)\, d\lambda$$

Let

$$\phi = \theta - 1 \Longrightarrow \theta = \phi + 1$$

$$f(x) = \frac{\beta(\phi+1)}{x!} \int_0^\infty \lambda^x e^{\lambda\phi} h(\lambda)\, d\lambda$$

But

$$\int_0^\infty \lambda^x e^{\lambda\theta} h(\lambda)\, d\lambda = \frac{\mu_x^{'}(\theta)}{\beta(\theta)}$$

Therefore,

$$f(x) = \frac{\beta(\phi+1)}{x!}\int_0^\infty \lambda^x e^{\lambda\phi} h(\lambda)\, d\lambda$$

$$= \frac{\beta(\phi+1)}{x!}\frac{\mu_x'(\phi)}{\beta(\phi)}$$

But

$$\phi = \theta - 1$$

therefore,

$$f(x) = \frac{\beta(\theta)}{x!\beta(\theta-1)}\mu_x'(\theta-1); x = 0, 1, 2, ... \tag{2.17}$$

where $\mu_x'(\theta-1)$ is the raw moment of order $X$ of the Linear Exponential Family with parameter $(\theta-1)$, (Sankaran, 1969).

# Chapter 3

## RECURSIVE RELATIONS FOR MIXED POISSON DISTRIBUTIONS

### 3.1 Introduction

A main difficulty with the use of Mixed Poisson distribution is that, with the exception of a few mixing distributions, their probability mass function $f(x)$ is difficult to evaluate (Albercht, 1984). One way of circumventing this problem is to express the mixed distributions in terms of recursive relations.

A number of methods for deriving such recursive relations have been developed, starting with the works of Katz (1965), Panjer (1981), Sundt (1992), Willmot (1993), etc. It is however interesting to note that the recursive models obtained earlier can also be derived by the use of Integration by Parts, which will be the main objective of this chapter.

A brief discussion is given on the previous works;

### 3.2 Panjer's Class of Recursive Relations

Pearson difference equation is given by

$$\frac{f(x+1)}{f(x)} = \frac{P(x)}{Q(x)} \tag{3.1}$$

where $f(\cdot)$ is the discrete probability distribution; $P(x)$ and $Q(x)$ are polynomials.

Katz (1965) considered the difference equation

$$\frac{f(x+1)}{f(x)} = \frac{\alpha+\beta x}{1+x}; x = 0, 1, 2, ... \tag{3.2}$$

Let $x+1=n$ and replace $f(n)$ by $p(n)$, then (3.2) becomes

$$\begin{aligned} p_n &= \left(\frac{\alpha+\beta(n-1)}{n}\right) p_{n-1} \\ &= \left(\frac{\alpha-\beta+\beta n}{n}\right) p_{n-1} \end{aligned}$$

Therefore,

$$p_n = \left(a+\frac{b}{n}\right) p_{n-1}; n=1,2,3,... \tag{3.3}$$

where $a=\beta$ and $b=\alpha-\beta$. Equation (3.3) is the Panjer's model for recursive relation.

By iteration or pgf technique, it can be shown that only Poisson, Binomial and Negative Binomial Distributions satisfy the Katz - Panjer model. (Sundt and Jewel, 1981; Katz, 1965).

Panjer's class of order k is defined by

$$\begin{aligned} \frac{f(x+1)}{f(x)} &= \frac{a+bx}{1+x}; \\ x &= k, k+1, k+2, ...; \\ x &\geq k \\ k &= 0,1,2,... \end{aligned}$$

(Hess et al, 2002).

## 3.3 Other Extensions

Panjer and Willmot (1982) considered the class of counting distributions which satisfy a recursion

$$p_n = p_{n-1} \frac{\sum_{t=0}^{k} a_t n^t}{\sum_{t=0}^{k} b_t n^t}; n=1,2,3,...$$

for some $k$.

Therefore

$$\begin{aligned} p_n \sum_{t=0}^{k} b_t n^t &= p_{n-1} \sum_{t=0}^{k} a_t n^t \\ &= p_{n-1} \sum_{t=0}^{k} a_t \left[1 + (n-1)\right]^t \\ &= p_{n-1} \sum_{t=0}^{k} a_t \left\{ \sum_{l=0}^{t} a_l \binom{t}{l} (n-1)^t \right\} \end{aligned}$$

Therefore

$$\begin{aligned} p_n \sum_{t=0}^{k} b_t n^t &= p_{n-1} \sum_{t=0}^{k} \sum_{l=0}^{t} a_t \binom{t}{k} (n-1)^l \\ &= p_{n-1} \sum_{t=0}^{k} \sum_{l=0}^{k} a_t \binom{t}{l} (n-1)^l \\ &= p_{n-1} \sum_{l=0}^{k} \sum_{t=0}^{k} a_t \binom{t}{l} (n-1)^l \\ &= p_{n-1} \sum_{l=0}^{k} \left\{ \sum_{t=0}^{k} a_t \binom{t}{l} \right\} (n-1)^l \\ &= p_{n-1} \sum_{l=0}^{k} c_l (n-1)^l \end{aligned}$$

where

$$c_l = \sum_{t=0}^{k} a_t \binom{t}{l}$$

which is due to Hasselager (1994).

Wang(1994) extended Hasselager model to

$$p_n \sum_{i=0}^{k} b_i n^i = \sum_{j=1}^{s} \left[ p_{n-j} \sum_{i=0}^{k} a_{ji} (n-j)^i \right] ; n = c, c+1, c+2, \ldots$$

## 3.4 Willmot's Approach

Willmot (1993) showed that, for several mixed Poisson distributions, a recursive formula can be obtained. Karlis and Xekalaki (2005) explain this approach as follows:

If the mixing density $g(\bullet)$ satisfies the relationship

$$\frac{d}{d\lambda} \ln g(\lambda) = \frac{\sum_{i=0}^{k} s_i \lambda^i}{\sum_{i=0}^{k} \omega_i \lambda^i}, \lambda > 0 \tag{3.4}$$

for some constants $s_i, \omega_i, i = 0, 1, ..., k$; $k > 0$, the probability function $P(x)$ of the Mixed Poisson with $g(\lambda)$ satisfies the recursive formula:

$$\sum_{n=-1}^{k} \{\varphi_n + m\omega_{n+1}\} (m+n)^{(n)} P(m+n) = 0 \tag{3.5}$$

where

$$a^{(b)} = a(a+1)(a+2)....(a+b+1) \tag{3.6}$$

and

$$\varphi_n = s_n + (n+1)\omega_{n+1} + \omega_n \tag{3.7}$$

with

$$\varphi_{-1} = 0$$

Appropriate modifications have been suggested by Willmot (1993) for different supports of $\lambda$. We should note that this iterative scheme requires calculation of the first k probabilities only.

Antzoulakos and Chadicontantinidis (2004) explain Willmot's Approach as follows:

Assume that the pdf $u(\bullet)$ satisfies,

$$\frac{d}{d\lambda}\log u(\lambda) = \frac{a(\lambda)}{b(\lambda)} = \frac{\sum_{i=0}^{r} a_i \lambda^i}{\sum_{i=0}^{r} b_i \lambda^i}, \lambda \in [\lambda_1, \lambda_2] \tag{3.8}$$

A random variable $L$ is said to belong to the class $W(r)$ if there exist a positive integer $r$ and constants $a_i$ and $b_i$ $(1 \leq i \leq r)$ such that the pdf $u(\bullet)$ of L satisfies (3.5) $(viz., L \in W(r)$ or $u(\bullet) \in W(r))$.

It is assumed that at least one of $a_i$ and $b_i$ is different from zero and the numerator in (3.5) are allowed to have common factors.

Willmot (1993) proposed a recursion for the evaluation of $p(\bullet)$ in the case where $L \in W(r)$. More specifically , he showed that for $n \geq 0$,

$$\sum_{i=-1}^{r} [a_i - b_i + (n+i+1)\, b_{i+1}]\, (n+i)^{(i)}\, p(n+1) = c_{\lambda_2}(n) - c_{\lambda_1}(n) \tag{3.9}$$

where

$$a^{(b)} = \prod_{i=1}^{b} (a+1-i)\,; \qquad p_{-1} = 0 \tag{3.10}$$

and

$$c_\lambda(n) = \left\{ \begin{array}{ll} b_0 u(0)\,, & \lambda = 0, n = 0 \\ 0, & \lambda = 0, n > 0 \\ b(\lambda)\, u(\lambda)\, p_\lambda(n)\,, & 0 < \lambda < \infty \\ 0, & \lambda = \infty \end{array} \right\} \tag{3.11}$$

Note:

$$p_n = \Pr(N = n) = \int_{\lambda_1}^{\lambda_2} p_\lambda(n)\, du(\lambda)\,; \quad n = 0, 1, 2, \ldots \tag{3.12}$$

and

$$p_\lambda(n) = \Pr(N_\lambda = n) = \frac{e^{-\lambda}\lambda^n}{n!}, \quad n = 0, 1, 2, \ldots \tag{3.13}$$

For Shifted Gamma Mixing Distribution,

$$u(\lambda) = \frac{\beta^{\alpha}(\lambda-\mu)^{\alpha-1}\exp[-\beta(\lambda-\mu)]}{\Gamma(\alpha)}; \quad \lambda \geq 0, \mu \geq 0$$

We observe that,

$$\frac{d}{d\lambda}\log u(\lambda) = \frac{\alpha - 1 + \beta\mu - \beta\lambda}{\lambda - \mu}$$

which corresponds to (3.5) with $r = 1$ and $a_0 = \alpha - 1 + \beta\mu$ , $a_1 = -\beta$ , $b_0 = -\mu$, $b_1 = 1$.

When $\mu = 0$, the shifted Gamma mixing distribution reduces to the usual Gamma mixing distribution.

For Scaled Beta Mixing Distribution,

$$\mu(\lambda) = \frac{\Gamma(\alpha+\beta)\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{\Gamma(\alpha)\Gamma(\beta)\mu^{\alpha+\beta-1}}; 0 \leq \lambda \leq \mu$$

$$\frac{d}{d\lambda}\log u(\lambda) = \frac{\mu(\alpha-1) - \lambda(\alpha+\beta-2)}{\lambda(\mu-\lambda)}$$

Which corresponds to (3.5) with $r = 2; a_0 = \mu(\alpha-1), a_1 = -(\alpha+\beta-2), a_2 = 0; b_0 = 0, b_1 = \mu, b_2 = -1$

$\alpha = 1 \Rightarrow$ Beta mixing distribution.

For Generalized Pareto Mixing Distribution,

$$u(\lambda) = \frac{\Gamma(\alpha+\beta)}{\Gamma(\alpha)\Gamma(\beta)}\frac{\mu^{\alpha}\lambda^{\beta-1}}{(\mu+\alpha)^{\alpha+\beta}}; \lambda \geq 0$$

$$\frac{d}{d\lambda}\log u(\lambda) = \frac{\mu(\beta-1) - \lambda(\alpha+1)}{\lambda(\lambda+\mu)}$$

Which corresponds to (3.5) with $r = 2$; $a_0 = \mu(\beta-1)$, $a_1 = -(\alpha+1)$, $a_2 = 0; b_0 = 0, b_1 = \mu, b_2 = 1$

$\beta = 1 \Rightarrow$ Pareto mixing distribution.

For Generalized Inverse Gaussian Mixing Distribution,

$$u(\lambda) = \frac{\mu^{-\alpha}\lambda^{\alpha-1}}{2K_\alpha(\mu\beta^{-1})} \exp\left[-\left(\frac{\lambda^2+\mu^2}{2\beta\lambda}\right)\right]; \lambda \geq 0$$

where $K_\alpha(x)$ denotes the modified Bessel function of the third kind with index $\alpha$.

$$\frac{d}{d\lambda}\log u(\lambda) = \frac{\mu^2 + 2\beta(\alpha-1)\lambda - \lambda^2}{2\beta\lambda^2}$$

which corresponds to (3.5) with $r = 2; a_0 = \mu^2, a_1 = 2\beta(\alpha-1), a_2 = -1; b_0 = 0, b_1 = 0, b_2 = 2\beta$

$\alpha = -\frac{1}{2} \Rightarrow$ Inverse Gaussian Mixing Distribution.

## 3.5 Sundt's Approach

Sundt (1992) extended Panjer Class of counting distributions to

$$p_n = \sum_{i=1}^{k}\left(a_i + \frac{b_i}{n}\right)p_{n-i}; n = 1, 2, \ldots \tag{3.14}$$

The probability generating function, pgf

$$G(s) = \sum_{n=0}^{\infty} p_n s^n$$

Therefore,

$$G'(s) = \sum_{n=0}^{\infty} np_n s^{n-1} = \sum_{n=1}^{\infty} np_n s^{n-1}$$

$$G'(s) = \sum_{n=1}^{\infty} ns^{n-1}p_n = \sum_{n=1}^{\infty}\left\{ns^{n-1}\sum_{i=1}^{k}\left(a_i + \frac{b_i}{n}\right)p_{n-i}\right\}$$

$$G'(s) = \sum_{n=1}^{\infty} \left\{ n s^{n-1} \sum_{i=1}^{k} \left( \frac{n a_i + b_i}{n} \right) p_{n-i} \right\}$$

$$G'(s) = \sum_{n=1}^{\infty} \left\{ s^{n-1} \sum_{i=1}^{k} (n a_i + b_i) p_{n-i} \right\}$$

$$G'(s) = \sum_{i=1}^{k} \sum_{n=1}^{\infty} (n a_i + b_i) p_{n-i} s^{n-1}$$

$$G'(s) = \sum_{i=1}^{k} \sum_{n=i}^{\infty} (n a_i + b_i) p_{n-i} s^{n-1}$$

$$G'(s) = \sum_{i=1}^{k} \left\{ [i a_i + b_i] p_0 s^{i-1} + [(i+1) a_i + b_i] p_1 s^{i} + \ldots + [(i+v) a_i + b_i] p_v s^{v+i-1} + \ldots \right\}$$

$$G'(s) = \sum_{i=1}^{k} \sum_{v=0}^{\infty} [(i+v) a_i + b_i] p_v s^{v+i-1}$$

$$G'(s) = \sum_{i=1}^{k} \left\{ \sum_{n=0}^{\infty} [(n+i) a_i + b_i] p_n s^{n+i-1} \right\}$$

$$G^{'}(s) = \sum_{i=1}^{k} \left\{ \sum_{n=0}^{\infty} \left[ na_i + ia_i + b_i \right] p_n s^{n+i-1} \right\}$$

$$G^{'}(s) = \sum_{i=1}^{k} \left\{ a_i \sum_{n=0}^{\infty} np_n s^{n+i-1} + (ia_i + b_i) s^{i-1} \sum_{n=0}^{\infty} p_n s^n \right\}$$

$$G^{'}(s) = \sum_{i=1}^{k} \left\{ a_i s^i \sum_{n=0}^{\infty} np_n s^{n-1} + (ia_i + b_i) s^{i-1} G(s) \right\}$$

$$G^{'}(s) = \sum_{i=1}^{k} \left\{ a_i s^i G^{'}(s) + (ia_i + b_i) s^{i-1} G(s) \right\}$$

Therefore,

$$\left[ 1 - \sum_{i=1}^{k} a_i s^i \right] G^{'}(s) = \sum_{i=1}^{k} (ia_i + b_i) s^{i-1} G(s)$$

Therefore,

$$\frac{G^{'}(s)}{G(s)} = \frac{\sum_{i=1}^{k} (ia_i + b_i) s^{i-1}}{1 - \sum_{i=1}^{k} a_i s^i}$$

Let

$$p(s) = \frac{G^{'}(s)}{G(s)} = \frac{d}{ds} \log G(s)$$

Therefore,

$$p(s) = \frac{\sum_{i=1}^{k} (ia_i + b_i) s^{i-1}}{1 - \sum_{i=1}^{k} a_i s^i} \tag{3.15}$$

which together with the initial condition $G(1) = 1$ determines the counting distribution $R_k(\underline{a}, \underline{b})$ satisfying (3.11) with

$$\underline{a} = (a_1, a_2, ..., a_k)$$

and

$$\underline{b} = (b_1, b_2, ..., b_k)$$

Panjer and Wang (1995) comment on the model (3.11) which Sundt (1992) extended further to a generalized class, namely

$$p_n = \sum_{i=1}^{k} \left( a_i + \frac{b_i}{n} \right) p_{n-i}; n = \omega + 1, \omega + 2, ... \tag{3.16}$$

## 3.6 Recursive Models based on Integration by Parts for Mixed Poisson Distributions

Integration by parts does not require assumptions given by Willmot (1993) or Sundt(1992). Thus it covers much more recursive relations including those obtained by Willmot and Sundt.

### 3.6.1 Rectangular Mixing Distribution

If the mixing distribution is U(a,b), then the recursive formula for the Poisson-Rectangular distribution becomes

$$f(x+1) = f(x) + \frac{1}{(x+1)!\,(b-a)} \left( e^{-a} a^{x+1} - e^{-b} b^{x+1} \right); x = 0, 1, 2, ... \tag{3.17}$$

with

$$f(0) = \frac{e^{-a} - e^{-b}}{b - a}$$

$$f(x+1) = f(x) - \frac{e^{-1}}{(x+1)!}; x = 0, 1, 2, \ldots \tag{3.18}$$

with

$$f(0) = 1 - e^{-1} \quad \text{when } a = 0 \quad \text{and} \quad b = 1$$

a) The mixing distribution is

$$g(\lambda) = \frac{1}{b - a}; a \leq \lambda \leq b \tag{3.19}$$

Therefore,

$$f(x) = \int_a^b \frac{e^{-\lambda}\lambda^x}{x!} \frac{1}{b-a} d\lambda$$

$$f(x) = \frac{1}{x!(b-a)} \int_a^b e^{-\lambda}\lambda^x d\lambda$$

$$f(x) = \frac{1}{x!(b-a)} \left[ \int_0^b e^{-\lambda}\lambda^x d\lambda - \int_0^a e^{-\lambda}\lambda^x d\lambda \right]$$

Let

$$\Gamma_b(x+1) = \int_0^b e^{-\lambda}\lambda^x d\lambda$$

and

$$\Gamma_a(x+1) = \int_0^a e^{-\lambda}\lambda^x d\lambda$$

which are both incomplete Gamma functions.

Let
$\mu = \lambda^x$ and $dv = e^{-\lambda} \Longrightarrow du = x\lambda^{x-1}$ and $v = -e^{-\lambda}$
Therefore,

$$\Gamma_b(x+1) = \left|-\lambda^x e^{-\lambda}\right|_0^b + \int_0^b e^{-\lambda} x\lambda^x d\lambda$$

$$\Gamma_b(x+1) = -\left[b^x e^{-b} - 0\right] + x\int_0^b \lambda^{x-1} e^{-\lambda} d\lambda$$

$$\Gamma_b(x+1) = -b^x e^{-b} + x\Gamma_b(x)$$

$$\Gamma_b(x+1) = -b^x e^{-b} + x\left[-b^{x-1}e^{-b} + (x-1)\Gamma_b(x-1)\right]$$

$$\Gamma_b(x+1) = -b^x e^{-b} - xb^{x-1}e^{-b} + x(x-1)\Gamma_b(x-1)$$

$$\begin{aligned}\Gamma_b(x+1) &= -b^x e^{-b} - xb^{x-1}e^{-b} \\ &\quad + x(x-1)\left[-b^{x-2}e^{-b} + (x-2)\Gamma_b(x-2)\right]\end{aligned}$$

$$\begin{aligned}\Gamma_b(x+1) &= -b^x e^{-b} - xb^{x-1}e^{-b} - x(x-1)b^{x-2}e^{-b} \\ &\quad + x(x-1)(x-2)\Gamma_b(x-2)\end{aligned}$$

$$\begin{aligned}\Gamma_b(x+1) &= -e^{-b}\left[b^x + xb^{x-1} + x(x-1)b^{x-2}\right] \\ &\quad +x(x-1)(x-2)\Gamma_b(x-2)\end{aligned}$$

$$\begin{aligned}\Gamma_b(x+1) &= -e^{-b}\left[b^x + xb^{x-1} + x(x-1)b^{x-2} + x(x-1)(x-2)b^{x-3}\right] \\ &\quad +x(x-1)(x-2)(x-3)\Gamma_b(x-3)\end{aligned}$$

$$\Gamma_b(x+1) = -e^{-b}\left[\begin{array}{c} b^x + xb^{x-1} + x(x-1)b^{x-2} + x(x-1)(x-2)b^{x-3} \\ +x(x-1)(x-2)(x-3)b^{x-4} + ... \\ ... + x(x-1)(x-2)(x-3)...[x-(x-1)]b^{x-x}\end{array}\right]$$

Therefore,

$$\frac{1}{x!(b-a)}\Gamma_b(x+1) = -\frac{e^{-b}}{(b-a)}\left\{\frac{b^x}{x!} + \frac{b^{x-1}}{(x-1)!} + \frac{b^{x-2}}{(x-2)!} + ... + \frac{b}{1!} + \frac{1}{0!}\right\}$$

Similarly,

$$\frac{1}{x!(b-a)}\Gamma_a(x+1) = -\frac{e^{-a}}{(b-a)}\left\{\frac{a^x}{x!} + \frac{a^{x-1}}{(x-1)!} + \frac{a^{x-2}}{(x-2)!} + ... + \frac{a}{1!} + \frac{1}{0!}\right\}$$

Therefore,

$$f(x) = \frac{1}{x!(b-a)}\left[\Gamma_b(x+1) - \Gamma_a(x+1)\right]$$

$$\begin{aligned}f(x) &= \frac{e^{-a}}{(b-a)}\left\{\frac{a^x}{x!} + \frac{a^{x-1}}{(x-1)!} + \frac{a^{x-2}}{(x-2)!} + ... + \frac{a}{1!} + \frac{1}{0!}\right\} \\ &\quad -\frac{e^{-b}}{(b-a)}\left\{\frac{b^x}{x!} + \frac{b^{x-1}}{(x-1)!} + \frac{b^{x-2}}{(x-2)!} + ... + \frac{b}{1!} + \frac{1}{0!}\right\}\end{aligned}$$

$$f(x)=\frac{1}{b-a}\left\{\begin{array}{l}\frac{\left(e^{-a}a^{x}-e^{-b}b^{x}\right)}{x!}+\frac{\left(e^{-a}a^{x-1}-e^{-b}b^{x-1}\right)}{(x-1)!}\\ \\ +\ldots+\frac{\left(e^{-a}a-e^{-b}b\right)}{1}+\left(e^{-a}-e^{-b}\right)\end{array}\right\}$$

$$f(x+1)=\frac{1}{b-a}\left\{\begin{array}{l}\frac{\left(e^{-a}a^{x+1}-e^{-b}b^{x+1}\right)}{(x+1)!}+\frac{\left(e^{-a}a^{x}-e^{-b}b^{x}\right)}{x!}+\frac{\left(e^{-a}a^{x-1}-e^{-b}b^{x-1}\right)}{(x-1)!}+\ldots\\ \ldots+\frac{\left(e^{-a}a-e^{-b}b\right)}{1}+\left(e^{-a}-e^{-b}\right)\end{array}\right\}$$

$$f(x+1)=\frac{1}{b-a}\left\{\frac{\left(e^{-a}a^{x+1}-e^{-b}b^{x+1}\right)}{(x+1)!}\right\}+f(x)$$

Therefore,

$$f(x+1)=f(x)+\frac{e^{-a}a^{x+1}-e^{-b}b^{x+1}}{(x+1)!\,(b-a)};x=0,1,2,...$$

and

$$f(0)=\frac{1}{b-a}\left[e^{-a}-e^{-b}\right]$$

b) When $a=0$ and $b=1$, then

$$f(x+1)=f(x)+\left[-\frac{e^{-1}}{(x+1)!}\right]=f(x)-\frac{e^{-1}}{(x+1)!};x=0,1,2,...$$

with initial condition

$$f(0)=1-e^{-1}$$

### 3.6.2 Poisson-Inverse Gaussian Distribution

If the Inverse Gaussian mixing distribution is given by

$$g(\lambda) = \left(\frac{\phi}{2\pi\lambda^3}\right)^{\frac{1}{2}} \exp\left\{-\frac{\phi(\lambda-\mu)^2}{2\mu^2\lambda}\right\}; \lambda > 0, \mu > 0 \text{ and } \phi > 0 \qquad (3.20)$$

then the recursive formula for Poisson-Inverse Gaussian distribution becomes

a)

$$\left(1+\frac{\phi}{2\mu^2}\right) x f(x+1) = x f(x) + \frac{\phi}{2}\frac{f(x-1)}{x+1}; x = 0, 1, 2, ...$$

with

$$f(-1) = 0$$

b)

$$(1+2\beta)\, n\,(n-1)\, p_n = \beta\,(n-1)\,(2n-3)\, p_{n-1} + \mu^2 p_{n-2}; \text{ for } n \geq 2$$

when $x = n-1$, $f(x+1) = p_n$ and $\phi = \frac{\mu^2}{\beta}$ with initial conditions

$$p_1 = \mu\,(1+2\beta)^{-\frac{1}{2}}\, p_0$$

where

$$p_0 = \exp\left\{-\frac{\mu}{2\beta}\left[(1+2\beta)^{\frac{1}{2}} - 1\right]\right\}$$

(Willmot, 1986)

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\left(\frac{\phi}{2\pi\lambda^3}\right)^{\frac{1}{2}} \exp\left[-\frac{\phi(\lambda-\mu)^2}{2\mu^2\lambda}\right] d\lambda$$

$$f(x) = \frac{1}{x!}\left(\frac{\phi}{2\pi}\right)^{\frac{1}{2}} \int_0^{\infty} e^{-\lambda} \frac{\lambda^x}{\lambda^{\frac{3}{2}}} \exp\left[-\frac{\phi}{2\mu^2\lambda}\left(\lambda^2 + \mu^2 - 2\mu\lambda\right)\right] d\lambda$$

$$f(x) = \frac{1}{x!}\left(\frac{\phi}{2\pi}\right)^{\frac{1}{2}} \int_0^{\infty} e^{-\lambda} \lambda^{x-\frac{3}{2}} \exp\left[-\frac{\phi}{2\mu^2}\lambda - \frac{\phi\mu^2}{2\mu^2\lambda} + \frac{\phi}{\mu}\right] d\lambda$$

$$f(x) = \frac{1}{x!}\left(\frac{\phi}{2\pi}\right)^{\frac{1}{2}} e^{\frac{\phi}{\mu}} \int_0^{\infty} e^{-\lambda\left(1+\frac{\phi}{2\mu^2}\right)-\frac{\phi}{2\lambda}} \lambda^{x-\frac{3}{2}} d\lambda$$

Therefore,

$$\left(\frac{\phi}{2\pi}\right)^{-\frac{1}{2}} e^{-\frac{\phi}{\mu}} x! f(x) = \int_0^{\infty} e^{-\lambda\left(1+\frac{\phi}{2\mu^2}\right)-\frac{\phi}{2\lambda}} \lambda^{x-\frac{3}{2}} d\lambda \approxeq I_x$$

Therefore,

$$I_x = \left(\frac{\phi}{2\pi}\right)^{-\frac{1}{2}} e^{-\frac{\phi}{\mu}} x! f(x)$$

and

$$I_x = \int_0^{\infty} e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}} \lambda^{x-\frac{3}{2}} d\lambda$$

Consider

$$I_x = \int_0^{\infty} e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}} \lambda^{x-\frac{3}{2}} d\lambda$$

Let

$$u = e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}} \text{ and } dv = \lambda^{x-\frac{3}{2}}$$

Therefore,

$$du = \left[-\left(1+\frac{\phi}{2\mu^2}\right)+\frac{\phi}{2\lambda^2}\right]e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}}d\lambda$$

and

$$v = \frac{\lambda^{x-\frac{1}{2}}}{x-\frac{1}{2}}$$

Therefore

$$I_x = -\int_0^\infty \frac{\lambda^{x+1-\frac{3}{2}}}{x+1-\frac{3}{2}}\left[-\left(1+\frac{\phi}{2\mu^2}\right)+\frac{\phi}{2\lambda^2}\right]e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}}d\lambda$$

$$= -\int_0^\infty -\frac{\lambda^{x+1-\frac{3}{2}}}{x+1-\frac{3}{2}}\left(1+\frac{\phi}{2\mu^2}\right)e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}}d\lambda$$

$$-\int_0^\infty \frac{\lambda^{x+1-\frac{3}{2}}}{x+1-\frac{3}{2}}\frac{\phi}{2\lambda^2}e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}}d\lambda$$

$$= \left(1+\frac{\phi}{2\mu^2}\right)\frac{1}{\left(x+1-\frac{3}{2}\right)}\int_0^\infty e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}}\lambda^{x+1-\frac{3}{2}}d\lambda$$

$$-\frac{\phi}{2\left(x+1-\frac{3}{2}\right)}\int_0^\infty e^{-\left(1+\frac{\phi}{2\mu^2}\right)\lambda-\frac{\phi}{2\lambda}}\lambda^{x-1-\frac{3}{2}}d\lambda$$

therefore

$$I_x = \left(1+\frac{\phi}{2\mu^2}\right)\frac{1}{\left(x+1-\frac{3}{2}\right)}I_{x+1}-\frac{\phi}{2\left(x-\frac{1}{2}\right)}I_{x-1}$$

This implies that

$$x!f(x) = \left(1+\frac{\phi}{2\mu^2}\right)\frac{(x+1)!}{\left(x+1-\frac{3}{2}\right)}f(x+1)$$

$$-\frac{\phi}{\left(x+1-\frac{3}{2}\right)}(x-1)!f(x-1)$$

$$xf(x) = \left(1 + \frac{\phi}{2\mu^2}\right) \frac{x(x+1)}{\left(x - \frac{1}{2}\right)} f(x+1) - \frac{\phi}{(2x-1)} f(x-1)$$

Therefore

$$\begin{aligned} \left(1 + \frac{\phi}{2\mu^2}\right) \frac{x(x+1)}{\left(x - \frac{1}{2}\right)} f(x+1) &= xf(x) \\ &\quad + \frac{\phi}{(2x-1)} f(x-1) \\ \text{for } ; x &= 0, 1, 2, \ldots \text{with } f(-1) = 0 \end{aligned} \tag{3.21}$$

When

$$x = n - 1$$

$$\left(1 + \frac{\phi}{2\mu^2}\right) \frac{n(n-1)}{\left(n - \frac{3}{2}\right)} p_n = (n-1) p_{n-1} + \frac{\phi}{[2n-3]} p_{n-2}; n = 2, 3, 4, \ldots$$

### 3.6.3 Poisson-Generalized Inverse Gaussian Distribution (Sichel Distribution)

If the Generalized Inverse Gaussian distribution is given by

$$g(\lambda) = \frac{\eta^{-\gamma} \lambda^{\gamma-1}}{2K_\gamma(\omega)} \exp\left\{-\frac{\omega\left(\eta^2 + \lambda^2\right)}{2\eta\lambda}\right\}; \lambda \geq 0, -\infty < \gamma < \infty, \eta > 0, \omega \geq 0 \tag{3.22}$$

where $K_\gamma(\omega)$ is the modified Bessel Function of the third kind with index $\gamma$, then the recursive formula for the Poisson-Generalized Inverse Gaussian distribution becomes

$$(2\eta + \omega) x(x+1) f(x+1) = 2\eta(x+\gamma) xf(x) + \omega\eta^2 f(x-1); x = 0, 1, 2, \ldots \tag{3.23}$$

and

$$f(-1) = 0$$

(Willmot & Panjer, 1987)

**Corollary 1**

When $\omega = \mu\beta^{-1}$, $\eta = \mu$, $x = n - 1$ and $f(x+1) = p_n$, then

$$\mu(1+2\beta)\, n(n-1)\, p_n = 2\beta(\eta+\gamma-1)(n-1)\, p_{n-1} + \mu^2 p_{n-2}; n = 2, 3, 4, \ldots$$

Further when $\gamma = -\frac{1}{2}$, then we have

$$\mu(1+2\beta)\, n(n-1)\, p_n = 2\beta\left(\eta - \frac{3}{2}\right)(n-1)\, p_{n-1} + \mu^2 p_{n-2}; n = 2, 3, 4, \ldots$$

with initial conditions

$$p_1 = \mu(1+2\beta)^{-\frac{1}{2}}\, p_0$$

where

$$p_0 = \exp\left\{-\frac{\mu}{\beta}\left[(1+2\beta)^{\frac{1}{2}} - 1\right]\right\}$$

### 3.6.4 Poisson-Exponential with One parameter

If the distribution for the exponential with one parameter is given by

$$g(\lambda) = \mu e^{-\mu\lambda}; \lambda > 0$$

then the recursive formula for the Poisson-Exponential with one parameter distribution becomes

$$f(x+1) = \left(\frac{1}{1+\mu}\right) f(x); x = 0, 1, 2, \ldots \tag{3.24}$$

Proof

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\mu e^{-\mu\lambda} d\lambda \\ &= \frac{\mu}{x!}\int_0^\infty e^{-(1+\mu)\lambda}\lambda^x d\lambda \end{aligned}$$

$$\frac{x!}{\mu} f(x) = \int_0^\infty e^{-(1+\mu)\lambda}\lambda^x d\lambda$$

Now,

$$I_x = \frac{x!}{\mu} f(x)$$

*and*

$$I_x = \int_0^\infty e^{-(1+\mu)\lambda}\lambda^x d\lambda$$

Let

$$u = e^{-(1+\mu)\lambda} \text{ and } dv = \lambda^x$$

$$du = -(1+\mu) e^{-(1+\mu)\lambda} d\lambda \text{ and } v = \frac{\lambda^{x+1}}{x+1}$$

Then

$$\begin{aligned} I_x &= \int_0^\infty \frac{\lambda^{x+1}}{x+1}(1+\mu) e^{-(1+\mu)\lambda} d\lambda \\ &= \frac{1+\mu}{x+1}\int_0^\infty \lambda^{x+1} e^{-(1+\mu)\lambda} d\lambda \end{aligned}$$

$$I_x = \left(\frac{1+\mu}{x+1}\right) I_{x+1}$$

$$I_{x+1} = \left(\frac{x+1}{1+\mu}\right) I_x$$

Now

$$\frac{(x+1)!}{\mu} f(x+1) = \left(\frac{x+1}{1+\mu}\right) \frac{x!}{\mu} f(x)$$

Therefore,

$$f(x+1) = \left(\frac{1}{1+\mu}\right) f(x); x = 0, 1, 2, ...$$

Verification
From the closed form of Poisson-Exponential distribution, given by (2.3),

$$f(x) = \left(\frac{\mu}{1+\mu}\right)\left(\frac{1}{1+\mu}\right)^x; x = 0, 1, 2, ...$$

Now

$$f(x+1) = \left(\frac{\mu}{1+\mu}\right)\left(\frac{1}{1+\mu}\right)^{x+1}; x = 0, 1, 2, ...$$

Therefore

$$\frac{f(x+1)}{f(x)} = \left(\frac{1}{1+\mu}\right)$$

Therefore,

$$f(x+1) = \left(\frac{1}{1+\mu}\right) f(x); x = 0, 1, 2, ...$$

which is the same as equation (3.24)

### 3.6.5 Poisson-Gamma with One parameter

If the distribution of Gamma with one parameter is given by

$$g(\lambda) = \frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}; \lambda > 0, \alpha > 0$$

then the recursive for Poisson-Gamma with one parameter becomes

$$f(x+1) = \frac{1}{2}\left(\frac{x+\alpha}{x+1}\right) f(x); x = 0, 1, 2, ... \tag{3.25}$$

Proof

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}d\lambda \\ &= \frac{1}{x!\Gamma(\alpha)}\int_0^\infty e^{-2\lambda}\lambda^{x+\alpha-1}d\lambda \end{aligned}$$

$$x!\Gamma(\alpha) f(x) = \int_0^\infty e^{-2\lambda}\lambda^{x+\alpha-1}d\lambda$$

$$\begin{aligned} I_x &= x!\Gamma(\alpha) f(x) \\ I_x &= \int_0^\infty e^{-2\lambda}\lambda^{x+\alpha-1}d\lambda \end{aligned}$$

Using integration by parts, let

$$u = e^{-2\lambda} \text{ and } dv = \lambda^{x+\alpha-1}$$

$$du = -2e^{-2\lambda}d\lambda, \quad v = \frac{\lambda^{x+\alpha}}{x+\alpha}$$

Therefore

$$\begin{aligned} I_x &= \int_0^\infty 2e^{-2\lambda}\frac{\lambda^{x+\alpha}}{x+\alpha}d\lambda \\ &= \frac{2}{x+\alpha}\int_0^\infty e^{-2\lambda}\lambda^{x+\alpha}d\lambda \end{aligned}$$

Now,

$$I_x = \left(\frac{2}{x+\alpha}\right) I_{x+1}$$

then

$$I_{x+1} = \left(\frac{x+\alpha}{2}\right) I_x$$

Now

$$(x+1)!\Gamma(\alpha) f(x+1) = \left(\frac{x+\alpha}{2}\right) x!\Gamma(\alpha) f(x)$$

$$(x+1) f(x+1) = \left(\frac{x+\alpha}{2}\right) f(x)$$

Therefore

$$f(x+1) = \frac{1}{2}\left(\frac{x+\alpha}{x+1}\right) f(x); x = 0, 1, 2, ...$$

Verification
From equation (2.5)

$$f(x) = \binom{x+\alpha-1}{x} \left(\frac{1}{2}\right)^{\alpha} \left(\frac{1}{2}\right)^{x}; x = 0, 1, 2, ...$$

$$f(x+1) = \binom{x+\alpha}{x+1} \left(\frac{1}{2}\right)^{\alpha} \left(\frac{1}{2}\right)^{x+1}; x = 0, 1, 2, ...$$

Now

$$\begin{aligned} \frac{f(x+1)}{f(x)} &= \frac{1}{2} \frac{\binom{x+\alpha}{x+1}}{\binom{x+\alpha-1}{x}} \\ &= \frac{1}{2} \frac{(x+\alpha)!\,(\alpha-1)!x!}{(\alpha-1)!\,(x+1)!\,(x+\alpha-1)!} \\ &= \frac{1}{2}\left(\frac{x+\alpha}{x+1}\right) \end{aligned}$$

Therefore,

$$f(x+1) = \frac{1}{2}\left(\frac{x+\alpha}{x+1}\right) f(x); x = 0, 1, 2, ...$$

which is the same as equation (3.25).

### 3.6.6 Poisson-Gamma with Two parameters

If the pdf of a Gamma distribution with two parameters is given by

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta\lambda} \lambda^{\alpha-1}; \lambda > 0, \alpha > 0, \beta > 0$$

then the recursive formula for Poisson-Gamma with two parameters becomes

$$f(x+1) = \left[\frac{(x+\alpha)}{(x+1)(1+\beta)}\right] f(x); x = 0, 1, 2, ... \tag{3.26}$$

(Panjer & Willmot, 1992)

Proof

$$\begin{aligned} f(x) &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta\lambda} \lambda^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha}}{x!\Gamma(\alpha)} \int_0^{\infty} e^{-(1+\beta)\lambda} \lambda^{x+\alpha-1} d\lambda \end{aligned}$$

Then

$$\frac{x!\Gamma(\alpha)}{\beta^{\alpha}} f(x) = \int_0^{\infty} e^{-(1+\beta)\lambda} \lambda^{x+\alpha-1} d\lambda$$

Now

$$I_x = \frac{x!\Gamma(\alpha)}{\beta^{\alpha}} f(x)$$

$$I_x = \int_0^{\infty} e^{-(1+\beta)\lambda} \lambda^{x+\alpha-1} d\lambda$$

Using integration by parts, let

$$u = e^{-(1+\beta)\lambda} \text{ and } dv = \lambda^{x+\alpha-1}$$

$$du = -(1+\beta) e^{-(1+\beta)\lambda} d\lambda \text{ and } v = \frac{\lambda^{x+\alpha}}{x+\alpha}$$

Therefore,

$$\begin{aligned} I_x &= \int_0^{\infty} (1+\beta) e^{-(1+\beta)\lambda} \frac{\lambda^{x+\alpha}}{x+\alpha} d\lambda \\ &= \frac{1+\beta}{x+\alpha} \int_0^{\infty} e^{-(1+\beta)\lambda} \lambda^{x+\alpha} d\lambda \end{aligned}$$

Now

$$I_x = \left(\frac{1+\beta}{x+\alpha}\right) I_{x+1}$$

$$I_{x+1} = \left(\frac{x+\alpha}{1+\beta}\right) I_x$$

$$\frac{(x+1)!\Gamma(\alpha)}{\beta^\alpha} f(x+1) = \left(\frac{x+\alpha}{1+\beta}\right) \frac{x!\Gamma(\alpha)}{\beta^\alpha} f(x)$$

$$(x+1) f(x+1) = \left(\frac{x+\alpha}{1+\beta}\right) f(x)$$

Therefore,

$$f(x+1) = \left[\frac{(x+\alpha)}{(x+1)(1+\beta)}\right] f(x); x = 0, 1, 2, ...$$

Verification
From equation (2.7)

$$f(x) = \binom{x+\alpha-1}{x} \left(\frac{\beta}{1+\beta}\right)^\alpha \left(\frac{1}{1+\beta}\right)^x; x = 0, 1, 2, ...$$

$$f(x+1) = \binom{x+\alpha}{x+1} \left(\frac{\beta}{1+\beta}\right)^\alpha \left(\frac{1}{1+\beta}\right)^{x+1}; x = 0, 1, 2, ...$$

Then

$$\begin{aligned} \frac{f(x+1)}{f(x)} &= \frac{\binom{x+\alpha}{x+1}}{\binom{x+\alpha-1}{x}} \left(\frac{1}{1+\beta}\right) \\ &= \left(\frac{x+\alpha}{x+1}\right) \left(\frac{1}{1+\beta}\right) \end{aligned}$$

Therefore,

$$f(x+1) = \left(\frac{x+\alpha}{x+1}\right)\left(\frac{1}{1+\beta}\right) f(x); x = 0, 1, 2, ...$$

which is the same as equation (3.26).

### 3.6.7 Poisson-Gamma with four parameters Distribution (Poisson-Generalized Gamma Distribution)

If the generalized gamma mixing distribution is given by

$$g(\lambda) = \frac{\alpha^{m-\delta} e^{-\alpha\lambda} \lambda^{m-1}}{\Gamma_\delta(m, \alpha n)(\lambda + n)^\delta}; \lambda \geq 0, m > 0, \alpha > 0, n > 0, \delta \geq 0 \qquad (3.27)$$

then the recursive formula for the Poisson - Generalized Gamma distribution is

$$(\alpha+1)x(x+1)f(x+1) = [x + m - \delta - (\alpha+1)n]xf(x) + (x+m-1)nf(x-1); x \geq 1$$

and

$$f(0) = \left(\frac{\alpha}{1+\alpha}\right)^{m-\delta} \frac{\Gamma_\delta(m, (1+\alpha)n)}{\Gamma_\delta(m, \alpha n)}$$

Proof

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda$$

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \frac{\alpha^{m-\delta}}{\Gamma_\delta(m,\alpha n)} \frac{e^{-\alpha\lambda}\lambda^{m-1}}{(\lambda+n)^\delta} d\lambda$$

$$f(x) = \frac{\alpha^{m-\delta}}{x!\Gamma_\delta(m,\alpha n)} \int_0^\infty \frac{e^{-\lambda(\alpha+1)}\lambda^{x+m-1}}{(\lambda+n)^\delta} d\lambda$$

Therefore,

$$\Gamma_\delta(m,\alpha n)\,\alpha^{\delta-m} x! f(x) = \int_0^\infty \frac{e^{-\lambda(\alpha+1)}\lambda^{x+m-1}}{(\lambda+n)^\delta} d\lambda$$

Put

$$\lambda = nt \implies d\lambda = ndt$$

Therefore,

$$\begin{aligned}\int_0^\infty \frac{e^{-\lambda(\alpha+1)}\lambda^{x+m-1}}{(\lambda+n)^\delta} d\lambda &= \int_0^\infty \frac{e^{-(\alpha+1)nt}(nt)^{x+m-1}}{n^\delta(1+t)^\delta} ndt \\ &= n^{x+m-\delta} \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-1}}{(1+t)^\delta} dt\end{aligned}$$

Therefore,

$$\Gamma_\delta(m,\alpha n)\,\alpha^{\delta-m} x! n^{\delta-m-x} f(x) = \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-1}}{(1+t)^\delta} dt$$

$$\begin{aligned}\Gamma_\delta(m,\alpha n)(\alpha n)^{\delta-m} \frac{x!}{n^x} f(x) &= \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-1}}{(1+t)^\delta} dt \\ &\approx I_x\end{aligned}$$

Consider

$$I_x = \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-1}}{(1+t)^\delta} dt$$

Put

$$\begin{aligned} u &= e^{-(\alpha+1)nt} t^{x+m-1} \\ \Longrightarrow du &= \left[ -(\alpha+1) n e^{-(\alpha+1)nt} t^{x+m-1} + (x+m-1) e^{-(\alpha+1)nt} t^{x+m-2} \right] dt \end{aligned}$$

$$dv = (1+t)^{-\delta} \Longrightarrow v = \frac{(1+t)^{-\delta+1}}{-\delta+1}$$

Therefore,

$$I_x = |uv|_0^\infty - \int_0^\infty v du = 0 - \int_0^\infty \frac{(1+t)^{-\delta+1}}{-\delta+1} du$$

Therefore,

$$\begin{aligned} I_x &= \int_0^\infty \frac{(1+t)^{-\delta+1}}{\delta-1} \left[ \begin{array}{c} -(\alpha+1) n e^{-(\alpha+1)nt} t^{x+m-1} \\ +(x+m-1) e^{-(\alpha+1)nt} t^{x+m-2} \end{array} \right] dt \\ &= \frac{-(\alpha+1) n}{\delta-1} \int_0^\infty (1+t)^{-\delta+1} e^{-(\alpha+1)nt} t^{x+m-1} dt \\ &\quad + \frac{x+m-1}{\delta-1} \int_0^\infty (1+t)^{-\delta+1} e^{-(\alpha+1)nt} t^{x+m-2} dt \\ &= \frac{-(\alpha+1) n}{\delta-1} \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-1}}{(1+t)^{\delta-1}} dt \\ &\quad + \frac{x+m-1}{\delta-1} \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-2}}{(1+t)^{\delta-1}} dt \\ &= \frac{-(\alpha+1) n}{\delta-1} \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-1}}{(1+t)^{\delta-1}} \frac{(1+t)}{(1+t)} dt \\ &\quad + \frac{x+m-1}{\delta-1} \int_0^\infty \frac{e^{-(\alpha+1)nt} t^{x+m-2}}{(1+t)^{\delta-1}} \frac{(1+t)}{(1+t)} dt \end{aligned}$$

Therefore,

$$I_x = \frac{-(\alpha+1)n}{\delta-1}\left\{\int_0^\infty \frac{\left[e^{-(\alpha+1)nt}t^{x+m-1}+e^{-(\alpha+1)nt}t^{x+m}\right]}{(1+t)^\delta}dt\right\}$$
$$+\frac{x+m-1}{\delta-1}\left\{\int_0^\infty \frac{\left[e^{-(\alpha+1)nt}t^{x+m-2}+e^{-(\alpha+1)nt}t^{x+m-1}\right]}{(1+t)^\delta}dt\right\}$$

Therefore,

$$I_x = \frac{-(\alpha+1)n}{\delta-1}\left[I_x + I_{x+1}\right] + \frac{x+m-1}{\delta-1}\left[I_{x-1}+I_x\right]$$

Therefore,

$$(\delta-1)I_x = -(\alpha+1)nI_x - (\alpha+1)nI_{x+1} + (x+m-1)I_{x-1}$$
$$+(x+m-1)I_x$$

Therefore,

$$(\alpha+1)nI_{x+1} = \left[x+m-\delta-(\alpha+1)n\right]I_x + (x+m-1)I_{x-1}$$

Using

$$I_x = \Gamma_\delta(m,\alpha n)(\alpha n)^{\delta-m}\frac{x!}{n^x}f(x)$$

we have

$$(\alpha+1)n\frac{(x+1)!}{n^{x+1}}f(x+1) = \left[x+m-\delta-(\alpha+1)n\right]\frac{x!}{n^x}f(x)$$
$$+(x+m-1)\frac{(x-1)!}{n^{x-1}}f(x-1)$$

Therefore

$$\begin{aligned}\frac{(\alpha+1)\,nx\,(x+1)}{n^2}f\,(x+1) &= [x+m-\delta-(\alpha+1)\,n]\,\frac{x}{n}f\,(x)\\ &\quad +(x+m-1)\,f\,(x-1)\end{aligned}$$

$$(\alpha+1)\,x\,(x+1)\,f\,(x+1)=[x+m-\delta-(\alpha+1)\,n]\,xf\,(x)+(x+m-1)\,nf\,(x-1)\,;x\geq 1 \tag{3.28}$$

When $x=0$, we have

$$\Gamma_\delta\,(m,\alpha n)\,(\alpha n)^{\delta-m}\,\frac{0!}{n^0}f\,(0)=\int_0^\infty \frac{e^{-(\alpha+1)nt}t^{m-1}}{(1+t)^\delta}dt$$

Therefore

$$\Gamma_\delta\,(m,\alpha n)\,(\alpha n)^{\delta-m}\,f\,(0)=\int_0^\infty \frac{e^{-(\alpha+1)nt}t^{m-1}}{(1+t)^\delta}dt$$

But

$$\begin{aligned}\int_0^\infty \frac{e^{-(\alpha+1)nt}t^{m-1}}{(1+t)^\delta}dt &= \int_0^\infty \frac{e^{-(\alpha+1)nt}t^{m-1}}{(1+t)^{m-m+\delta-1+1}}dt\\ &= \int_0^\infty \frac{e^{-(\alpha+1)nt}t^{m-1}}{(1+t)^{m-(m-\delta+1)+1}}dt\\ &= \Gamma\,(m)\,\varphi\,[m,(m-\delta+1)\,;(\alpha+1)\,n]\end{aligned}$$

Therefore

$$f\,(0)=\frac{\Gamma\,(m)}{\Gamma_\delta\,(m,\alpha n)}\,(\alpha n)^{m-\delta}\,\varphi\,[m,(m-\delta+1)\,;(\alpha+1)\,n]$$

### 3.6.8 Poisson-Inverse Gamma Distribution

If the Inverse Gamma distribution is given by

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} \frac{e^{-\frac{\beta}{\lambda}}}{\lambda^{\alpha+1}}; \lambda > 0, \alpha > 0, \beta > 0 \tag{3.29}$$

then the recursive formula for the Poisson-Inverse Gamma distribution becomes

$$x(x+1) f(x+1) = (x-\alpha) x f(x) + \beta f(x-1); x = 0, 1, 2, ...$$

with

$$f(-1) = 0$$

which implies that, when $x = n-1$,and $f(x+1) = p_n$, then,

$$n(n-1) p_n = (n-1-\alpha)(n-1) p_{n-1} + \beta p_{n-2}; n = 2, 3, ...$$

Proof

$$\begin{aligned} f(x) &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\beta^{\alpha}}{\Gamma(\alpha)} \frac{e^{-\frac{\beta}{\lambda}}}{\lambda^{\alpha+1}} d\lambda \\ &= \frac{\beta^{\alpha}}{x!\Gamma(\alpha)} \int_0^{\infty} e^{-\left(\lambda+\frac{\beta}{\lambda}\right)} \lambda^{x-\alpha-1} d\lambda \end{aligned}$$

$$\frac{x!\Gamma(\alpha)}{\beta^{\alpha}} f(x) = \int_0^{\infty} e^{-\left(\lambda+\frac{\beta}{\lambda}\right)} \lambda^{x-\alpha-1} d\lambda$$

Now

$$I_x = \frac{x!\Gamma(\alpha)}{\beta^{\alpha}} f(x)$$

and

$$I_x = \int_0^{\infty} e^{-\left(\lambda+\frac{\beta}{\lambda}\right)} \lambda^{x-\alpha-1} d\lambda \qquad (*)$$

Using integration by parts to solve (*), let

$$u = e^{-\left(\lambda+\frac{\beta}{\lambda}\right)}$$

and

$$dv = \lambda^{x-\alpha-1}d\lambda$$

Now

$$du = -\left(1-\frac{\beta}{\lambda^2}\right)e^{-\left(\lambda+\frac{\beta}{\lambda}\right)}d\lambda$$

$$v = \frac{\lambda^{x-\alpha}}{x-\alpha}$$

Therefore

$$I_x = \int_0^\infty \left(1-\frac{\beta}{\lambda^2}\right)e^{-\left(\lambda+\frac{\beta}{\lambda}\right)}\frac{\lambda^{x-\alpha}}{x-\alpha}d\lambda$$

$$= \frac{1}{x-\alpha}\int_0^\infty e^{-\left(\lambda+\frac{\beta}{\lambda}\right)}\lambda^{x-\alpha}d\lambda - \frac{\beta}{x-\alpha}\int_0^\infty e^{-\left(\lambda+\frac{\beta}{\lambda}\right)}\lambda^{x-\alpha-2}d\lambda$$

$$I_x = \left(\frac{1}{x-\alpha}\right)I_{x+1} - \left(\frac{\beta}{x-\alpha}\right)I_{x-1}$$

$$I_{x+1} = (x-\alpha)\,I_x + \beta I_{x-1}$$

Now

$$\frac{(x+1)!\Gamma(\alpha)}{\beta^\alpha}f(x+1) = (x-\alpha)\frac{x!\Gamma(\alpha)}{\beta^\alpha}f(x) + \beta\frac{(x-1)!\Gamma(\alpha)}{\beta^\alpha}f(x-1)$$

Therefore

$$x(x+1)\,f(x+1) = x(x-\alpha)\,f(x) + \beta f(x-1). \qquad (3.30)$$

which is the recursive relation of Poisson - Inverse Gamma distribution as given by Willmot (1993).

### 3.6.9 Poisson-Beta Distribution

If the Beta mixing distribution is given by

$$g(\lambda) = \frac{\lambda^{\alpha-1}(1-\lambda)^{\beta-1}}{B(\alpha,\beta)}; 0 \leq \lambda \leq 1 \tag{3.31}$$

then the recursive formula for the Poisson-Beta distribution is

$$x(x+1)f(x+1) = x(x+\alpha+\beta)f(x) - (x+\alpha-1)f(x-1);$$

for

$x = 0, 1, 2, \ldots$

with

$$f(-1) = 0$$

implying that

$$n(n-1)p_n = (n-1+\alpha+\beta)(n-1)p_{n-1} - (n+\alpha-2)p_{n-2};$$

for

$n = 2, 3, 4, \ldots$

when

$$x = n-1 \text{ and } f(x+1) = p_n$$

Proof

$$f(x) = \int_0^1 \frac{e^{-\lambda}\lambda^x}{x!} \frac{\lambda^{\alpha-1}(1-\lambda)^{\beta-1}}{B(\alpha,\beta)} d\lambda$$

$$= \frac{1}{x!B(\alpha,\beta)} \int_0^1 e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)^{\beta-1} d\lambda$$

Therefore

$$B(\alpha,\beta)\,x!f(x)=\int_0^1 e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)^{\beta-1}\,d\lambda \approxeq I_x(\alpha,\beta)$$

Let

$$\begin{aligned} u &= e^{-\lambda}\lambda^{x+\alpha-1} \\ &\text{and} \\ dv &= (1-\lambda)^{\beta-1}\,d\lambda \end{aligned}$$

Therefore

$$\begin{aligned} du &= \left[-e^{-\lambda}\lambda^{x+\alpha-1}+(x+\alpha-1)\,e^{-\lambda}\lambda^{x+\alpha-2}\right]d\lambda \\ &\text{and} \\ v &= \frac{(1-\lambda)^{\beta}}{-\beta} \end{aligned}$$

Therefore

$$\begin{aligned} I_x(\alpha,\beta) &= \left|\frac{(1-\lambda)^{\beta}}{-\beta}e^{-\lambda}\lambda^{x+\alpha-1}\right|_0^1 \\ &\quad -\int_0^1 \frac{(1-\lambda)^{\beta}}{-\beta}\left\{-e^{-\lambda}\lambda^{x+\alpha-1}+(x+\alpha-1)\,e^{-\lambda}\lambda^{x+\alpha-2}\right\}d\lambda \\ &= 0+\int_0^1 -\frac{1}{\beta}e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)^{\beta}\,d\lambda \\ &\quad +\frac{(x+\alpha-1)}{\beta}\int_0^1 e^{-\lambda}\lambda^{x+\alpha-2}(1-\lambda)^{\beta}\,d\lambda \\ &= -\frac{1}{\beta}\int_0^1 e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)^{\beta}\,d\lambda \\ &\quad +\frac{(x+\alpha-1)}{\beta}\int_0^1 e^{-\lambda}\lambda^{x+\alpha-2}(1-\lambda)^{\beta}\,d\lambda \end{aligned}$$

Therefore

$$
\begin{aligned}
I_x(\alpha,\beta) &= -\frac{1}{\beta}\int_0^1 e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)(1-\lambda)^{\beta-1}\,d\lambda \\
&\quad +\frac{(x+\alpha-1)}{\beta}\int_0^1 e^{-\lambda}\lambda^{x+\alpha-2}(1-\lambda)(1-\lambda)^{\beta-1}\,d\lambda \\
&= -\frac{1}{\beta}\left\{\int_0^1 e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)^{\beta-1}\,d\lambda - \int_0^1 e^{-\lambda}\lambda^{x+\alpha}(1-\lambda)^{\beta-1}\,d\lambda\right\} \\
&\quad +\frac{(x+\alpha-1)}{\beta}\left\{\int_0^1 e^{-\lambda}\lambda^{x+\alpha-2}(1-\lambda)^{\beta-1}\,d\lambda - \int_0^1 e^{-\lambda}\lambda^{x+\alpha-1}(1-\lambda)^{\beta-1}\,d\lambda\right\} \\
&= -\frac{1}{\beta}\{I_x(\alpha,\beta) - I_{x+1}(\alpha,\beta)\} + \frac{(x+\alpha-1)}{\beta}\{I_{x-1}(\alpha,\beta) - I_x(\alpha,\beta)\}
\end{aligned}
$$

therefore

$$
\begin{aligned}
\beta I_x(\alpha,\beta) &= -I_x(\alpha,\beta) + I_{x+1}(\alpha,\beta) + (x+\alpha-1)I_{x-1}(\alpha,\beta) \\
&\quad -(x+\alpha-1)I_x(\alpha,\beta)
\end{aligned}
$$

therefore

$$
I_{x+1}(\alpha,\beta) = (x+\alpha+\beta)I_x(\alpha,\beta) - (x+\alpha-1)I_{x-1}(\alpha,\beta)
$$

$$
(x+1)!f(x+1) = (x+\alpha+\beta)x!f(x) - (x+\alpha-1)(x-1)!f(x-1)
$$

$$
\begin{aligned}
(x+1)xf(x+1) &= (x+\alpha+\beta)xf(x) \\
&\quad -(x+\alpha-1)f(x-1); \qquad (3.32)\\
\text{for } x &= 0,1,2,... \\
\text{with } f(-1) &= 0
\end{aligned}
$$

implying that

$$
\begin{aligned}
n(n-1)p_n &= (n+\alpha+\beta-1)(n-1)p_{n-1} - (n+\alpha-2)p_{n-2}; \\
\text{for } n &= 2,3,4,... \\
&\text{where} \\
x &= n-1 \text{ and } f(x+1) = p_n
\end{aligned}
$$

### 3.6.10 Poisson - Inverted Beta Distribution

If the Inverted Beta mixing pdf is given by

$$g(\lambda) = \frac{\lambda^{\alpha-1}}{B(\alpha,\beta)(1+\lambda)^{\alpha+\beta}}; \lambda > 0, \alpha > 0, \beta > 0 \tag{3.33}$$

then the recursive formula for the Poisson-Inverted Beta distribution is given by

$$x(x+1)f(x+1) = x(x-\beta-1)f(x) + (x+\alpha-1)f(x-1);$$

for

$$x = 0, 1, 2, \ldots$$

with

$$f(-1) = 0$$

implying that

$$n(n-1)p_n = (n-\beta-2)(n-1)p_{n-1} + (n+\alpha-2)p_{n-2}; n = 2, 3, 4, \ldots$$

Proof

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \frac{\lambda^{\alpha-1}}{B(\alpha,\beta)(1+\lambda)^{\alpha+\beta}} d\lambda$$

$$= \frac{1}{x!B(\alpha,\beta)} \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}} d\lambda$$

$$x!B(\alpha,\beta)f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}} d\lambda$$

Now

$$I_x = x!B(\alpha,\beta)f(x)$$

and

$$I_x = \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}} d\lambda$$

Using integration by parts, let

$$\begin{aligned} u &= e^{-\lambda}\lambda^{x+\alpha-1} \\ &\text{and} \\ dv &= (1+\lambda)^{-(\alpha+\beta)}\,d\lambda \end{aligned}$$

Then

$$\begin{aligned} du &= (x+\alpha-1)\,e^{-\lambda}\lambda^{x+\alpha-2} - e^{-\lambda}\lambda^{x+\alpha-1}d\lambda \\ &\text{and} \\ v &= \frac{(1+\lambda)^{-(\alpha+\beta-1)}}{-(\alpha+\beta-1)} \end{aligned}$$

Therefore

$$\begin{aligned} I_x &= \frac{1}{(\alpha+\beta-1)}\int_0^\infty \left[\frac{(x+\alpha-1)\,e^{-\lambda}\lambda^{x+\alpha-2}}{(1+\lambda)^{\alpha+\beta-1}} - \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta-1}}\right]d\lambda \\ &= \frac{(x+\alpha-1)}{(\alpha+\beta-1)}\int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-2}}{(1+\lambda)^{\alpha+\beta-1}}\frac{(1+\lambda)}{(1+\lambda)}d\lambda \\ &\quad -\frac{1}{(\alpha+\beta-1)}\int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta-1}}\frac{(1+\lambda)}{(1+\lambda)}d\lambda \\ &= \frac{(x+\alpha-1)}{(\alpha+\beta-1)}\left\{\int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}}d\lambda + \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-2}}{(1+\lambda)^{\alpha+\beta}}d\lambda\right\} \\ &\quad -\frac{1}{(\alpha+\beta-1)}\left\{\int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha}}{(1+\lambda)^{\alpha+\beta}}d\lambda + \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}}d\lambda\right\} \end{aligned}$$

$$I_x = \frac{(x+\alpha-1)}{(\alpha+\beta-1)}(I_x + I_{x-1}) - \frac{1}{(\alpha+\beta-1)}(I_{x+1} + I_x)$$

$$(\alpha+\beta-1)\,I_x = (x+\alpha-1)\,(I_x + I_{x-1}) - (I_{x+1} + I_x)$$

$$(\beta-x+1)\,I_x = (x+\alpha-1)\,I_{x-1} - I_{x+1}$$

$$I_{x+1} = (x-\beta-1)\,I_x + (x+\alpha-1)\,I_{x-1}$$

But

$$I_x = x!B(\alpha,\beta)f(x)$$

Therefore

$$(x+1)!B(\alpha,\beta)f(x+1) = (x-\beta-1)x!B(\alpha,\beta)f(x)+(x+\alpha-1)(x-1)!B(\alpha,\beta)f(x-1)$$

Then

$$x(x+1)f(x+1) = x(x-\beta-1)f(x)+(x+\alpha-1)f(x-1) \qquad (3.34)$$

is the recursive relation for Poisson - Inverted Beta Distribution.

### 3.6.11 Poisson - Scaled Beta Distribution

If the Scaled Beta mixing distribution is given by

$$g(\lambda) = \frac{\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{B(\alpha,\beta)\mu^{\alpha+\beta-1}}; 0 \le \lambda \le \mu \qquad (3.35)$$

then the recursive formula for the Poisson-Scaled Beta distribution is

$$x(x+1)f(x+1) = x(x+\alpha+\beta+\mu-1)f(x)-\mu(x+\alpha-1)f(x-1);$$

for

$x = 0, 1, 2, ...$

with

$$f(-1) = 0$$

implying that

$$n(n-1)p_n = (n+\alpha+\beta+\mu-2)(n-1)p_{n-1}-\mu(n+\alpha-2)p_{n-2}; n = 2, 3, ...$$

(Willmot & Panjer, 1987)

Proof

$$
\begin{aligned}
f(x) &= \int_0^{\mu} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{B(\alpha,\beta)\,\mu^{\alpha+\beta-1}} g(\lambda) \\
&= \frac{1}{x!B(\alpha,\beta)} \int_0^{\mu} \frac{e^{-\lambda}\lambda^{x+\alpha-1}(\mu-\lambda)^{\beta-1}}{\mu^{\alpha+\beta-1}} d\lambda \\
&= \frac{1}{x!B(\alpha,\beta)} \int_0^{\mu} e^{-\lambda} \frac{\lambda^{x+\alpha-1}}{\mu^{\alpha}} \left(1-\frac{\lambda}{\mu}\right)^{\beta-1} d\lambda \\
&= \frac{1}{x!B(\alpha,\beta)} \int_0^{\mu} \frac{e^{-\lambda}}{\mu} \frac{\lambda^{x+\alpha-1}}{\mu^{\alpha-1}} \left(1-\frac{\lambda}{\mu}\right)^{\beta-1} d\lambda \\
&= \frac{1}{x!B(\alpha,\beta)} \int_0^{\mu} \frac{e^{-\lambda}}{\mu} \mu^x \left(\frac{\lambda}{\mu}\right)^{x+\alpha-1} \left(1-\frac{\lambda}{\mu}\right)^{\beta-1} d\lambda \\
&= \frac{\mu^x}{x!\mu B(\alpha,\beta)} \int_0^{\mu} e^{-\lambda} \left(\frac{\lambda}{\mu}\right)^{x+\alpha-1} \left(1-\frac{\lambda}{\mu}\right)^{\beta-1} d\lambda
\end{aligned}
$$

Put

$$
\frac{\lambda}{\mu} = z \Longrightarrow \lambda = \mu z \text{ and } d\lambda = \mu dz
$$

Therefore

$$
\begin{aligned}
f(x) &= \frac{\mu^x}{x!B(\alpha,\beta)} \int_0^1 \frac{e^{-\mu z}}{\mu} z^{x+\alpha-1}(1-z)^{\beta-1}\,\mu dz \\
&= \frac{\mu^x}{x!B(\alpha,\beta)} \int_0^1 e^{-\mu z} z^{x+\alpha-1}(1-z)^{\beta-1}\,dz
\end{aligned}
$$

Therefore

$$
B(\alpha,\beta)\frac{x!}{\mu^x} f(x) = \int_0^1 e^{-\mu z} z^{x+\alpha-1}(1-z)^{\beta-1}\,dz \approxeq I_x(\alpha,\beta)
$$

Put

$$
u = e^{-\mu z} z^{x+\alpha-1} \text{ and } dv = (1-z)^{\beta-1}\,dz
$$

Therefore

$$\begin{aligned} du &= -\mu e^{-\mu z} z^{x+\alpha-1} + e^{-\mu z}(x+\alpha-1) z^{x+\alpha-2} \\ &\text{and} \\ v &= \frac{(1-z)^{\beta}}{-\beta} \end{aligned}$$

Therefore

$$\begin{aligned} I_x(\alpha,\beta) &= \left| e^{-\mu z} z^{x+\alpha-1} \frac{(1-z)^{\beta}}{-\beta} \right|_0^1 \\ &\quad + \int_0^1 \frac{(1-z)^{\beta}}{-\beta} \left\{ -\mu e^{-\mu z} z^{x+\alpha-1} + e^{-\mu z}(x+\alpha-1) z^{x+\alpha-2} \right\} dz \\ &= 0 + \int_0^1 -\frac{\mu}{\beta} e^{-\mu z} z^{x+\alpha-1} (1-z)^{\beta} dz \\ &\quad + \frac{(x+\alpha-1)}{\beta} \int_0^1 e^{-\mu z} z^{x+\alpha-2} (1-z)^{\beta} dz \\ &= -\frac{\mu}{\beta} \int_0^1 e^{-\mu z} z^{x+\alpha-1} (1-z)(1-z)^{\beta-1} dz \\ &\quad + \frac{(x+\alpha-1)}{\beta} \int_0^1 e^{-\mu z} z^{x+\alpha-2} (1-z)(1-z)^{\beta-1} dz \\ &= -\frac{\mu}{\beta} \left\{ \int_0^1 e^{-\mu z} z^{x+\alpha-1} (1-z)^{\beta-1} dz - \int_0^1 e^{-\mu z} z^{x+\alpha} (1-z)^{\beta-1} dz \right\} \\ &\quad + \frac{(x+\alpha-1)}{\beta} \left\{ \int_0^1 e^{-\mu z} z^{x+\alpha-2} (1-z)^{\beta-1} dz - \int_0^1 e^{-\mu z} z^{x+\alpha-1} (1-z)^{\beta-1} dz \right\} \end{aligned}$$

Therefore

$$\begin{aligned} \beta I_x(\alpha,\beta) &= -\mu \{ I_x(\alpha,\beta) - I_{x+1}(\alpha,\beta) \} \\ &\quad + (x+\alpha-1) \{ I_{x-1}(\alpha,\beta) - I_x(\alpha,\beta) \} \\ &= -\mu I_x(\alpha,\beta) + \mu I_{x+1}(\alpha,\beta) + (x+\alpha-1) I_{x-1}(\alpha,\beta) \\ &\quad - (x+\alpha-1) I_x(\alpha,\beta) \end{aligned}$$

Therefore

$$\begin{aligned}\mu I_{x+1}(\alpha,\beta) &= [\beta+\mu+x+\alpha-1]\, I_x(\alpha,\beta) \\ &\quad -(x+\alpha-1)\, I_{x-1}(\alpha,\beta)\end{aligned}$$

$$\begin{aligned}\mu\frac{(x+1)!}{\mu^{x+1}}f(x+1) &= (x+\alpha+\beta+\mu-1)\frac{x!}{\mu^x}f(x) \\ &\quad -(x+\alpha-1)\frac{(x-1)!}{\mu^{x-1}}f(x-1)\end{aligned}$$

Therefore

$$\frac{x(x+1)f(x+1)}{\mu^x} = \frac{(x+\alpha+\beta+\mu-1)xf(x)}{\mu^x} - \frac{(x+\alpha-1)f(x-1)}{\mu^{x-1}}$$

$$\begin{aligned}x(x+1)f(x+1) &= (x+\alpha+\beta+\mu-1)xf(x) \\ &\quad -\mu(x+\alpha-1)f(x-1); \\ &\quad \text{for} \\ x &= 0,1,2,... \\ &\quad \text{with} \\ f(-1) &= 0\end{aligned} \tag{3.36}$$

implying that

$$\begin{aligned}n(n-1)p_n &= (n+\alpha+\beta+\mu-2)(n-1)p_{n-1} - \mu(n+\alpha-2)p_{n-2}; \\ &\quad \text{for} \\ n &= 2,3,4,...\end{aligned}$$

### 3.6.12 Poisson - Lomax Distribution

If the Lomax mixing distribution is given by

$$g(\lambda) = \frac{ak^a}{(\lambda+k)^{a+1}}; \lambda > 0 \tag{3.37}$$

then the recursive formula for the Poisson - Lomax distribution becomes:

$$(x+1) f(x+1) = (x-k-a) f(x) + k f(x-1); x = 0, 1, 2, ...$$

with

$$f(-1) = 0$$

which implies that

$$p_n = \left[1 - \left(\frac{a+k+1}{n}\right)\right] p_{n-1} + \frac{k}{n} p_{n-2}; n = 2, 3, 4, ...$$

with the boundary condition

$$p_1 = a - (a+k) p_0$$

Proof

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \frac{ak^a}{(\lambda+k)^{a+1}} d\lambda \\ &= \frac{ak^a}{x!} \int_0^\infty \frac{e^{-\lambda}\lambda^x}{(\lambda+k)^{a+1}} d\lambda \end{aligned}$$

Now, let

$$\lambda = kt \Longrightarrow d\lambda = kdt$$

$$\begin{aligned} f(x) &= \frac{ak^a}{x!} \int_0^\infty \frac{e^{-kt}(kt)^x}{(kt+k)^{a+1}} kdt \\ &= \frac{ak^x}{x!} \int_0^\infty \frac{e^{-kt}t^x}{(1+t)^{a+1}} dt \end{aligned}$$

$$\frac{x!}{ak^x} f(x) = \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^{a+1}} dt$$

Now

$$I_x = \frac{x!}{ak^x} f(x)$$

and

$$I_x = \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^{a+1}} dt$$

Using integration by parts, let

$$u = e^{-kt} t^x$$

and

$$dv = (1+t)^{-(a+1)} dt$$

then

$$du = xe^{-kt} t^{x-1} - ke^{-kt} t^x dt$$

and

$$v = \frac{(1+t)^{-a}}{-a}$$

Now,

$$I_x = \frac{1}{a} \left\{ \int_0^\infty \frac{xe^{-kt} t^{x-1}}{(1+t)^a} dt - \int_0^\infty \frac{ke^{-kt} t^x}{(1+t)^a} dt \right\}$$

$$= \frac{x}{a} \int_0^\infty \frac{e^{-kt} t^{x-1}}{(1+t)^a} dt - \frac{k}{a} \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^a} dt$$

$$= \frac{x}{a} \int_0^\infty \frac{e^{-kt} t^{x-1}}{(1+t)^a} \frac{(1+t)}{(1+t)} dt - \frac{k}{a} \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^a} \frac{(1+t)}{(1+t)} dt$$

$$= \frac{x}{a} \int_0^\infty \frac{e^{-kt} t^{x-1}}{(1+t)^{a+1}} (1+t)\, dt - \frac{k}{a} \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^{a+1}} (1+t)\, dt$$

$$= \frac{x}{a} \left[ \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^{a+1}} dt + \int_0^\infty \frac{e^{-kt} t^{x-1}}{(1+t)^{a+1}} dt \right]$$

$$- \frac{k}{a} \left[ \int_0^\infty \frac{e^{-kt} t^{x+1}}{(1+t)^{a+1}} dt + \int_0^\infty \frac{e^{-kt} t^x}{(1+t)^{a+1}} dt \right]$$

$$I_x = \frac{x}{a}(I_x + I_{x-1}) - \frac{k}{a}(I_{x+1} + I_x)$$

$$\left(\frac{x-k-a}{a}\right) I_x = \left(\frac{x}{a}\right) I_{x-1} - \left(\frac{k}{a}\right) I_{x+1}$$

$$kI_{x+1} = (x-k-a) I_x + xI_{x-1}$$

But

$$I_x = \frac{x!}{ak^x} f(x)$$

then

$$k\frac{(x+1)!}{ak^{x+1}} f(x+1) = (x-k-a)\frac{x!}{ak^x} f(x) + x\frac{(x-1)!}{ak^{x-1}} f(x-1)$$

Therefore

$$(x+1) f(x+1) = (x-k-a) f(x) + kf(x-1) \tag{3.38}$$

is the recursive relation for Poisson - Pareto Distribution as given by Willmot (1993).

### 3.6.13 Poisson - Generalized Pareto Distribution

If the Generalized Pareto mixing distribution is given by

$$g(\lambda) = \frac{\mu^\alpha \lambda^{\beta-1}}{B(\alpha,\beta)(\mu+\lambda)^{\alpha+\beta}}; \lambda \geq 0, \alpha > 0, \beta > 0, \mu > 0 \tag{3.39}$$

then the recursive formula for the Poisson-Generalized Pareto distribution is given by

$$x(x+1) f(x+1) = x(x-\mu-\alpha) f(x) + \mu(x+\beta-1) f(x-1);$$

for

$x = 0, 1, 2, \ldots$

with

$$f(-1) = 0$$

implying that

$$n(n-1)p_n = (n-1-\mu-\alpha)(n-1)p_{n-1}+\mu(n-2+\beta)p_{n-2};$$

for

$n = 2, 3, 4, \ldots$

Proof

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\frac{\mu^\alpha \lambda^{\beta-1}}{B(\alpha,\beta)(\mu+\lambda)^{\alpha+\beta}}d\lambda$$

$$= \frac{\mu^\alpha}{x!B(\alpha,\beta)}\int_0^\infty \frac{e^{-\lambda}\lambda^{x+\beta-1}}{(\mu+\lambda)^{\alpha+\beta}}d\lambda$$

Let

$$\lambda = \mu t \Longrightarrow d\lambda = \mu dt$$

Now

$$f(x) = \frac{\mu^\alpha}{x!B(\alpha,\beta)}\int_0^\infty \frac{e^{-\mu t}(\mu t)^{x+\beta-1}}{(\mu+\mu t)^{\alpha+\beta}}\mu dt$$

$$= \frac{\mu^x}{x!B(\alpha,\beta)}\int_0^\infty \frac{e^{-\mu t}t^{x+\beta-1}}{(1+t)^{\alpha+\beta}}dt$$

$$\frac{x!B(\alpha,\beta)}{\mu^x}f(x) = \int_0^\infty \frac{e^{-\mu t}t^{x+\beta-1}}{(1+t)^{\alpha+\beta}}dt$$

Now

$$I_x = \frac{x!B(\alpha,\beta)}{\mu^x}f(x)$$

and

$$I_x = \int_0^\infty \frac{e^{-\mu t}t^{x+\beta-1}}{(1+t)^{\alpha+\beta}}dt$$

Using integration by parts, let

$$\begin{aligned} u &= e^{-\mu t} t^{x+\beta-1} \\ &\text{and} \\ dv &= (1+t)^{-(\alpha+\beta)}\, dt \end{aligned}$$

then

$$\begin{aligned} du &= \left[(\beta+x-1)\, e^{-\mu t} t^{x+\beta-2} - \mu e^{-\mu t} t^{x+\beta-1}\right] dt \\ &\text{and} \\ v &= \frac{(1+t)^{-(\alpha+\beta-1)}}{-(\alpha+\beta-1)} \end{aligned}$$

Therefore,

$$\begin{aligned} I_x &= \frac{1}{(\alpha+\beta-1)} \left\{ \int_0^\infty \frac{(\beta+x-1)\, e^{-\mu t} t^{x+\beta-2}}{(1+t)^{\alpha+\beta-1}} dt - \int_0^\infty \frac{\mu e^{-\mu t} t^{x+\beta-1}}{(1+t)^{\alpha+\beta-1}} dt \right\} \\ &= \frac{(\beta+x-1)}{(\alpha+\beta-1)} \int_0^\infty \frac{e^{-\mu t} t^{x+\beta-2}}{(1+t)^{\alpha+\beta-1}} \frac{(1+t)}{(1+t)} dt \\ &\quad - \frac{\mu}{(\alpha+\beta-1)} \int_0^\infty \frac{e^{-\mu t} t^{x+\beta-1}}{(1+t)^{\alpha+\beta-1}} \frac{(1+t)}{(1+t)} dt \\ &= \frac{(\beta+x-1)}{(\alpha+\beta-1)} \left[ \int_0^\infty \frac{e^{-\mu t} t^{x+\beta-1}}{(1+t)^{\alpha+\beta}} dt + \int_0^\infty \frac{e^{-\mu t} t^{x+\beta-2}}{(1+t)^{\alpha+\beta}} dt \right] \\ &\quad - \frac{\mu}{(\alpha+\beta-1)} \left[ \int_0^\infty \frac{e^{-\mu t} t^{x+\beta}}{(1+t)^{\alpha+\beta}} dt + \int_0^\infty \frac{e^{-\mu t} t^{x+\beta-1}}{(1+t)^{\alpha+\beta}} dt \right] \end{aligned}$$

$$I_x = \frac{(\beta+x-1)}{(\alpha+\beta-1)} (I_x + I_{x-1}) - \frac{\mu}{(\alpha+\beta-1)} (I_{x+1} + I_x)$$

$$\mu I_{x+1} = (x-\alpha-\mu)\, I_x + (\beta+x-1)\, I_{x-1}$$

But

$$I_x = \frac{x! B(\alpha,\beta)}{\mu^x} f(x)$$

Therefore,

$$\frac{\mu(x+1)!B(\alpha,\beta)}{\mu^{x+1}}f(x+1) = \frac{(x-\alpha-\mu)x!B(\alpha,\beta)}{\mu^{x}}f(x) + \frac{(\beta+x-1)(x-1)!B(\alpha,\beta)}{\mu^{x-1}}f(x-1)$$

Therefore,

$$x(x+1)f(x+1) = x(x-\alpha-\mu)f(x) + \mu(\beta+x-1)f(x-1) \quad (3.40)$$

is the recursive relation of Poisson - Generalized Pareto Distribution as given by Willmot (1993).

### 3.6.14 Poisson-Confluent Hypergeometric Distribution

If the Confluent-Hypergeometric distribution is given by

$$g(\lambda) = \frac{e^{-k\lambda}\lambda^{a-1}}{\Gamma(a)\varphi(a,c;k)(1+\lambda)^{a-c+1}} \quad (3.41)$$

for $\lambda > 0$, $c > a > 0$ and

$$\varphi(a,c;k) = \frac{1}{\Gamma(a)}\int_0^{\infty} \frac{e^{-kt}t^{a-1}}{(1+t)^{a-c+1}}dt$$

then the recursive formula for Poisson-Confluent Hypergeometric distribution is

$$(1+k)x(x+1)f(x+1) = (x+c-k-2)xf(x) + (x+a-1)f(x-1) \quad (3.42)$$

for

$$x = 0, 1, 2, \ldots$$

with

$$f(-1) = 0$$

Proof

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \frac{e^{-k\lambda}\lambda^{a-1}}{\Gamma(a)\,\varphi(a,c;k)\,(1+\lambda)^{a-c+1}} d\lambda \\ &= \frac{1}{x!\Gamma(a)\,\varphi(a,c;k)} \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a-1}}{(1+\lambda)^{a-c+1}} d\lambda \end{aligned}$$

$$x!\Gamma(a)\,\varphi(a,c;k)\,f(x) = \int_0^\infty e^{-\lambda(1+k)}\lambda^{x+a-1}\,(1+\lambda)^{-(a-c+1)}\,d\lambda$$

Now

$$I_x = x!\Gamma(a)\,\varphi(a,c;k)\,f(x)$$

and

$$I_x = \int_0^\infty e^{-\lambda(1+k)}\lambda^{x+a-1}\,(1+\lambda)^{-(a-c+1)}\,d\lambda$$

Using integration by parts, let

$$\begin{aligned} u &= e^{-\lambda(1+k)}\lambda^{x+a-1} \\ &\text{and} \\ dv &= (1+\lambda)^{-(a-c+1)}\,d\lambda \end{aligned}$$

then

$$\begin{aligned} du &= \left\{(x+\alpha-1)\,e^{-\lambda(1+k)}\lambda^{x+a-2} - (1+k)\,e^{-\lambda(1+k)}\lambda^{x+a-1}\right\} d\lambda \\ &\text{and} \\ v &= \frac{(1+\lambda)^{-(a-c)}}{-(a-c)} \end{aligned}$$

Therefore

$$\begin{aligned} I_x &= \frac{1}{(a-c)} \int_0^\infty \frac{(x+a-1)\,e^{-\lambda(1+k)}\lambda^{x+a-2}}{(1+\lambda)^{a-c}} d\lambda \\ &\quad - \frac{1}{(a-c)} \int_0^\infty \frac{(1+k)\,e^{-\lambda(1+k)}\lambda^{x+a-1}}{(1+\lambda)^{a-c}} d\lambda \end{aligned}$$

$$I_x = \frac{(x+a-1)}{(a-c)} \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a-2}}{(1+\lambda)^{a-c}} \frac{(1+\lambda)}{(1+\lambda)} d\lambda$$
$$-\frac{(1+k)}{(a-c)} \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a-1}}{(1+\lambda)^{a-c}} \frac{(1+\lambda)}{(1+\lambda)} d\lambda$$

$$I_x = \frac{(x+a-1)}{(a-c)} \left[ \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a-1}}{(1+\lambda)^{a-c+1}} d\lambda + \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a-2}}{(1+\lambda)^{a-c+1}} d\lambda \right]$$
$$-\frac{(1+k)}{(a-c)} \left[ \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a}}{(1+\lambda)^{a-c+1}} d\lambda + \int_0^\infty \frac{e^{-\lambda(1+k)}\lambda^{x+a-1}}{(1+\lambda)^{a-c+1}} d\lambda \right]$$

$$I_x = \frac{(x+a-1)}{(a-c)} (I_x + I_{x-1}) - \frac{(1+k)}{(a-c)} (I_{x+1} + I_x)$$

$$(a-c) I_x = (x+a-1)(I_x + I_{x-1}) - (1+k)(I_{x+1} + I_x)$$

$$(1+k) I_{x+1} = (x+c-k-2) I_x + (x+a-1) I_{x-1}$$

But

$$I_x = x!\Gamma(a)\varphi(a,c;k) f(x)$$

therefore

$$(1+k)(x+1)! f(x+1) = (x+c-k-2)\, x! f(x) + (x+a-1)(x-1)! f(x-1)$$

then the recursive relation becomes

$$(1+k)\, x(x+1) f(x+1) = (x+c-k-2)\, x f(x) + (x+a-1) f(x-1)$$

### 3.6.15 Poisson - Truncated Normal Distribution

If the Truncated Normal probability distribution function is given by

$$g(\lambda) = \frac{2}{\sqrt{2\pi\sigma^2}} e^{-\frac{(\lambda-\mu)^2}{2\sigma^2}}; 0 < \lambda < \infty, -\infty < \mu < \infty, \sigma^2 > 0 \qquad (3.43)$$

then the recursive formula for Poisson-Truncated Normal distribution is

$$(x+2) f(x+2) = (\sigma^2 + \mu) f(x+1) - \sigma^2 f(x); x = 0, 1, 2, ...$$

Put

$$x = n - 2$$

therefore

$$np_n = (\sigma^2 + \mu) p_{n-1} - \sigma^2 p_{n-2}$$

therefore

$$p_n = \left(\frac{\sigma^2 + \mu}{n}\right) p_{n-1} - \frac{\sigma^2}{n} p_{n-2}; n = 2, 3, 4, ...$$

**Proof** The probability distribution function of Normal distribution is given as

$$f(x) = \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{(x-\mu)^2}{2\sigma^2}}; -\infty < x < \infty, -\infty < \mu < \infty, \sigma^2 > 0$$

Now

$$\int_{-\infty}^{\infty} \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{(x-\mu)^2}{2\sigma^2}} dx = 1$$

since it is a pdf. Therefore

$$\int_{0}^{\infty} \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{(x-\mu)^2}{2\sigma^2}} dx = \frac{1}{2}$$

Therefore

$$\int_{0}^{\infty} \frac{2}{\sqrt{2\pi\sigma^2}} e^{-\frac{(x-\mu)^2}{2\sigma^2}} dx = 1$$

Thus

$$\frac{2}{\sqrt{2\pi\sigma^2}} e^{-\frac{(x-\mu)^2}{2\sigma^2}}; 0 < x < \infty$$

is a pdf. This is the pdf of the truncated normal distribution, thus

$$g(\lambda) = \frac{2}{\sqrt{2\pi\sigma^2}} e^{-\frac{(\lambda-\mu)^2}{2\sigma^2}}; \lambda > 0, -\infty < \mu < \infty, \sigma^2 > 0$$

The mixed poisson distribution is given as

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda$$

$$\begin{aligned} f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \frac{2}{\sqrt{2\pi\sigma^2}} e^{-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \\ &= \frac{2}{x!\sqrt{2\pi\sigma^2}} \int_0^\infty \lambda^x e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \end{aligned}$$

Therefore

$$\frac{\sqrt{2\pi\sigma^2}}{2} x! f(x) = \int_0^\infty \lambda^x e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \approxeq I_x$$

Put

$$u = e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} \text{ and } dv = \lambda^x d\lambda$$

therefore

$$\begin{aligned} du &= \left[-1 + \frac{2(\lambda-\mu)}{2\sigma^2}\right] e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \\ &\text{and} \\ v &= \frac{\lambda^{x+1}}{x+1} \end{aligned}$$

$$
\begin{aligned}
I_x &= -\int_0^\infty \frac{\lambda^{x+1}}{x+1}\left[-1+\frac{(\lambda-\mu)}{\sigma^2}\right] e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \\
&= -\int_0^\infty \frac{\lambda^{x+1}}{x+1}(-1)\, e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda - \int_0^\infty \frac{\lambda^{x+1}}{x+1}\frac{(\lambda-\mu)}{\sigma^2} e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \\
&= \int_0^\infty \frac{\lambda^{x+1}}{x+1} e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda - \int_0^\infty \frac{\lambda^{x+1}}{x+1}\frac{(\lambda-\mu)}{\sigma^2} e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda \\
&= \frac{1}{x+1}\int_0^\infty \lambda^{x+1} e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda - \frac{1}{(x+1)\,\sigma^2}\int_0^\infty \lambda^{x+1}(\lambda-\mu)\, e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda
\end{aligned}
$$

$$
\begin{aligned}
I_x &= \left(\frac{1}{x+1}\right) I_{x+1} - \frac{1}{\sigma^2(x+1)}\left[\int_0^\infty \lambda^{x+2} e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda - \mu\int_0^\infty \lambda^{x+1} e^{-\lambda-\frac{(\lambda-\mu)^2}{2\sigma^2}} d\lambda\right] \\
&= \left(\frac{1}{x+1}\right) I_{x+1} - \frac{1}{\sigma^2(x+1)}\left\{I_{x+2} - \mu I_{x+1}\right\}
\end{aligned}
$$

Therefore

$$\sigma^2(x+1)\, I_x = \sigma^2 I_{x+1} - I_{x+2} + \mu I_{x+1}$$

$$I_{x+2} = \left(\sigma^2+\mu\right) I_{x+1} - \sigma^2(x+1)\, I_x$$

Therefore

$$(x+2)!f(x+2) = \left(\sigma^2+\mu\right)(x+1)!f(x+1) - \sigma^2(x+1)\, x!f(x)$$

$$(x+2)\, f(x+2) = \left(\sigma^2+\mu\right) f(x+1) - \sigma^2 f(x); \qquad (3.44)$$

for

$$x = 0, 1, 2, \ldots$$

Put

$$x = n - 2$$

therefore

$$np_n = \left(\sigma^2 + \mu\right) p_{n-1} - \sigma^2 p_{n-2}$$

therefore

$$p_n = \left(\frac{\sigma^2 + \mu}{n}\right) p_{n-1} - \frac{\sigma^2}{n} p_{n-2}; n = 2, 3, 4, \ldots$$

# Chapter 4

## MIXED POISSON DISTRIBUTIONS IN TERMS OF THE LAPLACE TRANSFORMS OF THE MIXING DISTRIBUTION

### 4.1 Introduction

In this chapter the derivation of the relationship between a mixed Poisson pmf $f(x)$ and the Laplace of the mixing distribution is given. Then the relationship between the pgf of $X$ and the Laplace Transform of the mixing distribution is derived. They are then applied to the Exponential mixing distribution, Gamma mixing distributions with one and two parameters.

#### 4.1.1 Relationship between a mixed Poisson and Laplace of mixing distribution

We should note that

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda = E\left[\frac{e^{-\lambda}\lambda^x}{x!}\right] \tag{4.1}$$

But the Laplace Transform of $\lambda$ is defined as:

$$L_\lambda(s) = E\left[e^{-\lambda s}\right] \tag{4.2}$$

Differentiating $L_\lambda(s)$ with respect to $s$, we have

$$L'_\lambda(s) = E\left[-\lambda e^{-\lambda s}\right]$$

and

$$L''_\lambda(s) = E\left[(-\lambda)^2 e^{-\lambda s}\right]$$

In general,

$$L_{\lambda}^{(x)}(s) = \frac{d^x}{ds^x} L_{\lambda}(s) = (-1)^x E\left[\lambda^x e^{-\lambda s}\right]$$

When $s = 1$, we have

$$L_{\lambda}^{(x)}(1) = (-1)^x E\left[\lambda^x e^{-\lambda}\right]$$

Therefore,

$$\begin{aligned} f(x) &= E\left[\frac{e^{-\lambda}\lambda^x}{x!}\right] \\ &= \frac{1}{x!} E\left[\lambda^x e^{-\lambda}\right] \end{aligned}$$

$$f(x) = \frac{1}{x!}(-1)^x L_{\lambda}^{(x)}(1) \tag{4.3}$$

This is the mixed Poisson distribution expressed in terms of the Laplace transform of the mixing distribution.

### 4.1.2 Relationship between pgf of X and Laplace Transform of mixing distribution

The probability generating function is given by

$$G_x(s) = \sum_{x=0}^{\infty} \left[\int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda\right] s^x \tag{4.4}$$

$$G_x(s) = \int_0^{\infty} \left[\sum_{x=0}^{\infty} \frac{e^{-\lambda}(\lambda s)^x}{x!}\right] g(\lambda)\, d\lambda \tag{4.5}$$

$$G_x(s) = L_{\lambda}(1 - s) \tag{4.6}$$

where $L_{\lambda}(\cdot)$ is the Laplace Transform of $\lambda$.

### 4.1.3 Relationship between mixed Poisson distribution and Moments of the mixing distribution

Karlis and Xekalaki (2005) in their Proposition 14 gave an alternative formula linking the probability function of a mixed Poisson distribution to the moments of the mixing distribution.

$$\begin{aligned}
f(x) &= \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda \\
&= \frac{1}{x!}\int_0^\infty \left(e^{-\lambda}\right)\lambda^x g(\lambda)\, d\lambda \\
&= \int_0^\infty \left(\sum_{r=0}^\infty \frac{(-\lambda)^r}{r!}\right)\frac{\lambda^x}{x!} g(\lambda)\, d\lambda \\
&= \sum_{r=0}^\infty \int_0^\infty \frac{(-1)^r\lambda^r}{r!}\frac{\lambda^x}{x!} g(\lambda)\, d\lambda \\
&= \sum_{r=0}^\infty \left\{\frac{(-1)^r}{r!x!}\int_0^\infty \lambda^{x+r} g(\lambda)\, d\lambda\right\} \\
&= \sum_{r=0}^\infty \left\{\frac{(-1)^r}{r!x!} E\left[\Lambda^{x+r}\right]\right\}
\end{aligned}$$

$$\begin{aligned}
f(x) &= \sum_{r=0}^\infty \frac{(-1)^r}{r!x!}\mu_{x+r}(\lambda) \\
&= \frac{1}{x!}\sum_{r=0}^\infty \frac{(-1)^r}{r!}\mu_{x+r}(\lambda)
\end{aligned}$$

## 4.2 Exponential with one parameter

$$g(\lambda) = \mu e^{-\mu\lambda}; \lambda \geq 0, \mu > 0, \text{ as in (2.2)}$$

$$
\begin{aligned}
L(s) &= E\left(e^{-\lambda s}\right) \\
&= \int_0^{\infty} e^{-\lambda s} g(\lambda)\, d\lambda \\
&= \int_0^{\infty} e^{-\lambda s} \mu e^{-\mu\lambda} d\lambda \\
&= \mu \int_0^{\infty} e^{-\lambda(s+\mu)} d\lambda \\
&= \mu \left| \frac{-e^{-\lambda(s+\mu)}}{(s+\mu)} \right|_0^{\infty}
\end{aligned}
$$

$$
L(s) = \frac{\mu}{s+\mu} \tag{4.7}
$$

This is the Laplace transform of the Exponential distribution.

Now, we get the first four derivatives of the Laplace Transform and hence in general get the $x^{th}$ derivative.

$$
L'(s) = \frac{-\mu}{(s+\mu)^2}
$$

$$
L''(s) = \frac{2\mu}{(s+\mu)^3}
$$

$$
L'''(s) = \frac{-6\mu}{(s+\mu)^4}
$$

$$
L^{iv}(s) = \frac{24\mu}{(s+\mu)^5}
$$

Hence,

$$
L^{(x)}(s) = \frac{(-1)^x\, x!\mu}{(s+\mu)^{x+1}}
$$

Replacing $s$ with 1, we have

$$L^{(x)}(1) = \frac{(-1)^x x!\mu}{(\mu+1)^{x+1}} \tag{4.8}$$

Now,

$$\begin{aligned} f(x) &= \frac{1}{x!}(-1)^x L^{(x)}(1) \\ &= \frac{1}{x!}(-1)^x \frac{(-1)^x x!\mu}{(\mu+1)^{x+1}} \\ &= \frac{\mu}{(\mu+1)^{x+1}} \end{aligned}$$

Therefore,

$$f(x) = \left(\frac{\mu}{\mu+1}\right)\left(\frac{1}{\mu+1}\right)^x ; x = 0, 1, 2, \ldots \text{ as in (2.3)}$$

Now, applying formula (4.6),

$$G_x(s) = L(1-s)$$

But

$$L(s) = \frac{\mu}{\mu+s}$$

Then

$$\begin{aligned} G_x(s) &= \frac{\mu}{\mu+(1-s)} \\ &= \frac{\mu}{1+\mu-s} \\ &= \frac{\mu}{(1+\mu)\left[1-\frac{s}{1+\mu}\right]} \\ &= \left(\frac{\mu}{1+\mu}\right)\left(1-\frac{s}{1+\mu}\right)^{-1} \\ &= \left(\frac{\mu}{1+\mu}\right)\sum_{x=0}^{\infty}\left(\frac{s}{1+\mu}\right)^x \end{aligned}$$

Therefore

$$f(x) = \left(\frac{\mu}{1+\mu}\right)\left(\frac{1}{1+\mu}\right)^x$$

Next, consider

$$\begin{aligned} E[\Lambda^r] &= \int_0^\infty \lambda^r \mu e^{-\mu\lambda} d\lambda \\ &= \mu \int_0^\infty e^{-\mu\lambda} \lambda^r d\lambda \end{aligned}$$

Put

$$y = \mu\lambda \Longrightarrow \lambda = \frac{y}{\mu} \text{ and } d\lambda = \frac{dy}{\mu}$$

Therefore

$$\begin{aligned} E[\Lambda^r] &= \mu \int_0^\infty e^{-y} \left(\frac{y}{\mu}\right)^r \frac{dy}{\mu} \\ &= \frac{1}{\mu^r} \int_0^\infty e^{-y} y^r dy \\ &= \frac{1}{\mu^r} \Gamma(r+1) \\ &= \frac{r!}{\mu^r} \end{aligned}$$

Now

$$
\begin{aligned}
f(x) &= \frac{1}{x!}\sum_{r=0}^{\infty}\frac{(-1)^r}{r!}\mu_{x+r}(\lambda) \\
&= \frac{1}{x!}\sum_{r=0}^{\infty}\frac{(-1)^r}{r!}\frac{(x+r)!}{\mu^{x+r}} \\
&= \sum_{r=0}^{\infty}(-1)^r\frac{(x+r)!}{x!r!}\frac{1}{\mu^{x+r}} \\
&= \sum_{r=0}^{\infty}(-1)^r\binom{x+r}{r}\frac{1}{\mu^{x+r}} \\
&= \sum_{r=0}^{\infty}(-1)^r\binom{x+r+1-1}{r}\frac{1}{\mu^{x+r}} \\
&= \sum_{r=0}^{\infty}(-1)^r\binom{(x+1)+r-1}{r}\frac{1}{\mu^{x+r}} \\
&= \sum_{r=0}^{\infty}\binom{-(x+1)}{r}\frac{1}{\mu^{x+r}}
\end{aligned}
$$

$$
\begin{aligned}
f(x) &= \frac{1}{\mu^x}\sum_{r=0}^{\infty}\binom{-(x+1)}{r}\left(\frac{1}{\mu}\right)^r \\
&= \frac{1}{\mu^x}\left(1+\frac{1}{\mu}\right)^{-(x+1)} \\
&= \frac{1}{\mu^x}\left(\frac{\mu+1}{\mu}\right)^{-(x+1)} \\
&= \frac{1}{\mu^x}\left(\frac{\mu}{\mu+1}\right)^{x+1} \\
&= \left(\frac{1}{\mu}\right)^x\left(\frac{\mu}{\mu+1}\right)^x\left(\frac{\mu}{\mu+1}\right)
\end{aligned}
$$

Therefore,

$$f(x) = \left(\frac{1}{1+\mu}\right)^x \left(\frac{\mu}{1+\mu}\right); x = 0, 1, 2, ...$$

## 4.3 Gamma with one parameter

$$g(\lambda) = \frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}; \lambda > 0, \alpha > 0 \text{ as in (2.4)}$$

$$\begin{aligned} L(s) &= E\left(e^{-\lambda s}\right) \\ &= \int_0^\infty e^{-\lambda s} g(\lambda)\, d\lambda \\ &= \int_0^\infty e^{-\lambda s} \frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)} d\lambda \\ &= \frac{1}{\Gamma(\alpha)} \int_0^\infty e^{-\lambda(s+1)} \lambda^{\alpha-1} d\lambda \end{aligned}$$

Let

$$y = \lambda(s+1) \Longrightarrow \lambda = \frac{y}{s+1}; d\lambda = \frac{dy}{s+1}$$

$$\begin{aligned} L(s) &= \frac{1}{\Gamma(\alpha)} \int_0^\infty e^{-y} \frac{y^{\alpha-1}}{(s+1)^\alpha} dy \\ &= \frac{1}{\Gamma(\alpha)} \frac{1}{(s+1)^\alpha} \Gamma(\alpha) \end{aligned}$$

Therefore,

$$L(s) = \frac{1}{(s+1)^\alpha} \tag{4.9}$$

This is the Laplace transform for the Gamma Distribution with one parameter.

Now,

$$L'(s) = \frac{-\alpha}{(s+1)^{\alpha+1}}$$

$$L''(s) = \frac{\alpha(\alpha+1)}{(s+1)^{\alpha+2}}$$

$$L'''(s) = \frac{-\alpha(\alpha+1)(\alpha+2)}{(s+1)^{\alpha+3}}$$

In general,

$$\begin{aligned} L^{(x)}(s) &= \frac{(-1)^x \alpha(\alpha+1)(\alpha+2)\ldots(\alpha+x-1)}{(s+1)^{\alpha+x}} \\ &= (-1)^x \frac{(\alpha+x-1)!}{(\alpha-1)!} \frac{1}{(s+1)^{\alpha+x}} \end{aligned}$$

When $s = 1$,

$$L^{(x)}(1) = (-1)^x \frac{(\alpha+x-1)!}{(\alpha-1)!} \frac{1}{2^{\alpha+x}} \qquad (4.10)$$

Now,

$$\begin{aligned} f(x) &= \frac{1}{x!}(-1)^x L^{(x)}(1) \\ &= \frac{1}{x!}(-1)^x (-1)^x \frac{(\alpha+x-1)!}{(\alpha-1)!} \frac{1}{2^{\alpha+x}} \\ &= \frac{(\alpha+x-1)!}{x!(\alpha-1)!} \frac{1}{2^{\alpha+x}} \end{aligned}$$

Therefore,

$$f(x) = \binom{\alpha+x-1}{x} \left(\frac{1}{2}\right)^{\alpha} \left(\frac{1}{2}\right)^{x}; x = 0, 1, 2, \ldots \qquad (4.11)$$

Now, applying formula (4.6),

$$\begin{aligned} G_x(s) &= L_\lambda(1-s) \\ &= \frac{1}{(1+1-s)^\alpha} \\ &= \frac{1}{(2-s)^\alpha} \\ &= \frac{1}{2^\alpha\left[1-\frac{s}{2}\right]^\alpha} \\ &= \frac{1}{2^\alpha}\left(1-\frac{s}{2}\right)^{-\alpha} \end{aligned}$$

Now

$$\begin{aligned} G_x(s) &= \frac{1}{2^\alpha}\sum_{x=0}^{\infty}\binom{-\alpha}{x}\left(-\frac{s}{2}\right)^x \\ &= \frac{1}{2^\alpha}\sum_{x=0}^{\infty}(-1)^x\binom{-\alpha}{x}\left(\frac{1}{2}\right)^x s^x \end{aligned}$$

Therefore

$$\begin{aligned} f(x) &= \frac{1}{2^\alpha}(-1)^x\binom{-\alpha}{x}\left(\frac{1}{2}\right)^x \\ &= \frac{1}{2^{x+\alpha}}\binom{\alpha+x-1}{x} \\ &= \binom{\alpha+x-1}{x}\left(\frac{1}{2}\right)^\alpha\left(\frac{1}{2}\right)^x ; x=0,1,2,\ldots \end{aligned}$$

Next

$$\begin{aligned} E[\Lambda^r] &= \int_0^\infty \lambda^r \frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}d\lambda \\ &= \frac{1}{\Gamma(\alpha)}\int_0^\infty e^{-\lambda}\lambda^{r+\alpha-1}d\lambda \\ &= \frac{1}{\Gamma(\alpha)}\Gamma(r+\alpha) \end{aligned}$$

Therefore,

$$E\left[\Lambda^{x+r}\right] = \frac{1}{\Gamma(\alpha)}\Gamma(x+r+\alpha)$$

Now

$$\begin{aligned} f(x) &= \frac{1}{x!}\sum_{r=0}^{\infty}\frac{(-1)^r}{r!}\frac{\Gamma(x+r+\alpha)}{\Gamma(\alpha)} \\ &= \sum_{r=0}^{\infty}\frac{(-1)^r}{x!r!}\frac{(x+r+\alpha-1)!}{(\alpha-1)!} \\ &= \sum_{r=0}^{\infty}\frac{(-1)^r}{x!r!}\frac{(x+r)!}{(x+r)!}\frac{(x+r+\alpha-1)!}{(\alpha-1)!} \\ &= \sum_{r=0}^{\infty}(-1)^r\frac{(x+r)!}{x!r!}\frac{(x+r+\alpha-1)!}{(x+r)!\,(\alpha-1)!} \end{aligned}$$

$$f(x) = \sum_{r=0}^{\infty}(-1)^r\binom{x+r}{r}\binom{x+r+\alpha-1}{x+r}$$

## 4.4 Gamma with two parameters

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)}e^{-\beta\lambda}\lambda^{\alpha-1}; \lambda > 0, \alpha > 0, \beta > 0 \tag{4.12}$$

$$\begin{aligned} L(s) &= E\left(e^{-\lambda s}\right) \\ &= \int_0^{\infty} e^{-\lambda s}g(\lambda)\,d\lambda \\ &= \int_0^{\infty} e^{-\lambda s}\frac{\beta^{\alpha}}{\Gamma(\alpha)}e^{-\beta\lambda}\lambda^{\alpha-1}d\lambda \\ &= \frac{\beta^{\alpha}}{\Gamma(\alpha)}\int_0^{\infty} e^{-\lambda(\beta+s)}\lambda^{\alpha-1}d\lambda \end{aligned}$$

Let

$$y = \lambda(\beta + s) \Longrightarrow \lambda = \frac{y}{\beta + s}; d\lambda = \frac{dy}{\beta + s}$$

$$\begin{aligned} L(s) &= \frac{\beta^{\alpha}}{\Gamma(\alpha)} \int_0^{\infty} e^{-y} \left(\frac{y}{\beta + s}\right)^{\alpha - 1} \frac{dy}{\beta + s} \\ &= \frac{\beta^{\alpha}}{\Gamma(\alpha)} \left(\frac{1}{\beta + s}\right)^{\alpha} \int_0^{\infty} e^{-y} y^{\alpha - 1} dy \\ &= \frac{\beta^{\alpha}}{\Gamma(\alpha)} \left(\frac{1}{\beta + s}\right)^{\alpha} \Gamma(\alpha) \end{aligned}$$

Therefore,

$$L(s) = \left(\frac{\beta}{\beta + s}\right)^{\alpha} \tag{4.13}$$

This is the Laplace Transform of Gamma distribution with two parameters.

Now,

$$L'(s) = \beta^{\alpha} \left[\frac{-\alpha}{(\beta + s)^{\alpha + 1}}\right]$$

$$L''(s) = \beta^{\alpha} \left[\frac{\alpha(\alpha + 1)}{(\beta + s)^{\alpha + 2}}\right]$$

$$L'''(s) = \beta^{\alpha} \left[\frac{-\alpha(\alpha + 1)(\alpha + 2)}{(\beta + s)^{\alpha + 3}}\right]$$

In general,

$$\begin{aligned} L^{(x)}(s) &= \beta^{\alpha} \left[\frac{(-1)^x \alpha(\alpha + 1)(\alpha + 2) \ldots (\alpha + x - 1)}{(\beta + s)^{\alpha + x}}\right] \\ &= \frac{(-1)^x \beta^{\alpha}}{(\beta + s)^{\alpha + x}} \frac{(\alpha + x - 1)!}{(\alpha - 1)!} \end{aligned}$$

$$L^{(x)}(1) = \frac{(-1)^x \beta^\alpha}{(1+\beta)^{\alpha+x}} \frac{(\alpha+x-1)!}{(\alpha-1)!}$$

Now,

$$\begin{aligned} f(x) &= \frac{1}{x!}(-1)^x L^{(x)}(1) \\ &= \frac{1}{x!}(-1)^x (-1)^x \frac{\beta^\alpha}{(1+\beta)^{\alpha+x}} \frac{(\alpha+x-1)!}{(\alpha-1)!} \\ &= \frac{(\alpha+x-1)!}{x!\,(\alpha-1)!} \left(\frac{\beta}{1+\beta}\right)^\alpha \left(\frac{1}{1+\beta}\right)^x \end{aligned}$$

$$f(x) = \binom{\alpha+x-1}{x} \left(\frac{\beta}{1+\beta}\right)^\alpha \left(\frac{1}{1+\beta}\right)^x ; x = 0, 1, 2, \ldots \qquad (4.14)$$

Applying formula (4.6),

$$\begin{aligned} G_x(s) &= L_\lambda(1-s) \\ &= \left(\frac{\beta}{\beta+1-s}\right)^\alpha \\ &= \left(\frac{\beta}{(1+\beta)\left(1-\frac{s}{1+\beta}\right)}\right)^\alpha \end{aligned}$$

Therefore,

$$\begin{aligned} G_x(s) &= \left(\frac{\beta}{1+\beta}\right)^\alpha \frac{1}{\left(1-\frac{s}{1+\beta}\right)^\alpha} \\ &= \left(\frac{\beta}{1+\beta}\right)^\alpha \left(1-\frac{s}{1+\beta}\right)^{-\alpha} \\ &= \left(\frac{\beta}{1+\beta}\right)^\alpha \sum_{x=0}^{\infty} \binom{-\alpha}{x} \left(-\frac{s}{1+\beta}\right)^x \\ &= \left(\frac{\beta}{1+\beta}\right)^\alpha \sum_{x=0}^{\infty} (-1)^x \binom{-\alpha}{x} \left(\frac{1}{1+\beta}\right)^x s^x \end{aligned}$$

Therefore,

$$\begin{aligned} f(x) &= (-1)^x \binom{-\alpha}{x} \left(\frac{\beta}{1+\beta}\right)^{\alpha} \left(\frac{1}{1+\beta}\right)^{x} \\ &= \binom{\alpha+x-1}{x} \left(\frac{\beta}{1+\beta}\right)^{\alpha} \left(\frac{1}{1+\beta}\right)^{x}; x = 0, 1, 2, \ldots \end{aligned}$$

Next,

$$\begin{aligned} E(\Lambda^r) &= \int_0^{\infty} \lambda^r \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta\lambda} \lambda^{\alpha-1} d\lambda \\ &= \frac{\beta^{\alpha}}{\Gamma(\alpha)} \int_0^{\infty} e^{-\beta\lambda} \lambda^{r+\alpha-1} d\lambda \end{aligned}$$

Put

$$y = \beta\lambda \Longrightarrow \lambda = \frac{y}{\beta} \Longrightarrow d\lambda = \frac{dy}{\beta}$$

Therefore,

$$\begin{aligned} E(\Lambda^r) &= \frac{\beta^{\alpha}}{\Gamma(\alpha)} \int_0^{\infty} e^{-y} \left(\frac{y}{\beta}\right)^{r+\alpha-1} \frac{dy}{\beta} \\ &= \frac{\beta^{\alpha}}{\Gamma(\alpha)\beta^{r+\alpha}} \int_0^{\infty} e^{-y} y^{r+\alpha-1} dy \\ &= \frac{\Gamma(r+\alpha)}{\Gamma(\alpha)\beta^r} \end{aligned}$$

Therefore

$$\begin{aligned} f(x) &= \sum_{r=0}^{\infty} \frac{(-1)^r}{x!r!} \frac{\Gamma(x+r+\alpha)}{\Gamma(\alpha)\beta^{x+r}} \\ &= \sum_{r=0}^{\infty} \frac{(-1)^r}{x!r!} \frac{(x+r+\alpha-1)!}{(\alpha-1)!\beta^{x+r}} \\ &= \sum_{r=0}^{\infty} \frac{(-1)^r}{\beta^{x+r}} \binom{x+r}{r} \binom{x+r+\alpha-1}{x+r}; x = 0, 1, 2, \ldots \end{aligned}$$

## 4.5 Other Mixing Distributions

Willmot (1986) obtained the following

### 4.5.1 Sichel Distribution - GIG Mixing Distribution

$$g(\lambda) = \frac{\mu^{-\alpha}\lambda^{\alpha-1}e^{-\left(\lambda^2+\mu^2\right)/2\beta\lambda}}{2K_\alpha\left(\mu\beta^{-1}\right)}$$

where $K_\alpha(\cdot)$ is the modified Bessel function of the third kind with index $\alpha$.

$$L_\lambda(s) = \frac{K_\alpha\left\{\mu\beta^{-1}\left(1+2\beta s\right)^{\frac{1}{2}}\right\}}{K_\alpha\left(\mu\beta^{-1}\right)}\left(1+2\beta s\right)^{-\frac{\alpha}{2}}$$

$$G_\lambda(s) = \frac{K_\alpha\left\{\mu\beta^{-1}\left[1-2\beta\left(s-1\right)\right]^{\frac{1}{2}}\right\}}{K_\alpha\left(\mu\beta^{-1}\right)}\left[1-2\beta\left(s-1\right)\right]^{-\frac{\alpha}{2}}$$

and

$$p_n = \frac{\mu^n}{n!}K_{\alpha+n}\left[\mu\beta^{-1}\left(1+2\beta\right)^{\frac{1}{2}}\right]\left(1+2\beta\right)^{-\left(\frac{\alpha+n}{2}\right)}; n = 0,1,2,...$$

For the Poisson - Inverse Gaussian distribution, it is a Poisson mixture with mixing distribution

$$g(\lambda) = \mu\left(2\pi\beta\lambda^3\right)^{-\frac{1}{2}}e^{-(\lambda-\mu)^2/2\beta\lambda}$$

which is obtained by substituting $\alpha = -\frac{1}{2}$ in the previous formulae.

The Laplace Transform is

$$L_\lambda(s) = \exp\left[-\frac{\mu}{\beta}\left\{\left(1+2\beta s\right)^{\frac{1}{2}}-1\right\}\right]$$

and the pgf of the resulting Poisson mixture is

$$G_\lambda(s) = \exp\left\{-\frac{\mu}{\beta}\left[1-2\beta\left(s-1\right)\right]^{\frac{1}{2}}-1\right\}$$

The probabilities are given by

$$p_n = p_0 \frac{\mu^n}{n!} \sum_{k=0}^{n-1} \frac{(n-1+k)!}{(n-1-k)!k!} \left(\frac{\beta}{2\mu}\right)^k (1+2\beta)^{-\frac{(n+k)}{2}}; n = 1, 2, 3, \ldots$$

where

$$p_0 = \exp\left\{-\frac{\mu}{\beta}\left[1+2\beta\right]^{\frac{1}{2}} - 1\right\}$$

### 4.5.2 Reciprocal Inverse Gaussian

$$g(\lambda) = \left(\frac{\theta}{2\pi\lambda}\right)^{\frac{1}{2}} \exp\left\{-\frac{\theta\mu^2}{2\lambda}\left(1-\frac{\lambda}{\mu}\right)^2\right\}$$

$$L_\lambda(s) = \left(1+\frac{2s}{\theta}\right)^{-\frac{1}{2}} \exp\left\{\theta\mu\left[1-\left(1+\frac{2s}{\theta}\right)^{\frac{1}{2}}\right]\right\}$$

which is the Laplace Transform of the convolution of a Gamma and an Inverse Gaussian distribution.

The pgf of a Poisson mixed over this distribution is

$$G(s) = \left[1-\frac{2}{\theta}(s-1)\right]^{-\frac{1}{2}} \exp\left(\theta\mu\left\{1-\left[1-\frac{2}{\theta}(s-1)\right]^{\frac{1}{2}}\right\}\right)$$

which is the convolution of a Negative Binomial and Poisson - Inverse Gaussian distribution.

# Chapter 5

## MIXED POISSON DISTRIBUTIONS IN TERMS OF SPECIAL FUNCTIONS

### 5.1 Introduction

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} g(\lambda)\, d\lambda$$

The above mixed Poisson distribution can also be obtained by expressing it in term of special functions. This will be the main objective of this chapter.

### 5.2 Mixing with Scaled Beta Distribution

In

$$f(x) = \frac{x^{\alpha-1}(1-x)^{\beta-1}}{B(\alpha,\beta)}; 0 < x < 1, \alpha > 0, \beta > 0$$

Put

$$x = \frac{\lambda}{\mu} \Longrightarrow dx = \frac{d\lambda}{\mu} \text{ and } \lambda = \mu x$$

Therefore,

$$g(\lambda) = \frac{1}{B(\alpha,\beta)} \left(\frac{\lambda}{\mu}\right)^{\alpha-1} \left(1 - \frac{\lambda}{\mu}\right)^{\beta-1} \frac{1}{\mu}$$

$$g(\lambda) = \frac{\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{\mu^{\alpha+\beta-1}B(\alpha,\beta)}; 0 < \lambda < \mu$$

which is a Scaled Beta Distribution.

The Mixed Poisson Distribution becomes

$$f(x) = \int_0^{\mu} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{\mu^{\alpha+\beta-1}B(\alpha,\beta)} d\lambda$$

Therefore,

$$G_x(s) = \int_0^{\mu} \frac{e^{\lambda(s-1)}\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{B(\alpha,\beta)\,\mu^{\alpha+\beta-1}} d\lambda$$

Let

$$\lambda = \mu t \Longrightarrow d\lambda = \mu dt \text{ and } t = \frac{\lambda}{\mu}$$

Therefore,

$$\begin{aligned} G_x(s) &= \int_0^1 \frac{e^{\mu(s-1)t}(\mu t)^{\alpha-1}(\mu-\mu t)^{\beta-1}}{B(\alpha,\beta)\,\mu^{\alpha+\beta-1}} \mu dt \\ &= \int_0^1 \frac{e^{\mu(s-1)t}t^{\alpha-1}(1-t)^{\beta-1}}{B(\alpha,\beta)} dt \\ &= \int_0^1 \frac{e^{-\mu(1-s)t}t^{\alpha-1}(1-t)^{\beta-1}}{B(\alpha,\beta)} dt \end{aligned}$$

$$G_x(s) =_1 F_1\{\alpha, \alpha+\beta, \mu(s-1)\} \tag{5.1}$$

which is a confluent Hypergeometric function, Willmot (1987).

## 5.3 Mixing with Inverted Beta Distribution

In

$$f(x) = \frac{x^{\alpha-1}(1-x)^{\beta-1}}{B(\alpha,\beta)}; 0 < x < 1$$

Let

$$x = \frac{\lambda}{1+\lambda} \Longrightarrow \lambda = \frac{x}{1-x} \text{ and } \frac{dx}{d\lambda} = \frac{1}{(1+\lambda)^2}$$

Therefore,

$$g(\lambda) = \frac{1}{B(\alpha,\beta)} \left(\frac{\lambda}{1+\lambda}\right)^{\alpha-1} \left[1 - \frac{\lambda}{1+\lambda}\right]^{\beta-1} \frac{1}{(1+\lambda)^2}$$

$$g(\lambda) = \frac{\lambda^{\alpha-1}}{(1+\lambda)^{\alpha+\beta} B(\alpha,\beta)}; 0 < \lambda < \infty$$

This is the Inverted Beta Distribution.

Now, the mixed poisson distribution becomes,

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!} \frac{\lambda^{\alpha-1}}{(1+\lambda)^{\alpha+\beta} B(\alpha,\beta)} d\lambda$$

$$f(x) = \frac{1}{x! B(\alpha,\beta)} \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}} d\lambda \tag{5.2}$$

But the Confluent Hypergeometric Distribution of the second kind is defined as;

$$\Psi(a,c;x) = \frac{1}{\Gamma(a)} \int_0^\infty \frac{e^{-xt} t^{a-1}}{(1+t)^{a-c+1}} dt$$

$$\Psi(a,c;1) = \frac{1}{\Gamma(a)} \int_0^\infty \frac{e^{-t} t^{a-1}}{(1+t)^{a-c+1}} dt$$

$$\Psi(x+\alpha,c;1) = \frac{1}{\Gamma(x+\alpha)} \int_0^\infty \frac{e^{-t} t^{x+\alpha-1}}{(1+t)^{x+\alpha-c+1}} dt \tag{5.3}$$

Comparing (5.2) and (5.3), we have

$$\begin{aligned} x+\alpha-c+1 &= \alpha+\beta \\ \beta &= x-c+1 \end{aligned}$$

Therefore

$$c = x - \beta + 1$$

Now

$$\Psi(x+\alpha, x-\beta+1; 1) = \frac{1}{\Gamma(x+\alpha)} \int_0^\infty \frac{e^{-\lambda}\lambda^{x+\alpha-1}}{(1+\lambda)^{\alpha+\beta}} d\lambda \tag{5.4}$$

## 5.4 Mixing with Lomax Distribution

The pdf of Lomax distribution is given as

$$g(\lambda) = \frac{\alpha\beta^{\alpha}}{(\lambda+\beta)^{\alpha+1}}; \alpha > 0, \beta > 0, \lambda > 0$$

Therefore the Poisson - Lomax distribution is given by,

$$\begin{aligned} f(x) &= \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!} \frac{\alpha\beta^{\alpha}}{(\lambda+\beta)^{\alpha+1}} d\lambda \\ &= \alpha\beta^{\alpha} \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!\,(\beta+\lambda)^{\alpha+1}} d\lambda \\ &= \alpha\beta^{\alpha} \int_0^{\infty} \frac{e^{-\lambda}\lambda^x}{x!\left[\beta\left(1+\frac{\lambda}{\beta}\right)\right]^{\alpha+1}} d\lambda \end{aligned}$$

Put

$$t = \frac{\lambda}{\beta} \Longrightarrow \lambda = \beta t, d\lambda = \beta dt$$

Therefore,

$$\begin{aligned} f(x) &= \alpha\beta^{\alpha} \int_0^{\infty} \frac{e^{-\beta t}(\beta t)^x}{x!\beta^{\alpha+1}(1+t)^{\alpha+1}} \beta dt \\ &= \frac{\alpha\beta^x}{x!} \int_0^{\infty} \frac{e^{-\beta t} t^x}{(1+t)^{\alpha+1}} dt \\ &= \frac{\alpha\beta^x}{x!} \int_0^{\infty} \frac{e^{-\beta t} t^{(x+1)-1}}{(1+t)^{\alpha+1}} dt \\ &= \frac{\alpha\beta^x}{x!} \Gamma(x+1)\,\Psi(x+1, c; \beta) \end{aligned}$$

where,

$$\begin{aligned} \alpha + 1 &= (x+1) - c + 1 \\ c &= x - \alpha + 1 \end{aligned}$$

Therefore,

$$f(x) = \frac{\alpha\beta^x}{x!} \Gamma(x+1)\,\Psi(x+1, x-\alpha+1; \beta)$$

$$f(x) = \alpha\beta^x \Psi(x+1, x-\alpha+1; \beta) \tag{5.5}$$

## 5.5 Mixing with Generalized Pareto Distribution

$$g(\lambda) = \frac{1}{\delta}\left[1+\frac{\xi\lambda}{\delta}\right]^{-\frac{1}{\xi}-1} ; \lambda > 0, \xi > 0, \delta > 0 \tag{5.6}$$

This is the pdf of Generalized Pareto distribution.

The mixed poisson distribution now becomes,

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\frac{1}{\delta}\left[1+\frac{\xi\lambda}{\delta}\right]^{-\frac{1}{\xi}-1} d\lambda$$

$$f(x) = \frac{\delta^2}{\xi^{x+1}}\Psi\left(x+1, x-\frac{1}{\xi}+1; \frac{\delta}{\xi}\right) \tag{5.7}$$

## 5.6 Mixing with Truncated Gamma Distribution

The pdf of Truncated Gamma distribution is given by,

$$g(\lambda) = \frac{\beta^\alpha e^{-\beta\lambda}\lambda^{\alpha-1}}{\gamma(\alpha, b\beta) - \gamma(\alpha, a\beta)}; 0 < a < \lambda < b < \infty, \alpha > 0, \beta > 0 \tag{5.8}$$

The mixed poisson distribution is

$$f(x) = \int_0^\infty \frac{e^{-\lambda}\lambda^x}{x!}\frac{\beta^\alpha e^{-\beta\lambda}\lambda^{\alpha-1}}{\gamma(\alpha, b\beta) - \gamma(\alpha, a\beta)} d\lambda$$

$$\begin{aligned} f(x) &= \frac{\beta^\alpha}{x!(\beta+1)^{x+\alpha}}\frac{\gamma[x+\alpha, b(\beta+1)] - \gamma[x+\alpha, a(\beta+1)]}{\gamma(\alpha, b\beta) - \gamma(\alpha, a\beta)}; \\ x &= 0, 1, 2, ..; \alpha > 0, \beta > 0, 0 < a < b \end{aligned} \tag{5.9}$$

# Chapter 6

## SUMMARY AND CONCLUSION

### 6.1 Summary in words

This work starts by constructing the Poisson distribution using

(i) Power series expansion

(ii) Binomial - Poisson mixture

(iii) Convolution and Compound Distribution of iid random variables

(iv) The Poisson process as a Pure Birth Process

(v) Recursive Relation based on Panjer's zero order class of distributions.

Then various mixing (prior) distributions were used. we could classify them under:

(i) Densities on $(0, 1)$ domain

(ii) Densities on $(0, \infty)$ domain

(iii) Shifted and Truncated densities.

We could further classify them as:

(i) Classical distributions

(ii) Generalized distributions

The resulting mixed distributions can be expressed in:

(i) the Explicit form

(ii) Recursive form

(iii) the form of pgf and using the Laplace transform for the mixing distribution

(iv) the form of Special Functions

Four methods of evaluating Mixed Poisson distributions for different mixing distributions were considered in this project with the aim of constructing as many as possible Mixed Poisson distributions.

According to this study, the method that resulted in a good number of Mixed Poisson distributions compared to the other methods was that of obtaining recursive relations using integration by parts. It is interesting to note that this method is straightforward as it does not have any conditions that need to be considered, that is, only the method of integration by parts is applied in the evaluation of the integral of the Mixed Poisson distribution given by equation (2.1).

Some mixed Poisson distributions can be obtained using more than one of the methods considered in this paper. For instance, to obtain Negative Binomial distribution, the method of explicit evaluation and that of using the Laplace Transform with Gamma as the mixing distribution were used. The two methods yielded the same result. This is a clear indication that there is no restriction on what kind of method to use for a particular given mixing distribution, that is, any method can be used wherever possible.

## 6.2 Framework: Summary in Figures

So in reviewing literature and working on mixed Poisson distributions the following two frameworks could be used:

**Figure 6.1: A General Framework For Poisson Mixture**

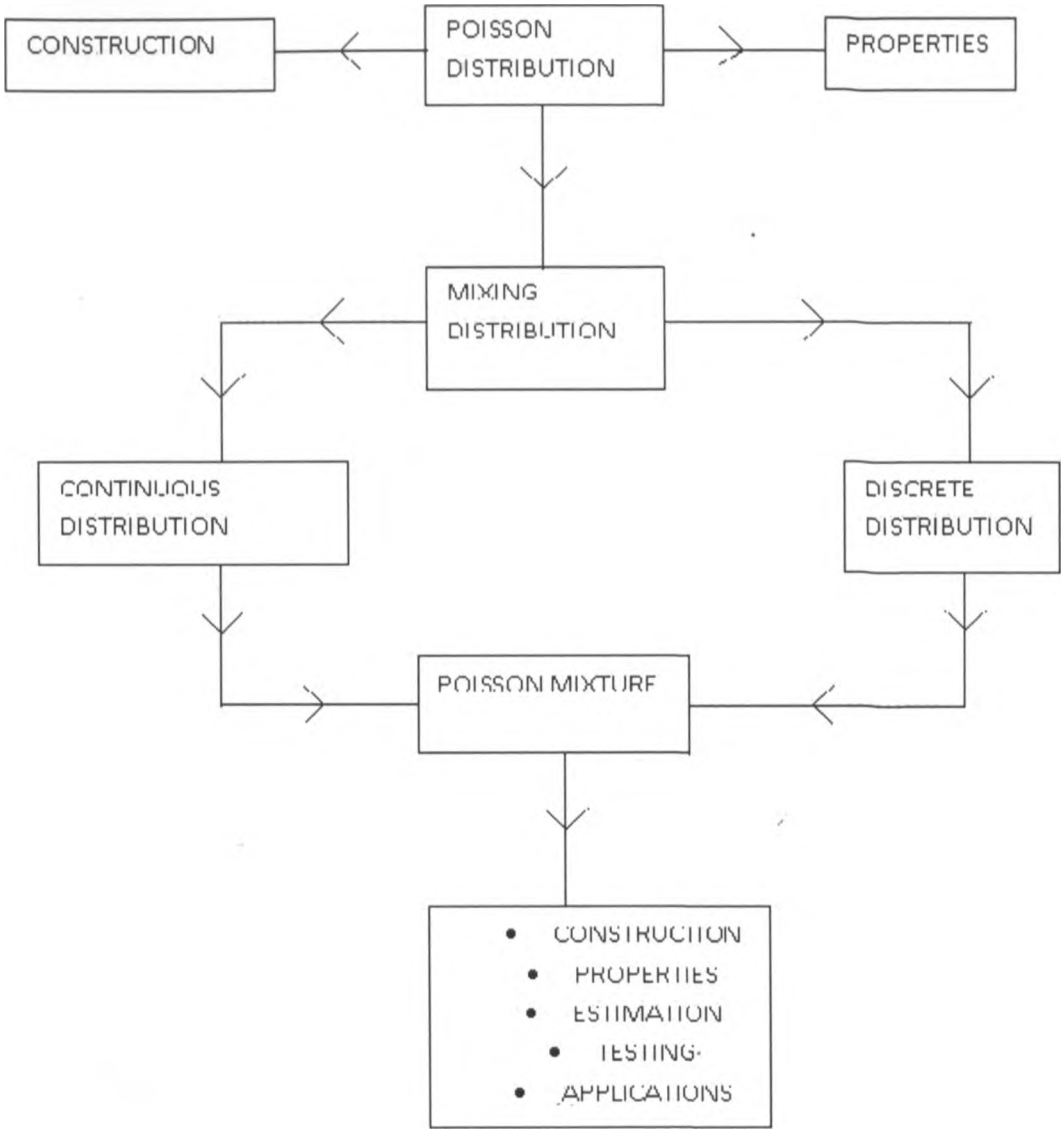
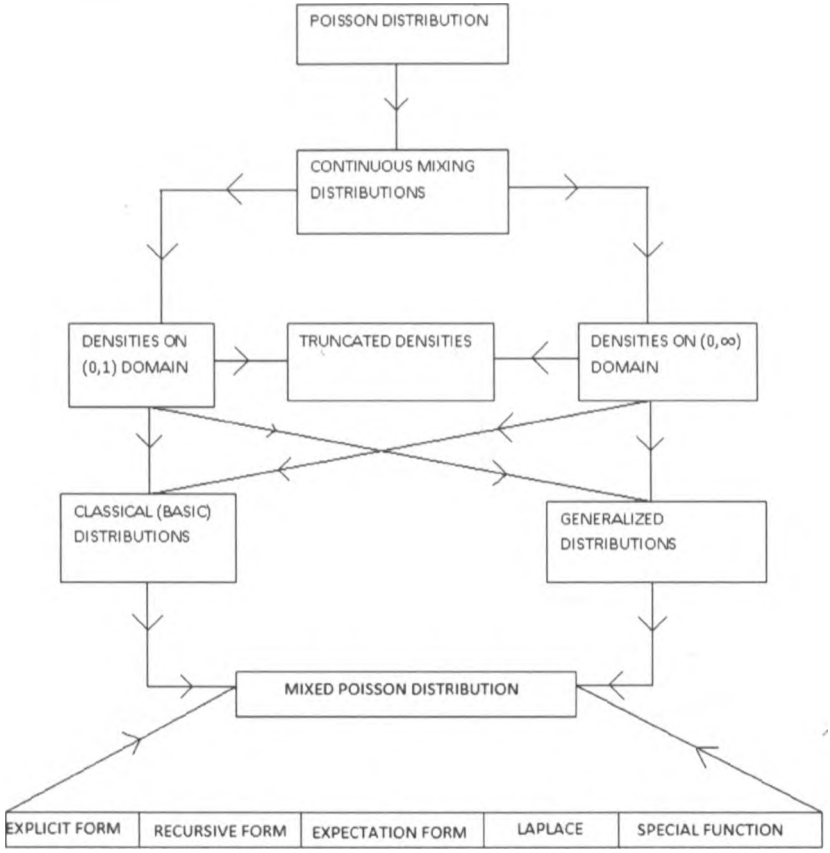


Figure 6.2: A Framework for Constructing Poisson Mixtures with Continuous Prior Distributions



## 6.3 Summary in a Table

Table 6.1: Identifying forms of Poisson Mixtures with corresponding mixing distributions

| | | FORMS OF MIXED POISSON DISTRIBUTION | | | |
|---|---|---|---|---|---|
| | MIXING DISTRIBUTION | EXPLICIT | RECURSION | LAPLACE | SPECIAL FUNCTION |
| | Densities on $(0,1);0<\lambda<1$ | | | | |
| 1 | Beta | | ✓ | | |
| | Densities on $(0,\infty);0<\lambda<\infty$ | | | | |
| 2 | Exponential | ✓ | ✓ | ✓ | |
| 3 | Gamma (1 parameter) | ✓ | ✓ | ✓ | |
| 4 | Gamma (2 parameters) | ✓ | ✓ | ✓ | |
| 5 | Inverse Gamma | | ✓ | | |
| 6 | Inverted Beta | | ✓ | | ✓ |
| 7 | Pareto | | | | |
| 8 | Lomax | | ✓ | | ✓ |
| 9 | Lindley | ✓ | | | |
| 10 | Linear Exponential Family | ✓ | | | |
| 11 | Inverse Gaussian | | ✓ | | |
| 12 | Rectangular | | ✓ | | |
| | Generalized Distributions | | | | |
| 13 | Generalized Exponential | | | | |
| 14 | Generalized Gamma | | ✓ | | |
| 15 | Generalized Lindley | ✓ | | | |
| 16 | Generalized Inverse Gaussian | | ✓ | | |
| 17 | Generalized Pareto | | ✓ | | ✓ |
| | Shifted and Truncated Densities | | | | |
| 18 | Zero Truncated Normal | ✓ | ✓ | | |
| 19 | Scaled Beta | | ✓ | | ✓ |
| 20 | Shifted Gamma | ✓ | | | |
| 21 | Rectangular (Uniform) | | | | |
| 22 | Truncated Gamma | | | | ✓ |
| | Distr. Based on Special Functions | | | | |
| 23 | Confluent Hypergeometric | | ✓ | | |

## 6.4 Formulae for mixing and mixed distributions

The following give a summary of the mixing distributions considered, their Laplace Transforms, the Mixed Poisson distribution in explicit form , the recursive relation of the Mixed Poisson distribution and the Mixed Poisson distribution expressed in Special Function.

### 6.4.1 Beta Distribution

$$g(\lambda) = \frac{\lambda^{\alpha-1}(1-\lambda)^{\beta-1}}{B(\alpha,\beta)}; 0 \leq \lambda \leq 1$$

$$x(x+1)f(x+1) = x(x+\alpha+\beta)f(x) - (x+\alpha-1)f(x-1);$$

for

$x = 0, 1, 2, ...$

### 6.4.2 Exponential Distribution

$$g(\lambda) = \mu e^{-\mu\lambda}; \lambda > 0$$

$$L(s) = \frac{\mu}{s+\mu}$$

$$L^{(x)}(s) = \frac{(-1)^x x!\mu}{(s+\mu)^{x+1}}$$

$$f(x) = \left(\frac{\mu}{1+\mu}\right)\left(\frac{1}{1+\mu}\right)^x; x = 0, 1, 2, ...$$

### 6.4.3 Gamma Distribution with one parameter

$$g(\lambda) = \frac{e^{-\lambda}\lambda^{\alpha-1}}{\Gamma(\alpha)}; \lambda > 0, \alpha > 0$$

$$L(s) = \frac{1}{(s+1)^{\alpha}}$$

$$L^{(x)}(s) = (-1)^{x} \frac{(\alpha+x-1)!}{(\alpha-1)!} \frac{1}{(s+1)^{\alpha+x}}$$

$$f(x) = \binom{x+\alpha-1}{x} \left(\frac{1}{2}\right)^{\alpha} \left(\frac{1}{2}\right)^{x}; x = 0, 1, 2, ...$$

$$f(x+1) = \frac{1}{2}\left(\frac{x+\alpha}{x+1}\right) f(x); x = 0, 1, 2, ...$$

### 6.4.4 Gamma Distribution with two parameters

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta\lambda}\lambda^{\alpha-1}; \lambda > 0, \alpha > 0, \beta > 0$$

$$L(s) = \left(\frac{\beta}{\beta+s}\right)^{\alpha}$$

$$L^{(x)}(s) = \frac{(-1)^{x}\beta^{\alpha}}{(\beta+s)^{\alpha+x}} \frac{(\alpha+x-1)!}{(\alpha-1)!}$$

$$f(x) = \binom{x+\alpha-1}{x} \left(\frac{\beta}{1+\beta}\right)^{\alpha} \left(\frac{1}{1+\beta}\right)^{x}; x = 0, 1, 2, ...$$

$$f(x+1) = \left[\frac{(x+\alpha)}{(x+1)(1+\beta)}\right] f(x); x = 0, 1, 2, ...$$

### 6.4.5 Inverse Gamma Distribution

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} \frac{e^{-\frac{\beta}{\lambda}}}{\lambda^{\alpha+1}}; \lambda > 0, \alpha > 0, \beta > 0$$

$$x(x+1) f(x+1) = (x-\alpha) x f(x) + \beta f(x-1); x = 0, 1, 2, ...$$

### 6.4.6 Inverted Beta Distribution

$$g(\lambda) = \frac{\lambda^{\alpha-1}}{B(\alpha, \beta)(1+\lambda)^{\alpha+\beta}}; \lambda > 0, \alpha > 0, \beta > 0$$

$$x(x+1) f(x+1) = x(x-\beta-1) f(x) + (x+\alpha-1) f(x-1);$$

for

$x = 0, 1, 2, ...$

### 6.4.7 Lomax Distribution

$$g(\lambda) = \frac{\alpha k^{\alpha}}{(\lambda+k)^{\alpha+1}}; \lambda > 0$$

$$f(x) = \alpha \beta^{x} \Psi(x+1, x-\alpha+1; \beta)$$

$$(x+1) f(x+1) = (x-k-a) f(x) + k f(x-1); x = 0, 1, 2, ...$$

### 6.4.8 Lindley Distribution

$$g(\lambda) = \frac{\theta^2}{(\theta+1)} (\lambda+1) e^{-\lambda\theta}; \lambda > 0, \theta > 0$$

$$f(x) = \frac{\theta^2 (\theta+2+x)}{(1+\theta)^{x+3}}; x = 0, 1, 2, ...$$

### 6.4.9 Linear Exponential Family

$$g(\lambda) = \beta(\theta) e^{\lambda\theta} h(\lambda); h(\lambda) \geq 0, -\infty < \theta < \infty, \lambda > 0$$

$$f(x) = \frac{\beta(\theta)}{x!\beta(\theta-1)} \mu_x^{'}(\theta-1); x = 0, 1, 2, ...$$

where $\mu_x^{'}(\theta-1)$ is the raw moment of order $X$ of the Linear Exponential Family with parameter $(\theta-1)$.

### 6.4.10 Inverse Gaussian Distribution

$$g(\lambda) = \left(\frac{\phi}{2\pi\lambda^3}\right)^{\frac{1}{2}} \exp\left\{-\frac{\phi(\lambda-\mu)^2}{2\mu^2\lambda}\right\}; \lambda > 0, \mu > 0 \text{ and } \phi > 0$$

$$\left(1 + \frac{\phi}{2\mu^2}\right) x f(x+1) = x f(x) + \frac{\phi}{2} \frac{f(x-1)}{x+1}; x = 0, 1, 2, ...$$

### 6.4.11 Rectangular Distribution

$$g(\lambda) = \frac{1}{b-a}; a \leq \lambda \leq b$$

$$f(x+1) = f(x) + \frac{1}{(x+1)!\,(b-a)}\left(e^{-a}a^{x+1} - e^{-b}b^{x+1}\right); x = 0, 1, 2, ...$$

### 6.4.12 Gamma Distribution with four parameters

$$g(\lambda) = \frac{\alpha^{m-\delta}e^{-\alpha\lambda}\lambda^{m-1}}{\Gamma_\delta(m, \alpha n)(\lambda+n)^\delta}; \lambda \geq 0, m > 0, \alpha > 0, n > 0, \delta \geq 0$$

$$(\alpha+1)\,x\,(x+1)\,f(x+1) = [x+m-\delta-(\alpha+1)\,n]\,xf(x) + (x+m-1)\,nf(x-1); x \geq 1$$

and

$$f(0) = \left(\frac{\alpha}{1+\alpha}\right)^{m-\delta}\frac{\Gamma_\delta(m, (1+\alpha)\,n)}{\Gamma_\delta(m, \alpha n)}$$

### 6.4.13 Generalized Lindley Distribution

$$g(\lambda) = \frac{\theta^2(\theta\lambda)^{\alpha-1}(\alpha+\lambda)e^{-\theta\lambda}}{(\theta+1)\,\Gamma(\alpha+1)}; \lambda > 0, \theta > 0, \alpha > 0$$

$$f(x) = \frac{\theta^{\alpha+1}\Gamma(x+\alpha)}{x!\,(\theta+1)^{x+\alpha+1}\,\Gamma(\alpha+1)}\left[\alpha + \frac{(x+\alpha)}{(\theta+1)}\right]; x = 0, 1, 2, ...; \theta > 0, \alpha > 0$$

### 6.4.14 Generalized Inverse Gaussian Distribution

$$g(\lambda) = \frac{\eta^{-\gamma}\lambda^{\gamma-1}}{2K_{\gamma}(\omega)} \exp\left\{-\frac{\omega(\eta^2+\lambda^2)}{2\eta\lambda}\right\}; \lambda \geq 0, -\infty < \gamma < \infty, \eta > 0, \omega \geq 0$$

where $K_{\gamma}(\omega)$ is the modified Bessel Function of the third kind with index $\gamma$.

$$(2\eta+\omega)x(x+1)f(x+1) = 2\eta(x+\gamma)xf(x) + \omega\eta^2 f(x-1); x = 0, 1, 2, ...$$

### 6.4.15 Generalized Pareto Distribution

$$g(\lambda) = \frac{\mu^{\alpha}\lambda^{\beta-1}}{B(\alpha,\beta)(\mu+\lambda)^{\alpha+\beta}}; \lambda \geq 0, \alpha > 0, \beta > 0, \mu > 0$$

$$x(x+1)f(x+1) = x(x-\mu-\alpha)f(x) + \mu(x+\beta-1)f(x-1);$$

for

$x = 0, 1, 2, ...$

### 6.4.16 Generalized Pareto Distribution

$$g(\lambda) = \frac{1}{\delta}\left[1+\frac{\xi\lambda}{\delta}\right]^{-\frac{1}{\xi}-1}; \lambda > 0, \xi > 0, \delta > 0$$

$$f(x) = \frac{\delta^2}{\xi^{x+1}}\Psi\left(x+1, x-\frac{1}{\xi}+1; \frac{\delta}{\xi}\right)$$

### 6.4.17 Shifted Gamma Distribution

$$g(\lambda) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} e^{-\beta(\lambda-\mu)} (\lambda-\mu)^{\alpha-1}; \lambda > 0, \alpha > 0, \beta > 0, \mu \gtrless 0$$

$$f(x) = \sum_{k=0}^{x} \frac{e^{-\mu}\mu^{x-k}}{(x-k)!} \frac{\Gamma(k+\alpha)}{\Gamma(k+1)\Gamma(\alpha)} \left(\frac{\beta}{1+\beta}\right)^{\alpha} \left(\frac{1}{1+\beta}\right)^{k}$$

### 6.4.18 Truncated Gamma Distribution

$$g(\lambda) = \frac{\beta^{\alpha} e^{-\beta\lambda} \lambda^{\alpha-1}}{\gamma(\alpha, b\beta) - \gamma(\alpha, a\beta)}; 0 < a < \lambda < b < \infty, \alpha > 0, \beta > 0$$

$$f(x) = \frac{\beta^{\alpha}}{x!(\beta+1)^{x+\alpha}} \frac{\gamma[x+\alpha, b(\beta+1)] - \gamma[x+\alpha, a(\beta+1)]}{\gamma(\alpha, b\beta) - \gamma(\alpha, a\beta)}; x \neq 0, 1, 2, \ldots$$

### 6.4.19 Zero-Truncated Normal Distribution

$$g(\lambda) = \frac{1}{\phi\left(\frac{\mu}{\sigma}\right)\sqrt{2\pi\sigma^2}} \exp\left[-\frac{(\lambda-\mu)^2}{2\sigma^2}\right]; 0 < \lambda < \infty, -\infty < \mu < \infty, \sigma^2 > 0$$

$$f(x) = \frac{1}{x!2\sqrt{\pi}} \frac{1}{\phi\left(\frac{\mu}{\sigma}\right)} \exp\left[-\left(\mu - \frac{\sigma^2}{2}\right)\right] \left(\mu - \sigma^2 + \sqrt{2\sigma^2}\right)^{x}$$
$$\sum_{r=0}^{x} \binom{x}{r} p^r (1-p)^{x-r} \left\{\Gamma\left(\frac{r+1}{2}\right)\left[1 - \Gamma\left(\frac{r+1}{2}\right)\right]\right\}$$

where

$$p = \frac{\sqrt{2\sigma^2}}{\mu - \sigma^2 + \sqrt{2\sigma^2}} \text{ and } 1 - p = \frac{\mu - \sigma^2}{\mu - \sigma^2 + \sqrt{2\sigma^2}}$$

$$(x+2)\,f(x+2) = \left(\sigma^2+\mu\right) f(x+1) - \sigma^2 f(x)\,; x = 0,1,2,...$$

### 6.4.20 Scaled Beta Distribution

$$g(\lambda) = \frac{\lambda^{\alpha-1}(\mu-\lambda)^{\beta-1}}{B(\alpha,\beta)\,\mu^{\alpha+\beta-1}}; 0 \le \lambda \le \mu$$

$$x(x+1)\,f(x+1) = x(x+\alpha+\beta+\mu-1)\,f(x) - \mu(x+\alpha-1)\,f(x-1);$$

for

$x = 0,1,2,...$

### 6.4.21 Confluent Hypergeometric Distribution

$$g(\lambda) = \frac{e^{-k\lambda}\lambda^{a-1}}{\Gamma(a)\,\varphi(a,c;k)\,(1+\lambda)^{a-c+1}}$$

for $\lambda > 0$, $c > a > 0$ and

$$\varphi(a,c;k) = \frac{1}{\Gamma(a)} \int_0^\infty \frac{e^{-kt}t^{a-1}}{(1+t)^{a-c+1}} dt$$

$$(a-c+1)\,x(x+1)\,f(x+1) = (k+1)\,xf(x) - (x+a-1)\,f(x-1);$$

for

$x = 0,1,2,...$

## 6.5 Routes to Poisson Mixtures via Continuous Mixing distributions

Figure 6.3: Direct Route

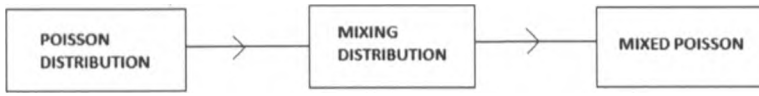


Figure 6.4: Expectation Route

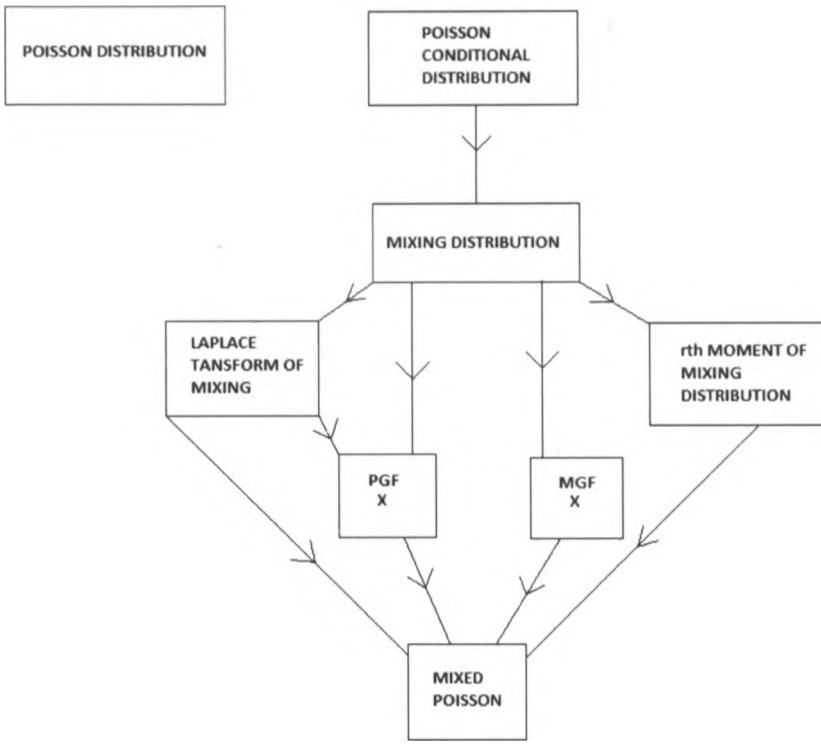
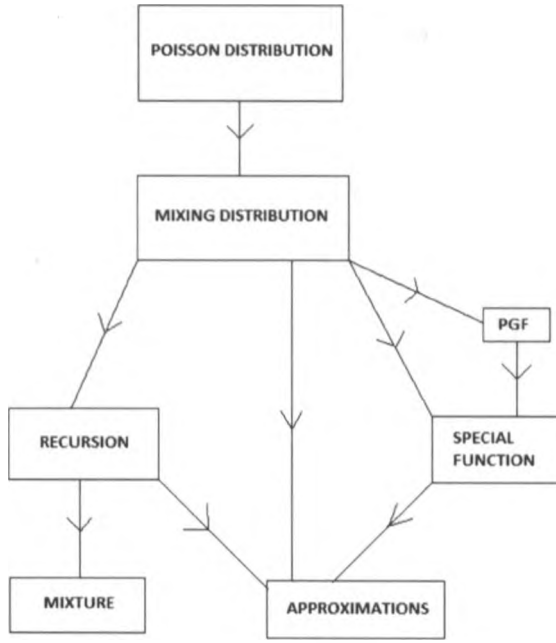


Figure 6.5: Approximation Route



## 6.6 Recommendations

The construction of Mixed Poisson distributions was not exhausted in this research. Therefore more work can be done using the methods of construction already used and also other methods can be studied or researched on.

Mixed Poisson distributions exhibit several interesting properties as given by Karlis and Xekalaki, (2005). These properties include; Identifiability, Modality and Shape properties, Infinite divisibility, Posterior Moments, etc. The study of these properties can form a good basis of further research on the Mixed Poisson distributions constructed in this paper.

In this study, only univariate Mixed Poisson distributions are considered but multivariate Mixed Poisson distributions are of equal interest to a researcher, hence, research can be carried out on the multivariate case.

This work concentrated on purely construction. There is therefore need to examine the following for each mixed Poisson distribution obtained:

(i) the behaviour of the mixed distribution

(ii) its properties

(iii) estimation

(iv) testing of hypothesis

(v) areas of application

There is also need to fill gaps that exist in Table 6.1.

(vi) Extending the work to other distributions in particular the class of densities in the (0,1) domain, truncated densities and densities based on special functions.

# REFERENCES


[1] Albrecht, P. (1984). Laplace Transforms, Mellin Transforms and Mixed Poisson Processes. Scandinavian Atuarial Journal , 11, 58-64.

[2] Antzoulakos, D.L. and S.Chadjiconstantinidis. (2004). On Mixed Poisson and Compound Mixed Poisson Distributions. Scandinavian Actuarial Journal , 161-188.

[3] Ashford, J.R. and R.G. Hunt. (1974). The Distribution of Doctor-Patient Contacts in the National Health Service. JRSS A , 137, 347-383.

[4] Bhattacharya, S.K. and M.S. Holla. (1965). On a Discrete Distribution with Special Reference to the Theory of Accident Proneness. JASA , 60, 1060-1066.

[5] Brown, S. and P. Holgate. (1971). Table of the Poisson-Lognormal Distribution. Sankhya B , 33, 235-248.

[6] Bulmer, M.G. (1974). On Fitting the Poisson Lognormal Distribution to Species Abundance Data. Biometrics , 30, 101-110.

[7] Burrel, Q. and V. Cane. (1982). The Analysis of Library Data. JRSS A , 145, 439-471.

[8] Chadjiconstantinidis, S. & Antzoulakos, D. (2002). Moments of Compound mixed Poisson distributions. Scandinavian Actuarial Journal , 3, 138-161.

[9] Chen, C., Randolph, P. & Liou, T. (2005). Using CUSUM Control Schemes for monitoring quality level in compound poisson production environment. The Geometric Poisson Process, Quality Engeneering , 17:2, 207-217.

[10] Cook, R. J. (2003). Conditional analysis of Mixed poisson processes with baseline counts: implications for trial design and analysis. Biostatistics , 4,3, 479-494.

[11] Greenwood, M. and G. Yule. (1920). An Inquiry into the Nature of Frequency Distributions Representative of Multiple Happenings with Particular Reference to the Occurrence of Multiple Attacks of Disease or of Repeated Accidents. JRSS A , 83, 255-279.

[12] Gupta, R. C. & Ong, S. H. (2005). Analysis of long-tailed count data by poisson mixtures. Communications in Statistics - Theory and Methods , 34:3, 557-573.

[13] Gurland, J. (1958). A Generalized Class of Contagious Distributions. Biometrics , 14, 229-249.

[14] Hegyi, S. (1998). H-Function extension of the NBD: further applications. Physics Letter B , 417, 186-192.

[15] Hess, K. T.; Liewald, A. and Schmidt, K. D. (2002). An extension of Panjer's recursion. ASTIN Bulletin, 32(2): 283-297.

[16] Hesselager, O. (1994). A Recursive Procedure for Calculation of some Compound Distributions. ASTIN Bulletin , 24, 19-32.

[17] Hougaard, P.; M.L.T. Lee and G.A. Whitmore. (1997). Analysis of Overdispersed Count Data by Mixtures of Poisson Variables and Poisson Processes. Biometrics , 53, 1225-1238.

[18] Johnson, N.L.; S. Kotz and A.W. Kemp. (1992). Univariate Discrete Distributions. New York: John Wiley and Sons .

[19] Karlis, D. and E. Xekalaki. (2005). Mixed Poisson Distribution. International Statistical Review , 73, 35-58.

[20] Katti, S. K. (1966). Interrelations among generalized distributions and their components. Biometrics , 22, 44-52.

[21] Katz, L. (1965). Unified treatment of a broad class of discrete probability distributions. In "Classical and Contagious Discrete Distributions", Pergamon Press, Oxford, 175-182.

[22] Kempton, R.A. (1975). A Generalized Form of Fisher's Logarithmic Series. Biometrika , 62, 29-38.

[23] Klugman, S. A.; Panjer, H. H. and Willmot, G. E. (1998). Loss Models: From Data to Decisions, Wiley, New York.

[24] Li, S. (2006). The distribution of the dividend payments in the compound poisson risk model perturbed by diffusion. Scandinavian Actuarial Journal , 2, 73-85.

[25] Lindley, D. V. (1958). Fiducial distributions and Bayes' theorem. J. Roy. Statist. Soc. , B 20, 102-107.

[26] Mahmoudi, E. & Zakerzadeh, H. (2010). Generalized Poisson - Lindley distribution. Communications in Statistics - Theory and Methods , 39:10, 1785-1798.

[27] McFadden, J. A. (1965). The Mixed Poisson Process. Sankhya: The Indian Journal of Statistics, Series A , 27, 83-92.

[28] Nadarajah, S. & Kotz, S. (2006). Compound Mixed Poisson distributions I. Scandinavian Actuarial Journal , 3, 141-162.

[29] Nadarajah, S. & Kotz, S. (2006). Compound Mixed Poisson distributions II. Scandinavian Actuarial Journal , 3, 163-181.

[30] Ong, S. H. (1998). A note on the mixed poisson formulation of the Poisson - Inverse Gaussian distribution. Communications in Statistics - Simulation and Computation , 27:1, 67-78.

[31] Panjer, H. and Wang, S. (1995). Computational aspects of Sundt's Generalized class. ASTIN Bulletin , 25(1), 5-17.

[32] Panjer, H. H. and Willmot, G. E. (1982). Recursions for Compound distributions. ASTIN Bulletin , 13, 1-11.

[33] Panjer, H. H. and Willmot, G. E. (1992). Insurance Risk models. Society of Actuaries, Schaumburg, Illinois.

[34] Panjer, H. H. (1981). Recursive evaluation of a family of compound distributions. ASTIN Bulletin , 12, 22-26.

[35] Patil, G.P. (1964). On Certain Compound Poisson and Compound Binomial Distributions. Sankhya A , 27, 293-294.

[36] Ruohonen, M. (1988). On a Model For the Claim Number Process. ASTIN BULLETIN , 18, 57-68.

[37] Sankaran, M. (1968). Mixtures by the Inverse Gaussian Distribution. Sankhya B , 30, 450-458.

[38] Sankaran, M. (1969). On Certain Properties of a Class of Compound Poisson Distributions. Sankhya B , 32, 353-362.

[39] Sankaran, M. (1970). The Discrete Poisson - Lindley Distribution. Biometrics , 26, 145-149.

[40] Sichel, H.S. (1975). On a Distribution Law for Word Frequencies. JASA , 70, 542-547.

[41] Sichel, H.S. (1974). On a Distribution Representing Sentence-Length in Written Prose. JRSS A , 137, 25-34.

[42] Sichel, H.S. (1982). Repeat - buying andd teh generalized inverse Gaussian - Poisson distribution. Appl. Statist. 31, 193-204

[43] Simon, P. (1955). On a Class of Skew Distributions. Biometrika , 42, 425-440.

[44] Steutel, F. & Van Harn, K. (1979). Discrete Analogues of self-decomposability. The Annals of Probability , 7, 893-899.

[45] Sundt, B. & Jewell, W. S. (1981). Further results on recursive evaluation of compound distributions. ASTIN Bulletin, 12.

[46] Sundt, B. (1992). On some Extensions of Panjer's class of distributions. ASTIN Bulletin , 22, 61-80.

[47] Wang, S. (1994). Further results on Hesselager's Recursive procedure for calculation of some compound distributions. ASTIN Bulletin , 24, 161-166.

[48] Willmot, G. E. (2011). On mixing, compounding and tail properties of a class of claim number distributions. Scandinavian Actuarial Journal , 1-21.

[49] Willmot, G. E. (1993). On recursive evaluation of mixed poisson probabilities and related quantities. Scandinavian Actuarial Journal , 2, 114-133.

[50] Willmot, G. E. (1987). Sundt and Jewell's family of discrete distributions. ASTIN Bulletin, 18, 17-29.

[51] Willmot, G., Drekic, S. & Cai, J. (2005). Equilibrium compound distributions and stop-loss moments. Scandinavian Actuarial Journal , 6-24.

[52] Willmot, G. E. and Panjer, H. H. (1987). Difference equation approaches in evaluation of Compound distributions. Insurance Math and Econ, 6, 43-56.

[53] Willmot, G.E. (1986). Mixed Compound Poisson Distribution. ASTIN Bulletin Supplement , 16, S59-S79.

[54] Zakerzadeh, H. & Dolati, A. (2010). Generalized Lindley distribution. Journal of Mathematical Extension .

# PREVALENCE AND RISK FACTORS OF CONDITIONS CAUSING LAMENESS IN SHEEP UNDER FREE RANGE GRAZING SYSTEM IN KAJIADO DISTRICT, KENYA.

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October 2011

## DECLARATION

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

02/11/2011

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This thesis has been submitted for examination with our approval as University supervisors

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# DEDICATION

To

My wife, Lucy and my children Teresiah and Timothy.

To God is all the glory and honour.

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## ACKNOWLEDGEMENTS

First, I sincerely thank the Almighty God for giving me the opportunity to undertake the postgraduate studies. I experienced divine protection and grace throughout the project period. Therefore, all glory and honour to Him forever and ever. Amen.

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## ABBREVIATIONS AND SYMBOLS

| | | |
|---|---|---|
| ASAL | - | Arid and Semi-Arid Lands |
| CODD | - | Contagious Ovine Digital Dermatitis |
| DEFRA | - | Department for Environment Food and Rural Affairs |
| FMD | - | Foot and Mouth Disease |
| GDP | - | Gross Domestic Product |
| NADIS | - | National Animal Diseases Information Service |
| VEIN | - | Veterinary Education and Information Network |
| Km | - | Kilometer |
| mm | - | Millimeters |
| df | - | Degrees of freedom |
| *et al* | - | At least three authors |
| P | - | P value |
| < | - | Greater than |
| > | - | Less than |
| ( | - | Opening bracket |
| ) | - | Closing bracket |
| % | - | Percentage |
| / | - | Division sign |

## ABSTRACT

Lameness can be a cause of negative economic output in sheep farming owing to its adverse effects on productivity, reproductive performance and poor growth performance in lambs. The extent of lameness in sheep and its associated predisposing causes in Kenyan sheep rearing systems has not been elucidated. This study was carried out in sheep under free-range grazing system in Kajiado District, Kenya, from March 2010 to June 2010 with the following objectives (1) to determine the prevalence of conditions causing lameness, (2) to determine the possible risk factors predisposing the sheep to lameness conditions.

This was a cross-sectional study in which each farm was visited several times, but each sheep on the farm was examined only once. Ten study farms were purposively selected from three divisions of Kajiado District, based on the willingness of the farmers to allow examination of their sheep and also on the stability of the farm's grazing routine from more nomadic tendencies. The data was collected either by filling a formal questionnaire with answers given through interviewing the farmers, farm managers or stockmen on animal-level factors, or by recording observations made on the farm regarding farm-level factors. The 10 farms had a total of 1916 sheep that met the study criteria. Out of these, 117 sheep were identified as lame during general locomotion scoring as they walked on a flat firm part of the ground. Each of these 117 sheep were examined closely for specific conditions or disorders causing lameness. Information on the actual disorders causing lameness was recorded. The location of the disorders on the limb, affected limbs whether fore or hind, and the affected claws whether lateral or medial were recorded in data

collection sheets. The lesions causing lameness were photographed. A mark was put on each examined sheep to avoid repeat examination.

Overall prevalence of lameness was 6.1% (117/1916), out of which the conditions with relatively higher percentages of occurrence were sole erosion (3.8%, 72/1916), overgrown claws (3.2%, 61/1916) and tick-bite dermatitis (1.6%, 30/1916). Infective conditions such as foot rot and interdigital dermatitis had prevalence of less than 1%. The rest of the conditions such as shelly hoof, soil-balling, over-trimming and bone problems were incidental findings each in a single sheep. The conditions causing lameness occurred on the foot in 94% (110/117) of the lame sheep and on proximal parts of the limb in 6% (7/117) of the cases. The distribution of the conditions among the lame sheep was 43.6% (51/117) on the hind limbs, 23.1% (27/117) on the forelimbs and 33.3% (39/117) affected both hind and fore limbs.

Although there were several animal-level factors evaluated, the only factors found to be significantly associated with higher locomotion score were the number of limbs with lesions ($\chi^2$ =11.15, p = 0.004), the affected limbs whether fore or hind ($\chi^2$ = 9.20, p = 0.010), the affected claw whether medial or lateral ($\chi^2$ = 16.98, p = 0.05) and the type of lesion ($\chi^2$ = 4.71, p = 0.030). The only farm-level factor that was significantly associated with higher locomotion score was presence of traumatic objects in the grazing grounds ($\chi^2$ = 11.01, p < 0.001).

This study concludes that the prevalence of lameness in sheep under free-range grazing system of dry zones such as Kajiado District is relatively low due to minimal farm-level risk factors. Similar prevalence studies should be carried out in high potential and wet areas of Kenya for comparison purposes.

# CHAPTER 1

## 1.0 INTRODUCTION

The livestock sector in Kenya contributes about 10% of the entire Gross Domestic Product (GDP) and 42% of the agricultural GDP (National Livestock Policy, 2008). Only one third of Kenya's land is suitable for agriculture while two-thirds is both arid and semi-arid (ASAL) in which the larger livestock population is reared. The arid and semi-arid lands support the pastoral communities in Kenya through livestock rearing which is the main source of their livelihood (Kariuki and Letitiya, 1996). The population of sheep in Kenya is estimated at about 17,129,606 million, most of which is under free-range grazing nomadic pastoralism and ranching systems (Kenya National Population and Housing Census Results, 2010).

Lameness is a major health problem in flocks of sheep worldwide. It is mostly associated with foot lesions (Gelasakis *et al.*, 2009). It impacts negatively on both welfare and economic productivity of individual sheep and entire flocks. Some of the negative effects of lameness include reduced weight gain, reduced birth weight of lambs, poor colostrum production by ewes and reduced reproductive performance (Henderson, 1990; Harwood *et al.*, 1997; Eze, 2002; DEFRA, 2003a). Lameness in sheep may be caused by many systemic and localized diseases, the commonest being foot rot, interdigital dermatitis, foot abscess and septic polyarthritis (Radostitis *et al.*, 2001; Vermunt and West, 2004; VEIN, 2008; The Merck Veterinary Manual, 2009). During the period between 1995 and 2008, annual reports of the Department of Veterinary Services in Kenya indicate that foot and mouth disease, black quarter, blue tongue, foot rot, fractures and arthritis are some of

the prevalent diseases contributing to sheep lameness (Department of Veterinary Services Annual Reports, 1995-2008).

The risk factors of lameness in sheep include; wetness of the environment, wet season, size and conformation of hooves, limb conformational defects and interdigital tick infestation (Bokko *et al.*, 2003; Azizi and Yakhchali, 2006).

This study was carried out in sheep under free-range grazing system with the purpose of determining the prevalence of conditions causing lameness and the possible risk factors predisposing the sheep to lameness conditions.

### 1.1 Justification

The status of lameness in sheep in Kenya is not known since no studies have been carried out previously. Sheep production forms part of the main livelihood of the pastoral communities in arid and semi-arid areas of Kenya and therefore a systematic study to establish the status of lameness was essential. The results of the study may give guidance for remedial and preventive measures and hence improve productivity of sheep and enhance the livelihood of these communities.

### 1.2 Objectives of the study

The study was therefore carried out with the following specific objectives:

1.2.1 To determine the prevalence of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya.

1.2.2 To determine the risk factors predisposing sheep to lameness conditions under free-range grazing system in Kajiado District, Kenya.

# CHAPTER 2

# 2.0 LITERATURE REVIEW

## 2.1. General overview and economic importance of lameness in sheep

Locomotion soundness is very vital for effective grazing, reproductive and production efficiency in all classes of livestock (Bokko *et al.*, 2003). Lameness is the alteration of gait and / or posture as a result of disease, limb disorders or trunk disorders. It is abnormal gait as a clinical sign, but not a disease in itself (Coulon *et al.*, 1996; Warnick *et al.*, 2001; Green *et al.*, 2002; Winter, 2004a; The Merck Veterinary Manual, 2009). Lameness is considered to be one of the most important health problems in sheep (Marshall *et al.*, 1991) and is an indication of pain, weakness, deformity, or other abnormalities in the musculo-skeletal system (The Merck Veterinary Manual, 2009). It can be divided into proximal limb lameness and foot lameness depending on the location of the lesion. Proximal limb lameness occurs when lesions are proximal to the fetlock joint, while foot lameness occurs when lesions are distal to the fetlock joint. The former has a lower prevalence rate compared to the latter (Hungerford, 1990).

Overgrown hooves, trauma, interdigital pouch inflammation, limb conformational defects, scalds, tick-bite dermatitis and fractures were reported to be among the causes of lameness in sheep in the arid zones of Nigeria but hoof overgrowth had the highest incidence (Eze, 2002; Bokko *et al.*, 2003). Ticks attached to the interdigital skin may cause lameness due to tissue damage and inflammatory reactions caused by their long mouth parts (Azizi and Yakhchali, 2006).

Lameness is also a major cause of economic loss in sheep as a result of poor or reduced production (Gatenby, 1986). The loss in production occurs through reduced weight gain in the fattening lambs, reduced wool growth and inadequate feed intake by the pregnant and lactating ewes resulting in pregnancy toxemia and neonatal diseases (Eze, 2002; DEFRA, 2003a). Lameness also affects reproduction by increasing the lambing interval and lowering of the ram's fertility (DEFRA, 2003a). The affected sheep have a significant fall in body weight and wool production during the period of lameness (Radostitis *et al.*, 2001). However the economic implication of lameness is difficult to quantify (Eze, 2002). Lameness is an important welfare determinant because it causes pain and discomfort (Offer *et al.*, 2000; DEFRA, 2003b). A survey carried out in the United Kingdom by the Royal Veterinary College established that the incidence of lameness in 547 farms was between 6 and 11% of all the sheep (DEFRA, 2003a). In Nigeria the incidence of lameness in sheep was found to be 15% (Eze, 2002).

**2.2 Normal functional anatomy of the ovine digit**

For purposes of description of lameness, the limb is divided into "proximal limb" and the "foot". Proximal limb is all parts of the limb proximal to the fetlock joint. The foot is all parts of the limb distal to the fetlock joint. The foot in the ovine is divided into two main digits and two accessory digits (dew claws). Each of the main digits is made up of three phalanges namely the proximal (P1), middle (P2) and distal (P3) phalanges. The ends of the digits are called "claws" or "hooves" and are covered by the horn capsule termed as the "hoof." The distal phalanx is inside the claw horn while the other two are outside the claw horn. The foot has two joints, which are the proximal interphalangeal joint (pastern

joint) and distal interphalangeal joint (coffin or pedal joint). The space between the two main digits is called the "interdigital space" which is made up of interdigital skin to which ticks attach and cause inflammation. The interdigital space can also accumulate dung (manure). These factors occurring in the interdigital space serve as predisposing causes for foot lameness (Berry, 1999; Clarkson and Faulli, 1990).

The claw is made up of the wall, sole, heel and white line. The claw wall refers to the hard horny structure (hoof) encasing the distal part of the digit on the dorsal, abaxial and the axial aspects. The walls particularly the dorsal and abaxial aspects are harder than the sole and the heel. The white line is the junction between the sole and the wall, while the coronet is the junction between the hoof wall and the skin. The horn is the epidermis of the claw while the corium is the dermis which contains the nerves and the vasculature. The corium produces the horn of the claw and so its damage results in defective horn production, which may lead to lameness. The main weight-bearing surfaces of the claw are the sole and the heel. The hardest parts of the claw that should naturally bear weight are the abaxial wall and the sole (Berry, 1999).

**2.3 Aetiology and predisposing factors of lameness**

The prevalence, type and severity of lameness in ruminants seem to vary from one region to another due to the prevailing predisposing factors in the region (Russell *et al.*, 1982). Foot lameness is considered to have multifactorial predisposing causes. Some of these are metabolic disturbances, trauma to the musculoskeletal system, lack of proper feet care and infections which are either systemic or localized to the limbs. The interdigital skin is

the primary site of invasion by infection, but this does not occur when the stratum corneum is dry and intact (Greenough, 1991; The Merck Veterinary Manual, 2009). Generally, the predisposing factors can be divided into three categories which include environmental factors, animal- and management-level factors.

### 2.3.1 Environmental factors

Environmental factors affect the prevalence of lameness within the flocks of sheep. Prevalence of lameness in a flock of sheep varies largely with pasture environment that can affect the feet (Clarkson and Ward, 1991). Lameness in sheep is more prevalent during the wet season and in the hind limbs (Mgasa and Arnbjerg, 1993). Wet environment causes softening of the hoof and maceration of interdigital skin, thus making it easy for penetration of foreign bodies and infection (Jubb and Malmo, 1991; Tranter *et al.*, 1993). For example sole erosion has a higher incidence during the rainy season than in dry season (Mgasa and Arnbjerg, 1993). Wetness of the pasture and animal rearing environment also favours proliferation of infectious agents especially *Fusobacterium necrophorum* and *Dichelobacter nodosus*, "the main causes of foot rot in sheep" (Greenough and Vermunt, 1991). Dry environment is cleaner and hygienic, hence reduces the incidence of foot lesions (Bergsten and Petterson, 1992). However, dry weather leads to desiccation of the hoof, which makes the horn hard, brittle and liable to cracking (Greenough, 1991). It has been reported that housing sheep greatly increases the incidence of lameness unless good husbandry practices are observed (Pugh, 2002).

### 2.3.2 Animal-level factors

Genetic factors attributed to individual animals predispose sheep to lameness (Gelasakis *et al.*, 2009). The incidence of lameness is higher in sheep that are less than four years of age. It decreases with age but the degree of lameness is more severe. Conformational defects which could have a genetic or inheritance factor in them also predispose to lameness (Bokko *et al.*, 2003).

### 2.3.3 Management-level factors

The management practices in the farms help to prevent or to treat the conditions that cause lameness, thus maintain and improve the efficiency of production (DEFRA, 2003a). Occurrence of lameness due to digital diseases in goats has been found to be related not only to climatic conditions but also to management factors (Nonga *et al.*, 2009). It has been reported that failure to practice foot-bathing and hoof trimming results in increased incidence of lameness in livestock (Arkins, 1981; Davis, 1982). Trimming of the hooves helps in the control of many of the lesions causing lameness (Tadich and. Hernández, 2000).

Other management factors that are associated with lameness include high stocking densities, failure to practice rotational grazing, lack of grass or concrete run, failure to add bacteriostats to dips and lack of mineral supplement (DEFRA, 2003a; Gelasakis *et al.*, 2009). Poorly maintained farm tracks with loose stones and trenches as well as overdriving of the animals by stockmen when herding them increase the risk to lameness (Clarkson and Ward, 1991). Nutrition is a fundamental factor associated with the health

of the foot and the animal in general. Hence, sheep fed on unbalanced rations suffer deficiencies in specific nutrients such as zinc that is involved in the keratinization of hoof wall and this could predispose to lameness (Gelasakis *et al.*, 2009).

### 2.4 Specific conditions causing lameness in sheep

The most common causes of lameness in sheep are infectious which could be systemic or localized in the foot, injuries and nutritional imbalances (The Merck Veterinary Manual, 2009). In this part of the literature, the frequently encountered conditions causing lameness are discussed.

#### 2.4.1 Foot rot

Foot rot is a highly contagious disease of sheep caused by dual infection with *Fusobacterium necrophorum* and *Dichelobacter nodosus* (Radostitis *et al.*, 2001), which are gram negative and anaerobic. *Fusobacterium necrophorum* is a normal residence of the sheep's environment, but *Dichelobacter nodosus* does not survive for more than a few days in the soil or pastures. Its long-term presence depends on the presence of infected animals (The Merck Veterinary Manual, 2009). Foot rot is the main infectious cause of lameness in sheep. It is characterized by inflammation of the skin at the skin-horn junction with severe lameness and occasionally resulting in animals walking on their knees. There is interdigital dermatitis, under-running of the hoof, foul odour of necrosis of the horn and in some cases all the four feet are affected (Radostitis *et al.*, 2001; Vermunt and West, 2004). Foot rot is initially caused by *Fusobacterium necrophorum* which starts as scald and later *Dichelobacter nodosus* invade the lesion. There are

different strains of *Dichelobacter nodosus* which have varying virulence. The synergistic presence of *Fusobacterium necrophorum* and *Dichelobacter nodosus* causes separation of the horn from the underlying structures of the foot. Depending on the strain of *Dichelobacter nodosus* involved, this separation may spread under the entire sole and up the wall of the hoof (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009). In the farm, exposure of the feet to wet pasture, hydration and hyperkeratosis of the stratum corneum of the interdigital skin and invasion of interdigital skin by *Fusobacterium necrophorum* lead to development of interdigital dermatitis (The Merck Veterinary Manual, 2009).

Acute foot rot is characterized by swelling, moistness of skin of the interdigital cleft and slight lameness that increases as necrosis under-runs the horn of the cleft (The Merck Veterinary Manual, 2009). Extensive under-running of the horn leads to severe lameness whereby the sheep carry up the leg. When the under-running affects more than one foot the sheep walks on its knees or remains recumbent. There is also a foul smelling discharge. Severely affected sheep sometimes are anorexic. Both *Fusobacterium necrophorum* and *Dichelobacter nodosus* survive in pasture for up to 12 days under favourable conditions, hence rotational grazing and isolation of infected animals can help in control of the disease (Radostitis *et al.*, 2001).

Foot rot should be viewed as a flock problem (DEFRA, 2003a). Management of foot rot in sheep involves both topical and parenteral treatment. Treatment methods include isolation of severely affected sheep, careful hoof paring and topical application of

bactericidal solutions such as formalin, copper sulphate or zinc sulphate solution. In severe cases the long-acting antibiotics such as oxytetracycline should be administered. Culling of any sheep that do not respond easily to treatment will help reduce the likelihood of future infections. Vaccination of affected sheep with a bacterin composed of *Dichelobacter nodosus* cells helps in the prevention and control. Most of the affected sheep recover with adequate treatment and when treated early (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009).

### 2.4.2 Interdigital dermatitis

Interdigital dermatitis is caused by an early mild infection with *Fusobacterium necrophorum*. Injuries to the interdigital epidermis may also result in interdigital dermatitis. This disease often predisposes and progresses to foot rot and foot abscess (The Merck Veterinary Manual, 2009). Interdigital dermatitis is characterized by an acute inflammatory condition of the interdigital skin, which has moist necrotic material, pitting and blanching of the horn, maceration and necrosis at the skin-horn junction. This results in separation of the horn at the heel with limited under-running of the horn and no odour. There is mild lameness (Radostitis *et al.*, 2001). Excessive moisture and heavy dung contamination of the environment are the most important predisposing factors (West, 1990; Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009).

Interdigital dermatitis should be viewed as a flock problem due to the common predisposing factor (DEFRA, 2003a). Most lesions heal rapidly when sheep are transferred to dry conditions. Topical applications of aerosol antibiotics and foot bathing

cauterizing agents such as 5% formaldehyde or 10% Zinc sulphate solution are quite effective in the treatment of interdigital dermatitis (DEFRA, 2003a; The Merck Veterinary Manual, 2009).

### 2.4.3 Foot abscess

Foot abscess affects adult sheep especially pregnant ewes and rams. It is particularly common in sheep that are driven to the pasture through roads with stony areas. The main bacteria involved in causing foot abscess in sheep are *Fusobacterium necrophorum* and *Actinomyces pyogenes* (The Merck Veterinary Manual, 2009). Foot abscess is an acute, suppurative infection, usually involving one digit of the foot. In most cases infection enters into the interdigital space causing interdigital dermatitis and extends deeper into one of the digits to involve the distal interphalangeal joint, associated ligaments and eventually the tendons. It may occur as toe abscess in which there is under-mining of the horn at the toe. Pain is severe and there may be swelling of the coronet, with eventual rupture oozing purulent discharge (Radostitis *et al.*, 2001). There is acute lameness (The Merck Veterinary Manual, 2009). When the abscess is exposed, pus and sinus tracts are observed (Vermunt and West, 2004). The lesion could also occur as heel abscess that results from extension of interdigital dermatitis into the soft tissues of the heel. When the abscess spreads deeper to involve interphalangeal joints, there is severe swelling at the caudal aspect of the foot which could rupture to discharge pus. When the abscess ruptures, there is marked reduction in pain and the gait improves tremendously due to relief of pressure to the underlying tissues of the claw (Radostitis *et al.*, 2001).

Treatment by surgical drainage, parenteral administration of Sodium Sulfadimidine solution and application of a local dressing is usually adequate (Radostitis *et al.*, 2001). However once the infection becomes established in the joint, treatment is of limited value (The Merck Veterinary Manual, 2009).

### 2.4.4 Contagious Ovine Digital Dermatitis

Contagious ovine digital dermatitis (CODD) is a highly contagious, erosive and proliferative infection of the epidermis proximal to the skin-horn junction in the flexor region of the interdigital space. Morbidity within a flock can be more than 90%. It affects any breed or age group but young sheep and sheep with poor immune response are most susceptible. Both the erosive and the proliferative lesions cause varying degrees of discomfort and give rise to severe lameness (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009). The essential difference between conventional foot rot and CODD is that CODD lesion starts at the coronary band. The ulcerative and proliferative lesions progress to under-running of the claw with complete detachment of the hoof in severe cases. The cause of the condition is not yet understood, but a variety of bacteria, including *Spirochaetes* have been identified in affected feet. Effective treatment involves use of antibiotics and footbaths (DEFRA, 2003a).

### 2.4.5 Claw deformities

Claw deformities are conditions of the foot where the claw overgrows or grows abnormally and may either directly cause lameness or predispose to other foot lesions. The common claw deformities are overgrown hooves and conformational defects. The

hoof overgrowth is characterized by increased length of the wall or sole that results in misshapen claws (VEIN, 2008). In some of the deformities, it is difficult to reshape the affected claws even by trimming. The documented claw deformities include the following:

### 2.4.5.1 Regular hoof overgrowth

Regular hoof overgrowth occurs mostly when sheep are reared on soft surfaces where little hoof wear takes place. This results in increased length of the wall of the claw or sole (Rhebun and Pearson, 1982; Mohammed *et al.*, 1996).

### 2.4.5.2 Beak claw

This is a claw deformity in which the dorsal surface of the claw is concave while the weight bearing surface is convex. The toes are turned upwards. This condition is reported in cattle (Rhebun and Pearson, 1982).

### 2.4.5.3 Corkscrew claw

This is a claw deformity characterized by medial spiraling of the abaxial claw wall towards the axial plane of the normal claw. It is probably an inherited problem and trimming cannot reshape the claws to normal shape. This condition is reported mainly in cattle (Rhebun and Pearson, 1982).

### 2.4.5.4 Scissor feet

This is manifested as an overlapping of the toes. It has been reported to be an inherited condition. This is mainly a condition of cattle (Rhebun and Pearson, 1982).

Management of claw deformities includes routine inspection of the feet of all sheep should be carried out at regular intervals. Foot trimming should be done by a skilled person. This reshapes the claws and eliminates the cracks and crevices that could trap mud and harbour foot rot bacteria (DEFRA, 2003a).

### 2.4.6 Shelly hoof (white line degeneration)

Shelly hoof results from separation of the hoof wall close to white line at the toe and is common in sheep grazing on lush pasture. Aetiology is thought to be nutritional (Winter, 2004a). The outer wall of the claw becomes loosened, forming a pocket between the hoof and the digit. A cavity forms in the hoof and is filled with soil and dung. Bacteria may enter and lead to abscess formation. It results in acute lameness. Unless the infection is present, management involves paring the feet and cleaning the dung and soil out of the cavity but if infection is present or suspected, the sheep should be foot-bathed with either copper sulphate, zinc sulphate or formalin solution (DEFRA, 2003a; VEIN, 2008).

### 2.4.7 Soil balling

Soil-balling is impaction of the interdigital space with a mixture of grass and manure or soil. The grass is matted by manure and soil, eventually becoming a lump stuck in the interdigital space. This accumulation causes lateral separation of the toes that leads to

mild pain and moderate lameness. The sheep shows discomfort as it walks until the lump falls off or is removed (Clarkson and Faulli, 1990; Winter, 2004b). Management involves the removal of the lump and this may need to be softened with water or cracked into pieces prior to removal (DEFRA, 2003a).

### 2.4.8 Septic polyarthritis

Septic polyarthritis is an acute or chronic arthritis of several joints of the limbs in lambs mainly caused by *Erysipelothrix rhusiopathiae*. There is sequestration of bacterial infection in the joints of both fore and hind limbs. It mainly affects the carpal, tarsal and interphalangeal joints. This affects lambs with umbilical infections or infection after docking and castration. Septic polyarthritis is predisposed by poor body condition of lambs at the time of surgery or adverse weather afterwards (The Merck Veterinary Manual, 2009). It is characterized by local pain, heat and swelling of the affected joints with severe lameness (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009).

Septic arthritis requires prompt treatment to avoid irreparable damage. Systemic broad-spectrum antibiotics are indicated, which could be administered both systemically and intra-articularly. Joint lavage, arthroscopic debridement and drainage could be done. Supportive treatment with Non-Steroidal Anti-inflammatory Drugs (NSAIDs) is also useful. The effectiveness of treatment should be monitored carefully with clinical signs and repeat synovial fluid analyses (The Merck Veterinary Manual, 2009).

### 2.4.9 Toe granuloma

Toe granuloma is a smooth strawberry-like growth at the site of damage on the sole or axial hoof wall. The overlying horn fails to grow back normally. This occurs after over enthusiastic foot paring which leads to bleeding. It also results after severe long-standing foot rot, toe abscess or puncture wounds. It may eventually cause overgrown misshapen hoof because the animal fails to bear full weight on the affected foot. Affected sheep are extremely lame. The strawberry-like growth becomes covered with loose horn but never heals properly and bleeds when touched (Scott and Henderson, 1991; Winter, 1998a). Management of toe granuloma involves surgical excision of the granulomatous tissue and the adjacent loose horn. Also cautery and repeated application of astringents such as copper sulphate are recommended (NADIS, 2003)

## 2.5 Nutritional causes of lameness

### 2.5.1 White muscle disease

White muscle disease is a degenerative muscle disease (Pugh, 2002). This is caused by selenium and vitamin E deficiency in sheep. The deficiency leads to muscular dystrophy and the sheep are unable to stand or walk. There is bilateral necrosis and calcification of limb muscles, leading to lameness (Radostitis *et al.*, 2001). Treating the cardiac form of white muscle disease is usually ineffective and the sheep that survive often do not thrive because of the residual cardiac damage. The muscular form of the disease can be treated with supplements of selenium and/or vitamin E (Pugh, 2002).

### 2.5.2 Laminitis

Laminitis results from aseptic inflammation of the sensitive laminae of the claws. It is predisposed by sudden introduction of high amounts of concentrate feeds to sheep. Clinically there is pain around the coronet leading to severe lameness (Radostitis *et al.*, 2001). There is also occurrence of septic laminitis referred to as the "Lamellar suppuration". This is an acute bacterial infection of laminar matrix of the hoof by *Fusobacterium necrophorum* and *Actinomyces pyogenes*. The infection is enhanced by impaction of interdigital space with mud and feces, overgrowth of the hoof or by separation of the wall after laminitis. The affected digit is hot and tender. Lameness is severe. This condition is more commonly observed on the fore limb. Affected sheep usually recover rapidly after paring of the horn to provide drainage (The Merck Veterinary Manual, 2009)

### 2.5.3 Photosensitization

In photosensitization the lightly pigmented parts of the skin are hyperactive to sunlight. This results primarily from consumption of plants with photodynamic agents. The photodynamic agents enter either through skin or gastro-intestinal tract and reach the skin unchanged. It is also associated with liver damage due to various poisonings. This is manifested by marked photosensitivity (The Merck Veterinary Manual, 2009). It leads to acute coronitis that causes lameness (Radostitis *et al.*, 201). However it is not a common condition in sheep. If photosensitization is diagnosed early and sheep immediately removed from the pastures to areas, sheltered from direct sunlight the sheep will normally recover well (The Merck Veterinary Manual, 2009).

## 2.6 Other general causes of lameness

The causes of lameness in this section are systemic diseases that may occasionally affect the limbs, particularly the feet.

### 2.6.1 Contagious pustular dermatitis (Orf)

This disease causes lesions on the lips, skin in the head region, muzzle and oral mucosa. Secondary lesions also occur on the limbs around the coronet, palmar and plantar surfaces of pastern joint and interdigital skin. Lesions can also extend to the tarsal and carpal joint areas with accompanying painful cellulitis and secondary infection leading to lameness. It affects lambs or non-immune adults (Radostitis *et al.*, 2001; Kitching, 2004). Sheep normally recover from orf within a week. Application of antibiotics and ensuring that infected sheep are supplemented with high quality feeds helps in the recovery. Isolation of the infected stock is advisable in order to slow down cross-transmission to healthy animals (Winter and Charmley, 1999).

### 2.6.2 Foot and Mouth Disease (FMD)

The disease is characterized by vesicles in the mouth and on the feet and teats, but oral lesions are not prevalent in sheep. Feet lesions commonly occur on the coronet, interdigital skin and the heel bulbs. FMD foot lesions can resemble foot rot, particularly if there is secondary bacterial infection. Lameness is severe and the morbidity is high (Caple, 1990; Radostitis *et al.*, 2001; VEIN, 2008). Management of foot and mouth disease involves slaughter of all affected and contacted sheep, quarantine of affected premises and vaccinations (The Merck Veterinary Manual, 2009).

### 2.6.3. Blue tongue disease

During the initial stages of infection with the bluetongue virus there is hyperaemia of the mucous membranes of the mouth and the skin of the feet around the coronet. Coronitis is severe with prominent haemorrhages which may be visible in the hooves. There is also separation of horn tissues from the coronary tissue. Laminitis may also result. Lameness when present is severe but is observed late in the syndrome (Radostitis *et al.,* 2001; Verwoerd and Erasmus, 2004; VEIN, 2008). There is no effective treatment. Prevention is effected through quarantine, inoculation with live modified virus vaccine and control of the vector (Gairdner, 2007; Abel, 2008).

### 2.6.4 Ulcerative dermatosis

Ulcerative dermatosis is characterized by destruction of the epidermal and subcutaneous tissues, development of raw granulating ulcers on the skin of the lips, limbs and external genital organs. Feet lesions occur in the interdigital space and above the coronet leading to lameness (Radostitis *et al.,* 2001). Management of ulcerative dermatosis includes isolation of affected sheep, removing the scabs and all necrotic tissues as well as treatment of foot lesions with copper sulphate or formaldehyde solutions in footbath troughs (The Merck Veterinary Manual, 2009).

### 2.6.5 Dermatophilosis

Dermatophilosis is caused by *Dermatophilus congolensis* zoospores that spread rapidly or from infected dipping tanks around the feet. It is characterized by proliferative dermatitis with exudative crusts and scab formation on the affected region of the body. The disease

affects the limbs from the coronet to stifle or hock. Mild lameness is observed (Zaria and Damin, 2004). The disease is predisposed by prolonged wetness, high humidity, high temperature and various ectoparasites. Acute cases of Dermatophilosis heal rapidly without treatment. However chronic cases can be effectively treated with penicillin. Also the clinically affected sheep should be isolated or culled (The Merck Veterinary Manual, 2009)

### 2.6.6 Post-dipping lameness

Post-dipping lameness is caused by *Erysipelothrix rhusiopathiae* and is observed in sheep of all ages. The disease is characterised by cellulitis at the coronary band and interdigital area affecting several animals 2-7 days after dipping. Most cases resolve after a few days, but in a few cases bacteraemia occurs resulting in joint swelling due to painful non-suppurative arthritis about 2–3 weeks after dipping (Radostitis *et al.,* 2001; NADIS, 2003). This arthritis may affect one or more joints. The treatment response in these cases is poor. The source of infection is faeces-contaminated dip, in which *E. rhusiopathiae* can multiply rapidly. Sheep should pass through water troughs and also walked over concrete before dipping to remove excess soil and faecal material from the feet. Dip-compatible bacteriostats should be added when it is necessary (Radostitis *et al.,* 2001; VEIN, 2008; The Merck Veterinary Manual, 2009).

## 2.7 Prevention of lameness in sheep

Prevention and control of lameness in sheep depend mainly on management or husbandry practices:

### 2.7.1 Good management practices

The management practices that will prevent the occurrence of lameness includes reduced stocking density (Elliot and Pinkus, 1993), regular foot bathing with 10% zinc sulphate or 10% copper sulphate solution (Parajuli and Goddard, 1989), avoiding long and dry pasture that may cause interdigital abrasions (Whittington, 1995), hoof trimming and proper genetic selection. Sheep that have foot infections should be separated from clean sheep (The Veterinary Formulary, 1998). Factors that enhance dry and clean environment also reduce the risk of spreading foot infections. These factors include adequate straw bedding that keeps the feet dry and clean as well as spreading lime on the floor especially around water troughs to help dry and sterilize the beddings (Henderson, 1990). It is important to cull persistently infected sheep which do not fully respond to treatment in order to minimize the source of infection to the rest of the flock (Winter, 1998a).

### 2.7.2 Vaccination

Foot rot vaccine can be used curatively as well as preventatively (The Veterinary Formulary, 1998).

### 2.7.3 Genetic selection

Studies in Australia have shown that genetic selection of sheep resistant to foot rot is possible (Raadsma *et al.*, 1990).

### 2.7.4 Hoof trimming

This is a skilled procedure and should be carried out on overgrown or misshapen feet. Routine trimming of all the feet is necessary. Trimming helps to eliminate cracks and crevices that could trap mud and harbour bacteria. Foot trimming allows penetration of footbath chemicals. Regular foot paring may prevent shelly hoof (Scott and Henderson, 1991; DEFRA, 2003b). Granuloma can be prevented by not over-paring the hooves (Winter, 1998b).

### 2.7.5 Foot bathing

Footbaths containing either 3% formalin or 10% copper sulphate solutions are recommended. Both have antimicrobial properties, but in addition formalin also hardens the claw horn (Arkins, 1981; Davis, 1982).

# CHAPTER 3

# 3.0 MATERIALS AND METHODS

## 3.1 Study area

Kajiado District is approximately 15,546 $km^2$ (Figure 3.1 and 3.2) with about 470,000 people and a population density of 30 people per $km^2$. The district has an estimated sheep population of 502,340. It is located in the semi-arid zone of Kenya, but has two rainy seasons, during March to May and October to November with annual rainfall ranges of 500 mm to 1250 mm. The sheep production among the pastoralists in this district is generally free-range grazing because of scarcity of pasture. The farms in which the study was carried out were located in Ngong, Ewaso Kedong and Isinya divisions within the district (Otieno, 2008).

## 3.2 Study design

This was a cross sectional study in which the farms were visited more than once, but in each farm every sheep that met the selection criteria was examined only once during the whole study.

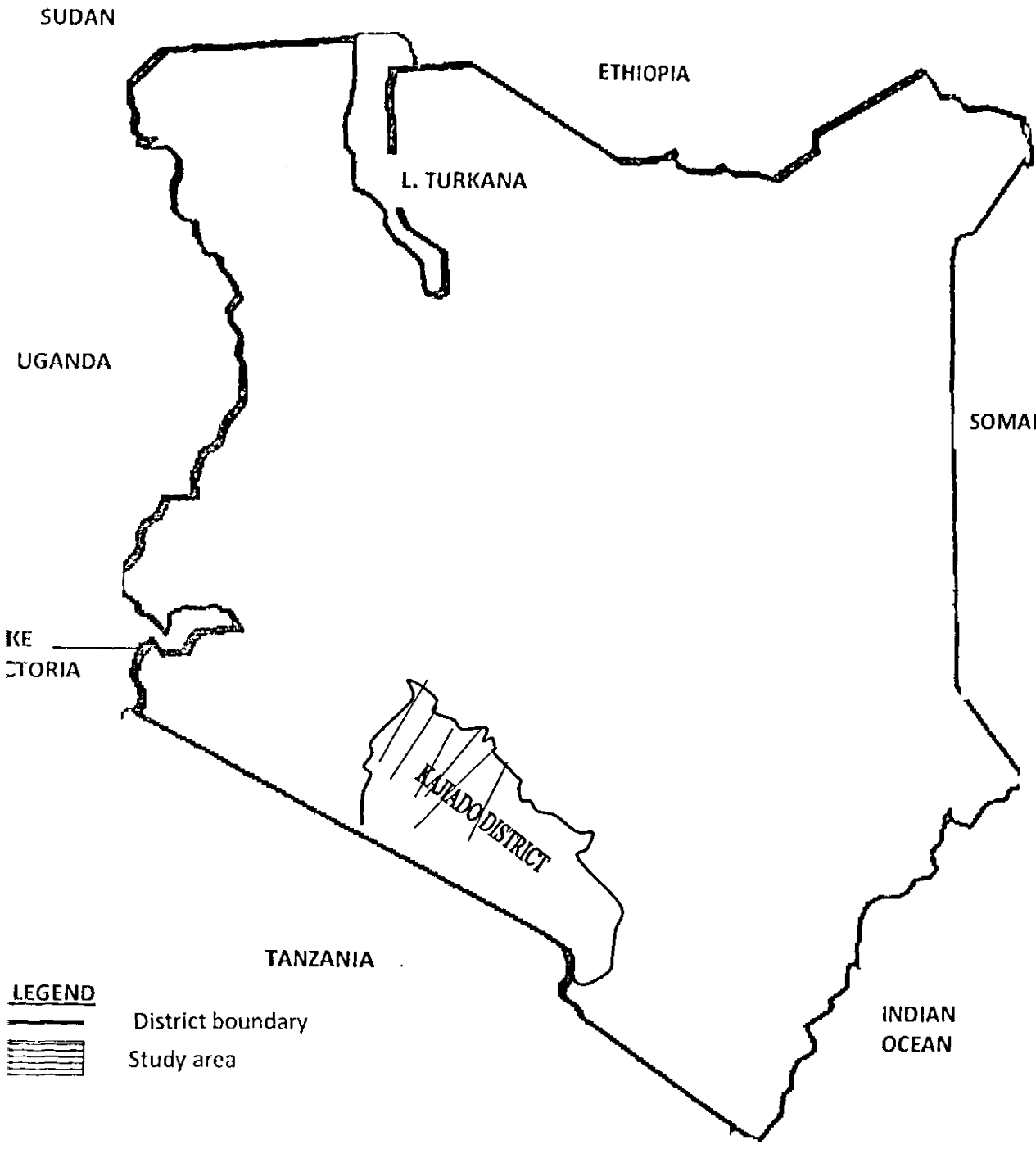


**Figure 3.1**: Map of Kenya showing Kajiado District in which the sheep examined for prevalence and risk factors of conditions causing lameness under free-range grazing system were reared (March 2010–June 2010).

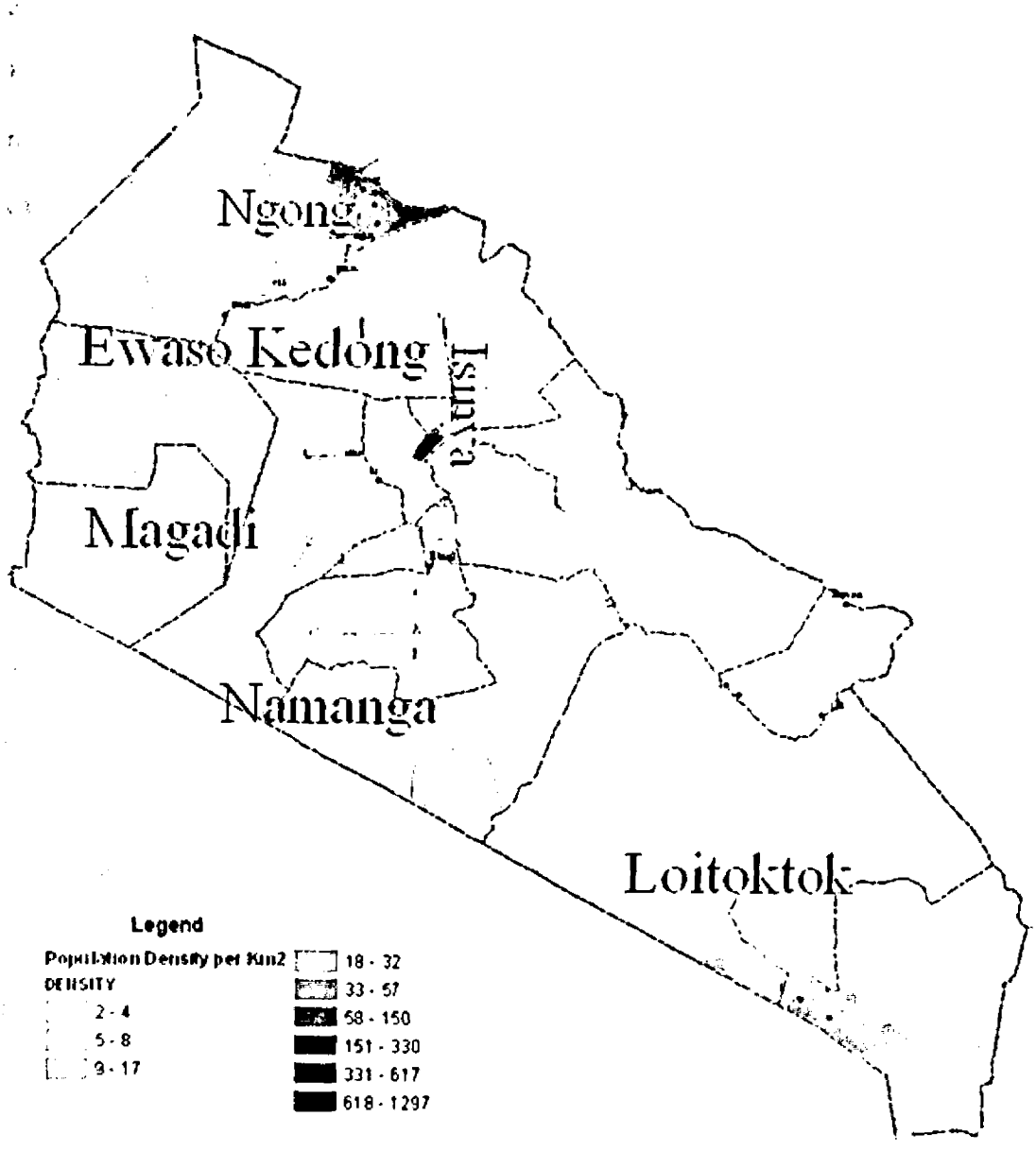


**Figure 3.2:** Map of Kajiado district, Kenya in which the farms included in the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system were located (March 2010–June 2010).

### 3.3 Farm selection

Farms selection was purposive for logistic reasons. It considered the number of sheep reared within the farms as well as willingness of the farmers to allow the study to be carried out on their sheep. Farms were identified with the help of local veterinary officers and animal health technicians. Farmer's consent for use of their farms and examination of their sheep was sought through local veterinarians or animal health technicians. Ten (10) farms, 4 in Ngong, 3 in Isinya and 3 in Ewaso Kedong divisions, each with a minimum of 100 sheep and 3 months of age or older were selected for the study.

### 3.4 Animal selection

A total of 1916 sheep were selected from the 10 farms. The selected sheep included both lame and non-lame, above three months of age, both sex and of varied breeds. The study in each farm was carried out early in the morning before the sheep were released from their night enclosures All sheep in each farm underwent general visual observation noting particularly the body and limb conformations while the sheep were at rest, in standing positions and during locomotion. Each sheep was made to walk on a flat and firm ground (Figure 3.3), the lame ones isolated and marked with a blue aerosol spray over the sacral region (Figure 3.3) for closer and specific limb examination.

### 3.5 Animal examination

#### 3.5.1 Visual observation

All the sheep in each farm were made to slowly walk through a firm ground area as the investigator observed them carefully to identify those with abnormal gait or showing

lameness (Figure3.3). The observation included the position of the back (level of dorsal column), placement of each limb on the ground, bearing of weight on the limbs and nature of the strides made. A locomotion score of 0 (not lame) to 4 (severely lame) as a locomotion scoring system (Table 3.1) was used to indicate the degree of lameness. Each sheep that was identified as lame was separated from the non-lame sheep for closer examination.

A B

**Figure 3.3**: **A**: Sheep walking out of a night enclosure and **B**: A lame sheep selected, marked and isolated. This was in one of the 10 farms during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

**Table 3.1:** Locomotion score scale used to assess lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010-June 2010).

| Score | Description of lameness | Conclusion |
|---|---|---|
| 0 | Normal gaits | Not lame |
| 1 | Gait is slightly abnormal | Mild lameness |
| 2 | Short strides on one or more legs | Moderate lameness |
| 3 | Favours one or more limbs by not bearing weight | Definite lameness |
| 4 | Complete refusal to bear weight on one or more limbs | Severe lameness |

### 3.5.2 Examination of lame sheep

Each lame sheep was restrained by a farm-worker and subjected to a thorough general physical examination with special emphasis on the lame limb(s) identified as the sheep walked to diagnose the specific lesion causing the lameness. The claws were thoroughly washed in order to clearly see the lesion in case claw structures were involved (Figure 3.4). If the cause of lameness was proximal to the foot, the whole region from the shoulder to the fetlock and from the hip to the fetlock was examined by deep hand palpations to locate the painful part. The joints were flexed and extended and presence of pain was indicated by the animal's reaction to these manipulations. Each lesion causing lameness was photographed using a digital camera (Sony DSC-W180, 10.1 Mega Pixels, Sony Corporation). The diagnosis or the condition causing the lameness for each sheep was recorded in data collection sheets. Bacteriological swab specimens were collected from exudative lesions for bacterial culture and identification. After examination a second mark was put on the back of each sheep cranial to the first mark using a blue aerosol spray to avoid repeat examination (Figure 3.5).

#### 3.5.2.1 Recording of findings

The conditions causing lameness were further classified into various categories during entry into the computer from the data collection sheets. During data entry the following parameters were clustered accordingly; location of the lesion on the limb, fore or hind limb, one or more limbs, lateral or medial claw or both (Appendix 1).

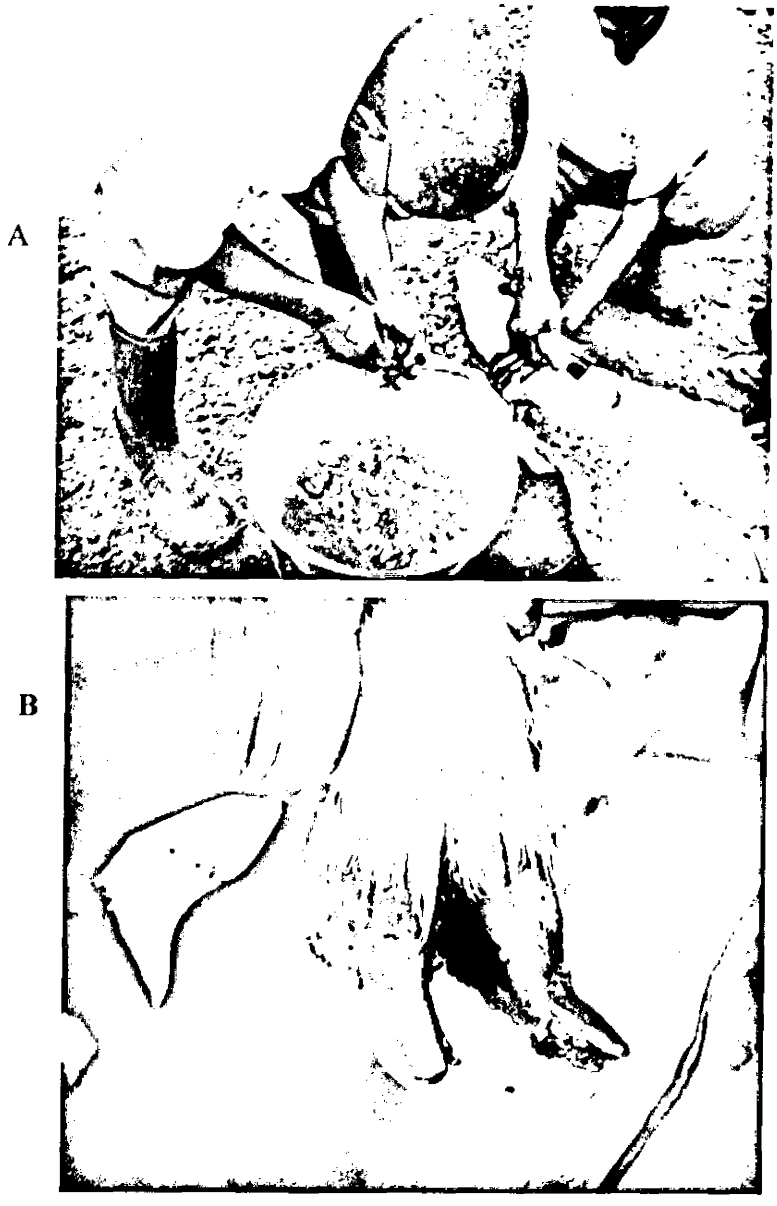


**Figure 3.4**: **A**: Thorough washing of the claws during individual animal examination. **B:** Taking a photograph of a thoroughly washed affected foot against a green sheet of cloth. This was done for all sheep with claw conditions in all the 10 farms during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

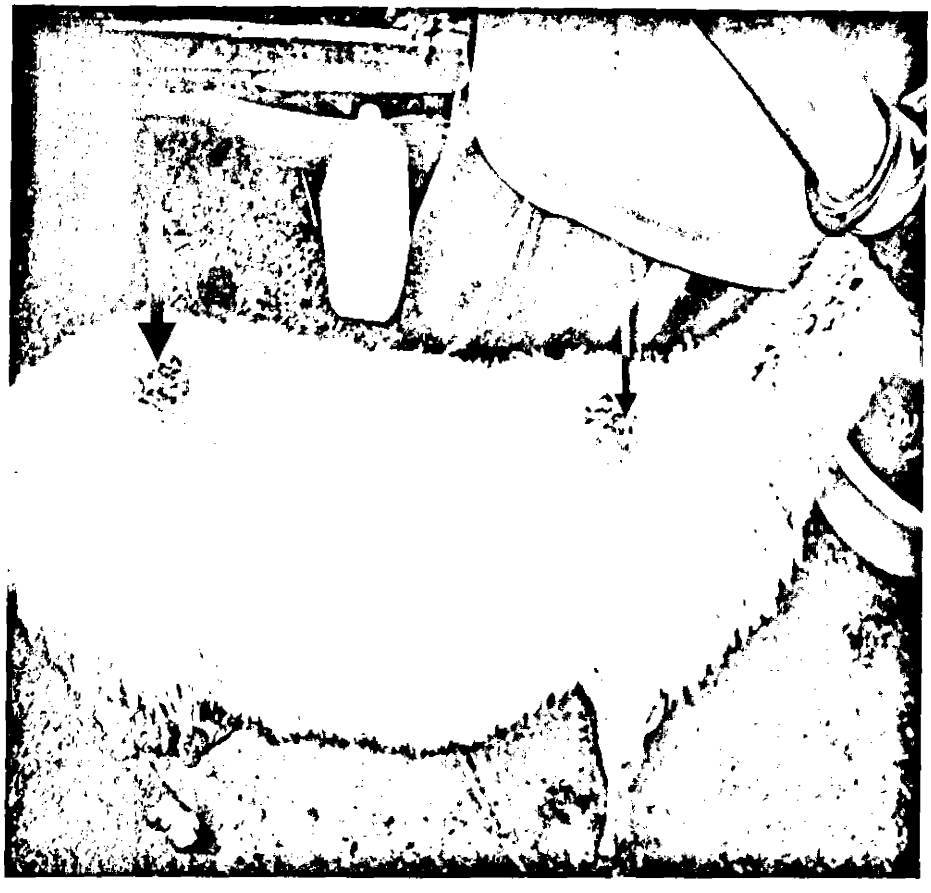

**Figure 3.5**: The first (caudal arrow) and second (cranial arrow) marks put on the back of the sheep before and after the Individual animal examination respectively in one of the 10 farms during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

### 3.6 Determination of the risk factors

#### 3.6.1 Farm-level factors

The farm environment was assessed during the visit. General observation of the sheep rearing environment was made in order to note any lameness predisposing factors. These included; the nature of terrain, grazing ground whether dry or marshy, presence of traumatic objects; state of farm tracks, type of pastures whether dry or green as well as the hygienic state of sheep night-resting enclosures. During data entry these observations were classified accordingly.

#### 3.6.2 Management-level factors

Data on the management practices was obtained by interviewing sheep owners, farm managers or stockmen. These included hoof trimming practices, tick control, feed supplementation, management of lameness cases and how stockmen handled the sheep. The data was collected by recording the important information in coded questionnaire forms (Appendix 1).

#### 3.6.3 Animal-level factors

Factors intrinsic to the animal that could predispose or enhance lameness were evaluated. These included estimated age, sex, breed, pregnancy status (when lambed, either less than or more than three months since lambing or pregnant) and limb conformation. Also evaluated was body condition score (BCS) of the lame sheep. The scores were evaluated as BCS 1 (poor), BCS 2 (fair), BCS 3 (good) and BCS 4 (very good) as suggested by Winter and Charmley (1999) and Suiter (2006). Some of these animal-level factors were

observed directly by the investigator and the rest were obtained by interviewing the farmers, stockmen, and farm managers. These were also recorded in the data sheets and questionnaire forms (Appendix 1).

### 3.7 Data handling

#### 3.7.1 Data recording

All data were written on data recording sheets that were designed and coded to capture the relevant information. Each sheep had a separate data sheet on which farm identification and flock size were indicated. The data sheet had three sections which included:(a) Animal-level section, (b) Interview section and (c) Farm environment section (Appendix1).

Data on animal-level factors were collected through questionnaires administered by the investigator interviewing the relevant persons at farm-level before the actual examination of each sheep. Data and information on management and farm-level factors were collected during visits to each of the 10 farms. This was achieved through observations by the investigator as well as administering of the questionnaires. Data recording was done separately for each sheep and for each farm.

#### 3.7.2 Data management

The data collected was stored in Microsoft office Excel 2003 (Microsoft Corporation, 2003). It was validated and verified to be correct as per the entries from the data record sheets. The data collected indicated presence or absence of a particular parameter. Coding

of each parameter for entry into computer was done. Parameters were coded as "1" (signifying "Yes" for presence of that parameter) and "2" (signifying "No" for absence of that parameter).

### 3.8 Data analysis

The data were imported into GENSTAT for windows discovery Edition 2 (VSN international). Descriptive statistics focusing on frequencies of occurrence of each parameter was done. Simple associations between lameness score and animal-level, farm-level and environmental factors were also computed. Chi-square ($\chi^2$) statistics were used to determine the associations between lameness and risk factors at $p < 0.05$ significance level. Prevalence of lameness was calculated as a percentage of lame sheep in the study population as follows:

$$\text{Prevalence of lameness (\%)} = \frac{\text{Total number of lame sheep}}{\text{Study population}} \times 100$$

Prevalence of each condition causing lameness in the study population was calculated as follows

$$\text{Prevalence of each condition among sheep examined (\%)} = \frac{\text{Total number with a specific condition}}{\text{Study population}} \times 100$$

Prevalence of each condition was also expressed as percentage of total number of lame sheep.

$$\text{Prevalence of each condition among lame sheep (\%)} = \frac{\text{Total number with a specific condition}}{\text{Total number of lame sheep}} \times 100$$

Chi-square ($\chi^2$) values were determined using 2×2 contingency table constituting 2 rows and 2 columns. In these associations, the chi-square ($\chi^2$) calculations were determined by evaluating each risk factor (variable) against each lameness condition (outcome) on the sheep. The degrees of freedom (df) in each case was standard, being calculated by

[(rows-1)(columns-1)], hence [(2-1) x (2-1) =1]

Therefore df was 1 for each association test

# CHAPTER 4

# 4.0 RESULTS

## 4.1 Descriptive statistics for the study farms

The study population in all the 10 farms was 1916 sheep, out of which 117 lame ones were examined. The 10 farms included in the study had an average flock size of 192 sheep which were all under free-range grazing system. The median number was 183 sheep. One of the farms was paddocked and the sheep were grazed within the paddocks (Figure 4.1). In 3 of the farms, the sheep were housed in roofed enclosures during the night after free-range grazing the whole day (Figure 4.2). The other 7 farms did not have any roof over the night enclosures. In 6 of these 7 farms, the night enclosures were made of timber and mesh wire sides (Figure 4.3), while the remaining one, the perimeter wall of the night resting area was secured with thorny tree branches (Figure 4.4).

In one of the farms, formalin solution was used as a foot-dip in a plastic container. The dipping of the feet into the formalin solution in this farm was done once per week. The rest of the farms had neither footbaths nor chemical foot-dips. Trimming of the hooves was routinely done in 5 farms only. In 3 of these farms, it was carried out by the owners or the stockmen while in the other 2, it was done by either a veterinary surgeon or an animal health assistant. In these 5 farms the trimming was done once a year, but in the other 5 farms hoof trimming was not done at all.

In the three-month period immediately preceding the study, there were cases of lame reportedly sheep observed in all the 10 farms. In 8 of them, the lame sheep were

reportedly treated during that period. In the other 2 farms, the lame sheep were not treated but were left to recover on their own or culled. Tick control was done by hand-spraying method using Knap-sack sprayers in all the 10 farms. Two years previously, dipping in a plunge-dip was employed in one of the farms. In this farm, the farm manager reported that there were more cases of lame sheep at the time of the study than during the period when plunge-dipping was being used.

**Figure 4.1**: Sheep grazing in a paddock in one of the 10 farms included in the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.2**: Roofed night-resting enclosure where sheep were held at night after free-range grazing during the day. This was the case in three of the 10 farms assessed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

A

B

**Figure 4.3**: **A**-Sheep in open-roofed enclosure in which they were held at night after free-range grazing during the day. **B**- The night-resting enclosure with manure accumulation. These were the situations in some of the farms studied for prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.4**: An enclosure where perimeter is secured with thorny tree branches where sheep were held at night in one of the 10 farms studied for the prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

### 4.2 Description of the lesions observed

#### 4.2.1 Prevalence of the foot conditions

It was reported in all the farms that lameness cases were more common during the wet seasons. The overall prevalence of lameness in the 10 farms was 6.1% (117/1916), while 93.9% (1799/1916) of the sheep were not lame. The prevalence rates of conditions causing lameness in a population of 1916 sheep are presented in Table 4.1. Sole erosion and overgrown claws were the most prevalent conditions at 3.8% (72/1916) and 3.2% (61/1916) respectively. Tick-bite dermatitis had a low prevalence of 1.6% (30/1916). The rest of the conditions had prevalence of less than 1% (Table 4.1) or were observed only in a single sheep as presented in Figure 4.5. Out of the 117 sheep that were lame, 81.2% had moderate to definite lameness, 12% had mild lameness and 6.8% were severely lame.

When calculated as a proportion of the population of sheep that were lame, the conditions with the highest percentage of occurrence were sole erosion 61.5% (72/117) and overgrown hooves at 52.1% (61/117). Those with moderate percentage of occurrence were tick-bite dermatitis at 25.6% (30/117) and, hoof fractures at 12.0% (14/117). The rest of the conditions had percentages of occurrence equal or lower than 5%. These percentages of occurrence among the lame sheep are presented in Table 4.2 and Figure 4.6. Some of the lame sheep had a single condition causing lameness, but others had more than one condition. The proportion of the sheep that had more than one lesion on their feet simultaneously was 67.5% of the lame sheep, hence the total percentage of more than 100%. Interdigital dermatitis was observed to invariably occur together with other lesions. The rest of the conditions causing lameness were observed only in one sheep each except shelly hoof that was observed in three sheep.

**Table 4.1**: Prevalence of conditions causing lameness in a population of 1916 sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March - June 2010)

| Conditions causing lameness | Number of sheep (n=1916) | Prevalence (%) $\frac{y}{n} \times 100$ (y = Number of sheep with each condition) |
|---|---|---|
| Sole erosion (bruising) | 72 | 3.8 |
| Overgrown hooves | 61 | 3.2 |
| Tick-bite dermatitis | 30 | 1.6 |
| Hoof cracks | 14 | 0.7 |
| Interdigital dermatitis | 6 | 0.3 |
| Shelly hoof | 3 | 0.2 |
| Soil balling | 1 | 0.1 |
| Osteomyelitis of metatarsal bone | 1 | 0.1 |
| Septic arthritis | 1 | 0.1 |
| Malunion of tibial bone | 1 | 0.1 |
| Hyperextension of fetlock joint | 1 | 0.1 |
| Overparing of medial claws | 1 | 0.1 |
| Foot rot | 1 | 0.1 |
| Foreign body penetration | 1 | 0.1 |

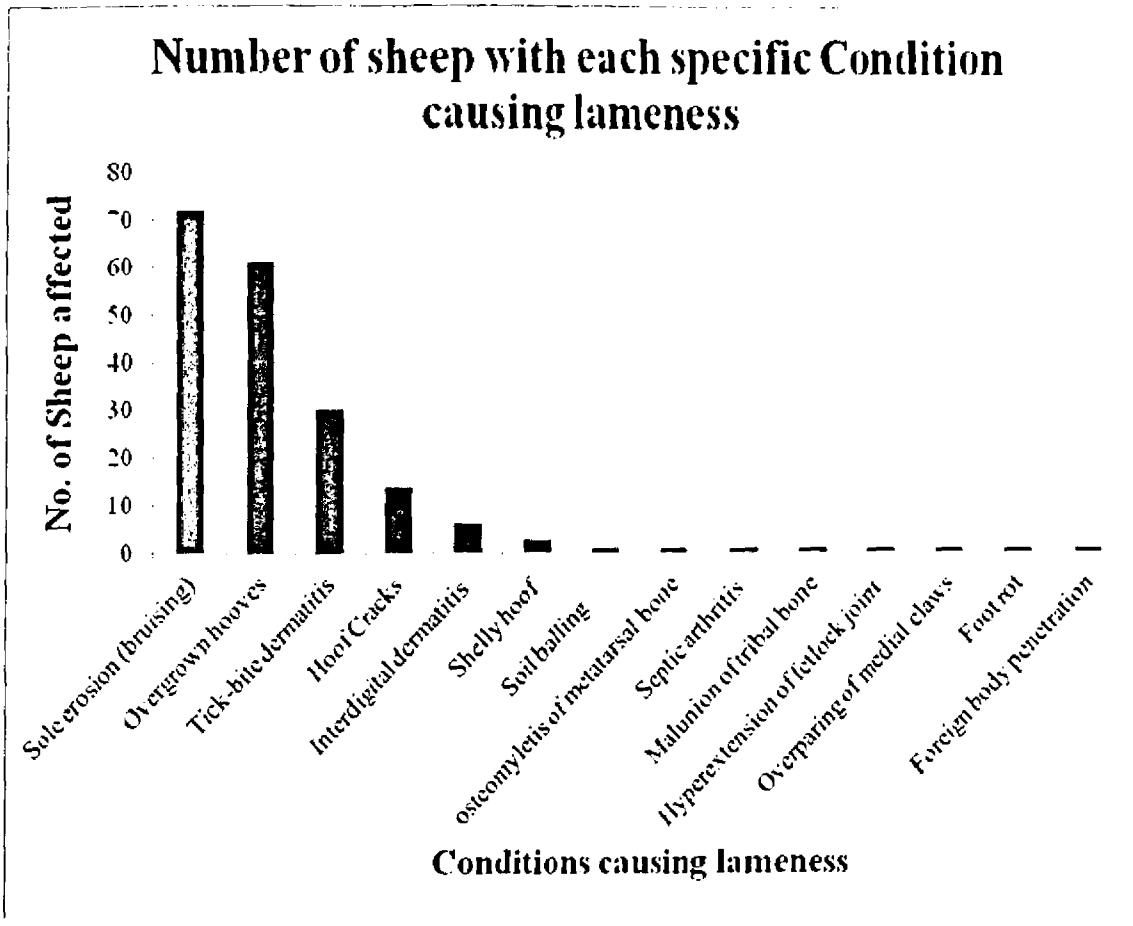


**Figure 4.5:** Conditions causing lameness according to the number of sheep affected in a population of 1916 sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March – June 2010)

**Table 4.2**: Percentages of conditions causing lameness in 117 sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March–June 2010).

| Conditions causing lameness | Number of sheep (n=117) | Prevalence (%) $\frac{y}{n} \times 100$ (y = Number of sheep with each condition) |
|---|---|---|
| Sole erosion(bruising) | 72 | 61.5 |
| Overgrown hooves | 61 | 52.1 |
| Tick-bite dermatitis | 30 | 25.6 |
| Hoof cracks | 14 | 12.0 |
| Interdigital dermatitis | 6 | 5.1 |
| Shelly hoof | 3 | 2.6 |
| Soil balling | 1 | 0.85 |
| Osteomyelitis of metatarsal bone | 1 | 0.85 |
| Septic arthritis | 1 | 0.85 |
| Malunion of tibial bone | 1 | 0.85 |
| Hyperextension of fetlock joint | 1 | 0.85 |
| Overparing of medial claw | 1 | 0.85 |
| Foot rot | 1 | 0.85 |
| Foreign body penetration | 1 | 0.85 |

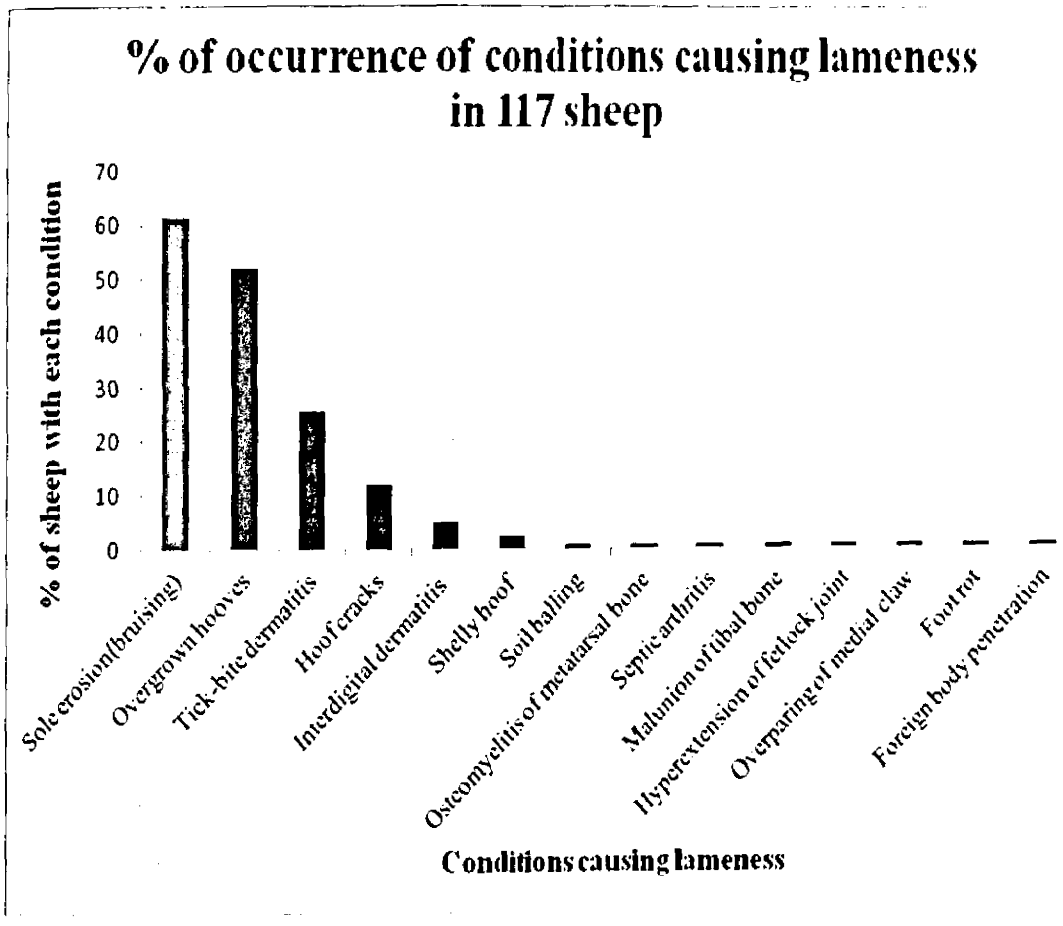


**Figure 4.6:** Percentage of occurrence of conditions causing lameness in a population of 117 lame sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March – June 2010)

### 4.2.2 Descriptive findings of the lameness conditions.

#### 4.2.2.1 Claw deformities

Normal claws were observed to have balanced growth of toe, sole and walls (Figure 4.7). However among the lame sheep, 52.1% (61/117) had various forms of deformities of the claws, most of which were related to hoof overgrowth. These included overgrowth and elongation of the toes, soles and claw walls. The overgrowth ranged from slight to excessive and also resulted in varying degrees of gait abnormalities. Out of the 61 sheep with claw deformities, 13.1% (8/61) had simple regular overgrowth, which mainly involved elongation of the sole and toes (Figure.4.8), but 86.9% (53/61) had varying degrees of excessive overgrowth of the hooves with some of them leading to misshapen claws. The excessive overgrowth with resulting misshaping of the claws included irregular elongation and widening of the hoof wall with some growing to cover the tread surface of the sole (Figure 4.9). Some toes were excessively elongated with resulting tendency to turning outward (lateral) or curving dorsally (Figure 4.10) and others were extremely splayed (Figure 4.11).

#### 4.2.2.2 Hoof wall cracks

Hoof wall cracks found in 11.9% (14/117) of the lame sheep, were mainly horizontal occurring either at the middle of lateral and dorsal wall, close to the toe or at the distal part of abaxial wall (Figure.4.12). In one sheep examined the claw wall was extensively overgrown extending and curving towards the sole (Figure 4.9).

**Figure 4.7**: Normal sheep claws both medial (Left brace) and lateral claws (Bold arrow) observed in the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

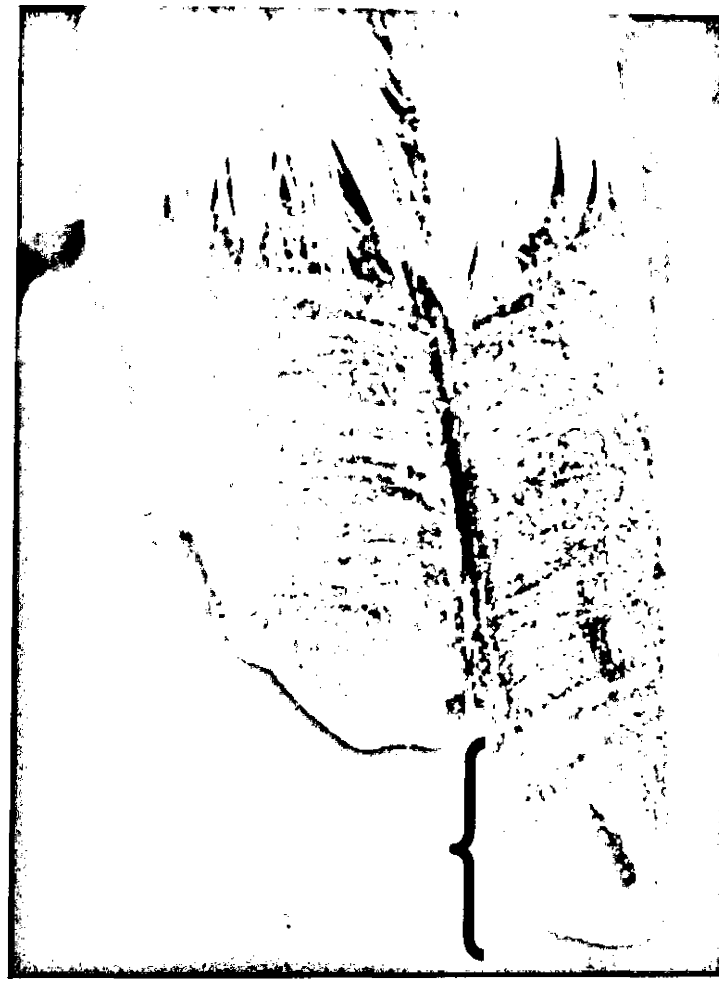

**Figure 4.8**: Regular overgrowth showing elongation of the toe (left brace) observed among some of the 117 lame sheep examined during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).


**Figure 4.9**: Excessive overgrowth of the hoof wall with resulting misshaping of the claws. **A**: Irregularly shaped claw wall (bold arrow). **B**: Widening of the claw wall with slight outward projection (right brace) and irregular outward growth (dotted arrow). **C**: Excessively overgrown lateral hoof wall covering the sole (dotted double headed arrow). These claw features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

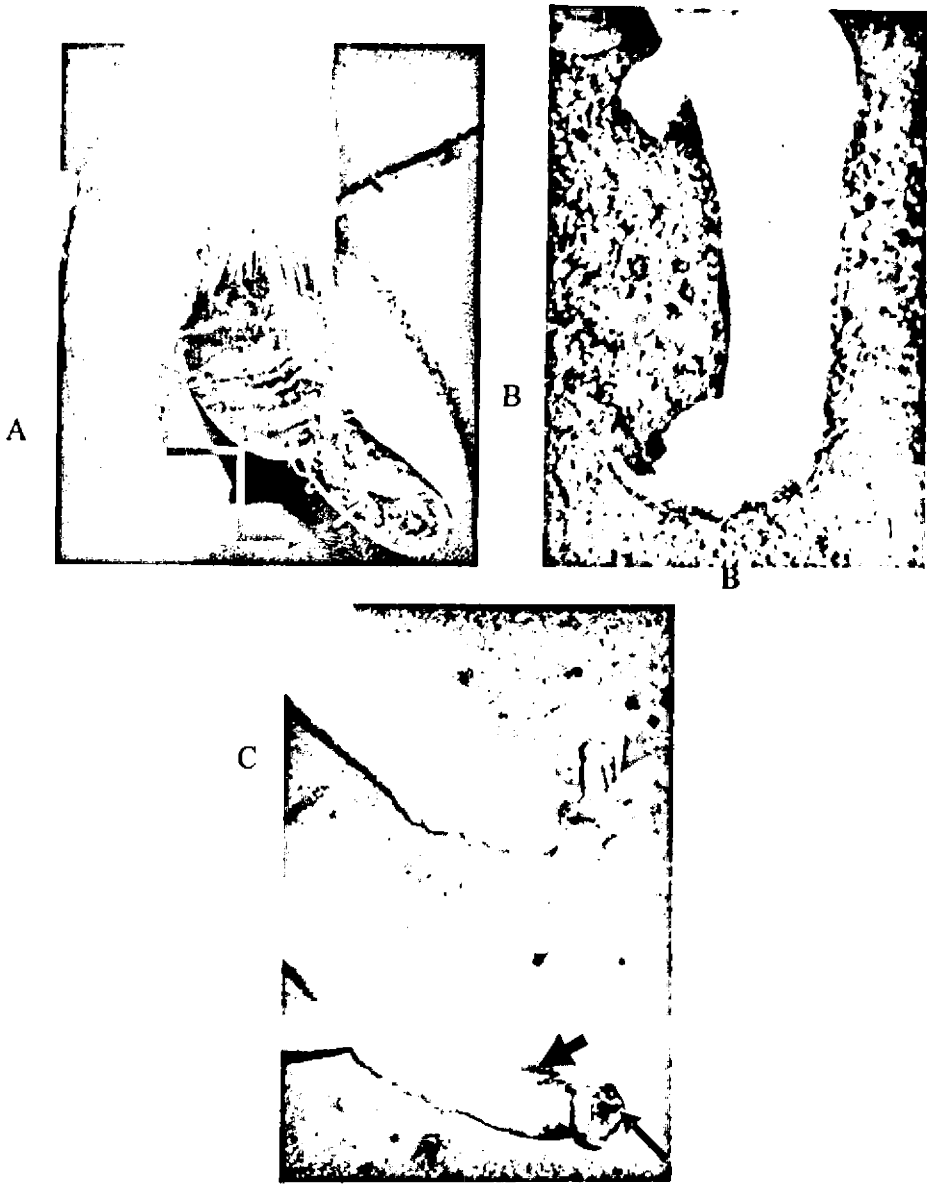


**Figure 4.10**: Excessively elongated and misshapen toes of the claw. **A**: Overgrown hoof walls and elongation of the toe in a lateral direction (angled arrow). **B**: Dorsal curvature of the toe (bold arrow). **C**: Dorsal curvature of the toe (dotted arrow), circularly coiled elongated toe (bold arrow) and over-short toe due to breakage (Arrow head). These toe features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

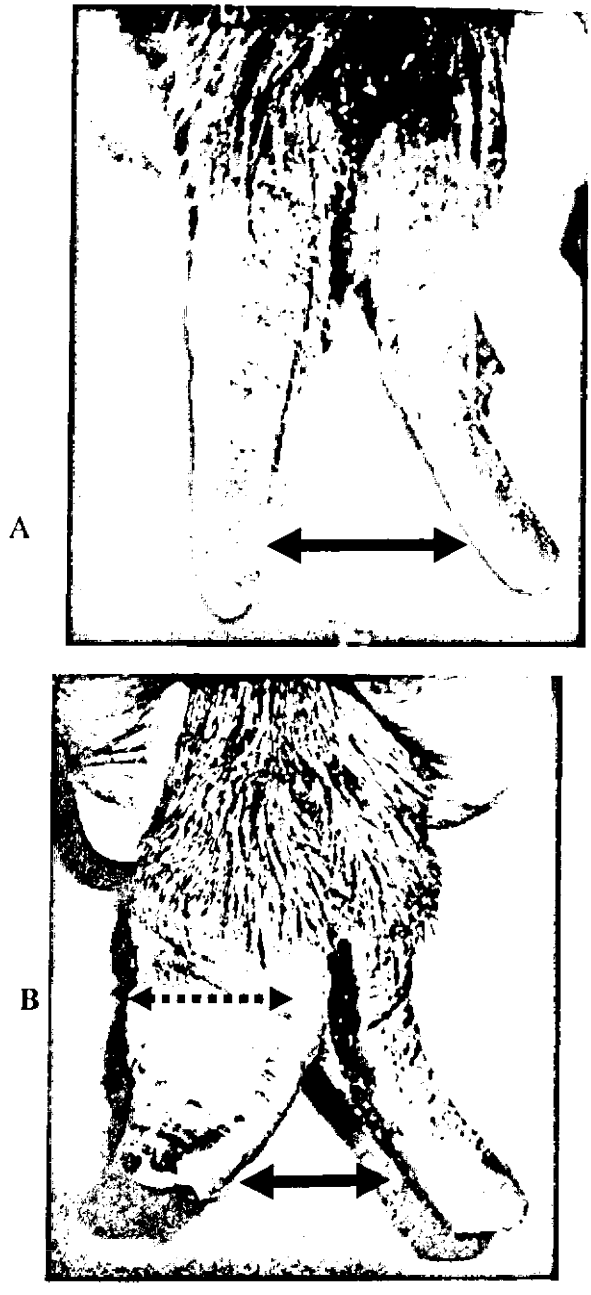


**Figure 4.11:** **A**: Regular elongation of the claw with excessively splayed toes; **B**: Elongation and splaying of the toes (double-headed bold arrow) with widening and flattening of the dorsal and lateral hoof wall (double-headed dotted arrow). These claw features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

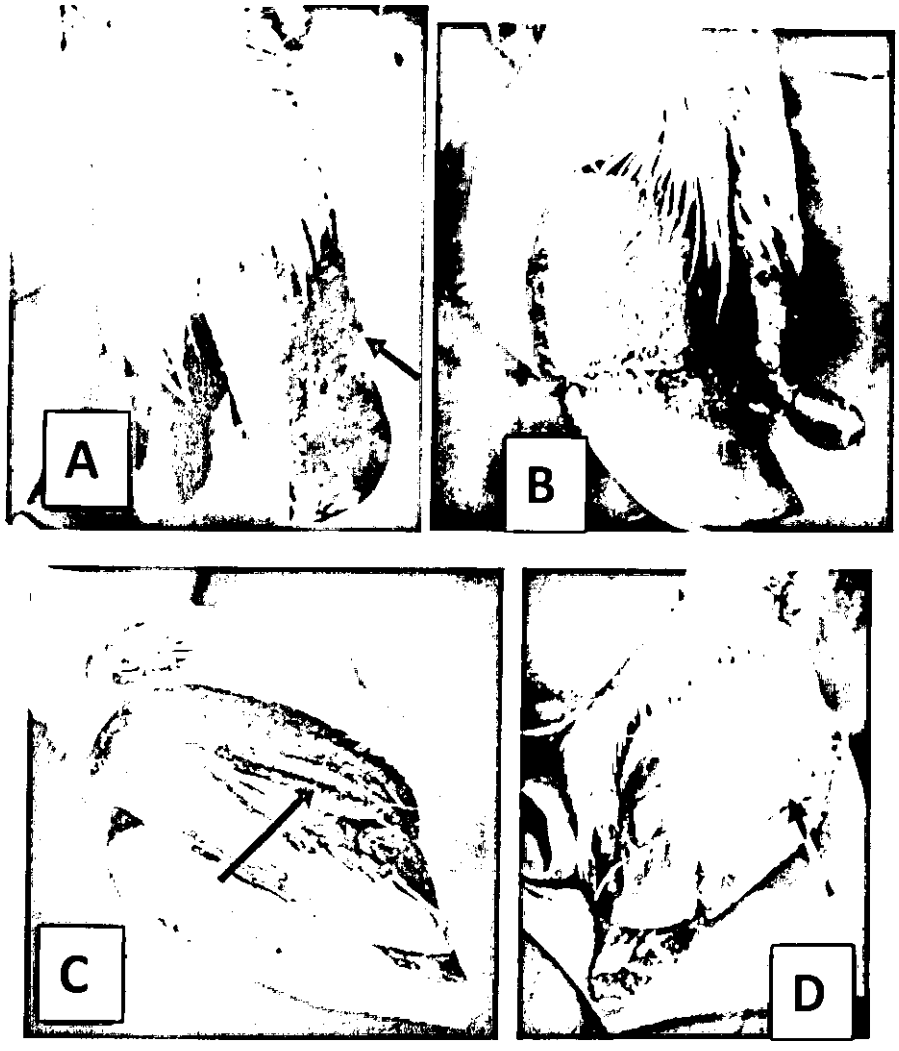


**Figure 4.12. A and B:** Horizontal hoof cracks at the middle of the lateral and dorsal claw walls. **C**: Horizontal hoof crack at the distal part of the abaxial wall. **D**: Horizontal hoof cracks at the toe of the claw. These claw features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.3 Sole erosion or bruising.

Out of the 117 lame sheep, 61.5% (72/117) had sole erosions, some of which were severe occurring with some degree of hoof overgrowth and others were mild. The severe sole erosion more invasively eroded the horn of the sole thus diminishing the thickness of the intact horn layer, but the mild sole bruising was only superficially erosive. The eroded horn of the sole appeared black and necrotic (Figure.4.13). The sole erosion lesions affected both medial and lateral claws. More cases of lameness involving sole erosion affected the hind limb (60%) and were mostly bilateral (57.8%). Painful responses were observed when pressure was applied on the eroded areas of the soles.

#### 4.2.2.4 Tick-bite dermatitis

Examination of affected lame sheep revealed heavy tick infestations on the limbs. Among the lame sheep, 25.6% (30/117) had tick bite dermatitis. The main sites of attachment of ticks were the skin on the plantar (caudal) aspect of foot between the dew claws and the coronet and also occasionally in the interdigital skin. The ticks were usually found aggregating together round a limited site. Severe inflammation was always observed and sheep were severely lame. In some of the sheep, the area of tick-bite was found to have developed dermatitis lesions with slight erythema (Figure 4.14). The sheep with large aggregates of tick attachments were moderately lame, but those with tick-bite dermatitis were definitely lame.

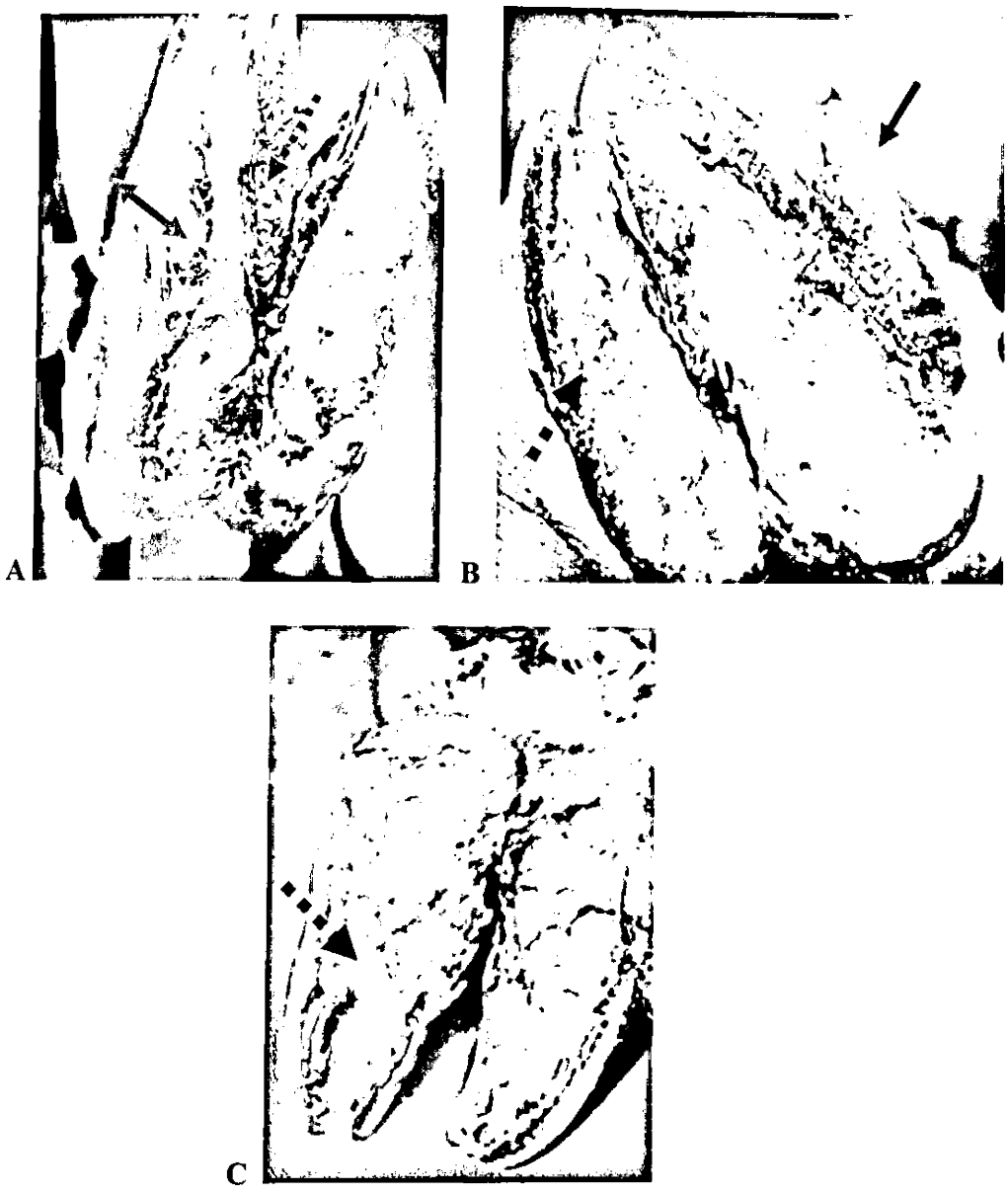


**Figure 4.13**: **A** and **B:** The invasive sole erosion with black necrotic horn (dotted arrows) occurring with overgrown hoof walls (double-headed and bold arrows). **C**: Superficial erosion of the sole (dotted arrow). These claw disorders were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

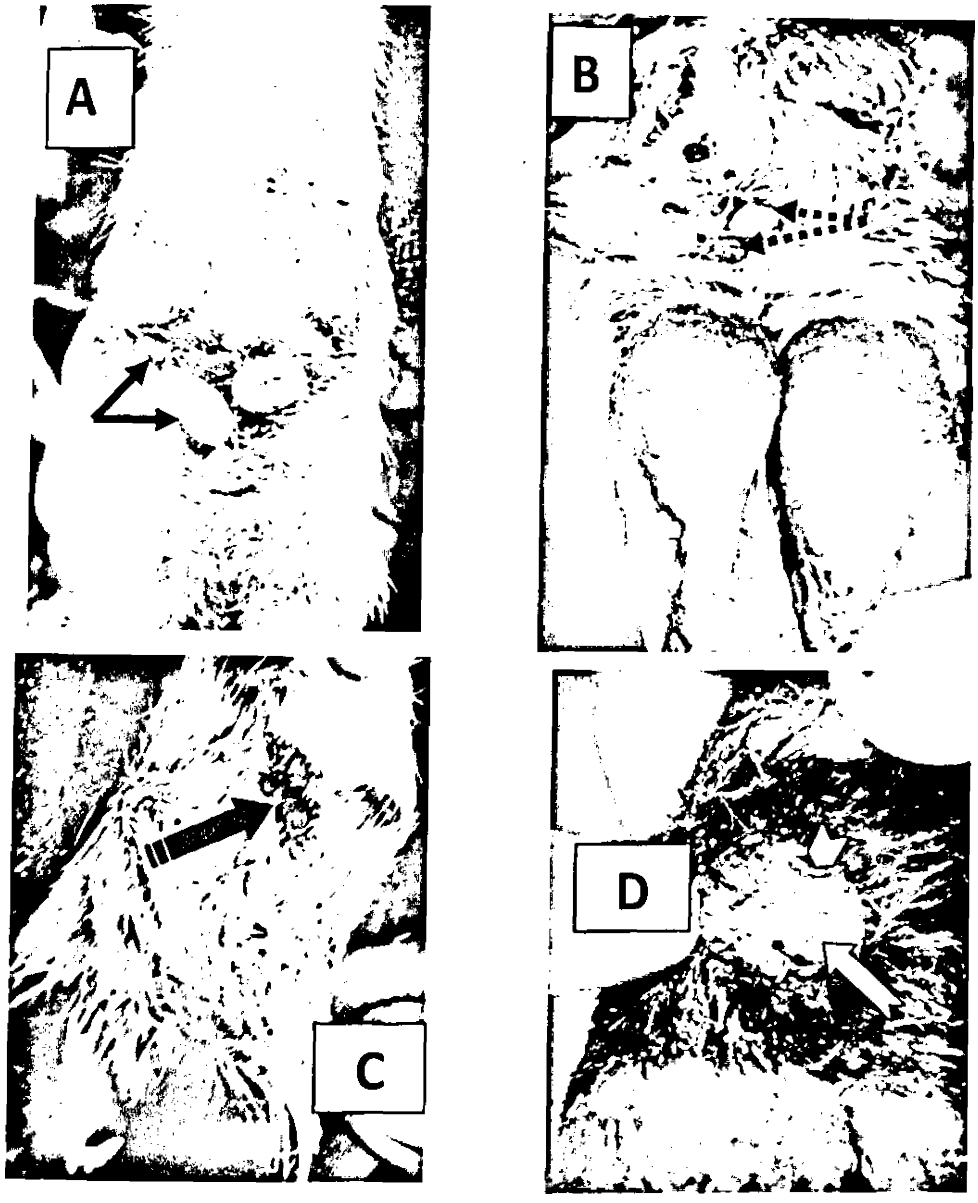


**Figure 4.14.** **A, B** and **C**: Several types of ticks attaching in a limited area distal to the dew claws (bold and dotted-V arrows). **D:** Tick-bite dermatitis lesion distal to the dew claws (notched arrow), with some ticks still attached (chevron). These were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.5 Interdigital dermatitis

Interdigital dermatitis was found in 5.1% (6/117) of the lame sheep. It was observed to be an acute inflammation of the interdigital skin. In some of the cases, the dermatitis lesion had ulcerated and the skin was erythematous, which resulted in moderate to severe lameness (Figure.4.15).

#### 4.2.2.6 Septic arthritis

Only one sheep was found to have septic arthritis involving one digit. The lesion was located at the proximal interphalangeal joint (pastern) of the foot. It was swollen, warmer than the surrounding tissues and discharging pus. The open parts of the lesion appeared necrotic with scab formation. The infection was observed to be affecting the deeper structures. The lesion was very painful and the sheep severely lame with the affected limb not bearing any weight (Figure 4.16). *Fusobacterium* species were isolated from culture of pus collected from the lesion.

#### 4.2.2.7 Foreign body penetration

A hard dry thorn was found as a foreign body penetrating the sole in one sheep. The horn of the sole around the penetrated area had dark-red discoloration and was slightly swollen (Figure 4.17). The sheep was severely lame and resisted bearing weight on the affected foot.

**Figure 4.15.** Granulating interdigital dermatitis lesion (bold arrow). Such lesions were found in 6 sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010)

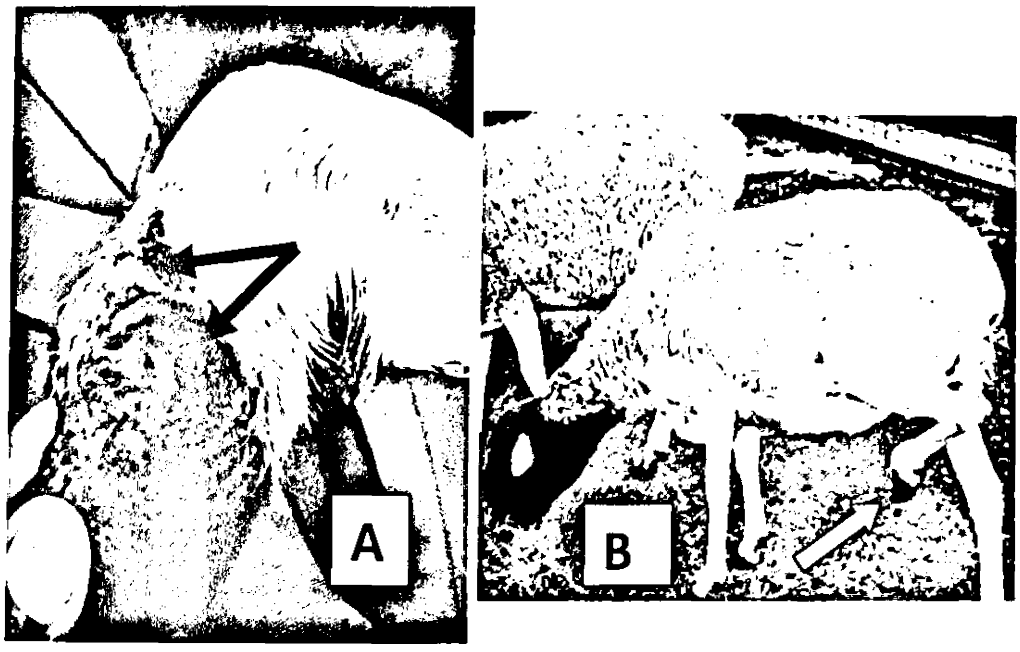


**Figure 4.16**. **A**: Septic arthritis of the proximal interphalangeal (pastern) joint with a necrotizing wound and scab forming wound (bold-V arrow). **B**: lifting of the left limb due to pain in a severely lame sheep (bold arrow). Such features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.17**: A penetrating foreign body and a resulting necrotizing wound in the sole of one claw with swelling and hyperemia around it (bold arrow). This was observed in one among the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.8 Interdigital soil-balling

Soil and grass stuck between the digits and formed into a hard lump commonly referred to as "soil or grass balling" was observed only in one among the lame sheep (Figure 4.18). The soil and grass balls were firmly attached to the underlying interdigital skin and the hooves. The sheep with soil balling manifested definite lameness.

#### 4.2.2.9 Foot rot

Only one sheep in this study had foot rot. The foot rot lesion included dermatitis in the interdigitum, slight under-running of the horn at the skin–horn junction and foul-smelling exudates (Figure 4.19). The sheep was severely lame.

#### 4.2.2.10 Shelly hoof

Shelly hoof was observed in one lame sheep. This sheep had hoof overgrowth with separation of walls and accumulation of dung material in the avulsed parts. Abaxial hoof walls had slight separation, but the axial walls had excessive separation (Figure 4.20). The sheep had moderate lameness on the affected foot.

**Figure 4.18**. Hard lumps (soil and grass balling) formed by prolonged accumulation of grass and soil in the interdigital space (bold arrows). These were firmly attached to the underlying interdigital skin and the hooves. This was observed in one among the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.19**: Interdigital foot rot lesion with some purulent discharge, necrosis, swelling and dung matting around it (bold arrow). The condition was seen in one among the lame sheep observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).


**Figure 4.20. A** and **B:** Separation of the hoof wall from the underlying parts of the claw, particularly the axial walls in "shelly hoof". Dung is accumulated between the separated structures (bold curved and straight arrows respectively). This disorder was found during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March-June 2010).

#### 4.2.2.11 Over-trimmed hooves

In one among the lame sheep, the hooves were found to have been excessively trimmed to the extent of traumatizing the sensitive laminae (Figure 4.21). The recommended trimming pattern was not followed, which resulted in vertical cutting-off of the toes. This was manifested as severe lameness of the affected limb.

#### 4.2.2.12 Malunion of tibial fracture

One sheep was reported to have had a fracture of right tibia. Examination revealed that the fracture had healed with an extreme malunion of the bone fragments, which resulted in deformation of tibia. The bone was curved medially near the hock joint. This resulted in adduction of the hock area and abduction of the foot leading to abnormal gait (Figure 4.22). The sheep had moderate lameness.

#### 4.2.2.13 Abnormal conformation of the foot (Fetlock hyperextension)

This was observed in one out of 117 sheep that were lame. The anomaly was observed at the fetlock joints of both hind limbs. The joints were flexed and mild lameness was observed (Figure. 4.23).

#### 4.2.2.14 Osteomyelitis of the metatarsal bone

Osteomyelitis was observed in one out of the 117 sheep that were lame. This affected the metatarsal bone. There was swelling and extreme pain. Lameness was definite.

**Figure 4.21**.Over-trimmed hooves affecting the sensitive laminae. The toes are completely cut off and the recommended trimming pattern was not followed (bold v-shaped arrows). This was observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.22**. Malunion of distal to mid tibia after fracture healing (chevron), leading to angled adduction of the hock joint area (dotted arrow) and extreme abduction of the foot (double-headed arrow). This was observed in one of the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

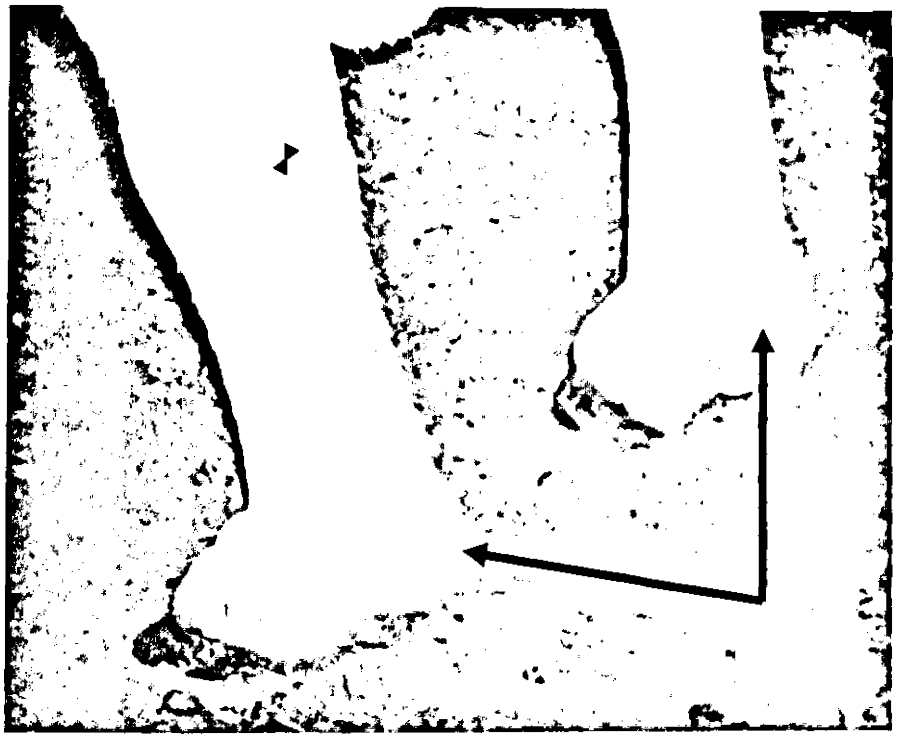

**Figure 4.23**: Abnormal conformation of the foot affecting the fetlock joints of the hind limbs (Arrows) resulting in mild lameness. This was observed in one of the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

### 4. 3 Distribution of lesions on the limbs among the lame sheep

Among the lame sheep, frequency of lesions on the hind limbs was 43.6%, on the fore limbs 23.1%, and on both hind and fore limbs simultaneously 33.3%. Among these lame sheep, the lesions were found on a single limb in 51 sheep (43.59%), on two limbs in 39 sheep (33.33%), on three limbs in 2 sheep (1.71%) and on all four limbs in 25 sheep (21.37%). The lesions causing lameness were located on the foot in 94% (110) of the lame sheep, among which 85.5% had lesions on both medial and lateral claws, 7.3% only on the lateral claws, 5.5% only on the medial claws and 1.7% located between the fetlock joint and the coronet. The remaining 6% (7) of the lame sheep had lesions located on the proximal parts of the limbs.

### 4.4 Description of possible risk factors of lameness

#### 4.4.1 Animal-level factors

These factors are presented in Table 4-3. Out of the 117 lame sheep, 76.9% (90) were females and 23.1% (27) were males. Among these lame sheep, the breeds were Dorpers (53.8%), crosses of Dorper and Red Maasai sheep (42.7%), crosses of Dorper and Merino (1.7%) and the Red Maasai sheep (1.7%). The body condition scores among the 117 lame sheep were as follows; very good (BCS 4) 17.95%, good (BCS 3) 63.25%, fair (BCS 2) 17.09% and poor (BCS 1.71%) . Most of the sheep (98.29%) were in BCS 2 to BCS 4. The percentage of lame sheep that were at least 3 months of age and above was 96.6% compared to 3.4% that were less than 3 months of age. Out of the 117 lame sheep, 11.9% (14) were in late gestation, 65.0% (76) were not pregnant and 23.1% (27) were males. Out of the 90 females, 58.9% had lambed more than the previous 3 months prior to the

study, 7.8% had lambed within the previous 3 months prior to the examination and 33.3% had not lambed. It is possible that some may have been pregnant or not pregnant at the time of the study. Pregnancy was not verified during this study.

**Table 4.3**: Animal-level factors observed in 117 lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

| Animal factors | Various levels of the animal factors | | | |
|---|---|---|---|---|
| Breed | Dorper | Dorper and Red Maasai cross | Dorper and Merino cross | Red Maasai |
| Percentage (%) | 54.84 | 42.74 | 1.71 | 1.71 |
| Weight (kg) | <20 | 20-30 | 31-50 | >50 |
| Percentage (%) | 1.71 | 17.09 | 63.25 | 17.95 |
| Age (Months) | <3 | ≥ 3 | -. | - |
| Percentage (%) | 3.4 | 96.6 | - | -. |
| Sex | Females | Males | - | - |
| Percentage (%) | 76.9 | 23.1 | - | - |
| Pregnancy | Pregnant | Not pregnant | Males | - |
| Percentage (%) | 11.9 | 65.0 | 23.1 | - |
| Lambing period | Current 3 months | More than previous 3 months | Not lambed | -. |
| Percentage (%) | 7.8 | 58.9 | 33.3 | - |

### 4.4.2 Farm-level factors

#### 4.4.2.1 State of the grazing areas

The areas that the sheep spent most of the time grazing in the 10 farms were of uneven bumpy terrain in 8 while flat ground in 2 of them. In one of these 8 farms, the ground was swampy and marshy. In 8 of the farms, there were traumatic objects such as thorny plants and small loose sharp pebbles of stones in the grazing areas. These loose sharp pebbles of stones were also found along the sheep walking tracks. The other 2 farms were free of any traumatic objects. The walking tracks had trench-like excavations in 6 of the 10 farms.

#### 4.4.2.2 Pastures and feeding

Since the study was carried out during the rainy season and immediately after the rains, the pastures were green in all the 10 farms. It was reported in 5 of the 10 farms that during drought when pastures were scarce, the sheep were supplemented with commercially available concentrates and hay. Four of these 5 farms supplemented with only grain concentrate, while the remaining one farm supplemented with only hay. The other 5 farms did not provide any supplements but the sheep were left to live on the scantily available pastures.

### 4.4.3 Management-level factors

#### 4.4.3.1 Hygienic state of the night-resting enclosures

In 5 of the 10 farms the night-resting enclosures were wet with manure accumulation while 5 were dry but also had manure accumulation.

#### 4.4.3.2 Hoof trimming

Hoof trimming was routinely carried out in 5 of the 10 farms and was either done by owners, stockmen, animal health assistants or veterinary surgeon. In three of the 5 farms the trimming was being carried out by unqualified personnel which predisposed them to over-trimming.

#### 4.4.3.3 Foot bathing

Only one of the 10 farms was carrying out foot bathing using formalin solution in a plastic container once a week.

#### 4.4.3.4 Ticks control

All the 10 farms practiced tick control methods by hand spraying using knap sack sprayers.

### 4.5 Association between possible risk factors and lameness

#### 4.5.1 Association between animal-level factors and lameness

The number of limbs affected was significantly associated with moderate to severe degrees of lameness ($\chi^2$ =11.15, p<0.05). The affected limb (whether fore or hind limb) ($\chi^2$ =9.20, p < 0.05) and the involved claws (whether lateral or medial) ($\chi^2$ =16.98, p < 0.05) were also significantly associated with degrees of lameness. There was significant but weak association between the presence of a lesion on the limb with mild to severe degrees of lameness ($\chi^2$ =4.71, p<0.05). The rest of the animal-level risk factors such as

sex, breed, body condition score and period when ewe lambed did not seem to influence the occurrence of lameness (Table 4.4)

### 4.5.2 Association between farm-level factors and lameness

There was significant association between the presence of traumatic objects in the farms and mild to severe degrees of lameness ($\chi^2 = 11.01$, $p < 0.05$). The other farm-level factors that were determined such as terrain, grazing ground, type of traumatic object and farm tracks did not show any statistically significant association with lameness (Table 4.5).

**Table 4.4**: Association between the locomotion score and animal-level factors in 117 sheep examined during a study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

| Animal-level risk factor | Chi-square ($\chi^2$) | p-value | Conclusion |
|---|---|---|---|
| Lesion | 4.71 | 0.030 | Associated |
| No of limbs affected | 11.15 | 0.004 | Associated |
| Affected limb | 9.20 | 0.010 | Associated |
| Involved claw | 16.98 | 0.051 | Associated |
| Claw deformity | 2.05 | 0.152 | No association |
| Type of claw deformity | 2.36 | 0.124 | No association |
| Sex | 0.97 | 0.325 | No association |
| Breed | 0.33 | 0.567 | No association |
| Weight | 2.25 | 0.324 | No association |
| BCS | 0.09 | 0.762 | No association |
| Pregnancy | 1.11 | 0.292 | No association |
| Lambed | 1.40 | 0.237 | No association |
| Location of the lesion | 3.13 | 0.792 | No association |

**Table 4.5**: Association between the locomotion score and farm-level factors in 117 sheep examined during a study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010)

| Farm-level Factors | Chi-square ($\chi^2$) | p-value | Conclusion |
|---|---|---|---|
| Traumatic objects | 11.01 | 0.0001 | Associated |
| Types of traumatic objects | 0.22 | 0.64 | No association |
| Terrain | 0.74 | 0.389 | No association |
| Grazing ground | 0.06 | 0.814 | No association |
| Farm tracks | 0.03 | 0.863 | No association |

# CHAPTER 5

## 5.0 DISCUSSION

The results of the current study revealed that the overall prevalence of lameness in sheep reared under free-range grazing system in the semi-arid district of Kajiado, Kenya is low at about 6.1%. This differs with findings in arid zones of Nigeria in which prevalence of lameness in sheep is higher (Bokko *et al.*, 2003). The differences in these prevalence's may in part be due to variations in the predisposing conditions in the arid and semi-arid climatic conditions in these two different regions. The main conditions causing lameness in sheep in the current study are non-infectious especially sole bruising, overgrown hooves and tick-bite dermatitis as has been reported previously (Eze, 2002; Bokko *et al.* 2003). The prevalence rate of 6.1% in this current study is within the range found in the United Kingdom (DEFRA, 2003a). However, it is lower than the range of 15-19.5% reported by others (Mohammed *et al.*, 1996; Eze, 2002; Bokko and Chaudhari, 2004). The low prevalence in the current study can probably be attributed to the fact that the semi-arid nature of the study area, provides a dry animal living-environment most of the year and almost all the risk factors that were observed (apart from presence of traumatic objects) were not significantly contributing to the occurrence of lameness. This differs with previous reports of arid and semi-arid conditions in Nigeria which resulted in slightly higher prevalence of lameness (Mohammed *et al,* 1996; Bokko and Chaudhari, 2004)

The finding of higher prevalence of foot lesions as compared to those in the proximal parts of the limbs in this study agrees with previous reports that indicated claw lesions as

the commonest cause of lameness in sheep (Bokko and Chaudhari, 2004). Distribution of foot lesions between the foot and proximal parts of the limbs as found in this study were similar to those reported for dairy cows (Cook *et al.*, 2004).

Painful responses leading to lameness was seen in the sheep with sole bruising in this study. This was probably due to the thinning of the horn of the sole in the bruised parts, which allowed transmission of pressure to the dermis of the claw when the sheep walked with their weight against the hard ground. Similar observations were made in sole bruising in cattle (Nguhiu-Mwangi, 2007; Nguhiu-Mwangi *et al.*, 2008).

In the current study, tick-bite dermatitis was the third most prevalent condition causing lameness. This can probably be attributed to the fact that most of the free-range grazing grounds in the study zone are likely to be tick-infested particularly from cattle, which are nomadically driven in search of pasture from place to place by the same sheep owners. The ease with which tick-dermatitis develops may be attributed to the density of aggregating ticks particularly on the plantar (caudal) aspect distal to the dew claws, as well as probable reaction to injected toxins by the ticks during the bites and tissue damage caused by the mouthparts of the ticks. All these lead to acute inflammation with pain and subsequent lameness. Similar dermatitis attributed to tissue damage by the large mouthparts of the ticks has been observed previously (Azizi and Yakhchali, 2006).

The rest of the conditions such as interdigital dermatitis, shelly hoof, soil balling, foreign body penetration, osteomyelitis of metatarsal bone, septic arthritis, malunion of tibial

bone, hyperextension of fetlock joint, over-trimmed hooves and foot rot were rare with prevalence of about 1% while some were incidental findings. The occurrences of some of these conditions such as foreign body penetration and fractures probably depended on accidental causes and others such as shelly hoof and soil-balling had low probability of occurrence depending on presence and suitability of the predisposing factors. The rampant presence of traumatic objects in the dry land pastures and accumulated manure in the night-resting enclosures increased the probability of occurrences of these conditions.

Infectious conditions such as foot rot were rare in the current study, possibly due to the harsh dry environment in which the causative agents could not propagate. However, the one sheep that had foot rot was severely lame because when these infectious conditions occur, the effects are destructive to the tissues and hence lameness is severe. This tends to support previous reports in cattle that dry environment reduces the incidence of foot lesions (Bergsten and Petterson, 1992). Although foot rot has been reported to be a flock problem which is highly contagious in sheep (Radostitis *et al.*, 2001; DEFRA, 2003a; The Merck Veterinary Manual, 2009), it was sharply contrasted by the low prevalence in the current study. The flock and contagious magnitude of foot rot is likely to be in sheep reared under persistent and prolonged wet conditions.

The few cases of interdigital dermatitis that were observed probably occurred owing to the fact that the study was carried out during the wet rainy season when the causative bacteria would easily multiply and the wet conditions of the foot environment would

enhance development of the lesions. This could also be due to the fact that interdigital dermatitis is more contagious than foot rot as has been reported previously (DEFRA, 2003a). It therefore might explain the reason more cases were seen with interdigital dermatitis than with foot rot.

The higher number of moderately lame sheep compared to the number with mild and severe lameness in this study could be attributed to the fact that the lameness conditions with the highest prevalence were found to be those that caused minimal pain or discomfort, such as sole bruising and hoof overgrowth. These more common conditions did not cause severe lameness, nevertheless their pain and discomfort exceeded mild degree of lameness, hence moderate lameness. The more painful infectious conditions had very low prevalence and hence the correspondingly low percentage of severely lame sheep. Similar findings have been reported in cattle (Nguhiu-Mwangi, 2007). A higher percentage of lame sheep were observed to have more than one foot affected simultaneously. This may be probably because the occurrence of the conditions with higher prevalence such as sole bruising, overgrown hooves and tick-bite dermatitis is most likely bilateral and thus involving more than one foot as well as several claws. The bilateral involvement of the limbs and claws observed in this study agrees with earlier reports (Mohammed *et al.*, 1996; Eze 2002; Bokko *et al.*, 2003).

The one sheep observed with over-trimmed hooves was definitely a management error or due to poor trimming skills. It caused lameness by the likelihood of exposed sensitive laminae treading directly on the ground or by the resulting interference with proper

weight distribution to the claws. It also may cause poor treading angle, which exerts pressure to limb structures that should not have much pressure. Similar observations of unskilled trimming in cattle leading to lameness have previously been cited (Blowey, 2002; Vermunt, 2004).

The one sheep found with interdigital soil-balling was predisposed by the manure stuck in the interdigital space, which was picked gradually little by little from what accumulated at the night-resting enclosure areas over prolonged time. The manure stuck in the interdigital spaces subsequently collects pieces of grass as the sheep grazes. Eventually that accumulated manure-grass mixture dries up within the interdigital spaces as "soil-balling" or "manure-balling" and leads to splaying of the toes as long as it remains on the claws. The splaying of the toes causes discomfort and pain that lead to mild lameness. Similar findings have been been reported previously (Clarkson and Faulli, 1990; Winter, 2004a). Although there were many sheep in the manure-accumulated night-resting enclosures, only one was found to have sustained the soil-balling lump. This is possible due to the fact that the probability of manure persistently getting stuck in the interdigital space may depend on individual variations of conformation of the claws and the space between them.

Dorper sheep or their crosses were found to be the preferred breeds by the Maasai community living in the study area, hence their higher numbers among the lame sheep relative to the other breeds. The tendency and the likelihood of selling off rams for slaughter and retaining the ewes for breeding of the flock is probably the reason why the

female sheep were more in number among the lame sheep. Similar observations have been made previously (Egwu *et al.*, 1994; Bokko and Chaudhari, 2004).

A higher percentage of the lame sheep was observed to be in good body condition probably corresponding to the higher percentage of the moderately lame which meant that the discomfort in these sheep was not severe enough to put them completely off feed. They were still able to move about slowly and feed, which resulted in maintenance of good body condition compared to poor body condition that would have resulted if the sheep had severe lameness. This deviates from observations by other researchers who reported poor body condition in majority of lame sheep probably because in their findings, the prevalence of severe lameness was also higher (Bokko and Chaudhari, 2004).

All animal-level factors including breed, age, sex, body condition score and lapse of time from lambing did not seem to significantly influence the occurrence of lameness. The prevailing uneven and bumpy terrain with a lot of stony pebbles in the majority of the evaluated farms, are likely to have predisposed the sheep to most of the lameness conditions affecting the claws. Similar observations on the influence of farm-level factors on occurrence of lameness have been reported (Clarkson and Faulli, 1990; Bokko and Chaudhari, 2004).

When more than one limb is affected by lameness conditions in any sheep, it is likely to cause much discomfort and pain that may precipitate difficulties in locomotion and

influence the locomotion score. This may explain the reason for strong significant association found between moderate to severe degrees of lameness and the number of limbs affected. A similar association that was found between the affected limb (fore or hind) or affected claw (lateral or medial), and the severity of lameness could probably be related to weight distribution in which the fore limbs bear more weight than the hind limbs, and the lateral claw bears more weight than the medial claw. These observations are similar to findings in cattle with more lameness on hind limbs, lateral claws of hind limbs and bilateral involvement (Tadich and Hernandez, 2000; Blowey, 2002; Vermunt, 2004). However, these observations sharply contrast previous reports which indicated that the fore limbs are subjected to more trauma than the hind limbs (Bokko and Chaudhari, 20004). Presence of several lesions on one single limb caused more discomfort and pain and this adversely affected the locomotion, resulting in the significant association found between presence of a lesion on the foot and mild to severe degrees of lameness.

The significant association between the presence of traumatic objects and the degree of lameness is probably attributed to difficulties in locomotion owing to discomfort and trauma caused by these objects on the treading surface of the claws. However, other farm-level factors such as grazing ground, type of traumatic object and farm tracks did not show any significant statistical association with lameness, nevertheless they could still have contributed to lameness by synergistically acting together with other predisposing factors. Similar findings have been reported in cattle (Greenough, 1991).

# CHAPTER 6

# 6.0 CONCLUSIONS AND RECOMMENDATIONS

## 6.1 Conclusions

The results of this study led to the following conclusions

6.1 1 Lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya is relatively low at 6.1%.

6.1.2 The main causes of lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya are non-infectious and infectious causes are negligibly low.

6.1.3 More than 90% of lameness in sheep involves the foot.

6.1.4 The main risk factor for lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya is presence of traumatic objects in the grazing ground.

## 6.2 Recommendations

The following recommendations can be made from the study as intervention measures to reduce the incidence of lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya:

6.2.1 Regular and skilled hoof trimming should be practiced.

6.2.2 Traumatic objects should be cleared from the grazing grounds.

6.2.3 Regular and effective methods of tick-control should be used.

6.2.3 Regular removal of manure from sheep night-resting enclosures should be encouraged.

6.2.4 Chemical footbaths will help to eliminate infectious causes of lameness and possibly use of formalin might harden the claw horn to prevent ease of bruising.

### 6.3 Areas for further research

There is a need to carry out further research related to the current study. These studies should include;

6.3.1 Studies in prevalence and risk factors of conditions causing lameness in high and medium potential lands of Kenya.

6.3.2 Controlled studies to verify the interactive role of various risk factors of sheep lameness.

6.3.3 Evaluation and quantification of the effect of lameness on financial economy in sheep enterprises

# CHAPTER 7

## REFERENCES


**Abel, C. (2008)**: "Bluetongue vaccine BTV8 questions answered" *Farmers Weekly.*

**Arkins, S. (1981)**: Lameness in dairy cows. *Irish veterinary Journal.* **35**:135-140

**Azizi, S. and Yakhchali, M. (2006)**: Transitory lameness in sheep due to Hyalomma infestation in Urmaia Iran. *Small Ruminant Research,* **63** (3):262-264.

**Bergsten, C. and Petterson, B. (1992)**: The cleanliness of cows tied in stalls and the health of their hooves as was influenced by the use of electric trainers. *Preventive Veterinary Medicine.* **13**:229-238.

**Berry, S. L. (1999)**: Hoof health. In Proceedings of the World Dairy Management Conference, Las Vegas, Nevada. pp 13-17. April 8-10, 1999.

**Blowey, R. W. (2002)**: Claw trimming: How should it be done? A comparison of two approaches. In Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando, FL, USA. pp 122-126, 9th -13th January 2002.

**Bokko, B. P; Adam, S. S. and Mohammed, A. (2003)**: Limb conditions that predispose sheep to lameness in the arid zone of Nigeria. *Small Ruminant Research.* **47** (2); 165-169.

**Bokko, B. P. and Chaudhari, S. U. R. (2004)**: Prevalence of lameness in sheep in the North East region of Nigeria. *International Journal of Agriculture and Biology.* **3-4**:519-521.

**Caple, I. W. (1990)** Vitamin D deficiency In Sheep Medicine, University of Sydney Post-graduate Committee in Veterinary Science, Proceedings No **141**, pp 381

**Clarkson, M. J. and Faulli, W. B. (1990)**: Lameness: A Handbook for the sheep Clinician, .4th Edition, Liverpool University Press, UK. pp 47-70.

**Clarkson, D. A. and Ward, W. R. (1991)**: Farm tracks, stockman's herding and lameness in dairy cattle. *Veterinary Record.* **129**:510-511.

**Cook, N. B; Nordlund, K. V. and Oetzel, G. R. (2004)**: Environmental influences on claw horn lesions associated with laminitis and sub acute ruminal acidosis in dairy cows. *Journal of Dairy Science.* **87**: E36-E46.

**Coulon, J. B; Lescouret, F. and Fonty, A. (1996)**: Effect of foot lesions on milk production by dairy cows. *Journal of Dairy Science* **79**: 44–49.

**Davis, R. C. (1982)**: Effects of regular formalin footbath on the incidence of foot lameness in dairy cattle. *Veterinary Record.* **111** (17):394.

**DEFRA. (2003a)**: Lameness in Sheep. DEFRA Publications. Available at: http://www.defra.gov.uk/animalh/welfare/pdf/sheeplameness.pdf April 2011.

**DEFRA (2003b)**: Code of Recommendations for the Welfare of Livestock: Sheep. DEFRA Publications.Availableat:http://www.defra.gov.uk/animalh/welfare/farmed/farmed/sheep/booklets/sheep.pdf April 2011.

**Department of Veterinary Services Annual Reports. (1995-2008)**. Ministry of Livestock Development, Republic of Kenya

**Egwu, G; Adamu, S. S; Ameh, J. A ; Ongeyili, P. A; Abana, P. S; Chaudhari, S. U. R. and RaboJ. S. (1994)**: Reproductive, clinic pathological and microbiological studies of interdigital pouch lameness in sheep in an arid zone of Nigeria. *Bulletin Animale production Africana.* **42**: 5-11.

**Elliot, M. and Pinkus, T. (1993)**: Homoeopathy, the Shepherd's Guide. Ainsworthy pharmacy, London. ISBN 0-9523411-2-3, pp 28.

**Eze, C, A. (2002)**: Lameness and reproductive performance in small ruminants in Nsukka Area of the Enugu State, Nigeria. *Small Ruminant Research.* **44**: 263–28867.

**Gairdner, J. (2007):** "Bluetongue outbreak in the UK. *Veterinary Record.* **161**: 534–5.

**Gatenby, R. M. (1986)**: Sheep production in the tropics and sub-tropics. Longman Singapore Publishers Ltd, London. pp 351.

**Gelasakis, A. I; Valergakis, G. E. and Arsenos, G. (2009)**: Predisposing factors of sheep lameness. *Journal of the Hellenic Veterinary Medical Society.* **6** (1): 63-74.

**Green, L. E; Hedges, V. J; Schukken, Y. H; Blowey, R. W. and Packington, A. J (2002)**: The impact of clinical lameness on the milk yield of dairy cows. *Journal of Dairy Science.* **85**: 2250–2256.

**Greenough, P. R. (1991)**: A review of factors predisposing to lameness in cattle. In: Breeding for Disease Resistance in Farm Animal, J. B, Owen and R. .F, Axford. Ed, CAB International, Wallingford, U.K. pp 371-393.32

**Greenough, P. R. and Vermunt, J. J. (1991)**: Evaluation of sub-clinical laminitis in a dairy herd and observations on associated nutritional and management factors. *Veterinary Record.* **128**: 11-17.

**Harwood, D. G; Cattell, J. H; Lewis, C. J. and Naylor, R. (1997):** Virulent foot rot in sheep. *Veterinary Record* **140**: (26) 687.

**Henderson, D. C. (1990)**: The veterinary book for sheep farmers. Farming Press Books, Ipswich, UK. pp 592-595.

**Hungerford, T. G. (1990):** Lameness. Hungerford's Diseases of Livestock, 9th Ed. McGraw-Hill Book Co. Sydney. pp 1059-60.

**Jubb, T. F. and Malmo, J. (1991):** Lacsions causing lameness requiring Veterinary treatment in pasture fed cattle in East Gippsland. *Australian Veterinary Journal.* **68**:21-24.

**Kariuki, D. P. and Letitiya. W. (1996):** Livestock production and health challenges in pastoral areas of Samburu district, Kenya Agricultural Research Institute (K.A.R.I.), Nairobi, Kenya.

**Kitching, R. P. (2004):** Contagious Pustular Dermatitis Infectious diseases of livestock.. Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University press, Cape Town, S A. pp 1282-1286.

**Marshall, D. J; Walker, R. I; Cullis, B. R. and Luff, M. F. (1991):** The effect of footrot on body weight and wool growth of sheep. *Australian Veterinary Journal.* **68**: 45–49.

**Mgasa, M. N. and Arnbjerg, J. (1993):** Occurrence of lameness and digital lesions in Tanzanian goats. *Small Ruminant Research.* **10**: 55-62.

**Mohammed, A. U. A; Badau, R. O. and Kene, R. O. C. (1996):** Lameness in sheep and goats in relation to hoof conditions in Sahel zone of Nigeria. *Bulletin. Animale Health Production. Africana.* **44**: 97-100.

**NADIS (2003):** Lameness not caused by Scald or Footrot. Sheep Disease Focus:

**National Livestock Policy. (2008).** Ministry of Livestock Development, Republic of Kenya.

**Nguhiu-Mwangi, J. (2007)**: Characteristics of laminitis and associated claw lesions in dairy cows in Nairobi and its environs under various management systems, PhD. Thesis, University of Nairobi, Kenya.

**Nguhiu-Mwangi, J; Mbithi, P. M. F; Wabacha, J. K. and Mbuthia, P. G. (2008):** Prognostic indicators and the importance of trimming in non-infective claw disorders in cattle. *The Kenya Veterinarian.* **32** (1) : 26-40.

**Nonga, H. E; Makungu, M; Bittegeko, S. B. P. and Mpanduji, D. G. (2009)**: Occurrence and management of lameness in goats: A case study of Magadu farm, Morogoro, Tanzania. *Small Ruminant Research,* **82:** 149-151.

**Offer, J. E; Mcnully, D. and Logue, D. N. (2000)**: Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. *Veterinary Record.* **147**: 105-109.

**Otieno, G. O. (2008):** Ministry for Development of Northern Kenya and other Arid Lands, Arid Lands Resource Management Project ii –Kajiado District

**Parajuli, B. and Goddard, P. J. (1989)**: A comparison of the efficacy of footbaths containing formalin or zinc sulphate in treating ovine foot rot under field conditions. *British Veterinary Journal.* **145**: 467-472.

**Pugh, D, G, (2002)**: Sheep & Goat Medicine. Philadelphia: Saunders. ISBN 0-7216-9052-1.

**Raadsma, H. W; Egerton, J. R; Outteridge, P. M; Nicholas, F. W; Brown, S. C. and Litchfield, A. M. (1990)**: An investigation into genetic aspects of resistance to foot rot in Merino sheep. *Wool Technology and Sheep Breeding.* **38**: 7-12.

**Radostitis, O. M; Gay, C. C; Blood, D. C. and Hinchcliff, K. W. (2001)**: A textbook of diseases of cattle, sheep, pigs, goats and horses, 9th edition. Paston press Ltd, London, Norfolk.

**Rhebun, W. C. and Pearson, F. G. (1982)**: Clinical management of bovine foot problems. *Journal of American Veterinary Medical. Association.*.**181**:572-579.

**Russell, A. M; Rowlands, G. J; Shaw, S. R. and Weaver, A. D. (1982)**: Survey of lameness in British dairy cattle. *Veterinary Record.* **111**:155-160.

**Scott, K. and Henderson, D. C. (1991)**: Foot Rot and Foot Conditions. In: Diseases of Sheep. 2nd edition.. W.B. Martin .and I. D. Aiken, Eds. Blackwell Scientific Publications, Oxford. pp 201-209.

**Suiter, J. (2006)**: Body condition scoring of sheep and goats, Department of Agriculture, Western Australia. Farmnote, 69/2006.

**Tadich, N. and Hernández. M. (2000)**: A survey on the prevalence of foot lesions in sheep from 25 small holdings in the province of Valdivia, *Archives Medicine Veterinary*. **32**.1.

**The Kenya National Population and Housing Census Results. (2010):** Ministry of State for Planning, National Development and Vision 2030.

**The Merck Veterinary Manual (2009)**: A Handbook of Diagnosis, Therapy and Disease Prevention and Control for the Veterinarian. 9th edition, Editor C. M, Fraser and Kahn, M.A. Published by Merck and Co. Inc. Railway, N. J, USA.

**The Veterinary Formulary. (1998)**: 4th Edition. Eds. Y. Bishop Pharmaceutical Press, London pp 395-406.

**Tranter, W. P; Morris, R. S; Dohoo, I. R. and Williamson, N. B. (1993)**: A case control study of lameness in dairy cows. *Preventive Veterinary Medicine*. **15**. 199-203.

**Vermunt, J. (2004):** Herd Lameness: A review, major causal factors, and guidelines for prevention and control. In: Proceedings of the 13th International Symposium and 5th Conference on Lameness in Ruminants. 11th -15th February 2004. Maribor, Slovenija. pp 1-15

**Vermunt, J. J. and West, D. M. (2004)**: Toe Fibroma, Toe Abscess and Foot Rot. In; Infectious Diseases of Livestock, Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University press, Cape Town, S A. pp 1725-1738.

**Verwoerd, D. W. and Erasmus, B. J. (2004)**: Blue Tongue Disease. In: Infectious Diseases of Livestock..Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University Press, Cape Town, S A. pp 1201-1215.

**Veterinary Education and Information Network (VEIN). (2008)**: Blue Tongue Disease. Foot and Mouth Disease. *Journal of Clinical Microbiology*. **47**(9): 2992-2994.

**Warnick, L, D; Janssen, D; Guard, C, L. and GröHn, Y, T. (2001)**: The effect of lameness on milk production in dairy cows. *Journal of Dairy Science*. **84:** 1988–1997.

**West, D. M. (1990)**: Foot abscess and other lameness problems of sheep. In: Sheep Medicine. Proceedings of the Study and Georgiana Reid Memorial refresher course for Veterinarians, 16th -20th July 1990. University of Sydney, Australia.

**Whittington, R. J. (1995)**: Observations on the indirect transmission of virulent ovine foot rot in sheep yards and its spread in sheep on unimproved pasture. *Australian Veterinary Journal.* **72** (4): 132-134.

**Winter, A. (1998a)**: Lameness in Sheep. The Moredun Foundation News Sheet. **3:** 1.

**Winter, A. (1998b)**: Virulent Foot rot in sheep. *Veterinary Record,* **141**: 1.

**Winter, A. (2004a)**: Lameness in sheep. *Diagnosis in Practice*, **26** (2): 58-63

**Winter, A. (2004b)**: Livestock Health and Welfare Division, Liverpool.

**Winter, A. and Charmley, J. (1999)**: The Sheep Keeper's Veterinary Handbook. Crowood Press Ltd, Marlborough, UK. ISBN 1-86126-235-3.

**Zaria, L. T. and Damin, J. (2004)**: Dermatophilosis. In: Infectious Diseases of Livestock. Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University press, Cape Town, S A. pp 2026-2035.

# CHAPTER 8

## APPENDICES

**Appendix 1:** Data collection sheets with parameters and their codes that were used during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**a) Animal-level factors**

1) Sheep code 1 Serial no up to 117
2) Farm ID 1= Farm no one 2= Farm no two ..... up to 10.
3) Flock size 1= 100-150 2= 151-200 3= 201- 250 4= 251-300 5= 301-350
4) Estimated age 1= Lamb 2= Adult
5) Sex 1= Male 2= Female
6) Breed 1=Dorper 2= Cross of Dorper and Maasai sheep 3= Cross of Dorper and Merino 4= Maasai sheep
7) Body condition score 1= Poor 2= Fair 3= Good 4= Very Good
8) Pregnancy status 1= Late pregnancy 2= Not pregnant
9) Recently lambed 1= Less than 3 months 2= Above 3 months
10) Lameness severity score 1= Mild 2= Moderate 3= Definite 4= Severe
11) Number of affected limbs 1= 1 2 = 2 3 = 3 4= 4
12) Affected limb 1= Fore limb 2= Hind limb 3= Both
13) Position of lesion 1= Proximal 2= Foot
14) Involved claw 1= Medial 2= Lateral 3= Both
15) Specific lesions causing lameness

1= Overgrown hoofs 2= Sole Erosion 3= Tick-bite dermatitis 4= Interdigital Dermatitis 5= Hoof Fracture 6= others.

16) Claw deformity 1= Present 2= Absent

17) If present, specify the type 1= Misshapen hoof 2= Hoof cracked 3= Not Applicable

18) Any bone or Joint involvement 1= Yes 2= No

19) Specimen(s) collected 1 =Yes 2= No

20) Biopsy for any swelling(s) 1= Yes 2= No

**b) Interview questionnaire**

21) Have you experienced any case of lameness in sheep on the farm in the last three months? 1= Yes 2= No

22) If yes, how many cases? 1 = 1-5 2 = 6-10 3 = >10.

23) Was the sheep treated? 1 = Yes 2 = No

24) Do you practice hoof trimming? 1 = Yes 2 = No

25) If yes, who does it 1 = Owner 2 = Stockman 3 = Vet surgeon / Animal Health Assistant

26) How often per year? 1 = Once 2 = More than once

27) How do you control ticks 1= Dipping 2= Hand spraying 3= Other method 3= None

28) Do you supplement the sheep during prolonged drought 1 = Yes 2 = No

29) If yes what type Of feed 1 = Concentrates 2 = Hay 3 = Not applicable

30) Have you observed more cases of lameness in certain lineage of sheep than in others

1 = Yes 2 = No

31) Have you seen cases of lameness in cattle at the same time with sheep

1 = Yes 2 = No

32) If yes, how many sheep were involved? 1 = Less than 10 2 = More than 10 3 = Whole herd.

33) Do you know of diseases that affect other parts of the body and cause lameness in sheep 1 = Yes 2 = No.

34) If yes, which organs were affected 1 = Rest of the skin 2 = Testis 3 = Head region 4 = Other parts specify .....

35) Which season are sheep mainly lame 1 = Dry season 2 = Wet season

36) Are there herdsmen who rear animals and they report more lameness cases than others 1 = Yes 2 = No.

37) If yes. what do you attribute this to 1 = Overdriving 2 = Witchcraft 3 = Not clear

**(c) Farm-level factors**

38) Terrain 1 = Bumpy 2 = Level

39) Grazing ground 1 = Dry 2 = Marshy

40) Traumatic objects 1 = Present 2 = None

41) If present, the type 1 = Dried thorns 2 = Loose stones 3 = Dried pastures.

42) State of Farm tracks 1 = Even 2 = Loose stones 3 = Trenches.

43) Pasture conditions 1 = Dry 2 = Green

44) Type of management 1 = Free-range 2 = Padlocking 3 = Migration.

45) State of sheep house or night-resting enclosures 1 = Wet and a lot of manure 2 = Dry and a lot of manure 3 = Dry and little manure 4 = Wet in the morning and little manure.

46) Observation of whether there is a footbath 1 = Present 2 = Absent.

47) If present, name of the chemical used  1 = Formalin  2 = Any other

# PREVALENCE AND RISK FACTORS OF CONDITIONS CAUSING LAMENESS IN SHEEP UNDER FREE RANGE GRAZING SYSTEM IN KAJIADO DISTRICT, KENYA.

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A thesis submitted in partial fulfillment of the requirements for Master of Science degree in Clinical Studies, University of Nairobi.

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October 2011

## DECLARATION

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

02/11/2011

Samuel Gitau Mugo (B.V.M)

This thesis has been submitted for examination with our approval as University supervisors

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## DEDICATION

To

My wife, Lucy and my children Teresiah and Timothy.

To God is all the glory and honour.

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## ABBREVIATIONS AND SYMBOLS

| | | |
|---|---|---|
| ASAL | - | Arid and Semi-Arid Lands |
| CODD | - | Contagious Ovine Digital Dermatitis |
| DEFRA | - | Department for Environment Food and Rural Affairs |
| FMD | - | Foot and Mouth Disease |
| GDP | - | Gross Domestic Product |
| NADIS | - | National Animal Diseases Information Service |
| VEIN | - | Veterinary Education and Information Network |
| Km | - | Kilometer |
| mm | - | Millimeters |
| df | - | Degrees of freedom |
| *et al* | - | At least three authors |
| P | - | P value |
| < | - | Greater than |
| > | - | Less than |
| ( | - | Opening bracket |
| ) | - | Closing bracket |
| % | - | Percentage |
| / | - | Division sign |

## ABSTRACT

Lameness can be a cause of negative economic output in sheep farming owing to its adverse effects on productivity, reproductive performance and poor growth performance in lambs. The extent of lameness in sheep and its associated predisposing causes in Kenyan sheep rearing systems has not been elucidated. This study was carried out in sheep under free-range grazing system in Kajiado District, Kenya, from March 2010 to June 2010 with the following objectives (1) to determine the prevalence of conditions causing lameness, (2) to determine the possible risk factors predisposing the sheep to lameness conditions.

This was a cross-sectional study in which each farm was visited several times, but each sheep on the farm was examined only once. Ten study farms were purposively selected from three divisions of Kajiado District, based on the willingness of the farmers to allow examination of their sheep and also on the stability of the farm's grazing routine from more nomadic tendencies. The data was collected either by filling a formal questionnaire with answers given through interviewing the farmers, farm managers or stockmen on animal-level factors, or by recording observations made on the farm regarding farm-level factors. The 10 farms had a total of 1916 sheep that met the study criteria. Out of these, 117 sheep were identified as lame during general locomotion scoring as they walked on a flat firm part of the ground. Each of these 117 sheep were examined closely for specific conditions or disorders causing lameness. Information on the actual disorders causing lameness was recorded. The location of the disorders on the limb, affected limbs whether fore or hind, and the affected claws whether lateral or medial were recorded in data

collection sheets. The lesions causing lameness were photographed. A mark was put on each examined sheep to avoid repeat examination.

Overall prevalence of lameness was 6.1% (117/1916), out of which the conditions with relatively higher percentages of occurrence were sole erosion (3.8%, 72/1916), overgrown claws (3.2%, 61/1916) and tick-bite dermatitis (1.6%, 30/1916). Infective conditions such as foot rot and interdigital dermatitis had prevalence of less than 1%. The rest of the conditions such as shelly hoof, soil-balling, over-trimming and bone problems were incidental findings each in a single sheep. The conditions causing lameness occurred on the foot in 94% (110/117) of the lame sheep and on proximal parts of the limb in 6% (7/117) of the cases. The distribution of the conditions among the lame sheep was 43.6% (51/117) on the hind limbs, 23.1% (27/117) on the forelimbs and 33.3% (39/117) affected both hind and fore limbs.

Although there were several animal-level factors evaluated, the only factors found to be significantly associated with higher locomotion score were the number of limbs with lesions ($\chi^2$ =11.15, p = 0.004), the affected limbs whether fore or hind ($\chi^2$ = 9.20, p = 0.010), the affected claw whether medial or lateral ($\chi^2$ = 16.98, p = 0.05) and the type of lesion ($\chi^2$ = 4.71, p = 0.030). The only farm-level factor that was significantly associated with higher locomotion score was presence of traumatic objects in the grazing grounds ($\chi^2$ = 11.01, p < 0.001).

This study concludes that the prevalence of lameness in sheep under free-range grazing system of dry zones such as Kajiado District is relatively low due to minimal farm-level risk factors. Similar prevalence studies should be carried out in high potential and wet areas of Kenya for comparison purposes.

# CHAPTER 1

## 1.0 INTRODUCTION

The livestock sector in Kenya contributes about 10% of the entire Gross Domestic Product (GDP) and 42% of the agricultural GDP (National Livestock Policy, 2008). Only one third of Kenya's land is suitable for agriculture while two-thirds is both arid and semi-arid (ASAL) in which the larger livestock population is reared. The arid and semi-arid lands support the pastoral communities in Kenya through livestock rearing which is the main source of their livelihood (Kariuki and Letitiya, 1996). The population of sheep in Kenya is estimated at about 17,129,606 million, most of which is under free-range grazing nomadic pastoralism and ranching systems (Kenya National Population and Housing Census Results, 2010).

Lameness is a major health problem in flocks of sheep worldwide. It is mostly associated with foot lesions (Gelasakis *et al.*, 2009). It impacts negatively on both welfare and economic productivity of individual sheep and entire flocks. Some of the negative effects of lameness include reduced weight gain, reduced birth weight of lambs, poor colostrum production by ewes and reduced reproductive performance (Henderson, 1990; Harwood *et al.*, 1997; Eze, 2002; DEFRA, 2003a). Lameness in sheep may be caused by many systemic and localized diseases, the commonest being foot rot, interdigital dermatitis, foot abscess and septic polyarthritis (Radostitis *et al.*, 2001; Vermunt and West, 2004; VEIN, 2008; The Merck Veterinary Manual, 2009). During the period between 1995 and 2008, annual reports of the Department of Veterinary Services in Kenya indicate that foot and mouth disease, black quarter, blue tongue, foot rot, fractures and arthritis are some of

the prevalent diseases contributing to sheep lameness (Department of Veterinary Services Annual Reports, 1995-2008).

The risk factors of lameness in sheep include; wetness of the environment, wet season, size and conformation of hooves, limb conformational defects and interdigital tick infestation (Bokko *et al.*, 2003; Azizi and Yakhchali, 2006).

This study was carried out in sheep under free-range grazing system with the purpose of determining the prevalence of conditions causing lameness and the possible risk factors predisposing the sheep to lameness conditions.

### 1.1 Justification

The status of lameness in sheep in Kenya is not known since no studies have been carried out previously. Sheep production forms part of the main livelihood of the pastoral communities in arid and semi-arid areas of Kenya and therefore a systematic study to establish the status of lameness was essential. The results of the study may give guidance for remedial and preventive measures and hence improve productivity of sheep and enhance the livelihood of these communities.

### 1.2 Objectives of the study

The study was therefore carried out with the following specific objectives:

1.2.1 To determine the prevalence of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya.

1.2.2 To determine the risk factors predisposing sheep to lameness conditions under free-range grazing system in Kajiado District, Kenya.

# CHAPTER 2

# 2.0 LITERATURE REVIEW

## 2.1. General overview and economic importance of lameness in sheep

Locomotion soundness is very vital for effective grazing, reproductive and production efficiency in all classes of livestock (Bokko *et al.*, 2003). Lameness is the alteration of gait and / or posture as a result of disease, limb disorders or trunk disorders. It is abnormal gait as a clinical sign, but not a disease in itself (Coulon *et al.*, 1996; Warnick *et al.*, 2001; Green *et al.*, 2002; Winter, 2004a; The Merck Veterinary Manual, 2009). Lameness is considered to be one of the most important health problems in sheep (Marshall *et al.*, 1991) and is an indication of pain, weakness, deformity, or other abnormalities in the musculo-skeletal system (The Merck Veterinary Manual, 2009). It can be divided into proximal limb lameness and foot lameness depending on the location of the lesion. Proximal limb lameness occurs when lesions are proximal to the fetlock joint, while foot lameness occurs when lesions are distal to the fetlock joint. The former has a lower prevalence rate compared to the latter (Hungerford, 1990).

Overgrown hooves, trauma, interdigital pouch inflammation, limb conformational defects, scalds, tick-bite dermatitis and fractures were reported to be among the causes of lameness in sheep in the arid zones of Nigeria but hoof overgrowth had the highest incidence (Eze, 2002; Bokko *et al.*, 2003). Ticks attached to the interdigital skin may cause lameness due to tissue damage and inflammatory reactions caused by their long mouth parts (Azizi and Yakhchali, 2006).

Lameness is also a major cause of economic loss in sheep as a result of poor or reduced production (Gatenby, 1986). The loss in production occurs through reduced weight gain in the fattening lambs, reduced wool growth and inadequate feed intake by the pregnant and lactating ewes resulting in pregnancy toxemia and neonatal diseases (Eze, 2002; DEFRA, 2003a). Lameness also affects reproduction by increasing the lambing interval and lowering of the ram's fertility (DEFRA, 2003a). The affected sheep have a significant fall in body weight and wool production during the period of lameness (Radostitis *et al.*, 2001). However the economic implication of lameness is difficult to quantify (Eze, 2002). Lameness is an important welfare determinant because it causes pain and discomfort (Offer *et al.*, 2000; DEFRA, 2003b). A survey carried out in the United Kingdom by the Royal Veterinary College established that the incidence of lameness in 547 farms was between 6 and 11% of all the sheep (DEFRA, 2003a). In Nigeria the incidence of lameness in sheep was found to be 15% (Eze, 2002).

**2.2 Normal functional anatomy of the ovine digit**

For purposes of description of lameness, the limb is divided into "proximal limb" and the "foot". Proximal limb is all parts of the limb proximal to the fetlock joint. The foot is all parts of the limb distal to the fetlock joint. The foot in the ovine is divided into two main digits and two accessory digits (dew claws). Each of the main digits is made up of three phalanges namely the proximal (P1), middle (P2) and distal (P3) phalanges. The ends of the digits are called "claws" or "hooves" and are covered by the horn capsule termed as the "hoof." The distal phalanx is inside the claw horn while the other two are outside the claw horn. The foot has two joints, which are the proximal interphalangeal joint (pastern

joint) and distal interphalangeal joint (coffin or pedal joint). The space between the two main digits is called the "interdigital space" which is made up of interdigital skin to which ticks attach and cause inflammation. The interdigital space can also accumulate dung (manure). These factors occurring in the interdigital space serve as predisposing causes for foot lameness (Berry, 1999; Clarkson and Faulli, 1990).

The claw is made up of the wall, sole, heel and white line. The claw wall refers to the hard horny structure (hoof) encasing the distal part of the digit on the dorsal, abaxial and the axial aspects. The walls particularly the dorsal and abaxial aspects are harder than the sole and the heel. The white line is the junction between the sole and the wall, while the coronet is the junction between the hoof wall and the skin. The horn is the epidermis of the claw while the corium is the dermis which contains the nerves and the vasculature. The corium produces the horn of the claw and so its damage results in defective horn production, which may lead to lameness. The main weight-bearing surfaces of the claw are the sole and the heel. The hardest parts of the claw that should naturally bear weight are the abaxial wall and the sole (Berry, 1999).

### 2.3 Aetiology and predisposing factors of lameness

The prevalence, type and severity of lameness in ruminants seem to vary from one region to another due to the prevailing predisposing factors in the region (Russell *et al.*, 1982). Foot lameness is considered to have multifactorial predisposing causes. Some of these are metabolic disturbances, trauma to the musculoskeletal system, lack of proper feet care and infections which are either systemic or localized to the limbs. The interdigital skin is

the primary site of invasion by infection, but this does not occur when the stratum corneum is dry and intact (Greenough, 1991; The Merck Veterinary Manual, 2009). Generally, the predisposing factors can be divided into three categories which include environmental factors, animal- and management-level factors.

### 2.3.1 Environmental factors

Environmental factors affect the prevalence of lameness within the flocks of sheep. Prevalence of lameness in a flock of sheep varies largely with pasture environment that can affect the feet (Clarkson and Ward, 1991). Lameness in sheep is more prevalent during the wet season and in the hind limbs (Mgasa and Arnbjerg, 1993). Wet environment causes softening of the hoof and maceration of interdigital skin, thus making it easy for penetration of foreign bodies and infection (Jubb and Malmo, 1991; Tranter *et al.*, 1993). For example sole erosion has a higher incidence during the rainy season than in dry season (Mgasa and Arnbjerg, 1993). Wetness of the pasture and animal rearing environment also favours proliferation of infectious agents especially *Fusobacterium necrophorum* and *Dichelobacter nodosus*, "the main causes of foot rot in sheep" (Greenough and Vermunt, 1991). Dry environment is cleaner and hygienic, hence reduces the incidence of foot lesions (Bergsten and Petterson, 1992). However, dry weather leads to desiccation of the hoof, which makes the horn hard, brittle and liable to cracking (Greenough, 1991). It has been reported that housing sheep greatly increases the incidence of lameness unless good husbandry practices are observed (Pugh, 2002).

### 2.3.2 Animal-level factors

Genetic factors attributed to individual animals predispose sheep to lameness (Gelasakis *et al.*, 2009). The incidence of lameness is higher in sheep that are less than four years of age. It decreases with age but the degree of lameness is more severe. Conformational defects which could have a genetic or inheritance factor in them also predispose to lameness (Bokko *et al.*, 2003).

### 2.3.3 Management-level factors

The management practices in the farms help to prevent or to treat the conditions that cause lameness, thus maintain and improve the efficiency of production (DEFRA, 2003a). Occurrence of lameness due to digital diseases in goats has been found to be related not only to climatic conditions but also to management factors (Nonga *et al.*, 2009). It has been reported that failure to practice foot-bathing and hoof trimming results in increased incidence of lameness in livestock (Arkins, 1981; Davis, 1982). Trimming of the hooves helps in the control of many of the lesions causing lameness (Tadich and. Hernández, 2000).

Other management factors that are associated with lameness include high stocking densities, failure to practice rotational grazing, lack of grass or concrete run, failure to add bacteriostats to dips and lack of mineral supplement (DEFRA, 2003a; Gelasakis *et al.*, 2009). Poorly maintained farm tracks with loose stones and trenches as well as overdriving of the animals by stockmen when herding them increase the risk to lameness (Clarkson and Ward, 1991). Nutrition is a fundamental factor associated with the health

of the foot and the animal in general. Hence, sheep fed on unbalanced rations suffer deficiencies in specific nutrients such as zinc that is involved in the keratinization of hoof wall and this could predispose to lameness (Gelasakis *et al.*, 2009).

### 2.4 Specific conditions causing lameness in sheep

The most common causes of lameness in sheep are infectious which could be systemic or localized in the foot, injuries and nutritional imbalances (The Merck Veterinary Manual, 2009). In this part of the literature, the frequently encountered conditions causing lameness are discussed.

#### 2.4.1 Foot rot

Foot rot is a highly contagious disease of sheep caused by dual infection with *Fusobacterium necrophorum* and *Dichelobacter nodosus* (Radostitis *et al.*, 2001), which are gram negative and anaerobic. *Fusobacterium necrophorum* is a normal residence of the sheep's environment, but *Dichelobacter nodosus* does not survive for more than a few days in the soil or pastures. Its long-term presence depends on the presence of infected animals (The Merck Veterinary Manual, 2009). Foot rot is the main infectious cause of lameness in sheep. It is characterized by inflammation of the skin at the skin-horn junction with severe lameness and occasionally resulting in animals walking on their knees. There is interdigital dermatitis, under-running of the hoof, foul odour of necrosis of the horn and in some cases all the four feet are affected (Radostitis *et al.*, 2001; Vermunt and West, 2004). Foot rot is initially caused by *Fusobacterium necrophorum* which starts as scald and later *Dichelobacter nodosus* invade the lesion. There are

different strains of *Dichelobacter nodosus* which have varying virulence. The synergistic presence of *Fusobacterium necrophorum* and *Dichelobacter nodosus* causes separation of the horn from the underlying structures of the foot. Depending on the strain of *Dichelobacter nodosus* involved, this separation may spread under the entire sole and up the wall of the hoof (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009). In the farm, exposure of the feet to wet pasture, hydration and hyperkeratosis of the stratum corneum of the interdigital skin and invasion of interdigital skin by *Fusobacterium necrophorum* lead to development of interdigital dermatitis (The Merck Veterinary Manual, 2009).

Acute foot rot is characterized by swelling, moistness of skin of the interdigital cleft and slight lameness that increases as necrosis under-runs the horn of the cleft (The Merck Veterinary Manual, 2009). Extensive under-running of the horn leads to severe lameness whereby the sheep carry up the leg. When the under-running affects more than one foot the sheep walks on its knees or remains recumbent. There is also a foul smelling discharge. Severely affected sheep sometimes are anorexic. Both *Fusobacterium necrophorum* and *Dichelobacter nodosus* survive in pasture for up to 12 days under favourable conditions, hence rotational grazing and isolation of infected animals can help in control of the disease (Radostitis *et al.*, 2001).

Foot rot should be viewed as a flock problem (DEFRA, 2003a). Management of foot rot in sheep involves both topical and parenteral treatment. Treatment methods include isolation of severely affected sheep, careful hoof paring and topical application of

bactericidal solutions such as formalin, copper sulphate or zinc sulphate solution. In severe cases the long-acting antibiotics such as oxytetracycline should be administered. Culling of any sheep that do not respond easily to treatment will help reduce the likelihood of future infections. Vaccination of affected sheep with a bacterin composed of *Dichelobacter nodosus* cells helps in the prevention and control. Most of the affected sheep recover with adequate treatment and when treated early (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009).

### 2.4.2 Interdigital dermatitis

Interdigital dermatitis is caused by an early mild infection with *Fusobacterium necrophorum*. Injuries to the interdigital epidermis may also result in interdigital dermatitis. This disease often predisposes and progresses to foot rot and foot abscess (The Merck Veterinary Manual, 2009). Interdigital dermatitis is characterized by an acute inflammatory condition of the interdigital skin, which has moist necrotic material, pitting and blanching of the horn, maceration and necrosis at the skin-horn junction. This results in separation of the horn at the heel with limited under-running of the horn and no odour. There is mild lameness (Radostitis *et al.*, 2001). Excessive moisture and heavy dung contamination of the environment are the most important predisposing factors (West, 1990; Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009).

Interdigital dermatitis should be viewed as a flock problem due to the common predisposing factor (DEFRA, 2003a). Most lesions heal rapidly when sheep are transferred to dry conditions. Topical applications of aerosol antibiotics and foot bathing

cauterizing agents such as 5% formaldehyde or 10% Zinc sulphate solution are quite effective in the treatment of interdigital dermatitis (DEFRA, 2003a; The Merck Veterinary Manual, 2009).

### 2.4.3 Foot abscess

Foot abscess affects adult sheep especially pregnant ewes and rams. It is particularly common in sheep that are driven to the pasture through roads with stony areas. The main bacteria involved in causing foot abscess in sheep are *Fusobacterium necrophorum* and *Actinomyces pyogenes* (The Merck Veterinary Manual, 2009). Foot abscess is an acute, suppurative infection, usually involving one digit of the foot. In most cases infection enters into the interdigital space causing interdigital dermatitis and extends deeper into one of the digits to involve the distal interphalangeal joint, associated ligaments and eventually the tendons. It may occur as toe abscess in which there is under-mining of the horn at the toe. Pain is severe and there may be swelling of the coronet, with eventual rupture oozing purulent discharge (Radostitis *et al.*, 2001). There is acute lameness (The Merck Veterinary Manual, 2009). When the abscess is exposed, pus and sinus tracts are observed (Vermunt and West, 2004). The lesion could also occur as heel abscess that results from extension of interdigital dermatitis into the soft tissues of the heel. When the abscess spreads deeper to involve interphalangeal joints, there is severe swelling at the caudal aspect of the foot which could rupture to discharge pus. When the abscess ruptures, there is marked reduction in pain and the gait improves tremendously due to relief of pressure to the underlying tissues of the claw (Radostitis *et al.*, 2001).

Treatment by surgical drainage, parenteral administration of Sodium Sulfadimidine solution and application of a local dressing is usually adequate (Radostitis *et al.*, 2001). However once the infection becomes established in the joint, treatment is of limited value (The Merck Veterinary Manual, 2009).

### 2.4.4 Contagious Ovine Digital Dermatitis

Contagious ovine digital dermatitis (CODD) is a highly contagious, erosive and proliferative infection of the epidermis proximal to the skin-horn junction in the flexor region of the interdigital space. Morbidity within a flock can be more than 90%. It affects any breed or age group but young sheep and sheep with poor immune response are most susceptible. Both the erosive and the proliferative lesions cause varying degrees of discomfort and give rise to severe lameness (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009). The essential difference between conventional foot rot and CODD is that CODD lesion starts at the coronary band. The ulcerative and proliferative lesions progress to under-running of the claw with complete detachment of the hoof in severe cases. The cause of the condition is not yet understood, but a variety of bacteria, including *Spirochaetes* have been identified in affected feet. Effective treatment involves use of antibiotics and footbaths (DEFRA, 2003a).

### 2.4.5 Claw deformities

Claw deformities are conditions of the foot where the claw overgrows or grows abnormally and may either directly cause lameness or predispose to other foot lesions. The common claw deformities are overgrown hooves and conformational defects. The

hoof overgrowth is characterized by increased length of the wall or sole that results in misshapen claws (VEIN, 2008). In some of the deformities, it is difficult to reshape the affected claws even by trimming. The documented claw deformities include the following:

#### 2.4.5.1 Regular hoof overgrowth

Regular hoof overgrowth occurs mostly when sheep are reared on soft surfaces where little hoof wear takes place. This results in increased length of the wall of the claw or sole (Rhebun and Pearson, 1982; Mohammed *et al.*, 1996).

#### 2.4.5.2 Beak claw

This is a claw deformity in which the dorsal surface of the claw is concave while the weight bearing surface is convex. The toes are turned upwards. This condition is reported in cattle (Rhebun and Pearson, 1982).

#### 2.4.5.3 Corkscrew claw

This is a claw deformity characterized by medial spiraling of the abaxial claw wall towards the axial plane of the normal claw. It is probably an inherited problem and trimming cannot reshape the claws to normal shape. This condition is reported mainly in cattle (Rhebun and Pearson, 1982).

#### 2.4.5.4 Scissor feet

This is manifested as an overlapping of the toes. It has been reported to be an inherited condition. This is mainly a condition of cattle (Rhebun and Pearson, 1982).

Management of claw deformities includes routine inspection of the feet of all sheep should be carried out at regular intervals. Foot trimming should be done by a skilled person. This reshapes the claws and eliminates the cracks and crevices that could trap mud and harbour foot rot bacteria (DEFRA, 2003a).

### 2.4.6 Shelly hoof (white line degeneration)

Shelly hoof results from separation of the hoof wall close to white line at the toe and is common in sheep grazing on lush pasture. Aetiology is thought to be nutritional (Winter, 2004a). The outer wall of the claw becomes loosened, forming a pocket between the hoof and the digit. A cavity forms in the hoof and is filled with soil and dung. Bacteria may enter and lead to abscess formation. It results in acute lameness. Unless the infection is present, management involves paring the feet and cleaning the dung and soil out of the cavity but if infection is present or suspected, the sheep should be foot-bathed with either copper sulphate, zinc sulphate or formalin solution (DEFRA, 2003a; VEIN, 2008).

### 2.4.7 Soil balling

Soil-balling is impaction of the interdigital space with a mixture of grass and manure or soil. The grass is matted by manure and soil, eventually becoming a lump stuck in the interdigital space. This accumulation causes lateral separation of the toes that leads to

mild pain and moderate lameness. The sheep shows discomfort as it walks until the lump falls off or is removed (Clarkson and Faulli, 1990; Winter, 2004b). Management involves the removal of the lump and this may need to be softened with water or cracked into pieces prior to removal (DEFRA, 2003a).

### 2.4.8 Septic polyarthritis

Septic polyarthritis is an acute or chronic arthritis of several joints of the limbs in lambs mainly caused by *Erysipelothrix rhusiopathiae*. There is sequestration of bacterial infection in the joints of both fore and hind limbs. It mainly affects the carpal, tarsal and interphalangeal joints. This affects lambs with umbilical infections or infection after docking and castration. Septic polyarthritis is predisposed by poor body condition of lambs at the time of surgery or adverse weather afterwards (The Merck Veterinary Manual, 2009). It is characterized by local pain, heat and swelling of the affected joints with severe lameness (Radostitis *et al.*, 2001; The Merck Veterinary Manual, 2009).

Septic arthritis requires prompt treatment to avoid irreparable damage. Systemic broad-spectrum antibiotics are indicated, which could be administered both systemically and intra-articularly. Joint lavage, arthroscopic debridement and drainage could be done. Supportive treatment with Non-Steroidal Anti-inflammatory Drugs (NSAIDs) is also useful. The effectiveness of treatment should be monitored carefully with clinical signs and repeat synovial fluid analyses (The Merck Veterinary Manual, 2009).

### 2.4.9 Toe granuloma

Toe granuloma is a smooth strawberry-like growth at the site of damage on the sole or axial hoof wall. The overlying horn fails to grow back normally. This occurs after over enthusiastic foot paring which leads to bleeding. It also results after severe long-standing foot rot, toe abscess or puncture wounds. It may eventually cause overgrown misshapen hoof because the animal fails to bear full weight on the affected foot. Affected sheep are extremely lame. The strawberry-like growth becomes covered with loose horn but never heals properly and bleeds when touched (Scott and Henderson, 1991; Winter, 1998a). Management of toe granuloma involves surgical excision of the granulomatous tissue and the adjacent loose horn. Also cautery and repeated application of astringents such as copper sulphate are recommended (NADIS, 2003)

## 2.5 Nutritional causes of lameness

### 2.5.1 White muscle disease

White muscle disease is a degenerative muscle disease (Pugh, 2002). This is caused by selenium and vitamin E deficiency in sheep. The deficiency leads to muscular dystrophy and the sheep are unable to stand or walk. There is bilateral necrosis and calcification of limb muscles, leading to lameness (Radostitis *et al.,* 2001). Treating the cardiac form of white muscle disease is usually ineffective and the sheep that survive often do not thrive because of the residual cardiac damage. The muscular form of the disease can be treated with supplements of selenium and/or vitamin E (Pugh, 2002).

### 2.5.2 Laminitis

Laminitis results from aseptic inflammation of the sensitive laminae of the claws. It is predisposed by sudden introduction of high amounts of concentrate feeds to sheep. Clinically there is pain around the coronet leading to severe lameness (Radostitis *et al.,* 2001). There is also occurrence of septic laminitis referred to as the "Lamellar suppuration". This is an acute bacterial infection of laminar matrix of the hoof by *Fusobacterium necrophorum* and *Actinomyces pyogenes.* The infection is enhanced by impaction of interdigital space with mud and feces, overgrowth of the hoof or by separation of the wall after laminitis. The affected digit is hot and tender. Lameness is severe. This condition is more commonly observed on the fore limb. Affected sheep usually recover rapidly after paring of the horn to provide drainage (The Merck Veterinary Manual, 2009)

### 2.5.3 Photosensitization

In photosensitization the lightly pigmented parts of the skin are hyperactive to sunlight. This results primarily from consumption of plants with photodynamic agents. The photodynamic agents enter either through skin or gastro-intestinal tract and reach the skin unchanged. It is also associated with liver damage due to various poisonings. This is manifested by marked photosensitivity (The Merck Veterinary Manual, 2009). It leads to acute coronitis that causes lameness (Radostitis *et al.,* 201). However it is not a common condition in sheep. If photosensitization is diagnosed early and sheep immediately removed from the pastures to areas, sheltered from direct sunlight the sheep will normally recover well (The Merck Veterinary Manual, 2009).

## 2.6 Other general causes of lameness

The causes of lameness in this section are systemic diseases that may occasionally affect the limbs, particularly the feet.

### 2.6.1 Contagious pustular dermatitis (Orf)

This disease causes lesions on the lips, skin in the head region, muzzle and oral mucosa. Secondary lesions also occur on the limbs around the coronet, palmar and plantar surfaces of pastern joint and interdigital skin. Lesions can also extend to the tarsal and carpal joint areas with accompanying painful cellulitis and secondary infection leading to lameness. It affects lambs or non–immune adults (Radostitis *et al.*, 2001; Kitching, 2004). Sheep normally recover from orf within a week. Application of antibiotics and ensuring that infected sheep are supplemented with high quality feeds helps in the recovery. Isolation of the infected stock is advisable in order to slow down cross-transmission to healthy animals (Winter and Charmley, 1999).

### 2.6.2 Foot and Mouth Disease (FMD)

The disease is characterized by vesicles in the mouth and on the feet and teats, but oral lesions are not prevalent in sheep. Feet lesions commonly occur on the coronet, interdigital skin and the heel bulbs. FMD foot lesions can resemble foot rot, particularly if there is secondary bacterial infection. Lameness is severe and the morbidity is high (Caple, 1990; Radostitis *et al.*, 2001; VEIN, 2008). Management of foot and mouth disease involves slaughter of all affected and contacted sheep, quarantine of affected premises and vaccinations (The Merck Veterinary Manual, 2009).

### 2.6.3. Blue tongue disease

During the initial stages of infection with the bluetongue virus there is hyperaemia of the mucous membranes of the mouth and the skin of the feet around the coronet. Coronitis is severe with prominent haemorrhages which may be visible in the hooves. There is also separation of horn tissues from the coronary tissue. Laminitis may also result. Lameness when present is severe but is observed late in the syndrome (Radostitis *et al.*, 2001; Verwoerd and Erasmus, 2004; VEIN, 2008). There is no effective treatment. Prevention is effected through quarantine, inoculation with live modified virus vaccine and control of the vector (Gairdner, 2007; Abel, 2008).

### 2.6.4 Ulcerative dermatosis

Ulcerative dermatosis is characterized by destruction of the epidermal and subcutaneous tissues, development of raw granulating ulcers on the skin of the lips, limbs and external genital organs. Feet lesions occur in the interdigital space and above the coronet leading to lameness (Radostitis *et al.*, 2001). Management of ulcerative dermatosis includes isolation of affected sheep, removing the scabs and all necrotic tissues as well as treatment of foot lesions with copper sulphate or formaldehyde solutions in footbath troughs (The Merck Veterinary Manual, 2009).

### 2.6.5 Dermatophilosis

Dermatophilosis is caused by *Dermatophilus congolensis* zoospores that spread rapidly or from infected dipping tanks around the feet. It is characterized by proliferative dermatitis with exudative crusts and scab formation on the affected region of the body. The disease

affects the limbs from the coronet to stifle or hock. Mild lameness is observed (Zaria and Damin, 2004). The disease is predisposed by prolonged wetness, high humidity, high temperature and various ectoparasites. Acute cases of Dermatophilosis heal rapidly without treatment. However chronic cases can be effectively treated with penicillin. Also the clinically affected sheep should be isolated or culled (The Merck Veterinary Manual, 2009)

### 2.6.6 Post-dipping lameness

Post-dipping lameness is caused by *Erysipelothrix rhusiopathiae* and is observed in sheep of all ages. The disease is characterised by cellulitis at the coronary band and interdigital area affecting several animals 2-7 days after dipping. Most cases resolve after a few days, but in a few cases bacteraemia occurs resulting in joint swelling due to painful non-suppurative arthritis about 2–3 weeks after dipping (Radostitis *et al.,* 2001; NADIS, 2003). This arthritis may affect one or more joints. The treatment response in these cases is poor. The source of infection is faeces-contaminated dip, in which *E. rhusiopathiae* can multiply rapidly. Sheep should pass through water troughs and also walked over concrete before dipping to remove excess soil and faecal material from the feet. Dip-compatible bacteriostats should be added when it is necessary (Radostitis *et al.,* 2001; VEIN, 2008; The Merck Veterinary Manual, 2009).

## 2.7 Prevention of lameness in sheep

Prevention and control of lameness in sheep depend mainly on management or husbandry practices:

### 2.7.1 Good management practices

The management practices that will prevent the occurrence of lameness includes reduced stocking density (Elliot and Pinkus, 1993), regular foot bathing with 10% zinc sulphate or 10% copper sulphate solution (Parajuli and Goddard, 1989), avoiding long and dry pasture that may cause interdigital abrasions (Whittington, 1995), hoof trimming and proper genetic selection. Sheep that have foot infections should be separated from clean sheep (The Veterinary Formulary, 1998). Factors that enhance dry and clean environment also reduce the risk of spreading foot infections. These factors include adequate straw bedding that keeps the feet dry and clean as well as spreading lime on the floor especially around water troughs to help dry and sterilize the beddings (Henderson, 1990). It is important to cull persistently infected sheep which do not fully respond to treatment in order to minimize the source of infection to the rest of the flock (Winter, 1998a).

### 2.7.2 Vaccination

Foot rot vaccine can be used curatively as well as preventatively (The Veterinary Formulary, 1998).

### 2.7.3 Genetic selection

Studies in Australia have shown that genetic selection of sheep resistant to foot rot is possible (Raadsma *et al.*, 1990).

### 2.7.4 Hoof trimming

This is a skilled procedure and should be carried out on overgrown or misshapen feet. Routine trimming of all the feet is necessary. Trimming helps to eliminate cracks and crevices that could trap mud and harbour bacteria. Foot trimming allows penetration of footbath chemicals. Regular foot paring may prevent shelly hoof (Scott and Henderson, 1991; DEFRA, 2003b). Granuloma can be prevented by not over-paring the hooves (Winter, 1998b).

### 2.7.5 Foot bathing

Footbaths containing either 3% formalin or 10% copper sulphate solutions are recommended. Both have antimicrobial properties, but in addition formalin also hardens the claw horn (Arkins, 1981; Davis, 1982).

# CHAPTER 3

# 3.0 MATERIALS AND METHODS

## 3.1 Study area

Kajiado District is approximately 15,546 $km^2$ (Figure 3.1 and 3.2) with about 470,000 people and a population density of 30 people per $km^2$. The district has an estimated sheep population of 502,340. It is located in the semi-arid zone of Kenya, but has two rainy seasons, during March to May and October to November with annual rainfall ranges of 500 mm to 1250 mm. The sheep production among the pastoralists in this district is generally free-range grazing because of scarcity of pasture. The farms in which the study was carried out were located in Ngong, Ewaso Kedong and Isinya divisions within the district (Otieno, 2008).

## 3.2 Study design

This was a cross sectional study in which the farms were visited more than once, but in each farm every sheep that met the selection criteria was examined only once during the whole study.

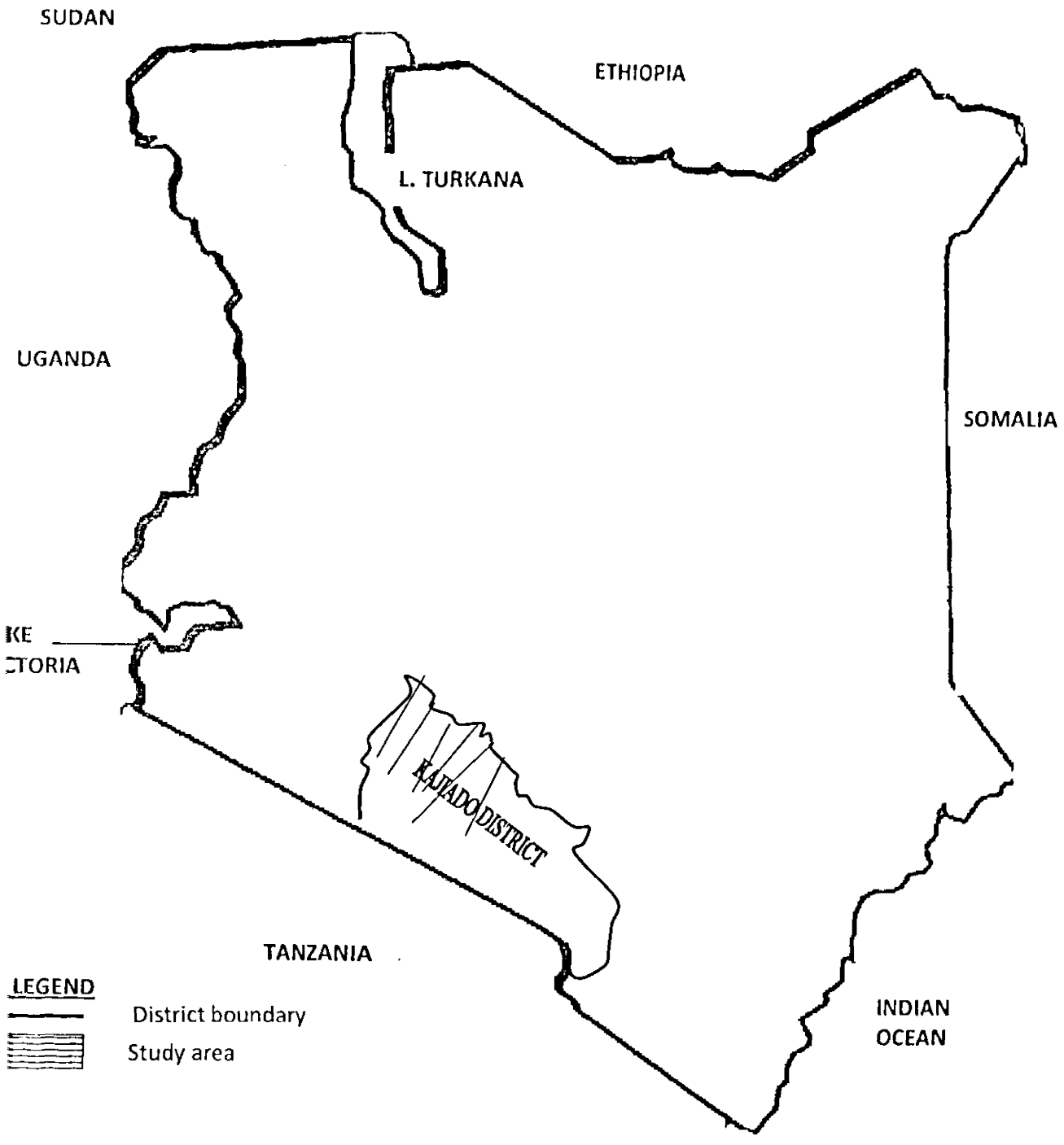


**Figure 3.1**: Map of Kenya showing Kajiado District in which the sheep examined for prevalence and risk factors of conditions causing lameness under free-range grazing system were reared (March 2010–June 2010).

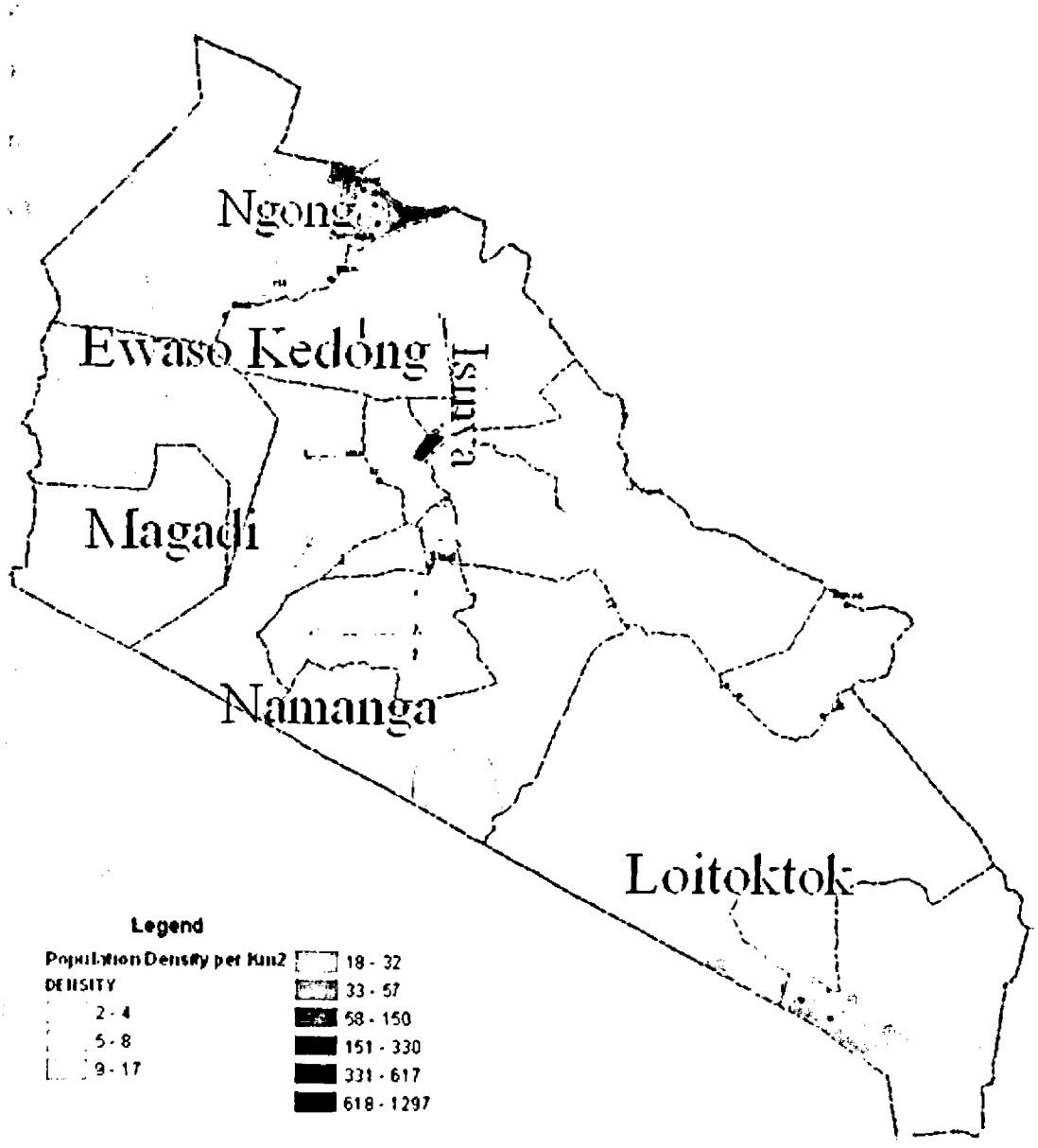


**Figure 3.2:** Map of Kajiado district, Kenya in which the farms included in the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system were located (March 2010–June 2010).

### 3.3 Farm selection

Farms selection was purposive for logistic reasons. It considered the number of sheep reared within the farms as well as willingness of the farmers to allow the study to be carried out on their sheep. Farms were identified with the help of local veterinary officers and animal health technicians. Farmer's consent for use of their farms and examination of their sheep was sought through local veterinarians or animal health technicians. Ten (10) farms, 4 in Ngong, 3 in Isinya and 3 in Ewaso Kedong divisions, each with a minimum of 100 sheep and 3 months of age or older were selected for the study.

### 3.4 Animal selection

A total of 1916 sheep were selected from the 10 farms. The selected sheep included both lame and non-lame, above three months of age, both sex and of varied breeds. The study in each farm was carried out early in the morning before the sheep were released from their night enclosures All sheep in each farm underwent general visual observation noting particularly the body and limb conformations while the sheep were at rest, in standing positions and during locomotion. Each sheep was made to walk on a flat and firm ground (Figure 3.3), the lame ones isolated and marked with a blue aerosol spray over the sacral region (Figure 3.3) for closer and specific limb examination.

### 3.5 Animal examination

#### 3.5.1 Visual observation

All the sheep in each farm were made to slowly walk through a firm ground area as the investigator observed them carefully to identify those with abnormal gait or showing

lameness (Figure3.3). The observation included the position of the back (level of dorsal column), placement of each limb on the ground, bearing of weight on the limbs and nature of the strides made. A locomotion score of 0 (not lame) to 4 (severely lame) as a locomotion scoring system (Table 3.1) was used to indicate the degree of lameness. Each sheep that was identified as lame was separated from the non-lame sheep for closer examination.

A B

**Figure 3.3**: **A**: Sheep walking out of a night enclosure and **B**: A lame sheep selected, marked and isolated. This was in one of the 10 farms during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

**Table 3.1:** Locomotion score scale used to assess lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010-June 2010).

| Score | Description of lameness | Conclusion |
|---|---|---|
| 0 | Normal gaits | Not lame |
| 1 | Gait is slightly abnormal | Mild lameness |
| 2 | Short strides on one or more legs | Moderate lameness |
| 3 | Favours one or more limbs by not bearing weight | Definite lameness |
| 4 | Complete refusal to bear weight on one or more limbs | Severe lameness |

### 3.5.2 Examination of lame sheep

Each lame sheep was restrained by a farm-worker and subjected to a thorough general physical examination with special emphasis on the lame limb(s) identified as the sheep walked to diagnose the specific lesion causing the lameness. The claws were thoroughly washed in order to clearly see the lesion in case claw structures were involved (Figure 3.4). If the cause of lameness was proximal to the foot, the whole region from the shoulder to the fetlock and from the hip to the fetlock was examined by deep hand palpations to locate the painful part. The joints were flexed and extended and presence of pain was indicated by the animal's reaction to these manipulations. Each lesion causing lameness was photographed using a digital camera (Sony DSC-W180, 10.1 Mega Pixels, Sony Corporation). The diagnosis or the condition causing the lameness for each sheep was recorded in data collection sheets. Bacteriological swab specimens were collected from exudative lesions for bacterial culture and identification. After examination a second mark was put on the back of each sheep cranial to the first mark using a blue aerosol spray to avoid repeat examination (Figure 3.5).

#### 3.5.2.1 Recording of findings

The conditions causing lameness were further classified into various categories during entry into the computer from the data collection sheets. During data entry the following parameters were clustered accordingly; location of the lesion on the limb, fore or hind limb, one or more limbs, lateral or medial claw or both (Appendix 1).

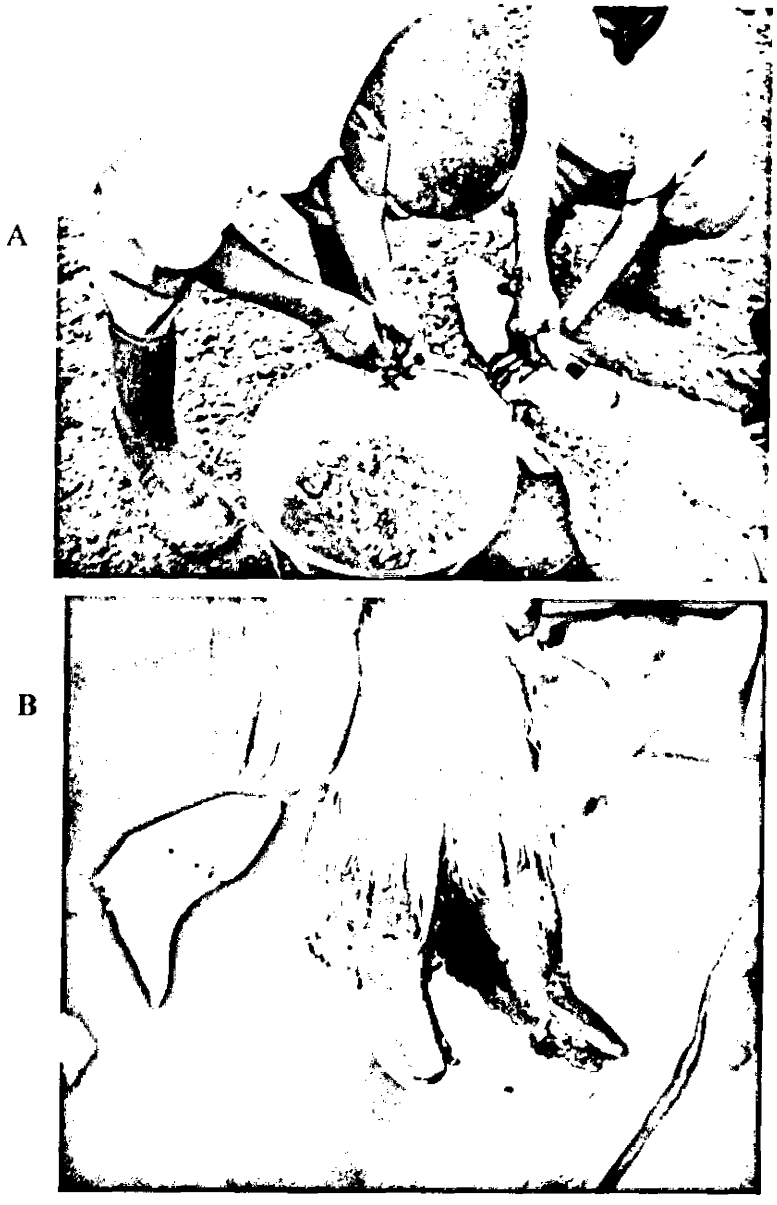


**Figure 3.4**: **A**: Thorough washing of the claws during individual animal examination. **B:** Taking a photograph of a thoroughly washed affected foot against a green sheet of cloth. This was done for all sheep with claw conditions in all the 10 farms during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

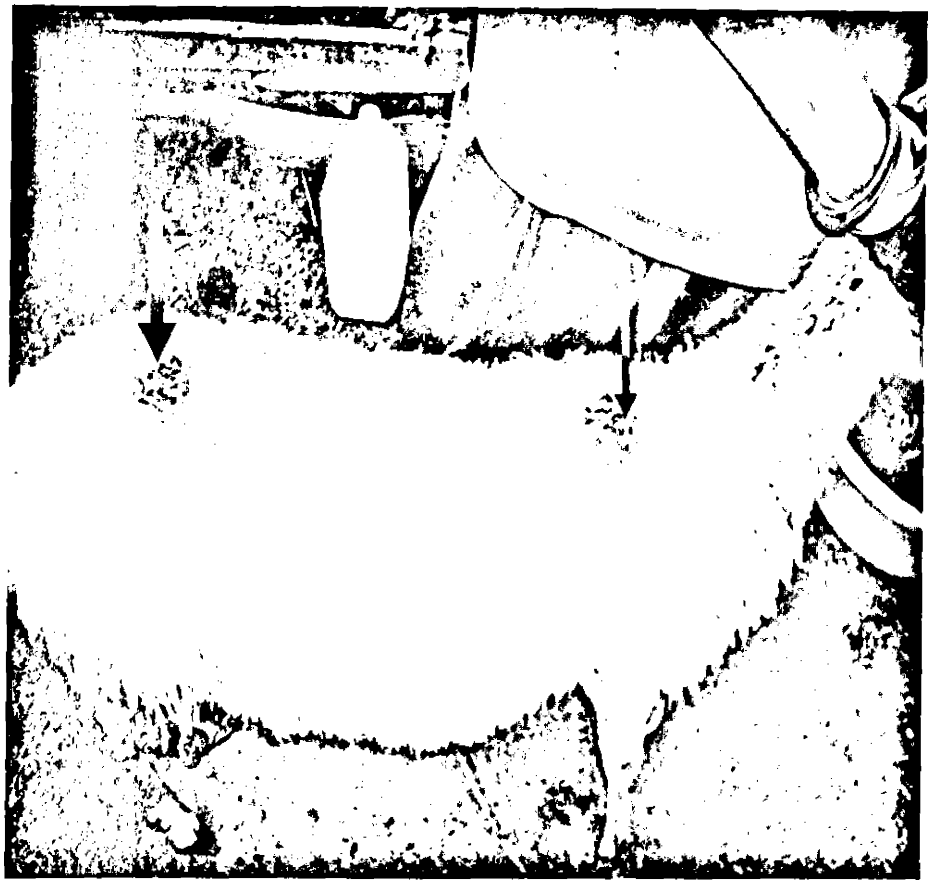

**Figure 3.5**: The first (caudal arrow) and second (cranial arrow) marks put on the back of the sheep before and after the Individual animal examination respectively in one of the 10 farms during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

### 3.6 Determination of the risk factors

#### 3.6.1 Farm-level factors

The farm environment was assessed during the visit. General observation of the sheep rearing environment was made in order to note any lameness predisposing factors. These included; the nature of terrain, grazing ground whether dry or marshy, presence of traumatic objects; state of farm tracks, type of pastures whether dry or green as well as the hygienic state of sheep night-resting enclosures. During data entry these observations were classified accordingly.

#### 3.6.2 Management-level factors

Data on the management practices was obtained by interviewing sheep owners, farm managers or stockmen. These included hoof trimming practices, tick control, feed supplementation, management of lameness cases and how stockmen handled the sheep. The data was collected by recording the important information in coded questionnaire forms (Appendix 1).

#### 3.6.3 Animal-level factors

Factors intrinsic to the animal that could predispose or enhance lameness were evaluated. These included estimated age, sex, breed, pregnancy status (when lambed, either less than or more than three months since lambing or pregnant) and limb conformation. Also evaluated was body condition score (BCS) of the lame sheep. The scores were evaluated as BCS 1 (poor), BCS 2 (fair), BCS 3 (good) and BCS 4 (very good) as suggested by Winter and Charmley (1999) and Suiter (2006). Some of these animal-level factors were

observed directly by the investigator and the rest were obtained by interviewing the farmers, stockmen, and farm managers. These were also recorded in the data sheets and questionnaire forms (Appendix 1).

### 3.7 Data handling

#### 3.7.1 Data recording

All data were written on data recording sheets that were designed and coded to capture the relevant information. Each sheep had a separate data sheet on which farm identification and flock size were indicated. The data sheet had three sections which included:(a) Animal-level section, (b) Interview section and (c) Farm environment section (Appendix1).

Data on animal-level factors were collected through questionnaires administered by the investigator interviewing the relevant persons at farm-level before the actual examination of each sheep. Data and information on management and farm-level factors were collected during visits to each of the 10 farms. This was achieved through observations by the investigator as well as administering of the questionnaires. Data recording was done separately for each sheep and for each farm.

#### 3.7.2 Data management

The data collected was stored in Microsoft office Excel 2003 (Microsoft Corporation, 2003). It was validated and verified to be correct as per the entries from the data record sheets. The data collected indicated presence or absence of a particular parameter. Coding

of each parameter for entry into computer was done. Parameters were coded as "1" (signifying "Yes" for presence of that parameter) and "2" (signifying "No" for absence of that parameter).

### 3.8 Data analysis

The data were imported into GENSTAT for windows discovery Edition 2 (VSN international). Descriptive statistics focusing on frequencies of occurrence of each parameter was done. Simple associations between lameness score and animal-level, farm-level and environmental factors were also computed. Chi-square ($\chi^2$) statistics were used to determine the associations between lameness and risk factors at $p < 0.05$ significance level. Prevalence of lameness was calculated as a percentage of lame sheep in the study population as follows:

$$\text{Prevalence of lameness (\%)} = \frac{\text{Total number of lame sheep}}{\text{Study population}} \times 100$$

Prevalence of each condition causing lameness in the study population was calculated as follows

$$\text{Prevalence of each condition among sheep examined (\%)} = \frac{\text{Total number with a specific condition}}{\text{Study population}} \times 100$$

Prevalence of each condition was also expressed as percentage of total number of lame sheep.

$$\text{Prevalence of each condition among lame sheep (\%)} = \frac{\text{Total number with a specific condition}}{\text{Total number of lame sheep}} \times 100$$

Chi-square ($\chi^2$) values were determined using 2×2 contingency table constituting 2 rows and 2 columns. In these associations, the chi-square ($\chi^2$) calculations were determined by evaluating each risk factor (variable) against each lameness condition (outcome) on the sheep. The degrees of freedom (df) in each case was standard, being calculated by

[(rows-1)(columns-1)], hence [(2-1) x (2-1) =1]

Therefore df was 1 for each association test

# CHAPTER 4

# 4.0 RESULTS

## 4.1 Descriptive statistics for the study farms

The study population in all the 10 farms was 1916 sheep, out of which 117 lame ones were examined. The 10 farms included in the study had an average flock size of 192 sheep which were all under free-range grazing system. The median number was 183 sheep. One of the farms was paddocked and the sheep were grazed within the paddocks (Figure 4.1). In 3 of the farms, the sheep were housed in roofed enclosures during the night after free-range grazing the whole day (Figure 4.2). The other 7 farms did not have any roof over the night enclosures. In 6 of these 7 farms, the night enclosures were made of timber and mesh wire sides (Figure 4.3), while the remaining one, the perimeter wall of the night resting area was secured with thorny tree branches (Figure 4.4).

In one of the farms, formalin solution was used as a foot-dip in a plastic container. The dipping of the feet into the formalin solution in this farm was done once per week. The rest of the farms had neither footbaths nor chemical foot-dips. Trimming of the hooves was routinely done in 5 farms only. In 3 of these farms, it was carried out by the owners or the stockmen while in the other 2, it was done by either a veterinary surgeon or an animal health assistant. In these 5 farms the trimming was done once a year, but in the other 5 farms hoof trimming was not done at all.

In the three-month period immediately preceding the study, there were cases of lame reportedly sheep observed in all the 10 farms. In 8 of them, the lame sheep were

reportedly treated during that period. In the other 2 farms, the lame sheep were not treated but were left to recover on their own or culled. Tick control was done by hand-spraying method using Knap-sack sprayers in all the 10 farms. Two years previously, dipping in a plunge-dip was employed in one of the farms. In this farm, the farm manager reported that there were more cases of lame sheep at the time of the study than during the period when plunge-dipping was being used.

**Figure 4.1**: Sheep grazing in a paddock in one of the 10 farms included in the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.2**: Roofed night-resting enclosure where sheep were held at night after free-range grazing during the day. This was the case in three of the 10 farms assessed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

A

B

**Figure 4.3**: A-Sheep in open-roofed enclosure in which they were held at night after free-range grazing during the day. **B**- The night-resting enclosure with manure accumulation. These were the situations in some of the farms studied for prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.4**: An enclosure where perimeter is secured with thorny tree branches where sheep were held at night in one of the 10 farms studied for the prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

## 4.2 Description of the lesions observed

### 4.2.1 Prevalence of the foot conditions

It was reported in all the farms that lameness cases were more common during the wet seasons. The overall prevalence of lameness in the 10 farms was 6.1% (117/1916), while 93.9% (1799/1916) of the sheep were not lame. The prevalence rates of conditions causing lameness in a population of 1916 sheep are presented in Table 4.1. Sole erosion and overgrown claws were the most prevalent conditions at 3.8% (72/1916) and 3.2% (61/1916) respectively. Tick-bite dermatitis had a low prevalence of 1.6% (30/1916). The rest of the conditions had prevalence of less than 1% (Table 4.1) or were observed only in a single sheep as presented in Figure 4.5. Out of the 117 sheep that were lame, 81.2% had moderate to definite lameness, 12% had mild lameness and 6.8% were severely lame.

When calculated as a proportion of the population of sheep that were lame, the conditions with the highest percentage of occurrence were sole erosion 61.5% (72/117) and overgrown hooves at 52.1% (61/117). Those with moderate percentage of occurrence were tick-bite dermatitis at 25.6% (30/117) and, hoof fractures at 12.0% (14/117). The rest of the conditions had percentages of occurrence equal or lower than 5%. These percentages of occurrence among the lame sheep are presented in Table 4.2 and Figure 4.6. Some of the lame sheep had a single condition causing lameness, but others had more than one condition. The proportion of the sheep that had more than one lesion on their feet simultaneously was 67.5% of the lame sheep, hence the total percentage of more than 100%. Interdigital dermatitis was observed to invariably occur together with other lesions. The rest of the conditions causing lameness were observed only in one sheep each except shelly hoof that was observed in three sheep.

**Table 4.1**: Prevalence of conditions causing lameness in a population of 1916 sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March - June 2010)

| Conditions causing lameness | Number of sheep (n=1916) | Prevalence (%) $\frac{y}{n} \times 100$ (y = Number of sheep with each condition) |
|---|---|---|
| Sole erosion (bruising) | 72 | 3.8 |
| Overgrown hooves | 61 | 3.2 |
| Tick-bite dermatitis | 30 | 1.6 |
| Hoof cracks | 14 | 0.7 |
| Interdigital dermatitis | 6 | 0.3 |
| Shelly hoof | 3 | 0.2 |
| Soil balling | 1 | 0.1 |
| Osteomyelitis of metatarsal bone | 1 | 0.1 |
| Septic arthritis | 1 | 0.1 |
| Malunion of tibial bone | 1 | 0.1 |
| Hyperextension of fetlock joint | 1 | 0.1 |
| Overparing of medial claws | 1 | 0.1 |
| Foot rot | 1 | 0.1 |
| Foreign body penetration | 1 | 0.1 |

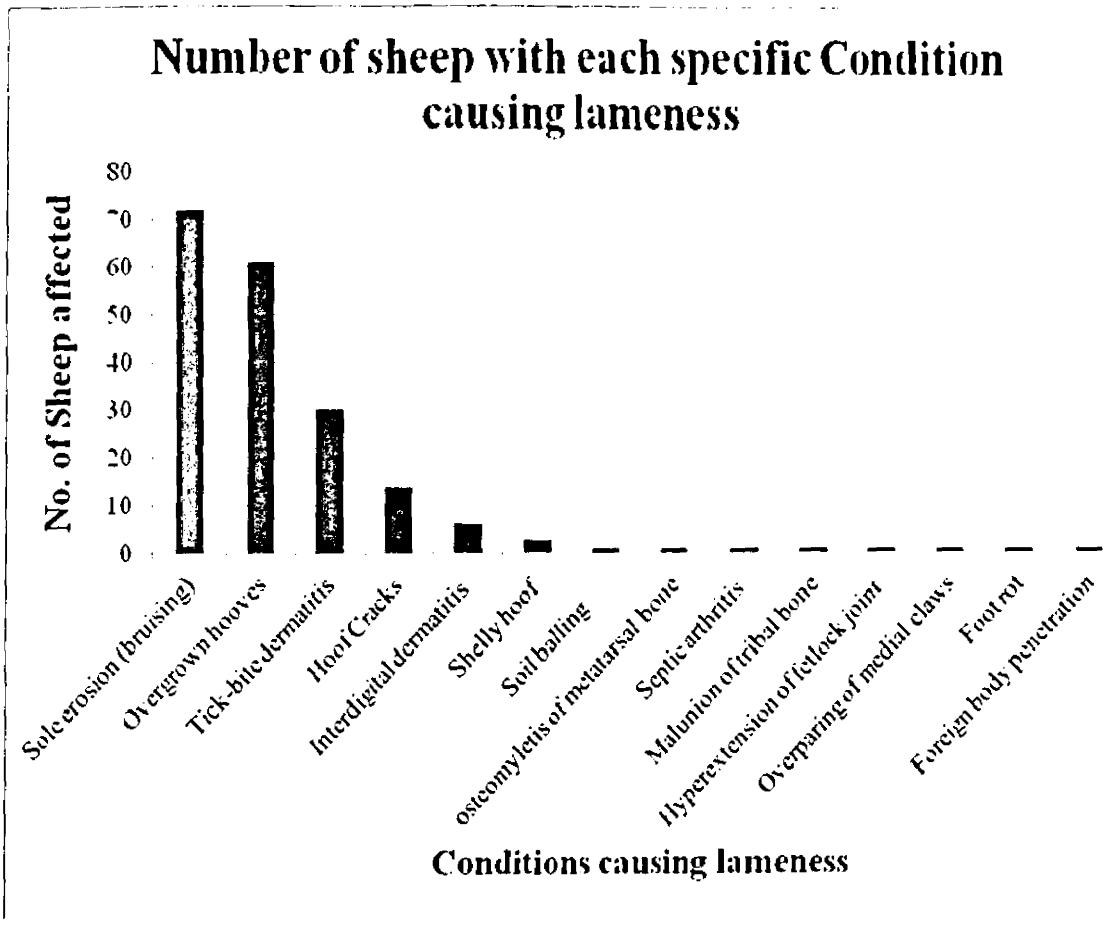


**Figure 4.5:** Conditions causing lameness according to the number of sheep affected in a population of 1916 sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March – June 2010)

**Table 4.2**: Percentages of conditions causing lameness in 117 sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March-June 2010).

| Conditions causing lameness | Number of sheep (n=117) | Prevalence (%) $\frac{y}{n} \times 100$ (y = Number of sheep with each condition) |
|---|---|---|
| Sole erosion(bruising) | 72 | 61.5 |
| Overgrown hooves | 61 | 52.1 |
| Tick-bite dermatitis | 30 | 25.6 |
| Hoof cracks | 14 | 12.0 |
| Interdigital dermatitis | 6 | 5.1 |
| Shelly hoof | 3 | 2.6 |
| Soil balling | 1 | 0.85 |
| Osteomyelitis of metatarsal bone | 1 | 0.85 |
| Septic arthritis | 1 | 0.85 |
| Malunion of tibial bone | 1 | 0.85 |
| Hyperextension of fetlock joint | 1 | 0.85 |
| Overparing of medial claw | 1 | 0.85 |
| Foot rot | 1 | 0.85 |
| Foreign body penetration | 1 | 0.85 |

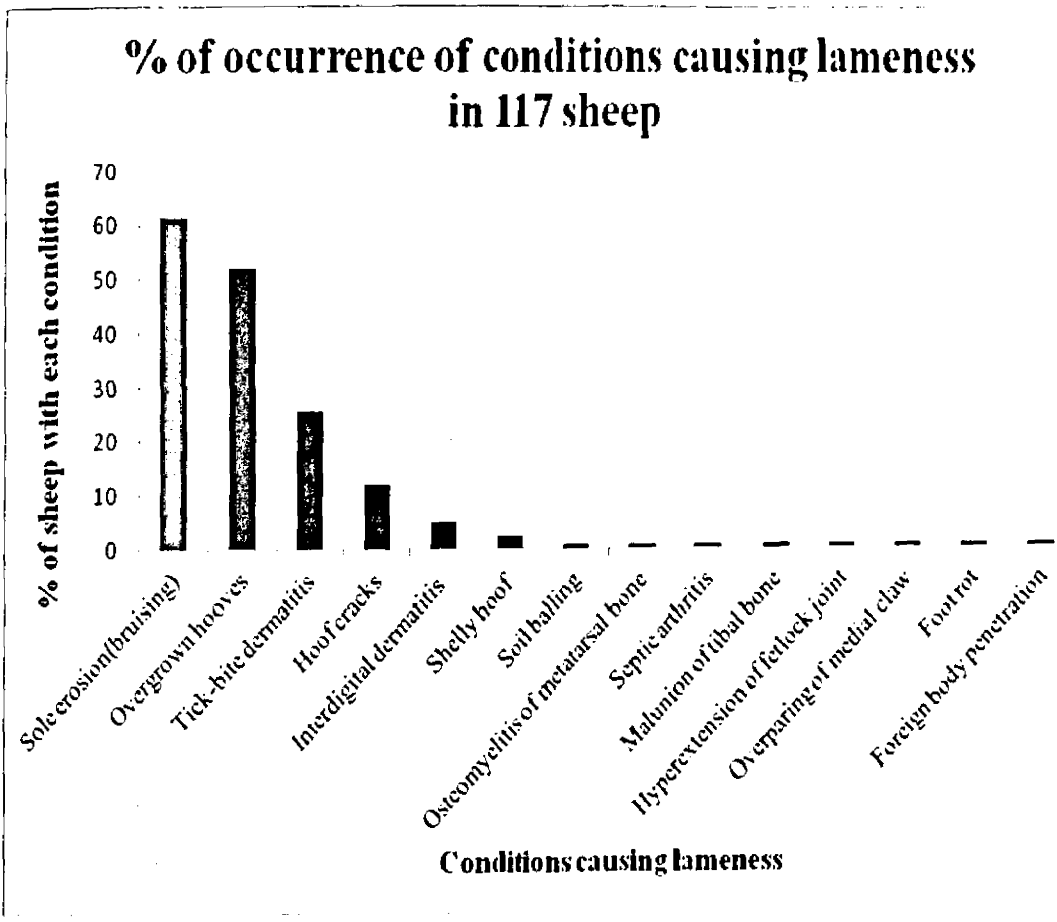


**Figure 4.6:** Percentage of occurrence of conditions causing lameness in a population of 117 lame sheep examined in 10 free-range grazing farms in Kajiado District, Kenya (March – June 2010)

### 4.2.2 Descriptive findings of the lameness conditions.

#### 4.2.2.1 Claw deformities

Normal claws were observed to have balanced growth of toe, sole and walls (Figure 4.7). However among the lame sheep, 52.1% (61/117) had various forms of deformities of the claws, most of which were related to hoof overgrowth. These included overgrowth and elongation of the toes, soles and claw walls. The overgrowth ranged from slight to excessive and also resulted in varying degrees of gait abnormalities. Out of the 61 sheep with claw deformities, 13.1% (8/61) had simple regular overgrowth, which mainly involved elongation of the sole and toes (Figure.4.8), but 86.9% (53/61) had varying degrees of excessive overgrowth of the hooves with some of them leading to misshapen claws. The excessive overgrowth with resulting misshaping of the claws included irregular elongation and widening of the hoof wall with some growing to cover the tread surface of the sole (Figure 4.9). Some toes were excessively elongated with resulting tendency to turning outward (lateral) or curving dorsally (Figure 4.10) and others were extremely splayed (Figure 4.11).

#### 4.2.2.2 Hoof wall cracks

Hoof wall cracks found in 11.9% (14/117) of the lame sheep, were mainly horizontal occurring either at the middle of lateral and dorsal wall, close to the toe or at the distal part of abaxial wall (Figure.4.12). In one sheep examined the claw wall was extensively overgrown extending and curving towards the sole (Figure 4.9).

**Figure 4.7**: Normal sheep claws both medial (Left brace) and lateral claws (Bold arrow) observed in the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.8**: Regular overgrowth showing elongation of the toe (left brace) observed among some of the 117 lame sheep examined during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).


**Figure 4.9**: Excessive overgrowth of the hoof wall with resulting misshaping of the claws. **A**: Irregularly shaped claw wall (bold arrow). **B**: Widening of the claw wall with slight outward projection (right brace) and irregular outward growth (dotted arrow). **C**: Excessively overgrown lateral hoof wall covering the sole (dotted double headed arrow). These claw features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

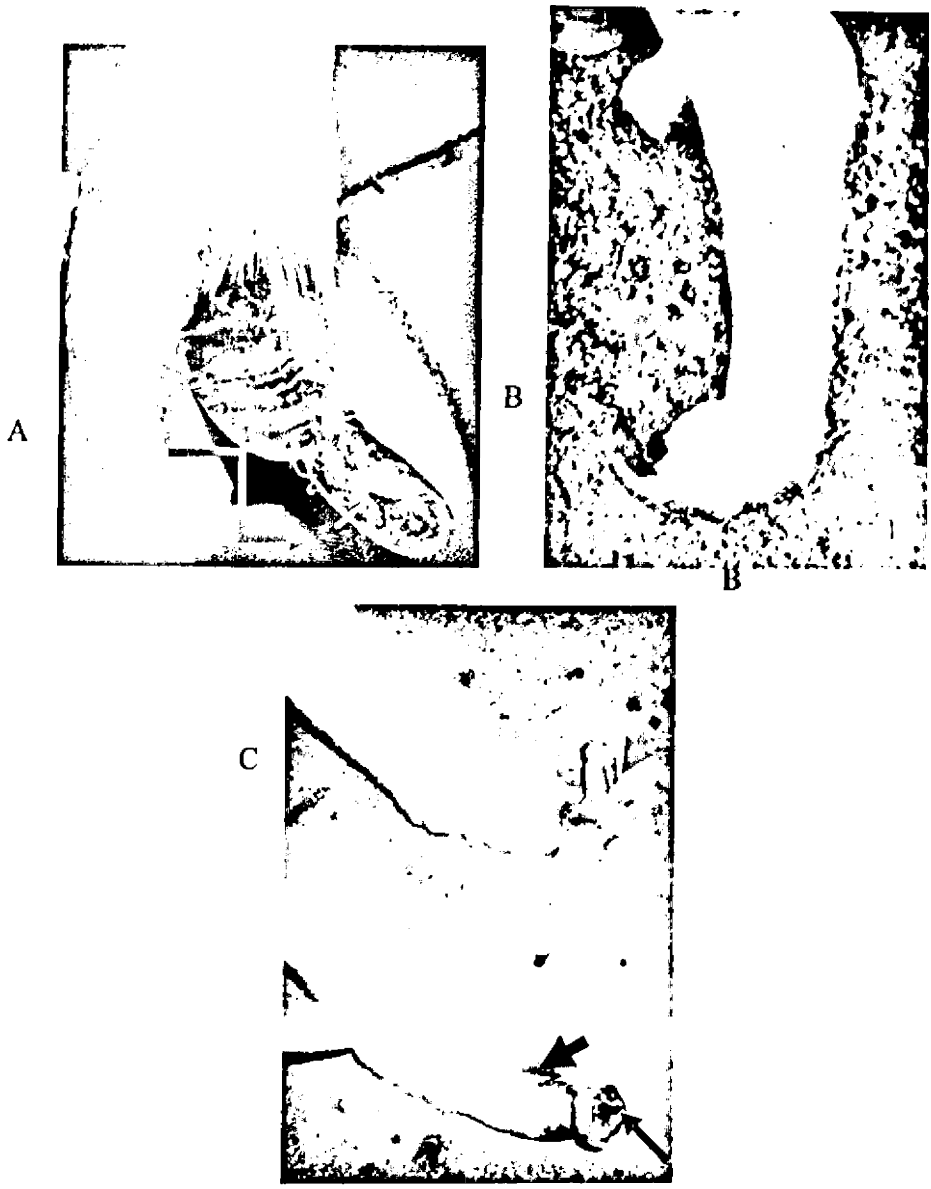


**Figure 4.10**: Excessively elongated and misshapen toes of the claw. **A**: Overgrown hoof walls and elongation of the toe in a lateral direction (angled arrow). **B**: Dorsal curvature of the toe (bold arrow). **C**: Dorsal curvature of the toe (dotted arrow), circularly coiled elongated toe (bold arrow) and over-short toe due to breakage (Arrow head). These toe features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

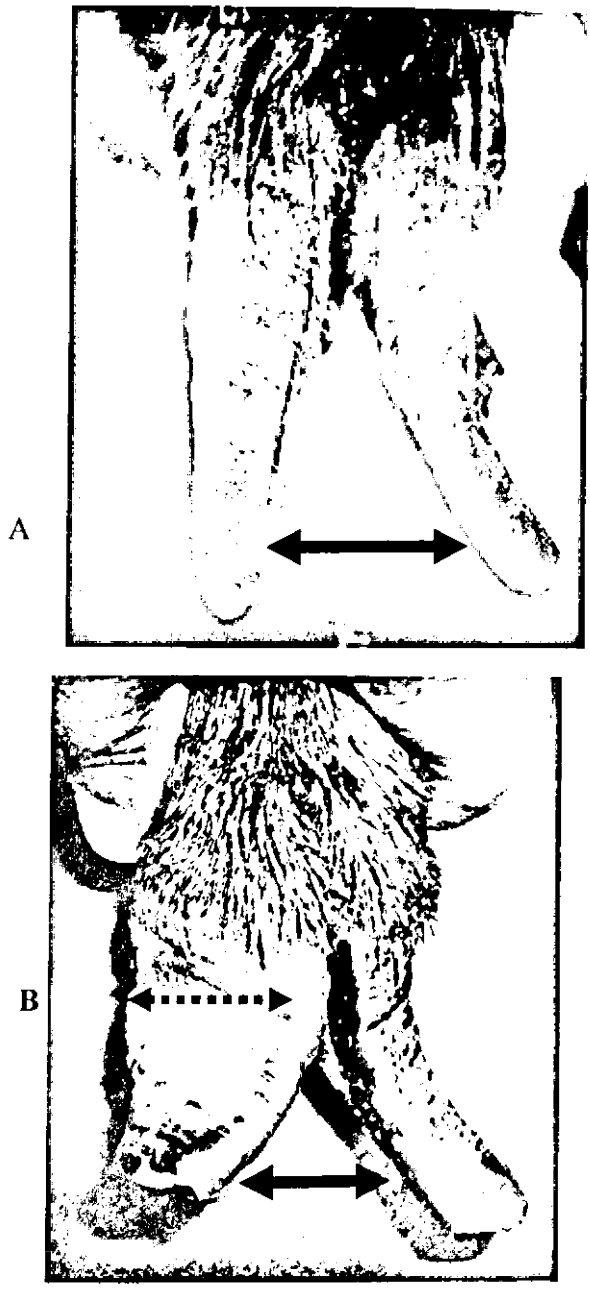


**Figure 4.11:** **A**: Regular elongation of the claw with excessively splayed toes; **B**: Elongation and splaying of the toes (double-headed bold arrow) with widening and flattening of the dorsal and lateral hoof wall (double-headed dotted arrow). These claw features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

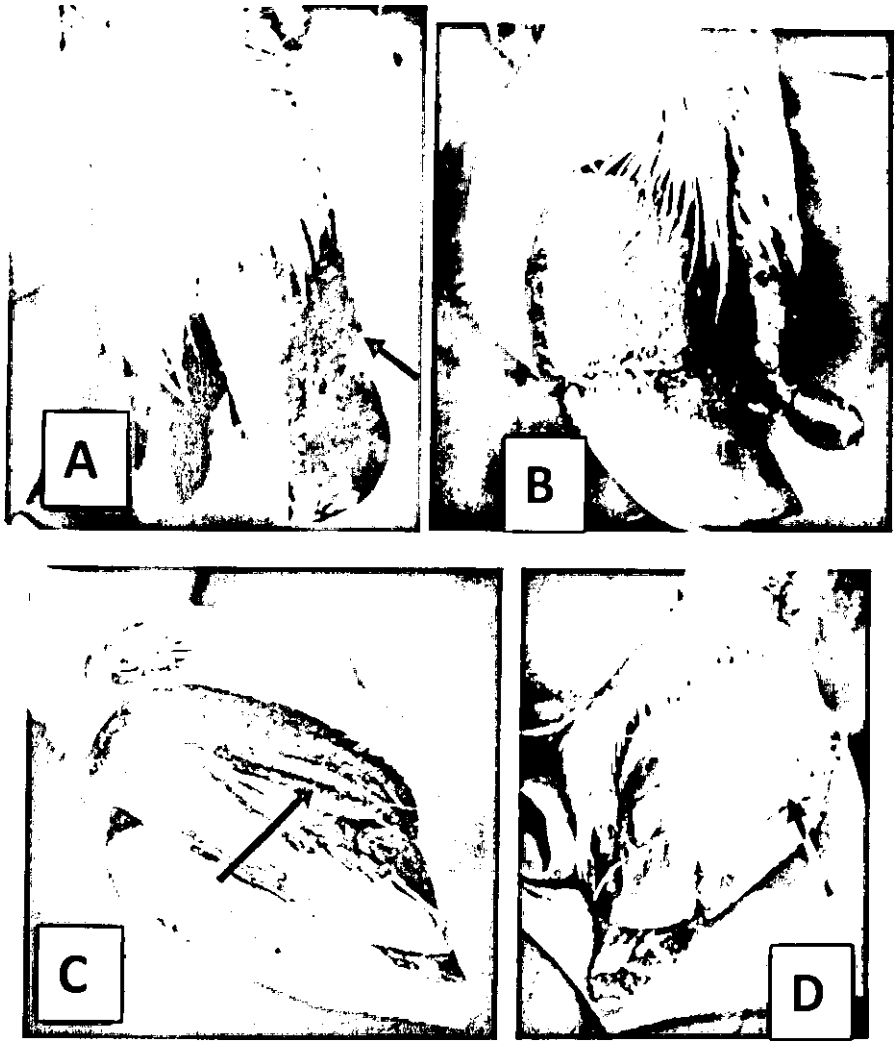


**Figure 4.12. A** and **B:** Horizontal hoof cracks at the middle of the lateral and dorsal claw walls. **C**: Horizontal hoof crack at the distal part of the abaxial wall. **D**: Horizontal hoof cracks at the toe of the claw. These claw features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.3 Sole erosion or bruising.

Out of the 117 lame sheep, 61.5% (72/117) had sole erosions, some of which were severe occurring with some degree of hoof overgrowth and others were mild. The severe sole erosion more invasively eroded the horn of the sole thus diminishing the thickness of the intact horn layer, but the mild sole bruising was only superficially erosive. The eroded horn of the sole appeared black and necrotic (Figure.4.13). The sole erosion lesions affected both medial and lateral claws. More cases of lameness involving sole erosion affected the hind limb (60%) and were mostly bilateral (57.8%). Painful responses were observed when pressure was applied on the eroded areas of the soles.

#### 4.2.2.4 Tick-bite dermatitis

Examination of affected lame sheep revealed heavy tick infestations on the limbs. Among the lame sheep, 25.6% (30/117) had tick bite dermatitis. The main sites of attachment of ticks were the skin on the plantar (caudal) aspect of foot between the dew claws and the coronet and also occasionally in the interdigital skin. The ticks were usually found aggregating together round a limited site. Severe inflammation was always observed and sheep were severely lame. In some of the sheep, the area of tick-bite was found to have developed dermatitis lesions with slight erythema (Figure 4.14). The sheep with large aggregates of tick attachments were moderately lame, but those with tick-bite dermatitis were definitely lame.

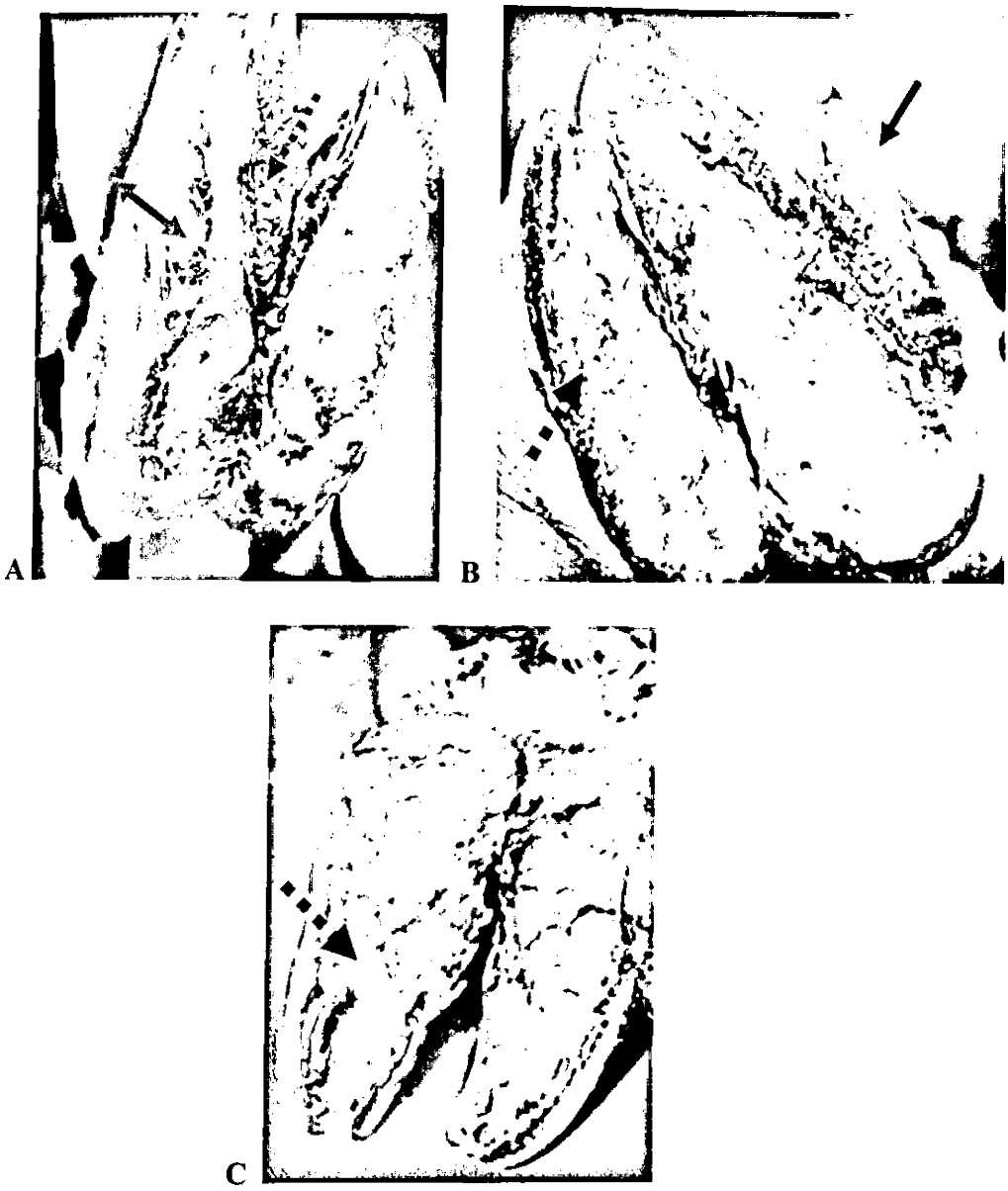


**Figure 4.13**: **A** and **B:** The invasive sole erosion with black necrotic horn (dotted arrows) occurring with overgrown hoof walls (double-headed and bold arrows). **C**: Superficial erosion of the sole (dotted arrow). These claw disorders were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

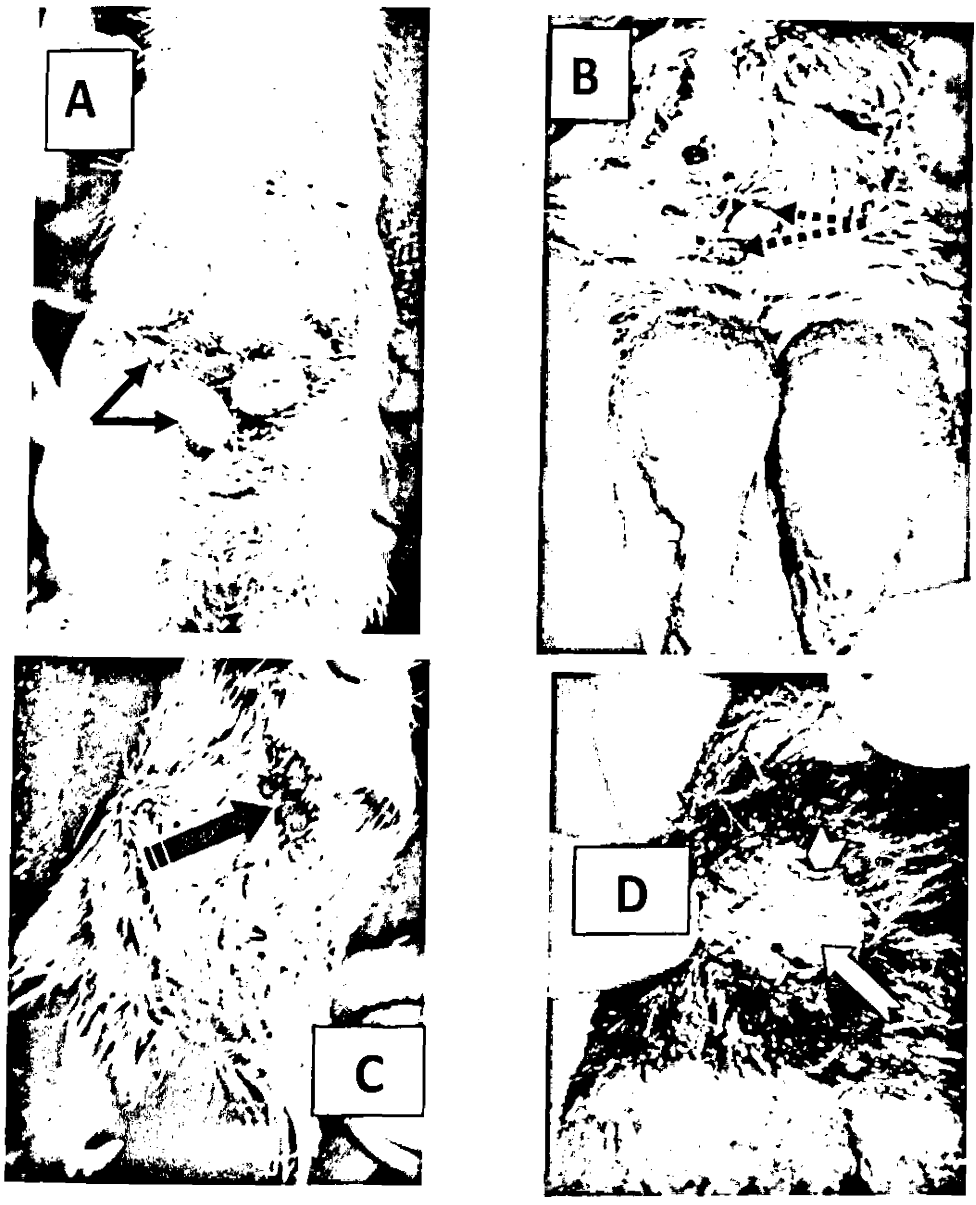


**Figure 4.14**. **A**, **B** and **C**: Several types of ticks attaching in a limited area distal to the dew claws (bold and dotted-V arrows). **D:** Tick-bite dermatitis lesion distal to the dew claws (notched arrow), with some ticks still attached (chevron). These were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.5 Interdigital dermatitis

Interdigital dermatitis was found in 5.1% (6/117) of the lame sheep. It was observed to be an acute inflammation of the interdigital skin. In some of the cases, the dermatitis lesion had ulcerated and the skin was erythematous, which resulted in moderate to severe lameness (Figure.4.15).

#### 4.2.2.6 Septic arthritis

Only one sheep was found to have septic arthritis involving one digit. The lesion was located at the proximal interphalangeal joint (pastern) of the foot. It was swollen, warmer than the surrounding tissues and discharging pus. The open parts of the lesion appeared necrotic with scab formation. The infection was observed to be affecting the deeper structures. The lesion was very painful and the sheep severely lame with the affected limb not bearing any weight (Figure 4.16). *Fusobacterium* species were isolated from culture of pus collected from the lesion.

#### 4.2.2.7 Foreign body penetration

A hard dry thorn was found as a foreign body penetrating the sole in one sheep. The horn of the sole around the penetrated area had dark-red discoloration and was slightly swollen (Figure 4.17). The sheep was severely lame and resisted bearing weight on the affected foot.

**Figure 4.15**. Granulating interdigital dermatitis lesion (bold arrow). Such lesions were found in 6 sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010)

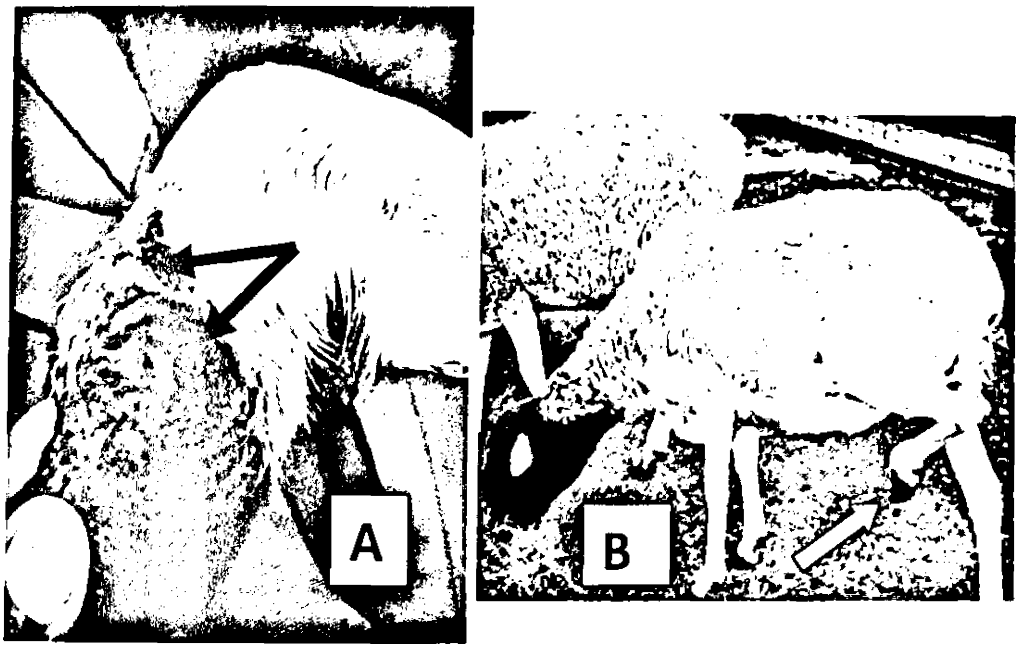


**Figure 4.16**. **A**: Septic arthritis of the proximal interphalangeal (pastern) joint with a necrotizing wound and scab forming wound (bold-V arrow). **B**: lifting of the left limb due to pain in a severely lame sheep (bold arrow). Such features were observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.17**: A penetrating foreign body and a resulting necrotizing wound in the sole of one claw with swelling and hyperemia around it (bold arrow). This was observed in one among the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.8 Interdigital soil-balling

Soil and grass stuck between the digits and formed into a hard lump commonly referred to as "soil or grass balling" was observed only in one among the lame sheep (Figure 4.18). The soil and grass balls were firmly attached to the underlying interdigital skin and the hooves. The sheep with soil balling manifested definite lameness.

#### 4.2.2.9 Foot rot

Only one sheep in this study had foot rot. The foot rot lesion included dermatitis in the interdigitum, slight under-running of the horn at the skin–horn junction and foul-smelling exudates (Figure 4.19). The sheep was severely lame.

#### 4.2.2.10 Shelly hoof

Shelly hoof was observed in one lame sheep. This sheep had hoof overgrowth with separation of walls and accumulation of dung material in the avulsed parts. Abaxial hoof walls had slight separation, but the axial walls had excessive separation (Figure 4.20). The sheep had moderate lameness on the affected foot.

**Figure 4.18**. Hard lumps (soil and grass balling) formed by prolonged accumulation of grass and soil in the interdigital space (bold arrows). These were firmly attached to the underlying interdigital skin and the hooves. This was observed in one among the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.19**: Interdigital foot rot lesion with some purulent discharge, necrosis, swelling and dung matting around it (bold arrow). The condition was seen in one among the lame sheep observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).


**Figure 4.20. A** and **B:** Separation of the hoof wall from the underlying parts of the claw, particularly the axial walls in "shelly hoof". Dung is accumulated between the separated structures (bold curved and straight arrows respectively). This disorder was found during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

#### 4.2.2.11 Over-trimmed hooves

In one among the lame sheep, the hooves were found to have been excessively trimmed to the extent of traumatizing the sensitive laminae (Figure 4.21). The recommended trimming pattern was not followed, which resulted in vertical cutting-off of the toes. This was manifested as severe lameness of the affected limb.

#### 4.2.2.12 Malunion of tibial fracture

One sheep was reported to have had a fracture of right tibia. Examination revealed that the fracture had healed with an extreme malunion of the bone fragments, which resulted in deformation of tibia. The bone was curved medially near the hock joint. This resulted in adduction of the hock area and abduction of the foot leading to abnormal gait (Figure 4.22). The sheep had moderate lameness.

#### 4.2.2.13 Abnormal conformation of the foot (Fetlock hyperextension)

This was observed in one out of 117 sheep that were lame. The anomaly was observed at the fetlock joints of both hind limbs. The joints were flexed and mild lameness was observed (Figure. 4.23).

#### 4.2.2.14 Osteomyelitis of the metatarsal bone

Osteomyelitis was observed in one out of the 117 sheep that were lame. This affected the metatarsal bone. There was swelling and extreme pain. Lameness was definite.

**Figure 4.21**.Over-trimmed hooves affecting the sensitive laminae. The toes are completely cut off and the recommended trimming pattern was not followed (bold v-shaped arrows). This was observed during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**Figure 4.22**. Malunion of distal to mid tibia after fracture healing (chevron), leading to angled adduction of the hock joint area (dotted arrow) and extreme abduction of the foot (double-headed arrow). This was observed in one of the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

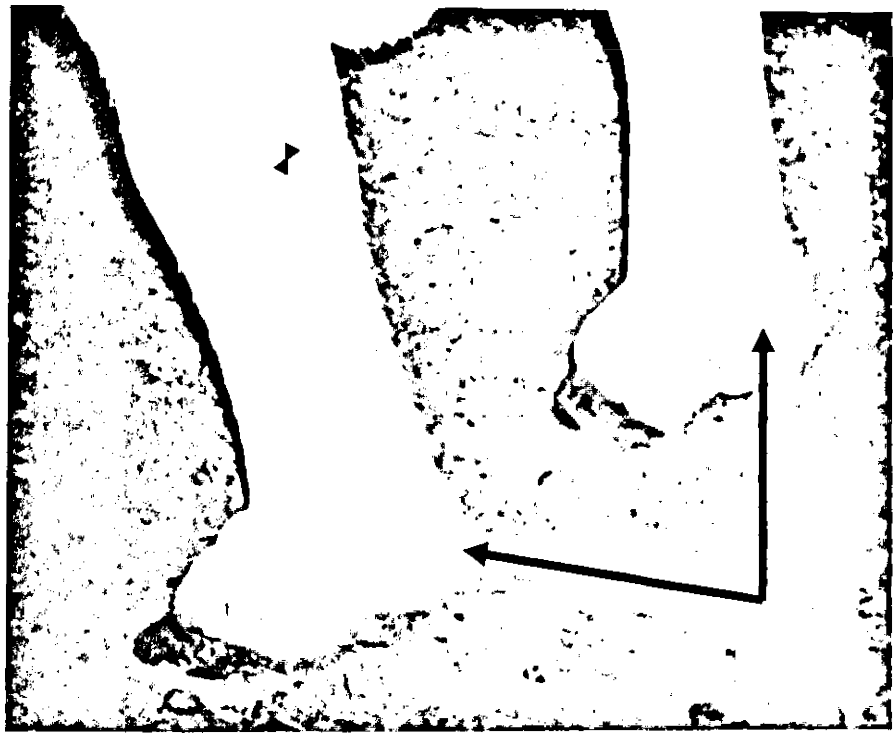

**Figure 4.23**: Abnormal conformation of the foot affecting the fetlock joints of the hind limbs (Arrows) resulting in mild lameness. This was observed in one of the lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

### 4. 3 Distribution of lesions on the limbs among the lame sheep

Among the lame sheep, frequency of lesions on the hind limbs was 43.6%, on the fore limbs 23.1%, and on both hind and fore limbs simultaneously 33.3%. Among these lame sheep, the lesions were found on a single limb in 51 sheep (43.59%), on two limbs in 39 sheep (33.33%), on three limbs in 2 sheep (1.71%) and on all four limbs in 25 sheep (21.37%). The lesions causing lameness were located on the foot in 94% (110) of the lame sheep, among which 85.5% had lesions on both medial and lateral claws, 7.3% only on the lateral claws, 5.5% only on the medial claws and 1.7% located between the fetlock joint and the coronet. The remaining 6% (7) of the lame sheep had lesions located on the proximal parts of the limbs.

### 4.4 Description of possible risk factors of lameness

#### 4.4.1 Animal-level factors

These factors are presented in Table 4-3. Out of the 117 lame sheep, 76.9% (90) were females and 23.1% (27) were males. Among these lame sheep, the breeds were Dorpers (53.8%), crosses of Dorper and Red Maasai sheep (42.7%), crosses of Dorper and Merino (1.7%) and the Red Maasai sheep (1.7%). The body condition scores among the 117 lame sheep were as follows; very good (BCS 4) 17.95%, good (BCS 3) 63.25%, fair (BCS 2) 17.09% and poor (BCS 1.71%) . Most of the sheep (98.29%) were in BCS 2 to BCS 4. The percentage of lame sheep that were at least 3 months of age and above was 96.6% compared to 3.4% that were less than 3 months of age. Out of the 117 lame sheep, 11.9% (14) were in late gestation, 65.0% (76) were not pregnant and 23.1% (27) were males. Out of the 90 females, 58.9% had lambed more than the previous 3 months prior to the

study, 7.8% had lambed within the previous 3 months prior to the examination and 33.3% had not lambed. It is possible that some may have been pregnant or not pregnant at the time of the study. Pregnancy was not verified during this study.

**Table 4.3**: Animal-level factors observed in 117 lame sheep during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

| Animal factors | Various levels of the animal factors | | | |
|---|---|---|---|---|
| Breed | Dorper | Dorper and Red Maasai cross | Dorper and Merino cross | Red Maasai |
| Percentage (%) | 54.84 | 42.74 | 1.71 | 1.71 |
| Weight (kg) | <20 | 20-30 | 31-50 | >50 |
| Percentage (%) | 1.71 | 17.09 | 63.25 | 17.95 |
| Age (Months) | <3 | ≥ 3 | -. | - |
| Percentage (%) | 3.4 | 96.6 | - | -. |
| Sex | Females | Males | - | - |
| Percentage (%) | 76.9 | 23.1 | - | - |
| Pregnancy | Pregnant | Not pregnant | Males | - |
| Percentage (%) | 11.9 | 65.0 | 23.1 | - |
| Lambing period | Current 3 months | More than previous 3 months | Not lambed | -. |
| Percentage (%) | 7.8 | 58.9 | 33.3 | - |

### 4.4.2 Farm-level factors

#### 4.4.2.1 State of the grazing areas

The areas that the sheep spent most of the time grazing in the 10 farms were of uneven bumpy terrain in 8 while flat ground in 2 of them. In one of these 8 farms, the ground was swampy and marshy. In 8 of the farms, there were traumatic objects such as thorny plants and small loose sharp pebbles of stones in the grazing areas. These loose sharp pebbles of stones were also found along the sheep walking tracks. The other 2 farms were free of any traumatic objects. The walking tracks had trench-like excavations in 6 of the 10 farms.

#### 4.4.2.2 Pastures and feeding

Since the study was carried out during the rainy season and immediately after the rains, the pastures were green in all the 10 farms. It was reported in 5 of the 10 farms that during drought when pastures were scarce, the sheep were supplemented with commercially available concentrates and hay. Four of these 5 farms supplemented with only grain concentrate, while the remaining one farm supplemented with only hay. The other 5 farms did not provide any supplements but the sheep were left to live on the scantily available pastures.

### 4.4.3 Management-level factors

#### 4.4.3.1 Hygienic state of the night-resting enclosures

In 5 of the 10 farms the night-resting enclosures were wet with manure accumulation while 5 were dry but also had manure accumulation.

#### 4.4.3.2 Hoof trimming

Hoof trimming was routinely carried out in 5 of the 10 farms and was either done by owners, stockmen, animal health assistants or veterinary surgeon. In three of the 5 farms the trimming was being carried out by unqualified personnel which predisposed them to over-trimming.

#### 4.4.3.3 Foot bathing

Only one of the 10 farms was carrying out foot bathing using formalin solution in a plastic container once a week.

#### 4.4.3.4 Ticks control

All the 10 farms practiced tick control methods by hand spraying using knap sack sprayers.

### 4.5 Association between possible risk factors and lameness

#### 4.5.1 Association between animal-level factors and lameness

The number of limbs affected was significantly associated with moderate to severe degrees of lameness ($\chi^2$ =11.15, $p<0.05$). The affected limb (whether fore or hind limb) ($\chi^2$ =9.20, $p < 0.05$) and the involved claws (whether lateral or medial) ($\chi^2$ =16.98, $p < 0.05$) were also significantly associated with degrees of lameness. There was significant but weak association between the presence of a lesion on the limb with mild to severe degrees of lameness ($\chi^2$ =4.71, $p<0.05$). The rest of the animal-level risk factors such as

sex, breed, body condition score and period when ewe lambed did not seem to influence the occurrence of lameness (Table 4.4)

### 4.5.2 Association between farm-level factors and lameness

There was significant association between the presence of traumatic objects in the farms and mild to severe degrees of lameness ($\chi^2 = 11.01$, $p < 0.05$). The other farm-level factors that were determined such as terrain, grazing ground, type of traumatic object and farm tracks did not show any statistically significant association with lameness (Table 4.5).

**Table 4.4**: Association between the locomotion score and animal-level factors in 117 sheep examined during a study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March 2010–June 2010).

| Animal-level risk factor | Chi-square ($\chi^2$) | p-value | Conclusion |
|---|---|---|---|
| Lesion | 4.71 | 0.030 | Associated |
| No of limbs affected | 11.15 | 0.004 | Associated |
| Affected limb | 9.20 | 0.010 | Associated |
| Involved claw | 16.98 | 0.051 | Associated |
| Claw deformity | 2.05 | 0.152 | No association |
| Type of claw deformity | 2.36 | 0.124 | No association |
| Sex | 0.97 | 0.325 | No association |
| Breed | 0.33 | 0.567 | No association |
| Weight | 2.25 | 0.324 | No association |
| BCS | 0.09 | 0.762 | No association |
| Pregnancy | 1.11 | 0.292 | No association |
| Lambed | 1.40 | 0.237 | No association |
| Location of the lesion | 3.13 | 0.792 | No association |

**Table 4.5**: Association between the locomotion score and farm-level factors in 117 sheep examined during a study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010)

| Farm-level Factors | Chi-square ($\chi^2$) | p-value | Conclusion |
|---|---|---|---|
| Traumatic objects | 11.01 | 0.0001 | Associated |
| Types of traumatic objects | 0.22 | 0.64 | No association |
| Terrain | 0.74 | 0.389 | No association |
| Grazing ground | 0.06 | 0.814 | No association |
| Farm tracks | 0.03 | 0.863 | No association |

# CHAPTER 5

## 5.0 DISCUSSION

The results of the current study revealed that the overall prevalence of lameness in sheep reared under free-range grazing system in the semi-arid district of Kajiado, Kenya is low at about 6.1%. This differs with findings in arid zones of Nigeria in which prevalence of lameness in sheep is higher (Bokko *et al.*, 2003). The differences in these prevalence's may in part be due to variations in the predisposing conditions in the arid and semi-arid climatic conditions in these two different regions. The main conditions causing lameness in sheep in the current study are non-infectious especially sole bruising, overgrown hooves and tick-bite dermatitis as has been reported previously (Eze, 2002; Bokko *et al.* 2003). The prevalence rate of 6.1% in this current study is within the range found in the United Kingdom (DEFRA, 2003a). However, it is lower than the range of 15-19.5% reported by others (Mohammed *et al.*, 1996; Eze, 2002; Bokko and Chaudhari, 2004). The low prevalence in the current study can probably be attributed to the fact that the semi-arid nature of the study area, provides a dry animal living-environment most of the year and almost all the risk factors that were observed (apart from presence of traumatic objects) were not significantly contributing to the occurrence of lameness. This differs with previous reports of arid and semi-arid conditions in Nigeria which resulted in slightly higher prevalence of lameness (Mohammed *et al,* 1996; Bokko and Chaudhari, 2004)

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The finding of higher prevalence of foot lesions as compared to those in the proximal parts of the limbs in this study agrees with previous reports that indicated claw lesions as

the commonest cause of lameness in sheep (Bokko and Chaudhari, 2004). Distribution of foot lesions between the foot and proximal parts of the limbs as found in this study were similar to those reported for dairy cows (Cook *et al.*, 2004).

Painful responses leading to lameness was seen in the sheep with sole bruising in this study. This was probably due to the thinning of the horn of the sole in the bruised parts, which allowed transmission of pressure to the dermis of the claw when the sheep walked with their weight against the hard ground. Similar observations were made in sole bruising in cattle (Nguhiu-Mwangi, 2007; Nguhiu-Mwangi *et al.*, 2008).

In the current study, tick-bite dermatitis was the third most prevalent condition causing lameness. This can probably be attributed to the fact that most of the free-range grazing grounds in the study zone are likely to be tick-infested particularly from cattle, which are nomadically driven in search of pasture from place to place by the same sheep owners. The ease with which tick-dermatitis develops may be attributed to the density of aggregating ticks particularly on the plantar (caudal) aspect distal to the dew claws, as well as probable reaction to injected toxins by the ticks during the bites and tissue damage caused by the mouthparts of the ticks. All these lead to acute inflammation with pain and subsequent lameness. Similar dermatitis attributed to tissue damage by the large mouthparts of the ticks has been observed previously (Azizi and Yakhchali, 2006).

The rest of the conditions such as interdigital dermatitis, shelly hoof, soil balling, foreign body penetration, osteomyelitis of metatarsal bone, septic arthritis, malunion of tibial

bone, hyperextension of fetlock joint, over-trimmed hooves and foot rot were rare with prevalence of about 1% while some were incidental findings. The occurrences of some of these conditions such as foreign body penetration and fractures probably depended on accidental causes and others such as shelly hoof and soil-balling had low probability of occurrence depending on presence and suitability of the predisposing factors. The rampant presence of traumatic objects in the dry land pastures and accumulated manure in the night-resting enclosures increased the probability of occurrences of these conditions.

Infectious conditions such as foot rot were rare in the current study, possibly due to the harsh dry environment in which the causative agents could not propagate. However, the one sheep that had foot rot was severely lame because when these infectious conditions occur, the effects are destructive to the tissues and hence lameness is severe. This tends to support previous reports in cattle that dry environment reduces the incidence of foot lesions (Bergsten and Petterson, 1992). Although foot rot has been reported to be a flock problem which is highly contagious in sheep (Radostitis *et al.*, 2001; DEFRA, 2003a; The Merck Veterinary Manual, 2009), it was sharply contrasted by the low prevalence in the current study. The flock and contagious magnitude of foot rot is likely to be in sheep reared under persistent and prolonged wet conditions.

The few cases of interdigital dermatitis that were observed probably occurred owing to the fact that the study was carried out during the wet rainy season when the causative bacteria would easily multiply and the wet conditions of the foot environment would

enhance development of the lesions. This could also be due to the fact that interdigital dermatitis is more contagious than foot rot as has been reported previously (DEFRA, 2003a). It therefore might explain the reason more cases were seen with interdigital dermatitis than with foot rot.

The higher number of moderately lame sheep compared to the number with mild and severe lameness in this study could be attributed to the fact that the lameness conditions with the highest prevalence were found to be those that caused minimal pain or discomfort, such as sole bruising and hoof overgrowth. These more common conditions did not cause severe lameness, nevertheless their pain and discomfort exceeded mild degree of lameness, hence moderate lameness. The more painful infectious conditions had very low prevalence and hence the correspondingly low percentage of severely lame sheep. Similar findings have been reported in cattle (Nguhiu-Mwangi, 2007). A higher percentage of lame sheep were observed to have more than one foot affected simultaneously. This may be probably because the occurrence of the conditions with higher prevalence such as sole bruising, overgrown hooves and tick-bite dermatitis is most likely bilateral and thus involving more than one foot as well as several claws. The bilateral involvement of the limbs and claws observed in this study agrees with earlier reports (Mohammed *et al.*, 1996; Eze 2002; Bokko *et al.*, 2003).

The one sheep observed with over-trimmed hooves was definitely a management error or due to poor trimming skills. It caused lameness by the likelihood of exposed sensitive laminae treading directly on the ground or by the resulting interference with proper

weight distribution to the claws. It also may cause poor treading angle, which exerts pressure to limb structures that should not have much pressure. Similar observations of unskilled trimming in cattle leading to lameness have previously been cited (Blowey, 2002; Vermunt, 2004).

The one sheep found with interdigital soil-balling was predisposed by the manure stuck in the interdigital space, which was picked gradually little by little from what accumulated at the night-resting enclosure areas over prolonged time. The manure stuck in the interdigital spaces subsequently collects pieces of grass as the sheep grazes. Eventually that accumulated manure-grass mixture dries up within the interdigital spaces as "soil-balling" or "manure-balling" and leads to splaying of the toes as long as it remains on the claws. The splaying of the toes causes discomfort and pain that lead to mild lameness. Similar findings have been been reported previously (Clarkson and Faulli, 1990; Winter, 2004a). Although there were many sheep in the manure-accumulated night-resting enclosures, only one was found to have sustained the soil-balling lump. This is possible due to the fact that the probability of manure persistently getting stuck in the interdigital space may depend on individual variations of conformation of the claws and the space between them.

Dorper sheep or their crosses were found to be the preferred breeds by the Maasai community living in the study area, hence their higher numbers among the lame sheep relative to the other breeds. The tendency and the likelihood of selling off rams for slaughter and retaining the ewes for breeding of the flock is probably the reason why the

female sheep were more in number among the lame sheep. Similar observations have been made previously (Egwu *et al.*, 1994; Bokko and Chaudhari, 2004).

A higher percentage of the lame sheep was observed to be in good body condition probably corresponding to the higher percentage of the moderately lame which meant that the discomfort in these sheep was not severe enough to put them completely off feed. They were still able to move about slowly and feed, which resulted in maintenance of good body condition compared to poor body condition that would have resulted if the sheep had severe lameness. This deviates from observations by other researchers who reported poor body condition in majority of lame sheep probably because in their findings, the prevalence of severe lameness was also higher (Bokko and Chaudhari, 2004).

All animal-level factors including breed, age, sex, body condition score and lapse of time from lambing did not seem to significantly influence the occurrence of lameness. The prevailing uneven and bumpy terrain with a lot of stony pebbles in the majority of the evaluated farms, are likely to have predisposed the sheep to most of the lameness conditions affecting the claws. Similar observations on the influence of farm-level factors on occurrence of lameness have been reported (Clarkson and Faulli, 1990; Bokko and Chaudhari, 2004).

When more than one limb is affected by lameness conditions in any sheep, it is likely to cause much discomfort and pain that may precipitate difficulties in locomotion and

influence the locomotion score. This may explain the reason for strong significant association found between moderate to severe degrees of lameness and the number of limbs affected. A similar association that was found between the affected limb (fore or hind) or affected claw (lateral or medial), and the severity of lameness could probably be related to weight distribution in which the fore limbs bear more weight than the hind limbs, and the lateral claw bears more weight than the medial claw. These observations are similar to findings in cattle with more lameness on hind limbs, lateral claws of hind limbs and bilateral involvement (Tadich and Hernandez, 2000; Blowey, 2002; Vermunt, 2004). However, these observations sharply contrast previous reports which indicated that the fore limbs are subjected to more trauma than the hind limbs (Bokko and Chaudhari, 20004). Presence of several lesions on one single limb caused more discomfort and pain and this adversely affected the locomotion, resulting in the significant association found between presence of a lesion on the foot and mild to severe degrees of lameness.

The significant association between the presence of traumatic objects and the degree of lameness is probably attributed to difficulties in locomotion owing to discomfort and trauma caused by these objects on the treading surface of the claws. However, other farm-level factors such as grazing ground, type of traumatic object and farm tracks did not show any significant statistical association with lameness, nevertheless they could still have contributed to lameness by synergistically acting together with other predisposing factors. Similar findings have been reported in cattle (Greenough, 1991).

# CHAPTER 6

# 6.0 CONCLUSIONS AND RECOMMENDATIONS

## 6.1 Conclusions

The results of this study led to the following conclusions

6.1 1 Lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya is relatively low at 6.1%.

6.1.2 The main causes of lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya are non-infectious and infectious causes are negligibly low.

6.1.3 More than 90% of lameness in sheep involves the foot.

6.1.4 The main risk factor for lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya is presence of traumatic objects in the grazing ground.

## 6.2 Recommendations

The following recommendations can be made from the study as intervention measures to reduce the incidence of lameness in sheep under free-range grazing system in arid and semi-arid zones of Kenya:

6.2.1 Regular and skilled hoof trimming should be practiced.

6.2.2 Traumatic objects should be cleared from the grazing grounds.

6.2.3 Regular and effective methods of tick-control should be used.

6.2.3 Regular removal of manure from sheep night-resting enclosures should be encouraged.

6.2.4 Chemical footbaths will help to eliminate infectious causes of lameness and possibly use of formalin might harden the claw horn to prevent ease of bruising.

### 6.3 Areas for further research

There is a need to carry out further research related to the current study. These studies should include;

6.3.1 Studies in prevalence and risk factors of conditions causing lameness in high and medium potential lands of Kenya.

6.3.2 Controlled studies to verify the interactive role of various risk factors of sheep lameness.

6.3.3 Evaluation and quantification of the effect of lameness on financial economy in sheep enterprises

# CHAPTER 7

## REFERENCES


**Abel, C. (2008)**: "Bluetongue vaccine BTV8 questions answered" *Farmers Weekly.*

**Arkins, S. (1981)**: Lameness in dairy cows. *Irish veterinary Journal.* **35**:135-140

**Azizi, S. and Yakhchali, M. (2006)**: Transitory lameness in sheep due to Hyalomma infestation in Urmaia Iran. *Small Ruminant Research,* **63** (3):262-264.

**Bergsten, C. and Petterson, B. (1992)**: The cleanliness of cows tied in stalls and the health of their hooves as was influenced by the use of electric trainers. *Preventive Veterinary Medicine.* **13**:229-238.

**Berry, S. L. (1999)**: Hoof health. In Proceedings of the World Dairy Management Conference, Las Vegas, Nevada. pp 13-17. April 8-10, 1999.

**Blowey, R. W. (2002)**: Claw trimming: How should it be done? A comparison of two approaches. In Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando, FL, USA. pp 122-126, 9th -13th January 2002.

**Bokko, B. P; Adam, S. S. and Mohammed, A. (2003)**: Limb conditions that predispose sheep to lameness in the arid zone of Nigeria. *Small Ruminant Research.* **47** (2); 165-169.

**Bokko, B. P. and Chaudhari, S. U. R. (2004)**: Prevalence of lameness in sheep in the North East region of Nigeria. *International Journal of Agriculture and Biology.* **3-4**:519-521.

**Caple, I. W. (1990)** Vitamin D deficiency In Sheep Medicine, University of Sydney Post-graduate Committee in Veterinary Science, Proceedings No **141**, pp 381

**Clarkson, M. J. and Faulli, W. B. (1990)**: Lameness: A Handbook for the sheep Clinician, .4th Edition, Liverpool University Press, UK. pp 47-70.

**Clarkson, D. A. and Ward, W. R. (1991)**: Farm tracks, stockman's herding and lameness in dairy cattle. *Veterinary Record.* **129**:510-511.

**Cook, N. B; Nordlund, K. V. and Oetzel, G. R. (2004)**: Environmental influences on claw horn lesions associated with laminitis and sub acute ruminal acidosis in dairy cows. *Journal of Dairy Science.* **87**: E36-E46.

**Coulon, J. B; Lescouret, F. and Fonty, A. (1996)**: Effect of foot lesions on milk production by dairy cows. *Journal of Dairy Science* **79**: 44–49.

**Davis, R. C. (1982)**: Effects of regular formalin footbath on the incidence of foot lameness in dairy cattle. *Veterinary Record.* **111** (17):394.

**DEFRA. (2003a)**: Lameness in Sheep. DEFRA Publications. Available at: http://www.defra.gov.uk/animalh/welfare/pdf/sheeplameness.pdf April 2011.

**DEFRA (2003b)**: Code of Recommendations for the Welfare of Livestock: Sheep. DEFRA Publications.Availableat:http://www.defra.gov.uk/animalh/welfare/farmed/farmed/sheep/booklets/sheep.pdf April 2011.

**Department of Veterinary Services Annual Reports. (1995-2008)**. Ministry of Livestock Development, Republic of Kenya

**Egwu, G; Adamu, S. S; Ameh, J. A ; Ongeyili, P. A; Abana, P. S; Chaudhari, S. U. R. and RaboJ. S. (1994)**: Reproductive, clinic pathological and microbiological studies of interdigital pouch lameness in sheep in an arid zone of Nigeria. *Bulletin Animale production Africana.* **42**: 5-11.

**Elliot, M. and Pinkus, T. (1993)**: Homoeopathy, the Shepherd's Guide. Ainsworthy pharmacy, London. ISBN 0-9523411-2-3, pp 28.

**Eze, C, A. (2002)**: Lameness and reproductive performance in small ruminants in Nsukka Area of the Enugu State, Nigeria. *Small Ruminant Research.* **44**: 263–28867.

**Gairdner, J. (2007):** "Bluetongue outbreak in the UK. *Veterinary Record.* **161**: 534–5.

**Gatenby, R. M. (1986)**: Sheep production in the tropics and sub-tropics. Longman Singapore Publishers Ltd, London. pp 351.

**Gelasakis, A. I; Valergakis, G. E. and Arsenos, G. (2009)**: Predisposing factors of sheep lameness. *Journal of the Hellenic Veterinary Medical Society.* **6** (1): 63-74.

**Green, L. E; Hedges, V. J; Schukken, Y. H; Blowey, R. W. and Packington, A. J (2002)**: The impact of clinical lameness on the milk yield of dairy cows. *Journal of Dairy Science.* **85**: 2250–2256.

**Greenough, P. R. (1991)**: A review of factors predisposing to lameness in cattle. In: Breeding for Disease Resistance in Farm Animal, J. B, Owen and R. .F, Axford. Ed, CAB International, Wallingford, U.K. pp 371-393.32

**Greenough, P. R. and Vermunt, J. J. (1991)**: Evaluation of sub-clinical laminitis in a dairy herd and observations on associated nutritional and management factors. *Veterinary Record.* **128**: 11-17.

**Harwood, D. G; Cattell, J. H; Lewis, C. J. and Naylor, R. (1997):** Virulent foot rot in sheep. *Veterinary Record* **140**: (26) 687.

**Henderson, D. C. (1990)**: The veterinary book for sheep farmers. Farming Press Books, Ipswich, UK. pp 592-595.

**Hungerford, T. G. (1990):** Lameness. Hungerford's Diseases of Livestock, 9th Ed. McGraw-Hill Book Co. Sydney. pp 1059-60.

**Jubb, T. F. and Malmo, J. (1991):** Laesions causing lameness requiring Veterinary treatment in pasture fed cattle in East Gippsland. *Australian Veterinary Journal.* **68**:21-24.

**Kariuki, D. P. and Letitiya. W. (1996):** Livestock production and health challenges in pastoral areas of Samburu district, Kenya Agricultural Research Institute (K.A.R.I.), Nairobi, Kenya.

**Kitching, R. P. (2004):** Contagious Pustular Dermatitis Infectious diseases of livestock.. Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University press, Cape Town, S A. pp 1282-1286.

**Marshall, D. J; Walker, R. I; Cullis, B. R. and Luff, M. F. (1991):** The effect of footrot on body weight and wool growth of sheep. *Australian Veterinary Journal.* **68**: 45–49.

**Mgasa, M. N. and Arnbjerg, J. (1993):** Occurrence of lameness and digital lesions in Tanzanian goats. *Small Ruminant Research.* **10**: 55-62.

**Mohammed, A. U. A; Badau, R. O. and Kene, R. O. C. (1996):** Lameness in sheep and goats in relation to hoof conditions in Sahel zone of Nigeria. *Bulletin. Animale Health Production. Africana.* **44**: 97-100.

**NADIS (2003):** Lameness not caused by Scald or Footrot. Sheep Disease Focus:

**National Livestock Policy. (2008).** Ministry of Livestock Development, Republic of Kenya.

**Nguhiu-Mwangi, J. (2007)**: Characteristics of laminitis and associated claw lesions in dairy cows in Nairobi and its environs under various management systems, PhD. Thesis, University of Nairobi, Kenya.

**Nguhiu-Mwangi, J; Mbithi, P. M. F; Wabacha, J. K. and Mbuthia, P. G. (2008):** Prognostic indicators and the importance of trimming in non-infective claw disorders in cattle. *The Kenya Veterinarian.* **32** (1) : 26-40.

**Nonga, H. E; Makungu, M; Bittegeko, S. B. P. and Mpanduji, D. G. (2009)**: Occurrence and management of lameness in goats: A case study of Magadu farm, Morogoro, Tanzania. *Small Ruminant Research,* **82:** 149-151.

**Offer, J. E; Mcnully, D. and Logue, D. N. (2000)**: Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. *Veterinary Record.* **147**: 105-109.

**Otieno, G. O. (2008):** Ministry for Development of Northern Kenya and other Arid Lands, Arid Lands Resource Management Project ii –Kajiado District

**Parajuli, B. and Goddard, P. J. (1989)**: A comparison of the efficacy of footbaths containing formalin or zinc sulphate in treating ovine foot rot under field conditions. *British Veterinary Journal.* **145**: 467-472.

**Pugh, D, G, (2002)**: Sheep & Goat Medicine. Philadelphia: Saunders. ISBN 0-7216-9052-1.

**Raadsma, H. W; Egerton, J. R; Outteridge, P. M; Nicholas, F. W; Brown, S. C. and Litchfield, A. M. (1990)**: An investigation into genetic aspects of resistance to foot rot in Merino sheep. *Wool Technology and Sheep Breeding.* **38**: 7-12.

**Radostitis, O. M; Gay, C. C; Blood, D. C. and Hinchcliff, K. W. (2001)**: A textbook of diseases of cattle, sheep, pigs, goats and horses, 9th edition. Paston press Ltd, London, Norfolk.

**Rhebun, W. C. and Pearson, F. G. (1982)**: Clinical management of bovine foot problems. *Journal of American Veterinary Medical. Association..***181**:572-579.

**Russell, A. M; Rowlands, G. J; Shaw, S. R. and Weaver, A. D. (1982)**: Survey of lameness in British dairy cattle. *Veterinary Record.* **111**:155-160.

**Scott, K. and Henderson, D. C. (1991)**: Foot Rot and Foot Conditions. In: Diseases of Sheep. 2nd edition.. W.B. Martin .and I. D. Aiken, Eds. Blackwell Scientific Publications, Oxford. pp 201-209.

**Suiter, J. (2006)**: Body condition scoring of sheep and goats, Department of Agriculture, Western Australia. Farmnote, 69/2006.

**Tadich, N. and Hernández. M. (2000)**: A survey on the prevalence of foot lesions in sheep from 25 small holdings in the province of Valdivia, *Archives Medicine Veterinary.* **32**.1.

**The Kenya National Population and Housing Census Results. (2010):** Ministry of State for Planning, National Development and Vision 2030.

**The Merck Veterinary Manual (2009)**: A Handbook of Diagnosis, Therapy and Disease Prevention and Control for the Veterinarian. 9th edition, Editor C. M, Fraser and Kahn, M.A. Published by Merck and Co. Inc. Railway, N. J, USA.

**The Veterinary Formulary. (1998)**: 4th Edition. Eds. Y. Bishop Pharmaceutical Press, London pp 395-406.

**Tranter, W. P; Morris, R. S; Dohoo, I. R. and Williamson, N. B. (1993)**: A case control study of lameness in dairy cows. *Preventive Veterinary Medicine*. **15**. 199-203.

**Vermunt, J. (2004):** Herd Lameness: A review, major causal factors, and guidelines for prevention and control. In: Proceedings of the 13th International Symposium and 5th Conference on Lameness in Ruminants. 11th -15th February 2004. Maribor, Slovenija. pp 1-15

**Vermunt, J. J. and West, D. M. (2004)**: Toe Fibroma, Toe Abscess and Foot Rot. In; Infectious Diseases of Livestock, Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University press, Cape Town, S A. pp 1725-1738.

**Verwoerd, D. W. and Erasmus, B. J. (2004)**: Blue Tongue Disease. In: Infectious Diseases of Livestock..Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University Press, Cape Town, S A. pp 1201-1215.

**Veterinary Education and Information Network (VEIN). (2008)**: Blue Tongue Disease. Foot and Mouth Disease. *Journal of Clinical Microbiology*. **47**(9): 2992-2994.

**Warnick, L, D; Janssen, D; Guard, C, L. and GröHn, Y, T. (2001)**: The effect of lameness on milk production in dairy cows. *Journal of Dairy Science*. **84**: 1988–1997.

**West, D. M. (1990)**: Foot abscess and other lameness problems of sheep. In: Sheep Medicine. Proceedings of the Study and Georgiana Reid Memorial refresher course for Veterinarians, 16th -20th July 1990. University of Sydney, Australia.

**Whittington, R. J. (1995)**: Observations on the indirect transmission of virulent ovine foot rot in sheep yards and its spread in sheep on unimproved pasture. *Australian Veterinary Journal.* **72** (4): 132-134.

**Winter, A. (1998a)**: Lameness in Sheep. The Moredun Foundation News Sheet. **3:** 1.

**Winter, A. (1998b)**: Virulent Foot rot in sheep. *Veterinary Record,* **141**: 1.

**Winter, A. (2004a)**: Lameness in sheep. *Diagnosis in Practice*, **26** (2): 58-63

**Winter, A. (2004b)**: Livestock Health and Welfare Division, Liverpool.

**Winter, A. and Charmley, J. (1999)**: The Sheep Keeper's Veterinary Handbook. Crowood Press Ltd, Marlborough, UK. ISBN 1-86126-235-3.

**Zaria, L. T. and Damin, J. (2004)**: Dermatophilosis. In: Infectious Diseases of Livestock. Edited by J. W, Coetzer/ and R. C, Tustin, 2nd Edition, Oxford University press, Cape Town, S A. pp 2026-2035.

# CHAPTER 8

## APPENDICES

**Appendix 1:** Data collection sheets with parameters and their codes that were used during the study of prevalence and risk factors of conditions causing lameness in sheep under free-range grazing system in Kajiado District, Kenya (March–June 2010).

**a) Animal-level factors**

1) Sheep code 1 Serial no up to 117
2) Farm ID 1= Farm no one 2= Farm no two ..... up to 10.
3) Flock size 1= 100-150 2= 151-200 3= 201- 250 4= 251-300 5= 301-350
4) Estimated age 1= Lamb 2= Adult
5) Sex 1= Male 2= Female
6) Breed 1=Dorper 2= Cross of Dorper and Maasai sheep 3= Cross of Dorper and Merino 4= Maasai sheep
7) Body condition score 1= Poor 2= Fair 3= Good 4= Very Good
8) Pregnancy status 1= Late pregnancy 2= Not pregnant
9) Recently lambed 1= Less than 3 months 2= Above 3 months
10) Lameness severity score 1= Mild 2= Moderate 3= Definite 4= Severe
11) Number of affected limbs 1= 1 2 = 2 3 = 3 4= 4
12) Affected limb 1= Fore limb 2= Hind limb 3= Both
13) Position of lesion 1= Proximal 2= Foot
14) Involved claw 1= Medial 2= Lateral 3= Both
15) Specific lesions causing lameness

1= Overgrown hoofs 2= Sole Erosion 3= Tick-bite dermatitis 4= Interdigital Dermatitis 5= Hoof Fracture 6= others.

16) Claw deformity 1= Present 2= Absent

17) If present, specify the type 1= Misshapen hoof 2= Hoof cracked 3= Not Applicable

18) Any bone or Joint involvement 1= Yes 2= No

19) Specimen(s) collected 1 =Yes 2= No

20) Biopsy for any swelling(s) 1= Yes 2= No

**b) Interview questionnaire**

21) Have you experienced any case of lameness in sheep on the farm in the last three months? 1= Yes 2= No

22) If yes, how many cases? 1 = 1-5 2 = 6-10 3 = >10.

23) Was the sheep treated? 1 = Yes 2 = No

24) Do you practice hoof trimming? 1 = Yes 2 = No

25) If yes, who does it 1 = Owner 2 = Stockman 3 = Vet surgeon / Animal Health Assistant

26) How often per year? 1 = Once 2 = More than once

27) How do you control ticks 1= Dipping 2= Hand spraying 3= Other method 3= None

28) Do you supplement the sheep during prolonged drought 1 = Yes 2 = No

29) If yes what type Of feed 1 = Concentrates 2 = Hay 3 = Not applicable

30) Have you observed more cases of lameness in certain lineage of sheep than in others 1 = Yes 2 = No

31) Have you seen cases of lameness in cattle at the same time with sheep 1 = Yes 2 = No

32) If yes, how many sheep were involved? 1 = Less than 10 2 = More than 10 3 = Whole herd.

33) Do you know of diseases that affect other parts of the body and cause lameness in sheep 1 = Yes 2 = No.

34) If yes, which organs were affected 1 = Rest of the skin 2 = Testis 3 = Head region 4 = Other parts specify .....

35) Which season are sheep mainly lame 1 = Dry season 2 = Wet season

36) Are there herdsmen who rear animals and they report more lameness cases than others 1 = Yes 2 = No.

37) If yes. what do you attribute this to 1 = Overdriving 2 = Witchcraft 3 = Not clear

**(c) Farm-level factors**

38) Terrain 1 = Bumpy 2 = Level

39) Grazing ground 1 = Dry 2 = Marshy

40) Traumatic objects 1 = Present 2 = None

41) If present, the type 1 = Dried thorns 2 = Loose stones 3 = Dried pastures.

42) State of Farm tracks 1 = Even 2 = Loose stones 3 = Trenches.

43) Pasture conditions 1 = Dry 2 = Green

44) Type of management 1 = Free-range 2 = Padlocking 3 = Migration.

45) State of sheep house or night-resting enclosures 1 = Wet and a lot of manure 2 = Dry and a lot of manure 3 = Dry and little manure 4 = Wet in the morning and little manure.

46) Observation of whether there is a footbath 1 = Present 2 = Absent.

47) If present, name of the chemical used  1 = Formalin  2 = Any other