GENETIC EVALUATION OF TICK RESISTANCE IN SOUTH AFRICAN BONSMARA CATTLE

MUTSHINYA ANANIAS BUDELI

BSC AGRIC. (UNIVEN)

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SUPERVISOR: PROF. D NORRIS

CO-SUPERVISOR: DR A. MAIWASHE

CO-SUPERVISOR: DR K.A. NEPHAWE

CO-SUPERVISOR: PROF. J.W. NGAMBI

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DECLARATIONS

I declare that the mini dissertation hereby submitted to the university of Limpopo, for the degree of Master of Science in Agriculture (Animal Breeding and Genetics) degree has not been previously submitted by me for a degree at this or any another University, that it is my work in design and in execution, and that all material contained herein has been duly acknowledged.

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Budeli Mutshinya Ananias(Mr) Date

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To my wife Ndivhudzannyi Joyce Budeli, thank you for your understanding and support during my studies. To Ngoako William Selapa, thank you for your time and assistance.

I will not end until I give thanks to my creator the Heavenly Father for giving me life, divine heath and sound mind.

ABSTRACT

The objectives of the study were to estimate genetic parameters for tick resistance and to evaluate the effect of the level of tick infestation on the estimates of genetic parameters in South African Bonsmara cattle. Field data of repeated tick count records (n = 11 280) on 1 176 animals were collected between 1993 and 2005 by ten breeders participating in the National Beef Recording and Improvement Scheme. The distribution of tick count records were normalized using a Box-Cox transformation. Data were divided into 7 sub-data sets based on the mean tick count per contemporary group, to facilitate the investigation of the effect of level of tick infestation on the derived genetic parameters. A repeatability animal model including the fixed effects of contemporary group and age of animal at tick counting and random effects of the direct additive genetic, permanent environmental and residual effects was used to estimate genetic parameters using REML procedures. The additive genetic variances for tick count ranged from 0.01 to 0.08. Variances for the permanent environment ranged from 0.00 to 0.03. Phenotypic variance decreased with increasing mean tick count level while additive genetic variance increased with increasing mean tick count level. The heritability also increased with mean tick count level until a mean tick count level of ≥30. The highest heritability estimate obtained in the current study was 0.17 for data with mean tick count level ≥25. These results suggest that sufficient genetic variation for tick count exists in the Bonsmara cattle. Therefore genetic selection for tick resistance is feasible even though genetic progress may be slow.

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Chapter 1

General Introduction

1.1. Background

Losses in livestock production due to parasites have long been of concern to livestock producers, government and researchers (Seifert, 1984). Tick-borne diseases are one of the major constraints to livestock improvement throughout the tropics. One million cattle are estimated to have died of East Coast fever in sub-Saharan Africa during 1989 alone (Mukhebi *et al*., 1992). In South Africa, tick-borne diseases cost the livestock industry about R70-R200 million per year (Bigalke, 1980).

Various tick-borne disease control methods have been employed in South Africa (Bigalke *et al.*, 1976; De Vos, 1979; Purnell and Schroder, 1984). Historically, the earliest tick control trials with dipping agents in South Africa started in 1889. Shortly after the discovery in the USA in 1893 that ticks transmit the causal piroplasm of redwater in cattle; the foundation was laid for the control of ticks and tick-borne diseases with chemical toxins (Hayward, 1981). Increase in acaricides prices and drug resistance pose a challenge in the application of these methods, because it is expensive and takes time to develop new drugs.

Hayward (1981) stated that the best solution (to high acaricides prices and resistance to drugs) might be the identification of naturally resistant breeds and encouragement of their use. Natural disease resistance refers to the inherent capacity of an animal to resist disease when exposed to pathogens, without prior exposure or immunization (Adams and Templeton, 1998). Utech *et al*. (1978) defined tick resistance as the ability of cattle to limit the number of ticks that survive to maturity.

Although some of the observed variation in natural tick resistance is related to environmental factors, a significant component of variation in natural disease resistance appears to be heritable and, therefore, to be passed stably from parent to offspring (Adams and Templeton, 1998). Several studies have been conducted on genetic determination of tick resistance (Utech *et al.,* 1978; Spickett *et al.,* 1989;

Rechav and Kostrzewski, 1991). Tick resistance has been shown to be heritable (Hewetson, 1972). Davis (1993) reported the heritability for tick resistance to be about 34%, thus indicating that genetic improvement through selection should be effective. Information on the resistance status of the various breeds of cattle is needed to provide a basis for selection, by either breeding animals for tick resistance, or culling cattle with low tick resistance, or both. There is a genetic basis for variation in tick resistance and it varies within and among breeds (Utech *et al.,* 1978). Indigenous breeds have been found to be more resistant than exotic breeds (Latif, 2006). All cattle are tick-resistant to some degree, acquiring individual levels of tick resistance in response to tick challenge (Roberts, 1968; Wagland, 1975).

It was reported in Australia that *Bos indicus* cattle (e.g. Zebu) and their crosses in particular, develop a high degree of tick resistance to blue-tick infestation, thereby preventing the parasites from having any noteworthy effect on production (Seifert, 1971). In South Africa, breeders have made a remarkable progress by developing and improving the indigenous Afrikaner and Drakensberger cattle, and in recent years with the breeding of the Bonsmara (Latif, 2006). These breeds are more resistant to ticks than most of the exotic breeds in the country.

1.2. Problem statement

The use of acaricides has been the most commonly appied method to control tick infestation and tick population. This method is costly and many farmers cannot afford to use acaricides. Additionally, ticks develop resistance to acaricides. Though ticks pose enormous challenges to livestock production, very little work has been done to seek alternative ways of controlling them. Such alternative methods may include selecting animals based on their genetic potential, if sufficient genetic variation exists for tick resistance.

1.3. Aims and objectives of the study

1.3.1. Aim

To develop a model for genetic selection tool for tick resistance in South African beef cattle.

1.3.2. Objectives

i. To develop an operational statistical model for genetic analysis of tick resistance in beef cattle.
ii. To estimate genetic parameters for tick resistance in Bonsmara cattle.

1.4. Hypothesis

Null Hypothesis: The heritability for tick resistance (tick count) is not different from zero (i.e. there is not sufficient genetic variation to enable genetic selection for tick resistance).

Alternative Hypothesis: The heritability for tick resistance (tick count) is greater than zero (i.e. there is sufficient genetic variation to enable genetic selection for tick resistance).

1.5. Organization of the dissertation

This dissertation is organised as follows: The aim of the study is presented in Chapter 1. Chapter 2 is a review of the relevant literature on tick resistance in beef cattle as well as the genetic basis of tick resistance for genetic selection programs; Chapter 3 is presented in a form of a self-contained article following the South Africa Journal of Animal Science format; Chapter 4 presents conclusions and implications to the livestock industry.

Chapter 2

LITERATURE REVIEW

2.1. Background

Parasitic diseases are a global problem and considered as a major obstacle to the health and product performance of animals. These may be due to endo-parasites that live inside the body or ecto-parasites such as ticks, mites, flies, fleas, midges, etc, which attack the body surface (Rajput *et al.*, 2006). Ticks are of great economic importance as vectors of several diseases of domestic livestock and of commercially farmed wildlife in sub-Saharan Africa (Norval and Horak, 1994). Ticks are external blood-sucking and cause illnesses themselves as well as being efficient carriers of diseases (Green and Burton, 1985). Tick-borne protozoan diseases (e.g. Theileriosis and Babesiosis) rickettsial diseases (e.g. Anaplasmosis), cowdriosis and tick-associated dermatophilosis are major health and management problems for livestock producers in many developing countries (Rajput *et al.*, 2006). There are 690 species and subspecies of ticks that are recognised globally (Norval and Horak, 1994).

Between 1974 and 1979 an estimated one million cattle died in Zimbabwe, mainly of tick-borne diseases (Lawrence *et al.,* 1980). Three hundred million cattle in tropical and subtropical regions are at the risk of infection with tick-borne diseases (Wright, 1990) and the economic losses inflicted by these diseases in South Africa alone are estimated to be R70 to R200 million per annum (Bigalke, 1980).

Different types of ticks cause different types of diseases, for example blue tick causes redwater and gallsickness, heart water is caused by bont ticks and corridor disease by brown ear ticks. Ticks are divided into two groups: soft bodied ticks (Argasidae) and hard bodied species (Ixodidae). Hard ticks feed for extended periods of time on their hosts, varying from several days to weeks, depending on factors such as life stage, host type, and species of tick (Rajput *et al.*, 2006).Soft ticks go through multiple nymphal stages , gradually increasing in size until the final molt to the adult stage. The time to completion of the entire life cycle is generally longer than that of hard ticks, lasting over several years. In addition many soft ticks have resistance to

starvation, and can survive for many years without a blood meal (Furman and Loomis, 1984). Ticks are classified according to their life cycles, i.e. one-host, two-host and three-host ticks. One-host ticks (e.g. blue tick) feed and live on one animal for most of their life cycle. Other ticks feed on two different animals in their lifetime (e.g. brown ear tick); these are referred to as two-host ticks. Three-host ticks (e.g. bont tick) feed on three different animals in their lifetime (Norval, 1994). Scholtz *et al.* (1991) reported that loss in weaning mass of calves from cows infested predominately with one-host ticks *(B. decoloratus*) to be higher and in the region of 8.0-9.0 g for each engorged female.

2.2. Conventional methods to control ticks

Normally, tick borne diseases are controlled by acaricides, which are chemicals that kill ticks or prevent their attachment. Acaricides can be used as sprays, dips, pour-ons, spot treatment or injectable drugs, and thereafter the ticks which are on them drop off or are killed. However, the development of resistance in ticks to successive acaricide compounds has been a major problem. This has been compounded by the increasing cost of acaricides, illegal cattle movement, civil unrest, poor management and inadequate maintenance of infrastructure. The devastating extent of droughts in Africa has made many dip-tanks non-operational due to lack of water. Another complication associated with the use of acaricides is that they are environmental pollutants and may also contaminate milk and meat (Latif, 2006). Animal well-being has become a significant concern among consumers who expect food animals to be well treated, raised in idyllic environments and free of disease. Consumers also expect their meat products to be free of residual antibiotics and therapeutic drugs (Snowder, 2006).

Strict acaricide application results in heavy losses in highly susceptible cattle when tick control breaks down. An extreme example was the breakdown of dipping infrastructure during the war of independence in Zimbabwe, where a compulsory dipping policy had been in force since 1914. Between 1974 and 1979 an estimated one million cattle died, mainly of tick-borne diseases (Latif, 2006). Strict tick control is also difficult to maintain in many countries and more rigorous methods for controlling ticks and tick-borne diseases are being investigated. The broad approach has been to use integrated control measures, which include the natural exposure to

Anaplasma, Babesia or *Cowdria* organisms while animals are very young. Other means include immunizing older animals with live vaccines, immunization against East Coast Fever by infection and treatment, chemotherapy and strategic acaricide application to control overwhelming tick infestation or disease challenge (Latif, 2006).

2.3. Genetic resistance to ticks

Despite traditional disease control measures, losses attributable to infectious diseases continue to impede the livestock industries (Adams and Templeton, 1998; Detilleux, 2001). As mentioned earlier, bacterial pathogens develop resistance to antibiotics and thus other methods are desperately needed to counter diseases previously treated by conventional antibiotics. One approach is to improve genetic resistance to infectious pathogens (Detilleux, 2001). Genetic disease resistance involve both immune and non-immune mechanism, which is the inherent capacity of a previously unexposed animal to resist disease when challenged by pathogens. Genetic differences in the effects of parasitic burdens on growth are evident, and the genotypes least affected are those best adapted to the climatic environment (Detilleux, 2001). Consideration should therefore be given to further improvement of resistant genotypes by selection. When the nutritional environment is adequate, high levels of infestation has little effect on any of the genotypes, and it may be the most economic policy to treat animals to parasites only during periods of stress (Seifert, 1971).

Although the nurturing environment influences variability in disease expression, natural resistance has been found to be heritable and is transmitted from parent to offspring. Thus, an alternative approach to enhancing animal health management systems is to increase the overall level of genetic resistance at herd and population level by using selective breeding programmes (Adams and Templeton, 1998). The most important element in this control package is the use of breeds of cattle that are genetically resistant to tick infestations. Resistant animals carry fewer ticks and require less dipping, thus making control relatively easy and cheaper (Latif, 2006). Resistance to tick infestation varies among individuals and breeds of cattle (Rajput *et al.,* 2006). Most indigenous cattle in areas where tick-borne diseases occur possess a natural resistance to these diseases (Utech *et al*., 1978). It has long been recognised

that some animals, or whole breeds, consistently carry fewer ticks than others kept in the same environment (Roberts, 1968a; Wagland, 1975). Host resistance expressed by an animal's ability to prevent the maturing of large numbers of ticks, and disease immunity, are survival mechanisms for the host and for external and internal parasites (Rajput *et al.,* 2006). Resistant animals consistently carry fewer ticks than susceptible animals. Female ticks completing engorgement are fewer and smaller on resistant animals than on susceptible animals (Latif, 2006). Such differences are caused by variation in the animals' abilities to respond immunologically to tick infestation (Roberts, 1968b). The ability to develop resistance is heritable (Hewetson, 1972; Seifert, 1984) and the actual manifestation is acquired (Riek, 1962; Roberts, 1968a). It is stable over longer periods, although stresses such as lactation or sickness cause a drop in resistance (Wharton *et al.,* 1970; Seifert, 1971; Utech *et al.*, 1978). Different levels of resistance occur in all breeds, but Zebu cattle and their crosses are more resistant (Riek, 1962; Wilkinson, 1962; Wharton *et al.*, 1969; Seifert, 1971; Hewetson, 1979). Improved tick control following the use of tick-resistant cattle has been demonstrated in various breeds of cattle and crossbreds (Riek, 1962). For example, cross between Zebu and Taurine cattle was shown to carry fewer ticks and required less dipping than temperate breeds of cattle on similar pastures (Wharton *et al*., 1969).

Bonsma, in the 1940s, made observations on cattle mortality due to heartwater and its relation to the number of ticks on animals of different breeds. Such observations formed the basis of the studies on host-resistance to tick infestations in South Africa (Bonsma, 1981). The study showed that Africander cattle carried far fewer ticks than British beef cattle and had a far lower mortality rate than the British cattle (6% vs 60%). Assessment and quantification of host-resistance were carried out on several African indigenous breeds of cattle and majority of these breeds are tick-resistant and of high productivity (Trail and Gregory, 1981; Saeed *et al.,* 1987).

In South Africa, the resistance of Bonsmara cattle to ticks was reported to be better than that of Simmental and Sanata Gertudis breeds but not as high as Afrikaner, Brahman or Nguni (Rechav and Kostrzewski, 1991). In Australia, Burns *et al*. (1997) reported superior tick resistance of the Belmont Red when compared with Simmental and Hereford breeds. Studies by Seifert (1971) and Frisch and O'Neill (1998*b*)

indicated that the Belmont Red was more resistant to ticks than *Bos taurus* but not as resistant as *Bos indicus* breeds. Spickett *et al.* (1989) conducted a study comparing resistance of ticks by Nguni, Bonsmara and Hereford breeds. The result showed Nguni to have a higher potential to develop tick resistance than the other two breeds. They also found that tick resistance varies within breeds and this suggests that culling or selection would improve tick resistance within the breed.

Scholtz *et al.* (1991) conducted a study comparing the effects of tick infestation on the productivity of Hereford, Bonsmara and Nguni. The results indicated that, with no dipping, Hereford weaned calves with significantly lower body weight than those of the other two breeds. There were no significant differences between the weaning weights of calves from the undipped Bonsmara and Nguni cows. Calves from the Nguni dipped group were 7 kg heavier than those from undipped Nguni group, but the difference was not significant. When all animals were dipped the Bonsmara weaned heavier calves than the two breeds and the difference was significant. Ticks had a minor effect on the productivity of Nguni cows as measured by the weaning weights of their calves but the effect was severe on the productivity of Hereford cows and intermediate for Bonsmara (Scholtz *et al*., 1991).

Scholtz *et al.* (1991) conducted a study on tick counts dividing the ticks into one-host (*Boophilus decoloratus*) and multi-host group (*A. hebraeum*; *Hyalomma truncatum*; *Rhipicephalus evertsi evertsi*). They found that the incidence of multi-host engorged female ticks was very low and averaged 0.8, 0.8 and 0.4 ticks per count on Hereford, Bonsmara and Nguni respectively. Very high infestation of *B. decoloratus* was experienced during the months of October, November and December. The counts were, therefore, separated into a peak infestation period (October, November, and December) and infestation over the whole suckling period (October-April). Breed had a marked effect on tick infestation with Herefords being most susceptible and Nguni the least. They also found that there were no differences in tick infestation level between lactating cows and bulls. Sub-fertile heifers, however, had much higher tick burdens than lactating cows and bulls, suggesting that the heifers were either highly susceptible to tick infestation because of their physiological status or that they did not conceive because of high tick burdens.

2.4. Modes of tick infestation

Ticks prefer to attach themselves to the areas of the body where the hide is thin and less exposed to solar radiation, and also to areas where the skin movement to impulse is less. The skin reaction of cattle to external parasites varies according to the degree of adaptability. Rigorous skin movement at the slightest irritation in adapted cattle serve as an effective repellent. This type of reaction is always found in cattle with thick hides and short, smooth and well developed subcutaneous muscle development. The subcutaneous muscle lies transversely across the hide like whip wales. This characteristic is dominant and is inherited in cattle such as *Bos indicus x Bos taurus* crossbred cattle which have thick hides with short glossy hair and well developed subcutaneous muscles. The thick vascular hide of adapted cattle freely admits exudation and clotting of blood at the site of insect and tick bites. This reduces the ability of parasites to attack the skin effectively (Bonsma, 1981). The woolly-coated animal becomes hyperthermic on hot days and, as a result of stress, stands or lies in the shade where the incidence of ticks is very much higher than in the open (Bonsma, 1981).

Knowledge of the mode of disease infection and host response is essential to comprehend the complexity of selecting for disease resistance. A simplistic explanation is given here. First, the pathogen must be present in the host's environment. The pathogen must penetrate host cell barriers in sufficient numbers, attack target cells and replicate. Sub-clinical or clinical expression of the disease is dependent on the pathogen's virulence and the interaction between pathogen and host characteristics (Snowder, 2006).

There are three immune defences against infection: natural, innate, and acquired immunity. All three must be present and functioning to keep the animal healthy. Natural immunity is the first barrier and is comprised of skin, hair, mucous membranes, secretions (tears, urine, stomach, saliva, mucous, skin secretions, etc.), grooming behaviour (licking, dust rolling, tail swishing, etc.) and favourable microorganisms that compete directly or indirectly against pathogens. There are also nutritional components to natural immunity (Snowder, 2006). Dehydration and malnutrition can decrease natural secretions making some tissues more susceptible to

infection. Vitamin and mineral deficiencies result in suppressed immune systems. There are genetic components to natural immunity that can be identified. For example, some pigs are fully resistant to bacteria-induced diarrhoea (E. coli) because they lack an intestinal cell receptor for the bacteria to attach to (Gibbons *et al.,* 1977). Hair/wool length, skin secretions, and hide thickness can affect fly infestation of livestock.

The innate immunity refers to the immune system one is born with and is the initial response by the body to eliminate microbes and prevent infection. It involves white blood cells (natural killer cells, neutrophils, eosinophils, monocytes, and macrophages), complement proteins (C1 - C4) that adhere to pathogens, and cytokines (interferons and chemokines) that attract immune cells to the site of infection. The innate immune system searches for antigens (bacteria, fungi, and viruses). When an antigen is discovered, the innate system attacks it or "elicits" inflammation to attract immune cells. The innate system is not specific to any one type of pathogen and has no memory of previous exposure to a pathogen or antigen. Breed differences in the innate immune system have been reported. A higher haemolytic complement activity in *Bos indicus* breeds is associated with their higher resistance to tick infestation and subsequent tick borne diseases when compared to *Bos taurus* breeds (Wambura *et al.,* 1998).

The acquired immune system is developed from previous exposure to pathogens or vaccines and can recognize pathogens previously exposed to. Acquired immunity is antigen specific. There are two types of acquired immunity: the cell mediated immunity which is comprised of immune cells that directly attack pathogen infected cells, and the humoral immunity which is made up of antibodies (specific immune proteins) that are directed at the pathogens themselves. The acquired immune system is comprised of T and B cells, which are specialized white blood cells. The T cells destroy pathogen-infected cells whilst the B cells develop into specific antibody producing cells (Snowder, 2006).

Acquired immunity occurs in two forms: passive and active. Passive or maternal immunity is passed from the cow to the calf via colostrum containing high levels of antibodies. Passive immunity is temporary. Disease resistance of very young calves is

highly dependent on passive immunity. This type of protection is short lived because soon after birth, the calf's intestinal tract has a significant reduction in its ability to absorb immunoglobulins (antibodies), and the cow's production of colostrums decreases as lactation progresses. Half of the colostrum antibodies absorbed by the calf will be excreted, broken down or absorbed at 8 to 16 days postpartum and most will be gone by 30 to 60 days postpartum (Besser *et al*., 1988). There are genetic components of passive immunity in cattle and, recently, DNA markers associated with failure of passive immunity have been reported (Laegreid *et al*., 2002; Clawson *et al*.,2004). Therefore, it is important that the calf's own immune system (active immune system) develops at an early age to produce cell-mediated immunity and antibodies in response to antigens and vaccines to take over when passive or maternal immunity diminishes (Snowder, 2006).

An understanding of immune systems forms a base for genetic selection programs for disease resistance. For example, if the breeding goal is to reduce bacterial diarrhoea in young calves, then selection traits might include the dam's genetic potential for producing specific colostrum antibodies (passive immunity) and the calf's genetic potential for developing an innate and acquired immune system early in life that responds to the diarrhoea causing pathogen (Snowder, 2006).

2.5. Genetic relationship between tick counts and other traits

Previous studies on genetic relationship between tick counts with other traits are limited. Genetic correlations between tick counts and weights and gains of cattle measured in the presence of those parasites and other environmental stresses are, on average, mostly negligible to weakly positive (unfavorable) (Davis, 1993) except for low negative, (i.e. favorable) correlations of tick count with live weight at 400 days (-0.27) and dry season gain (-0.22) (Table 2.1). Estimates from individual studies were variable and this variation tended to be related to the level of resistance of the breed under study (Mackinnon *et al*., 1990a) although given the imprecise nature of some estimates, it is difficult to be certain of the trend. The separate breed parameters suggest that selection for growth in breeds with relatively high levels of resistance will lead to no change or increases parasite counts, i.e. reduced resistance, while

selection for growth in a less resistant genotype would result in reduced parasites counts, i.e. increased resistance.

Burns *et al.* (1997) found that age at weaning was significantly correlated with tick burdens with older calves having greater tick counts. Sularsas (1985) observed that an association between tick counts and age was influenced by seasonal tick burdens, tick counts increased with age only at times of high levels of tick infestation. This is in conflict with what Burns *et al.* (1997) found in their study; they found that an association between age and tick count was detected even though tick burdens were never very severe. This association between age at weaning and tick count may be explained by younger calves still receiving maternal antibodies from their dams compared with older calves, which would be partially weaned before weaning and therefore largely independent of their dam's milk supply.

Genetic correlations between tick and worm egg counts (r_g=0.30), and tick burdens were also influenced by the age of the dam with heifers from young dams having lower tick burdens than those of mature or old dams (Burns *et al*., 1997).

Table 2.1. Average of published genetic correlations between traits measured on Bos indicus and zebu cross animals in northern Australia expressed as a percentage (rgX100). Adapted from Davis (1993).

Trait[1]	WW	W400	PW	PO	SC	TK
WW		0.90	0.94	-0.24	0.29	0.02
W400			0.80	0.30	0.43	-0.01
PW				0.08	0.09	-0.05
PO					0.09	0.15
SC						0.02

[1]WW = weaning weight; W400 = weight at 400 days; PW = pre-weaning gain; PO = post-weaning gain; SC = scrotal circumference; TK = tick count.

2.6. Conclusions

It is evident from this literature review that although conventional methods have been extensively utilized to control ticks in livestock production systems, there is a need to explore genetic variation amongst breeds for tick resistance and utilize this information in genetic selection programs. Indigenous livestock breeds of South Africa such as Nguni and Bonsmara cattle are known to be adaptable to the harsh environmental conditions, as well as tick tolerant and disease resistant as compared to their exotic counterparts. However estimates of genetic parameters for these traits to support these contentions have been limited due to lack of data. The current study will explore genetic variability for tick tolerance as an indicator of tick resistance within the Bonsmara cattle breed.

Chapter 3

GENETIC PARAMETER ESTIMATES FOR TICK RESISTANCE IN BONSMARA CATTLE[#]

3.1. Introduction

Tick-borne diseases are one of the major constraints to livestock production throughout the tropics and sub-tropics. Losses in livestock production due to external parasites have long been a major concern to livestock producers in the tropics and subtropics (Seifert, 1984). One million cattle are estimated to have died of East Coast fever in sub-Saharan Africa during 1989 alone (Mukhebi *et al*., 1992). In South Africa, tick-borne diseases have been estimated to cost the livestock industry about R70 to R200 million per year (Bigalke, 1980).

Various tick-borne disease control methods have been employed in South Africa (Bigalke *et al*., 1976; De Vos, 1979; Purnell and Schroder, 1984). Historically, the earliest tick control trials with dipping agents in South Africa started in 1889. These trials were prompted by the discovery in the USA in 1893 that ticks transmit the causal piroplasm of redwater in cattle (Hayward, 1981). Increase in acaricides prices and drug resistance pose challenges in the application of these methods because the application processes are associated with an increase in input costs, while it takes time to develop new drugs.

Hayward (1981) stated that the best solution to high acaricide prices and resistance to drugs might be the identification of naturally resistant breeds and the encouragement of their use. Natural disease resistance refers to the inherent capacity of an animal to resist disease when exposed to pathogens, without prior exposure or immunization (Adams and Templeton, 1998). Utech *et al*. (1978) defined tick resistance as the ability of cattle to limit the number of ticks that survive to maturity.

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Although some of the observed variation in natural tick resistance is related to environmental factors, a significant component of variation in natural disease resistance appears to be of genetic origin (Adams and Templeton, 1998). Several studies have been conducted on genetic determination of tick resistance (Utech *et al*., 1978; Spickett *et al*., 1989; Rechav *et al*., 1990). Tick resistance has been shown to be heritable (Hewetson, 1972). Davis (1993) reported a heritability estimate of 34% for tick resistance, indicating that genetic improvement through selection should be effective. Information on resistance status within the various breeds of cattle is needed to provide a basis for selection, by either breeding from animals with resistance, or culling cattle with low tick resistance, or both.

The South African National Beef Recording and Improvement Scheme of the Agricultural Research Council initiated a tick count data collection pilot project in 1993 in conjunction with the South African Bonsmara Cattle Breeders Society. The short-term objective of the project was to collect data that could be used as a management tool (i.e. to determine when to dip the animals) and for phenotypic selection. The long-term objective was to collect data that will ultimately form the basis for the development and implementation of a genetic improvement program for tick resistance. The primary objective of the current study is therefore to assess the level of genetic variation for tick resistance in South African Bonsmara cattle by estimating genetic parameters for tick count. The secondary objective is to evaluate the effect of the level of tick infestation on the genetic parameter estimates for tick count.

3.2. Materials and Methods

Data used in the current study were obtained from the National Beef Recording and Improvement Scheme (NBRIS). The data included tick count records on Bonsmara cattle from 10 stud herds that participated in the tick count data collection pilot project. The breeders participating in the project were located in Limpopo, North-West and Western Cape provinces of South Africa and their participation in the project ranged from one to nine years (1993 to 2005). Participants in the project (i.e. the breeders) were responsible for collection of tick count data following the guidelines of the NBRIS (NBRIS, 2008). Briefly, the guidelines states that animals

should not be dipped during the testing period. In case were dipping is necessary tick count records must be collected prior to dipping. A minimum period of three weeks must be allowed between two dates of tick counting. Frisch and O'Neill (1998b) and Burns *et al*. (1997) used the same period between counts in their studies on tick resistance.

The guidelines further states that the same person should record tick count in a given herd and date. It is also important that the person recording tick counts be experienced or under the supervision of an experienced person. In the study by Seifert (1971) it was reported that errors due to observer were shown to be heterogeneous and were greater when a temporary engaged novice made duplicate counts than when they were made by an experienced person.

The following information was recorded at tick counting: animal identification, tick count, and the sex of animal. In some herds tick counts were taken throughout the year while others concentrated on certain months (i.e. when ticks were prevalent). The original data set consisted of 11 280 repeated measurements of tick count from 1 176 animals. Other pieces of information necessary for genetic parameter estimation such as national animal identification, date of birth, and pedigree information were obtained from Integrated Registration and Genetic Information System (INTERGIS) database.

Data were edited to exclude tick count records (1) on animals younger than 250 days, and (2) extreme tick count records i.e. more than five standard deviations above the mean. Furthermore, contemporary groups (the concatenation of herd, sex and year, month and day of tick counts) with less than 5 animals were excluded. Preliminary analysis of the data showed that variation in tick counts among animals in a contemporary group depended on the level of tick infestation. That is, the coefficient of variation in tick count increased with the mean tick count in a contemporary group. In the study by Burrow (2001) a minimum of 15 ticks per side was required for the records to be considered useful. In the current study, to investigate the effect of level of tick infestation (i.e. mean tick count in a contemporary group) on genetic parameter estimates, a total of seven data sets were created. The seven data sets included records from contemporary groups with mean tick count ≥ 5 (Data 1), ≥ 10 (Data 2), ≥ 15

(Data 3), ≥ 20 (Data 4), ≥ 25 (Data 5), ≥ 30 (Data 6), and ≥ 35 (Data 7). The number of contemporary groups per data set ranged from 36 to 210. Visual inspection of the frequency distributions revealed that tick count had a non-Gaussian or non-normal distribution. Thus, the data were normalized using a Box-Cox family of power transformations (Box & Cox, 1964). The skewness for data sets 1 to 7 before (after) transformation was as follows: 2.689 (-0.071), 2.333 (-0.193), 1.844 (-0.283), 1.565 (-0.364), 1.287 (-0.670), 1.130 (-0.919) and 0.846 (-1.209). The skewness of 0 indicates that the distribution is symmetric. In general, the transformation enhanced the normality of the data except for data set 7. The Box-Cox transformation is given by $y' = (y^k - 1)/k$ for $k \neq 0$ or $y' = \log(y)$ for $k = 0$; where y and y' are the raw and transformed tick count data, respectively. The maximum likelihood estimate of the parameter k was obtained using an algorithm proposed by Hyde (1999). Silva *et al*. (2006) and Gasparin *et al*. (2006) also used a Box-Cox transformation in the analysis of faecal egg count in Angus cattle and tick count in experimental population, respectively. Handlesman (2002) pointed out that a power transformation performs better than logarithmic transformation in normalising sperm concentration data. It should be noted that data transformation does not guarantee that the transformed data are normal. However, transformation may improve properties of estimates, predictions, and inferences (Sonstegard *et al*., 2006). All results are presented in the transformed scale and all references to tick counts or scores imply transformed counts unless specific reference is made to raw data.

A repeatability animal model was used to analyse the data. The model included the fixed effects of the contemporary group and age of the animal at tick count data collection and random additive genetic, permanent environment and residual effects. The matrix representation of the model equation is as follows:

$$y = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wc} + e$$

where **y** is a vector of normalised tick count observations, **b** is a vector of fixed effects of contemporary group and age (fitted as a quadratic regression), **a** is a vector of random direct additive genetic effects of the animals, **c** is a vector of random permanent environmental effects of the animals, **e** is a vector of random residual

effects unique to each observation and **X**, **Z** and **W** are known incidence matrices relating the fixed and random effects, respectively, to observations in **y**. The random effects were assumed to be normally distributed with the following first and second moments:

$$E\begin{bmatrix} a \\ c \\ e \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix}$$

and

$$\text{Var}\begin{bmatrix} a \\ c \\ e \end{bmatrix} = \begin{bmatrix} A\sigma_a^2 & 0 & 0 \\ 0 & I_c\sigma_c^2 & 0 \\ 0 & 0 & I_e\sigma_e^2 \end{bmatrix}$$

where A is the numerator relationship matrix, I_c is an identity matrix of the order equal to the number of animals with records, I_e is an identity matrix of the order equal to the number of records. The variance components σ_a^2 , σ_c^2 and σ_e^2 are the direct additive genetic, permanent environment and residual variances respectively.

Variance components and their corresponding ratios to the phenotypic variance were estimated using the Variance Components Estimation version 6.0.2 package (VCE6) of Groeneveld *et al*. (2008). The VCE estimates variance components by the REML procedure use analytical gradients and Gibbs sampling. The method of analytical gradient was used in the current study. The software package for multivariate Prediction and Estimation (PEST Version 4.2) of Groeneveld *et al*. (1990) was used to format the data for VCE6. A three-generation pedigree was built around the data for each of the seven data sets considered. The pedigrees ranged from 1 583 to 2 747 animals in different data sets.

3.3. Results and discussions

A comprehensive summary statistics of the final data sets is presented in Table 3.1. The smallest data set included records on 492 animals while the largest data set included records on 1 104 animals. The number of sires and dams in the different data sets ranged from 75 to 130 and 364 to 754, respectively. The minimum and maximum number of herds in the different data sets was 6 and 10 respectively. The variation in the age of the animals at tick count data collection in the current study was considerable (i.e. from 250 to 5 521 days). The average age of the animals ranged from 864 to 964 days in the different data sets. Corbet *et al*. (2006) analysed tick count data from animals that had a narrower age range than considered in the current study (i.e. from 360 to 2 920 days). Burrow (2001) estimated genetic parameters for tick count using monthly records collected from weaning (at six month) to 18 months of age.

Table 3.1. Summary statistics of the different data sets[1]

Item	Number of records	Number of animals	Mean	Std Dev	CV (%)
	Data set 1: Mean tick count ≥5				
Tick count	7 671	1137	18.60 (2.56)	19.95 (0.94)	107.26 (36.72)
Age of animals, days			951.89	676.67	71.09
	Data set 2: Mean tick count ≥10				
Tick count	5 333	1025	23.58 (2.87)	21.87 (0.85)	92.75 (29.62)
Age of animals, days			963.87	708.89	73.55
	Data set 3: Mean tick count ≥15				
Tick count	3 109	903	31.66 (3.22)	24.89 (0.76)	78.62 (23.60)
Age of animals, days			922.37	702.40	76.15
	Data set 4: Mean tick count ≥20				
Tick count	2 211	762	37.67 (3.43)	26.58 (0.70)	70.56 (20.41)
Age of animals, days			960.68	729.61	75.95
	Data set 5: Mean tick count ≥25				
Tick count	1 563	750	44.32 (3.61)	28.21 (0.68)	63.65 (18.84)
Age of animals, days			908.12	754.44	83.08
	Data set 6: Mean tick count ≥30				
Tick count	1 246	713	48.56 (3.71)	29.20 (0.67)	60.13 (18.06)
Age of animals, days			863.91	761.19	88.11
	Data set 7: Mean tick count ≥35				
Tick count	762	514	58.67 (3.94)	30.66 (0.60)	52.26 (15.23)
Age of animals, days			934.48	904.76	96.82

[1]The numbers in brackets are the transformed tick counts.

The untransformed tick count for all the respective data sets were similar and ranged from 0 to 155 (data not shown) while untransformed averages ranged from 18.60 to 58.67 (Table 3.1). The level of tick infestation in the current study was comparable to what has been found in other studies. Corbet *et al*. (2006) found a mean of 37 ticks from 622 animals with tick count ranging from 1 to 150. Turner and Short (1972) compared tick infestation of different breeds and the mean tick count per side for the Afrikaner and Brahman breeds on natural infestation was 20-30 ticks whereas the Shorthorn breed carried 75-100 ticks per side. In the study by Regitano *et al.* (2006) the mean tick count from artificial infestation was 21.52.

The estimates of variance components and corresponding ratios for transformed tick count are presented in Table 3.2. The additive genetic variance increased with an increase in the mean tick count per contemporary group to a maximum at ≥30 after which a sharp decline was observed. The lowest estimate of genetic variance was observed for data with mean tick count of ≥35. The drastic decline in the additive genetic variance for Data set 7 (or mean tick count of ≥35) could be ascribed to the low number of animals in the data set. Variances for permanent environment decreased with an increase in mean tick count. This was expected since the number of records per animal decreased with an increase in the mean tick count per contemporary group (Table 3.1). Thus, the amount of information available to estimate the permanent environmental effect was limited in data sets with a high mean tick count per contemporary group. Phenotypic and residual variances decreased with an increase in mean level of tick count per contemporary group.

Table 3.2. Estimates of variance components for normalised tick count and their ratios (±s.e.) to the phenotypic variance

Parameter	Data Set (Mean tick count)						
	≥5	≥10	≥15	≥20	≥25	≥30	≥35
σ_a^2	0.0322	0.0322	0.0412	0.0581	0.0770	0.0763	0.0129
σ_c^2	0.0013	0.0021	0.0116	0.0271	0.0009	0.0000	0.0047
σ_e^2	0.6363	0.4693	0.4157	0.3796	0.3867	0.3820	0.3959
σ_p^2	0.6698	0.5036	0.4685	0.4648	0.4647	0.4583	0.4136
h^2	0.05±0.01	0.06±0.02	0.09±0.03	0.13±0.04	0.17±0.05	0.17±0.04	0.03±0.06
c^2	0.00±0.01	0.00±0.01	0.03±0.02	0.06±0.03	0.00±0.04	0.00±0.00	0.01±0.08
e^2	0.95±0.01	0.93±0.01	0.89±0.03	0.82±0.03	0.83±0.03	0.83±0.04	0.96±0.06

σ_a^2 - direct additive genetic variance, σ_c^2 - permanent environmental variance, σ_e^2 - residual variance, σ_p^2 - phenotypic variance, h^2 - heritability or ratio of the direct additive genetic variance to the phenotypic variance, c^2 - ratio of the permanent environmental variance to the phenotypic variance, e^2 - ratio of the residual variance to the phenotypic variance.

The heritability estimates for the different data sets are presented in Table 3.2. These estimates increased with the mean tick count per contemporary group and stabilised when the mean tick count per contemporary group was ≥25 and <31. The estimates ranged from 0.05 to 0.17 for data with mean tick count of ≥5 and ≥30, respectively. The low estimate of heritability for data with mean tick count of ≥35 corresponded with its lowest genetic variance. These results also indicate that as more cattle are infested with ticks, increased levels of genetic variation between cattle would be observed. Heritability estimates from this study are lower compared to results from other similar studies. A study on Belmont Red cattle in Australia reported a heritability estimate of log-transformed tick count to be 0.42 (Burrow, 2001). Seifert (1971) evaluated variations in resistance of cattle tick between and within breeds of cattle and reported a heritability of 0.48 for the Shorthorn x Hereford line and an estimate of 0.82 for Zebu crossbreds. Davis (1993) reported a heritability estimate of 0.34 for log-transformed tick counts. Wharton *et al.* (1970) reported heritability estimates of 0.39 for dam-calf correlations and 0.49 for full sib correlations. In the same study heritability was further estimated based on winter and summer seasons. The heritability estimates for dam-calf were 0.42 and 0.07 whereas for full sibs were 0.64 and -0.17 for summer and winter respectively. Separating data according to

season resulted in increased heritability in summer and a low to zero heritability in winter. It was suggested that this might be due to either seasonal change in the intensity of expression of component of host resistance or a seasonal change in the sensitivity of ticks to some mechanism of host resistance. It is important to note that, at the time of the year when discrimination is poor, it is not a matter of the same variation in inherent susceptibility being obscured by some extra error of variation: the total variation (animal+error) is reduced at this time and inherent differences in resistance produce small effects. No suggestion was offered as to the cause of the seasonal effect on discrimination. It is therefore important that the existence of the effect should be defined under any circumstances where research into resistance, or selection for resistance, may be undertaken.

Wharton *et al.* (1970) suggested that the high heritability estimates from full-sib correlations compared to those from dam-calf correlations may due to inflation by maternal effects but apart from the possibility of short-lived transfer of passive immunity, the mechanism of maternal effects are not obvious.

Henshall (2004) reported heritability an estimate of 0.41 for transformed data and emphasized that tick count should be recorded at the time when animals have had sufficient exposure to ticks to ensure that resistance has been acquired. In the study by Hewetson (1968) heritability estimates from 5 levels of artificial infestation of sires increased from 0.28 to 0.42 between the fourth and fifth infestation, whereas there was zero heritability at the first infestation. Hewetson (1968) emphasised the importance of acquired resistance in genetic analyses of tick counts.

The animal permanent environment variance ratios were lower for all ranges of mean tick counts considered in the current study, thus indicating that permanent environment had little or no effect on variation of tick counts. It was mentionable only in data set 4 (i.e. mean tick count of ≥20) where it contributed only 6% of the total variation. In the study by Burrow (2001) the animal permanent environment accounted for 18% of the phenotypic variation, which was higher than in the present study. The residual ratios were higher for data sets considered in the current study indicating that there is still a lot of unknown phenotypic variation that was not

accounted for by the effects in the model. The high proportion of residual variation observed in the current study could be due to differences in the data collection process among the different participating herds. Standardisation of the tick count data collection process should be considered in the National Beef Recording and Improvement Scheme to obtain data more suitable to estimating the heritability of tick counts.

3.4. Conclusions

The results from the current study indicate that sufficient levels of genetic variation for tick count exists in the South African Bonsmara population. Thus, selection for tick resistance using estimated breeding values for tick counts is a viable option even though genetic progress may be slow. Special attention should be given to the data collection process to enhance the quality of the data. It is recommended that records from contemporary groups with mean tick count of at least 20 ticks per animal should be considered for genetic evaluation purposes. It is therefore important that under natural infestation, tick count recording be done at the time when tick population is high.

Chapter 4

GENERAL CONCLUSIONS AND RECOMMENDATIONS

Genetic improvement of beef cattle production efficiency and profitability requires a comprehensive national genetic evaluation system. That is, breeding values should be estimated for all traits of economic importance. While the current national beef genetic evaluation system in South Africa is quite advanced, breeding values for adaptability traits or their indicators are not available. In order to bridge this gap, the current study investigated the possibility of developing a genetic selection tool for tick resistance using tick count as an indicator trait.

A proper implementation of a national genetic evaluation requires the development of an operational statistical model and estimation of genetic parameters for the traits under consideration. The operational model for genetic analysis of tick count was successfully developed in the current study. Furthermore, genetic parameters (genetic and environmental variances) were also successfully estimated. The additive genetic variance obtained in the current study indicates that sufficient genetic variation for tick count exists in the Bonsmara cattle breed.

The heritability estimate for tick count was found to be low. This estimate of heritability indicates that selection for tick count would lead to slow genetic progress in resistance to ticks in Bonsmara cattle. It is important to note that large amount of tick count data will be required to obtained accurate estimates of breeding values. It is therefore recommended that a new breeding value for tick count be implemented in the national beef genetic evaluation system. Initial implementation should commence with the Bonsmara breed since sufficient tick count data for breeding value estimation is already available.

Selection for tick resistance through the use of breeding values for tick count will be associated with correlated response in traits that are genetically correlated to tick count. To assess the correlated response that could result from selection for tick count, knowledge of genetic correlations between tick count and other traits of economic importance will be required. It is therefore important that future research focus on

obtaining estimates of genetic correlations between tick count and other traits of economic importance.

The current investigation also evaluated the impact of tick infestation on estimates of genetic parameters. The highest estimate of heritability was obtained when the tick infestation was higher than 20 ticks per animal. These results indicate that individual animal genetic differences for tick load are only expressed at high levels of tick infestations. It is therefore recommended that records from contemporary groups with mean tick count of at least 20 ticks per animal should be considered for genetic evaluation purposes.

The high proportion of residual variation observed in the current study could be due to differences in the data collection process as among the different participating herds. Standardisation of the tick count data collection process should be considered in the National Beef Recording and Improvement Scheme to obtain data more suitable to estimating the heritability of tick counts.

Chapter 5

REFERENCES

Adams, L.G. and Templeton, J.W. 1998. Genetic resistance to bacterial diseases of animals. *Scientific and Technical Review.* 17: 200-219.

Besser, T.E., McGuire, T.C., Gay, C.C. and Pritchett, L.C. 1988. Transfer of functional immunoglobulin G (IgG) antibody into the gastrointestinal tract accounts for IgG clearance in calves. *Journal of Virology.* 62: 2234-2237.

Bigalke, R.D., De Vos, A.J. and Barrowman, P.P. 1976. The control of some tick-borne disease in S.A. Bulletin de l' office International des Epizooties. 86: 89-100.

Bigalke, R.D. 1980. The control of ticks and tick-bone disease of cattle in South Africa. *Zimbambwe Veterinary Journal.* 11: 20-21.

Bonsma, J.C. 1981. Breeding tick repellent cattle. In Whitehead, G.B. and Gibson, J.D (eds), Tick biology and control. Tick Research Unit, Rhodes University, Grahamstown South Africa. pp 67-69.

Box, G.E.P. and Cox, D.R. 1964. An analysis of transformations. *Journal of Royal Statistical Society. B.* 26: 211-246.

Burns, B.M., Reid, D.J. and Taylor, J.F. 1997. An evaluation of growth adaptive traits of different cattle genotypes in subtropical environment. *Australian Journal of Experimental Agriculture.* 37: 399-405. doi: 10.107/EA 96092.

Burrow, H.M. 2001. Variances and covariances between productive and adaptive traits and temperament in a composite breed of tropical beef cattle. *Livestock Production Science.* 70: 213-233.

Clawson, M.L., Heaton, M.P., Chitko-McKown, C.G., Fox, J.M., Smith, T.P.L., Snelling, W.M., Keele, J.W. and Laegreid, W.W. 2004. Beta-2 microglobulin haplotypes in the U.S. beef cattle and association with failure of passive transfer in newborn calves. *Mammalian Genome*.15: 227-236.

Corbert, N.J., Sherpherd, R.K., Burrow H.M., Prayaga, K.C., Van der Westhuizen, J. and Bosma, D.J. 2006. Evaluation of Bonsmara and Belmont Red cattle in South Africa 2. Genetic parameters for growth and fertility. *Australian Journal of Experimental Agriculture.* 46: 213-223.

Davis, P. 1993. Genetic parameters for tropical beef cattle in Northern Australia: A Review. *Australian Journal of Agricultural Research.* 44: 179-198.

Detilleux, J. 2001. Genetic improvement of resistance to infectious diseases in livestock. *Journal of Dairy Science*. 84 (E.suppl.): E39-46.

De Vos, A.J. 1979. Epidemiology of bovine babesiosis in South Africa. *Onderstepoort Journal of Veterinary Research.* 50: 357-362.

Furman, D.P. and. Loomis, E.C. 1984. The Ticks of California. University of California Publications, Bulletin of the California Insect Survey, Vol. 25. University of California Press, California.

Frisch, J.E. and O'Neill, C.J. 1998*b.* Comparative evaluation of beef cattle breeds of African, European and Indian origins. 2. Resistance to cattle and gastrointernal nematodes. *Animal Science.* 67: 39-48.

Gasparin, G., Miyata, M., Coutinho, L.L., Martinez, M.L., Machado, M.A., Silva, M.V.G.B., Campos, A.L., Sonstegard, T.S. and Regitano, L.C.A. 2006. Detection of a QTL associated with tick resistance on bovine chromosome 7 (BTA7) using a F_2 experimental population. *8th World Congress on Genetics Applied to Livestock Production*, August 13-18, Belo Horizonte, MG, Brasil.

Green, P.E. and Burton, D.W. 1985. Identifying ticks of veterinary interest. *Queensland Agricultural Journal.* January-February .

Groeneveld, E., Kovac, M. and Mielenz, N. 2008. VCE User's Guide and Reference Manual Version 6.0. Institute of Farm Animal Genetics, Mariensee, Germany.

Groeneveld, E., Kovac, M. and Wavy, T. 1990. Pest software package for multivariate Prediction and estimation version 4.2. Department of Animal Science, University of Illinois, USA.

Gibbons, R.A., Selwood, R., Burrows, M. and Hunter, P.A. 1977. Inheritance of resistance to neonatal *Escherichia* diarrhea in the pig: Examination of the genetic system. *Theoretical and Applied Genetics*. 55: 65-70.

Handlesman, D.J. 2002. Optimal power transformations for analysis of sperm concentration and other semen variables. *Journal Andrology*. 23: 629-634.

Hayward, S. 1981. Opening address. In: Whitehead, G.B. and Gibson, J.D (eds), Tick biology and control. Tick Research Unit, Rhodes University, Grahamstown South Africa. pp xiii –xiv.

Henshall, J.M. 2004. A genetic analysis of parasites resistance traits in a tropically adapted line of *Bos taurus. Australian Journal of Agricultural Research*. 55: 1109-1116.

Hewetson, R.W. 1968. Resistance of cattle to tick, *Boophilus microplus* II. The inheritance of resistance to experimental infestations. *Australian Journal of Agricultural Research* 19: 497-505.

Hewetson, R.W. 1979. Results of selection for cattle tick resistance in cattle. *Proceedings of the 56th Annual Conference of Australian Veterinary Association*. pp 91-93.

Hewetson, R.W. 1972. The inheritance of resistance by cattle to cattle tick. *Australia veterinary Journal.* 48: 299-303.

Hyde, S. 1999. Likelihood based inference on the Box-Cox family of transformations: SAS and MATLAB programs. MSc thesis, Montana State University, USA.

Laegreid, W.W., Heaton, M.P., Keen, J.E., Grosse, W.M., Chitko-McKown, C.G., Smith, T.P.L., Keele, J.W.C., Bennet, J.L. and Besser, T.E. 2002. Association of bovine neonatal Fc receptor alphacahin(FCGRT) haplotypes with serum IgG concentration in newborn calves*. Mammalian Genome.* 13: 704-710.

Latif, A.A. 2006. Sustainable control methods for ticks and tick-borne diseases in Africa .*Veterinary Research Laboratory,* Harare, Zimbabwe.

Lawrence, J.A., Fogging, C.M. and Norval, R.A.I. 1980. The effects of war on the control of diseases of livestock in Rhodesia(Zimbabwe). *Veterinary Record* 107: 82-85.

Mackinnon, M.J, Meyer, K. and Hetzel, D.J.S. 1990a. Genetic variation and covariation for growth, parasite resistance and heat tolerance in tropical cattle. *Livestock Production Science*. 27: 105-22.

Mukhebi A.W., Perry, B.D and Kruska, R. 1992. Estimated economics of theileriosis control in Africa. *Preventative Veterinary Medicine.* 12: 73-85.

National Beef Recording and Improvement Scheme. Rules and Guidelines. V2008. 31 December 2008. Irene Regional Office Private Bag X2, Irene 0062. Tel. (012) 672 9081.

Norval**,** R. A. I. and I.G. Horak. 1994**.** Vectors**:** Ticks**.** In Coetzer**,** J.A.W. and R.C. Tustin**,** editors. (Eds). Infectious diseases of livestock Vol**.** 1**.** pp. 3–42.

Purnell, R.E. and Schroder, J. 1984. Herd Prophylatis of tick-borne diseases in susceptible cattle in South Africa: *Proceedings of XIIIth World Congress on Diseases of Cattle,* 17-24 Sepetember, Durban, pp 488-493.

Rajput, Z.I., Hu. S., Chen W., Arijo, A.G. and Xiao C. 2006. Importance of ticks and their chemical and immunilogical control in livestock. *J Zhejiang University of Science B* 7: 912-921.

Rechav, Y.,Dauth, J. And Els, D.A. 1990.Resistance of Brahman and Simmentaler cattle to Southern African ticks. *Ondersteproot Journal of Veterinary Research.* 57: 7-12.

Rechav, Y., and Kostrzewski, M.W. 1991. Relative resistance of six cattle breeds to the tick *Boophilus decoloratus* in South Africa. *Ondersteproot Journal of Veterinary Research.* 58: 181-186.

Regitano, L.C.A., Martinez, M.L. and Machado, M.A. 2006. Molecular aspects of bovine tropical adaptation. *8th World Congress on Genetics Applied to Livestock Production*, August 13-18, Belo Horizonte, MG, Brasil.

Riek, R.F. 1962. Studies on the reaction of animals to infestation with ticks. VI resistance of cattle to infestation with the tick *Boophilus microplus*. *Australian Journal of Agricultural Research.* 13: 532-551.

Roberts, J.A. 1968a. Acquisition by the host of resistance to the cattle tick, *Boophilus microplus* (Canestrini). *Journal of Parasitology.* 54: 657-662.

Roberts, J.A. 1968b. Resistance of cattle to the tick *Boophilus microplus* (Canestrni). II. Stages of the parasite against which resistance is manifest. *Journal of Parasitology.* 54: 667-673.

Saeed, A.M., Ward, P.N., Light, D., Durkin, J.W. and Wilson, R.T. 1987. Characterization of Kenana cattle at Um Banein, Sudan. International Livestock Centre for Africa Research Report 16.Addis Ababa. Ethiopia.46 pp.

Scholtz, M.M., Spickett, A.M., Lombard, P.E. and Enslin, C.B. 1991. The effect of tick infestation on the productivity of cows of three breeds of cattle. *Onderstepoort Journal of Veterinary Research.* 58: 71-74.

Seifert, G.W. 1984a. Research and practical experience in selection for resistance to the cattle tick (*Bos microplus*) and gastrointestinal helminths in northern Australia beef cattle. *Proceedings of World Sheep Beef cattle Breed. Pretoria, South Africa.* pp. 149-159.

Seifert, G.W. 1971. Variations between and within breeds of cattle in resistance to field infestation on the cattle tick. *Boophilus microplus*. *Australian Journal of Agricultural Research.* 22: 159-168.

Seifert, G.W. 1984b. Selection of beef cattle in Northern Australia for resistance to the cattle tick. (*Boophilus microplus*); research and application. *Preventative Veterinary Medicine.* 2: 553-558.

Silva, M.V.G.B., Van Tassell, C.P., Sonstegard, T.S., Cobuci, J.A. and Gasbarre, L. 2006. Estimates of genetic parameters using random regression models for gastrointestinal Helminthoid data in Angus cattle. *8th World Congress on Genetics Applied to Livestock Production*, August 13-18, Belo Horizonte, MG, Brasil.

Snowder, G. 2006, Genetic selection for disease resistance: Challenges and Opportunities. USDA, ARS, MARC, Clay Center, NE 68933.

Sonstegard, T.S., Silva, M.V.G.B., Gasbarre, L.C. and Van Tassell, C.P. 2006. Use of Box-Cox transformation in analysis of fecal egg count data. *8th World Congress on Genetics Applied to Livestock Production*, August 13-18, Belo Horizonte, MG, Brasil.

Spickett, A.M., De Klerk, D., Enslin, C.B. and Scholtz, M.M. 1989. Resistance of Nguni, Bonsmara and Hereford cattle to ticks in a bushveld region of South Africa. *Ondersteproot Journal of Veterinary Research.* 56: 245-250.

Sularsas, D. 1985. Genetic variation and correlations between body heights, growth, adaptive and reproductive traits in tropical beef cattle. PhD Thesis, James Cook traits University of North Queensland, Townsville.

Trail, J.C.M. and Gregory, K.E. 1981.Characterization of the Boran and Sahiwal breeds of cattle for economic characterers. *Journal of Animal Science* 52: 1286-1293.

Turner, H.G. and Short, A.J. 1972. Effects of field infestations of gastrointestinal helminths and of the cattle tick (*Boophilus Microplus*) on growth of the three breeds of cattle. *Australian Journal of Agricultural Research*. 23: 177-193.

Utech, K.B.W., Wharton, R.H. and Kerr, J.D. 1978. Resistance to *Boophilus microplus* in different breeds of cattle. *Australian Journal of Agricultural Research.*29: 885-895.

Wagland, B.M. 1975. Host resistance to cattle tick *Boophilus microplus* Brahman (*Bos indicus)*. I. Response of previously unexposed cattle to four infestation with 20, 000 larvae. *Australian Journal of Agricultural Research.* 26: 1073-1080.

Wharton, R.H., Harry, K.R.S., Wilkinson, P.R., Utech, K.B. and Kelly, B.M. 1969. A comparison of cattle tick control by pasture spelling planned dipping and tick resistance cattle. *Australian Journal of Agricultural Research.* 20: 783-793.

Wharton, R.H., Utech, K.B.W. and Turner, H.G. 1970. Resistance to the cattle tick, *Boophilus microplus* in a herd of Australian Illawarra Shorthorn cattle: Its assessment and heritability. *Journal of Agricultural Research.* 21: 163-181.

Wambura, P.N.,Gwaisa, P.S., Silayo, R.S. and Rugaimukamu, E.A. 1998. Breed-association resistance to tick infestation in *Bos indicus* and their crosses with *Bos Taurus*. *Veterinary Parasitology*. 77:63-70.

Wilkinson, P.R. 1962. Selection of cattle for tick resistance and the effect of herds susceptibility on Boophilus population. Australian *Journal of . Agricultural Research., 13: 974-983.*

Wright, I.G. 1990. Immunodiagnosis of immunoprophylaxis against the haemoparasite Babesia *sp* and *anaplasma sp* in domestic animals. *Revenue Scientifique Office International des Epizootics.* 9: 35-356.