

Significant veterinary research on the dromedary camels of Kenya: Past and Present

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Abstract

The dromedary camel (*Camelus dromedarius*) is extremely well adapted to hot and arid environments owing to its unique anatomical, physiological and behavioural features. Dromedaries do not only survive where other domestic species do not, but they also carry out work and produce valuable food products for the benefit of people. Dromedaries offer the only means of utilizing large areas of arid lands in many countries around the globe. The camel's feed and water needs are special. They browse and graze on vegetation that is inaccessible or unpalatable to other livestock and many other animals, and they have a remarkable tolerance to dehydration. The virtues of the dromedary camels in Kenya and their importance in food security of pastoralist people have been more and more recognized over the last decades. Pastoralism is replacing agro-pastoralism in some areas of sub-Saharan Africa due to climate change. In areas where the growing period will be too short and no longer support crop cultivation, pastoralism may become the only sustainable source of food production. A compilation of significant research done on the dromedaries of Kenya during recent decades is reviewed, particularly regarding veterinary aspects of the most prevalent diseases that have been and are being studied. Trypanosomosis (surra) has received most attention, followed by camelpox, mastitis and milk hygiene, camel calf losses, sarcoptic mange and other skin diseases, helminthosis, haemorrhagic septicaemia and respiratory infections.

Key words: dromedary camels, veterinary research, Kenya

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Introduction

Throughout history the dromedary camel (*Camelus dromedarius*) has been a domestic animal of great importance, serving

large numbers of people representing various cultures throughout vast tracts of arid and semiarid lands. In Africa, the dromedary camel is generally kept by nomadic

pastoralists and is an important part of the livelihood of these people, essential to their subsistence economy (Fig 1). The camels are of great importance socially and culturally as well as economically (Wako et al., 2012a)¹, a cornerstone in the social organisation of many of the camel-keeping societies. With an increasing human population, regular droughts and declining per capita production of food in Africa, there is a need to develop sustainable resources in the marginal semi-arid and arid rangelands.

Despite the historical importance of camels, research interest in this very special species has been very small compared to research on other domestic animals. Kenya like many other countries concentrated its veterinary, animal husbandry and breeding activities on cattle and on the fertile parts of the country (SIDA, 1982). Around the late 1970s and early 1980s, the focus shifted towards development of small ruminants. Only in recent years has the attitude towards the dromedary camel slowly changed into one of appreciation (Perry et al., 2002). The scientific community in Kenya, in

Figure 1. A herd of dromedary camels (*Camelus dromedarius*) of the ethnic group, Rendille, of Kenya.

¹Underlined authors signifies that their studies, at least partly, were made on dromedaries in Kenya.

spite of the previous lack of interest by the general public, acknowledged the `virtues of the camels` and the challenges of understanding the causes of negative impacts to the health and productivity of these extraordinary animals and have acted upon this. They have done a great amount of research on various aspects of the dromedary. This was manifested at the very first international workshop on camelids in 1979 in Khartoum (Ross-Cockrill, 1984).

This paper will review some of the important scientific contributions on the study of Kenyan dromedaries (*Camelus dromedarius*) particularly regarding veterinary aspects during the last decades.

Significance of camels in the East African region

The camel population worldwide is estimated to be about 18.9 million (FAO-STAT, 2004) of which about 95% are dromedaries. Of these about 73% (15 million) are found in Africa (18 countries). Camel production is of significant importance in the arid lowlands of eastern Africa including the Horn of Africa where about 11 million camels represent a significant part of the domestic livestock of the region (Kaufmann, 1998). In the arid and semi-arid areas of Kenya (ASAL) camels constitute 25% of the domestic herbivore biomass (DHB)

and about 6% of Kenya's total DHB (Schwartz and Dioli, 1992).

Dromedaries constitute the most important livestock for a significant number of households (Wilson, 1984; Wako et al., 2012a).

Between the 10th and 13th centuries A.D. camel herding people began migrating into northern Kenya, traces are found around the Chalbi desert (Stiles, 1987). The Rendille, Gabra, Sakuye, Degodia, Ajuran and Garre (the latter four are clans of Somali origin) are the ethnic communities with the longest tradition of camel husbandry in Kenya. The Turkana came to adopt it later, about 100 years ago (Kaufmann, 1998). In East Africa, this expansion of camel keeping is slowly continuing today even into Tanzania (Pelant et al., 1997) and into Northeast Uganda (in Karamoja). Traditional cattle cultures such as the Boran, Oromo, Samburu, Maasai and Pokot, have gradually introduced camels into their traditional herds. Wooded areas within the jurisdiction of these communities may be utilized more efficiently and in a more sustainable manner when camels share browsing and grazing with other livestock. A decade ago, the camel population in Kenya was estimated to be about 830,000, 5-6% of Africa's camel population (FAO-STAT 2002). Although no credible livestock census has been done in Kenya since 1969, there are estimates stating that

the camel population has increased to about one million (Njiru et al., 2004). Camels are kept in at least 16 districts of the country, mostly in the arid and semi-arid areas (ASAL), covering over 80% of the total land surface of Kenya, where about 25% of the human population of Kenya lives. ASAL harbours in all little over half of the country's livestock population. According to Mitaru (2002) 54% of the camels are found in the North Eastern Province, 29% in Eastern Province, 13% in Rift Valley Province and 4% in Coast Province.

Camel husbandry systems differ between the different camel herding ethnic communities (mainly nomadic pastoralists) in Kenya. In addition, small numbers of dromedary herds are kept on research institutes, private commercial ranches and a limited number of private entrepreneurs. To all of them, milk is the most important produce. Not all use camels as a beast of burden. The Turkana, for example employ donkeys only for such work (Kaufmann and Binder, 2002; Mochabo et al., 2006). Another very important feature of the dromedaries is that they are both grazers and browsers and can reach feeding resources to a height of around 3.5 m in bushes and trees (Fig 2). In many environments dromedaries do not compete with other ruminant livestock on the range (Field, 1979; Evans and Powy's, 1984). This

inspired ranchers in Kenya that traditionally bred cattle and small ruminants to introduce dromedaries. Between 1974 and 1978 camels were introduced to four ranches (Evans and Powy's, 1984). The positive outcome of this pioneering initiative inspired more Kenyan ranchers to also diversify their herds of livestock.

Increasing importance of camel milk to the pastoral communities

Camel milk is the most important product of the camels in Kenya and contributes between 50 - 60% of the nutrient intake of some of the pastoralist communities of sub-Saharan Africa (Simpkin et al., 1997; Kaufmann, 1998). In northern Kenya camels supply up to 70% of the milk consumed by the pastoral communities (Schwartz and Schwartz, 1985). With increasing aridity of the ranges, camels prevail in the herds because they are better adapted than other livestock to water and feed stress. A significant feature of camels is their capacity to maintain milk production throughout dry seasons, during periods when milk from other milk producing livestock, such as cattle, goats and sheep, becomes scarce and unreliable. Under the same arid conditions, the camel produces much more milk than the zebu cow and for a longer time per lactation (average lactation periods of camels are 12-18 months). Studies in Kenya have

Figure 2. Camels browsing and grazing.

shown that during the dry season one lactating Rendille camel produces as much milk as five Samburu zebu cows produce during the wet season (Field and Simpkin, 1985). In similar semi-arid environments, a herd of 100 camels produced four times as much milk per annum as a herd of 100 cattle (Stiles, 1995). Similar findings of the milk yields of milk producing animals of the nomadic Afar people in the neighbouring country of Ethiopia were reported (Richard and Gerard, 1985).

There are considerable variations in calf and milking management between the different pastoralist groups (Simpkin et al., 1997). Management practices significantly influence total milk production. In recent decades, there have been substantial changes in the utilization of camel milk, from simply supplying the needs of camel

keeping communities to the development of substantial marketing and distribution networks for camel milk, which is now sold in rural towns and settlements and in peri-urban and urban markets (Farah and Younan, 2005). The milk of camels often commands a much higher price than that of cattle, particularly during the dry season.

Several studies on milk production, lactation, milk composition and chemistry, milk processing and products have been done on Kenyan camels; (Simpkin et al., 1997; Bachmann and Schulthess, 1987; Bansal et al., 2009; Farah et al., 1992; Farah and Atkins, 1992; Kappeler et al., 1998, 2003, 2004; Wangoh et al., 1998), as well as on mastitis and milk hygiene (Younan et al., 2001; Younan, 2002; Younan and Kenyanjui, 2003; Younan et al., 2003; Younan and Abdurahman,

Figure 3. Woman selling camel milk at the market.

2004; Younan et al., 2006a). In addition, Kenyan researchers have shown interest in nutrition vis à vis milk yield and growth (Kuria et al., 2004). Factors affecting the milk yield of camels are similar to those common to all dairy animals: genetic potential, health status and nutrition, and good management. It is possible to breed dairy camels with uniform mammary glands producing 30-40 litres of milk per day (Wernery, 2006).

Meat – a product of increasing demand

Due to the increasing population of Somalis to urban centers and in particular to Eastley of Nairobi, camel milk as well as meat of camels are growing in demand. Thus also meat and meat products are attracting research interest. During the last decades research on

processing of camel meat into marketable products was conducted in Kenya (Farah and Fischer, 2004).

In this context it can be mentioned that several studies are published on preferred plant species of Kenyan dromedaries and the nutritive value of these fodder species (Tubei, 1984; Rutagwenda et al., 1990; Kuria et al., 2005).

Gender issues and camel productivity

In the Horn of Africa and in Kenya camels are usually herded and milked by boys and young men, but the milk of the camels is the property of women. Female micro-enterprises dominate in the informal camel milk marketing chains (SNV, 2008; Younan and Mwangi, 2010) and women reap most of the benefits from handling, distributing and

selling of the product (Fig 3). Optimal production of milk means better nutrition for children and for camel calves. It also contributes to a better standard of living for the family and community because a cash income for women is generated. Any improvement, i.e. increase of camel milk production for the individual household by controlling diseases which influence milk production negatively will thus benefit the women. In addition, an increase in numbers of baggage animals, as a result of better disease control, will also ease the burden of transport for women.

The loss of a lactating dam and/or a single young female camel has - in the short and long perspective - significant consequences for the productivity of the herd (Dahl and Hjort, 1976). The consequences may be disastrous for a family that owns only a few camels.

Disease as a limiting factor

The most important factor in limiting livestock production generally is disease (Perry et al., 2002). This applies also to camel production (Richard, 1979; Simpkin, 1983; Dabelo, 2012). Diseases may be overcome by better husbandry, management, disease control and prevention (Bornstein, 1987). It was shown in a study that the productivity of Rendille camels in Kenya, extensively managed,

improved by simple veterinary interventions (Rutagwenda, 1982). Although camels are resistant to some of the most severe infections of other livestock, such as rinderpest, contagious bovine pleuropneumonia and foot-and-mouth disease (Wernery et al., 2002), there are other common diseases, which have long term negative effects on the health and productivity of camels, such as trypanosomosis and camelpox.

Trypanosomosis was found to reduce the milk yield of camel herds by 35-57% (Njiru et al., 2002a). Camelpox affects a large proportion of individuals in infected herds and affected female camels may abort, cease milk production (Wilson et al., 1982) and are also prone to mastitis (Younan et al., 2001). Pox infection may have a severe impact on the growth and weight gains of infected calves (Dioli and Stimmelmayer, 1992). Gastro-intestinal (G-I) parasite infections are also common in camels and claimed to be the third most important production impediment in camels (Rutagwenda, 1982; Perry et al., 2002). It is also claimed that a significant part of the high mortalities of camel calves aged up to one year (>30%) is due to diarrhoea (Kaufmann, 1998). There are several reports that list the most important and prevalent diseases among dromedary camels in eastern Africa (Mochabo et al., 2005) based on questionnaire surveys, participa-

tory epidemiology and group discussions with camel herders. Among the ‘‘top ten’’ of these diseases one always finds, with some variation in the order of priorities as number one trypanosomosis (with very few exceptions), followed by helminthosis, camel pox, orf and other skin conditions particularly sarcoptic mange, then calf diarrhoea, respiratory disease, camel haemorrhagic septicaemia (HS), sometimes not differentiated from Swollen Glands or ‘Khanid’ (Rendille name of a disease/syndrome of unknown cause), lymphangitis, wry neck, mastitis etc. Many of these conditions are poorly documented or studied partly due to the poor accessibility of the camels, husbanded as they are in remote dry lands.

Textbooks and manuals on camel husbandry and disease only concerned with Eastern African context have been published (Schwartz and Dioli, 1992; Evans, et al., 1995). In addition, a manual on camels edited by Köhler-Rollefson et al. (2001) contains information on Kenyan dromedaries as do the manuals; ‘Camel Husbandry - a practical guide to camel husbandry’ (Gitao, 2006) and ‘Camel manual for service providers’ (Younan et al., 2011), respectively.

Camel Trypanosomosis

Kenyan researchers recognized early that camel trypanosomosis (surra) was the most important and prevalent disease in camels (Wilson et al., 1983) and they have published considerable work on the causative parasite, *Trypanosoma evansi*, the most widespread and pathogenic trypanosome in the world (Njiru et al., 2004). *Trypanosoma evansi* has been and is still recognized as the most important pathogen of dromedary camels in Kenya (Nantulya, 1990). Apart from *T. evansi*, Wilson et al., (1983) found *T. brucei* and *T. congolense* (tsetse-transmitted trypanosomes) to be present in varying degrees in four Kenyan camel herds, all causing trypanosomosis. In addition, tsetse transmitted *T. vivax* (Bennet, 1933) and *T. simiae* (Röttcher et al., 1987; Mihok et al., 1994) have also been associated with the disease in Kenyan camels. *Trypanosoma simiae* has also been reported as a camel pathogen in Somalia (Pellegrini, 1948; Dirie et al., 1989). There is a definite correlation between the seasonal outbreaks of *T. evansi* infections and the increase of *Tabanus* spp. during the rains (Mahmoud and Gray, 1980; Njiru et al., 2002b). *Trypanosoma evansi* lacks the genes necessary for mitochondrial development (Gibson et al., 1983; Borst et al., 1987; Songa et al., 1990) and is thought to have originated from *T. brucei* by

adaptation to a non-cyclical mode of transmission in the insect vector (Hoare, 1957). Many camels have a mild and protracted *T. evansi* infection persisting for several years, eventually leading to emaciation and death (Bornstein, 2002a). In *T. evansi* infected camels, necrotic foci in the liver and spleen and generalised lymphoid tissue hyperplasia are commonly seen at necropsy (Röttcher et al., 1987).

North of the tsetse belt in Africa the prevalence of surra varies from 6.6% in Morocco to over 58% in Somalia (Bornstein, 2002a). In Kenya, the prevalence of *T. evansi* among 2000 tested camels was 48% (Olaho-Mukani and Wilson, 1983), and 79% in a smaller herd of 174 camels (Rutagwenda, 1984). In screening 450 camels of 8 different herds in Kenya, Olaho-Mukani et al. (1996a) found 13-54.9% of the camels positive by micro-haematocrit centrifugation test (MHCT), by mouse inoculation (MI), and by Ag-ELISA and monoclonal antibody based card latex agglutination test (Suratex®), respectively. In a pilot study in Lakipia district of Kenya, *T. evansi* prevalence ranged in camels from 11.1% by MHCT to 28.1% by Suratex® and 37.9% using the Card Agglutination Test (CATT/*T. evansi*) for trypanosomosis (Njiru et al., 2002a). Current or past trypanosome prevalence rates of 72-95% were

recently found in 18 herds in northern Kenya (Oyieke, 2003).

Prevalence data are based on several different tests with large variation of sensitivities and specificities, thus figures must be regarded with caution and interpreted as rough estimates. The demonstration and identification of *T. evansi* in the blood is difficult due to the often low and fluctuating parasitaemia, particularly in chronic and subclinical cases. Some of the traditional parasitological diagnostic techniques have a very low sensitivity of around 50% (Monzon et al., 1990; Nantulya, 1990; Luckins, 1992; Bornstein, 2002a). The micro-haematocrit centrifugation test (Woo, 1969; Woo, 1971) detected trypanosomes in camel blood 6 to 10 days earlier than was possible by wet or thick blood films (Kelley and Schillinger, 1983a,b). Mouse Inoculation (MI) is a fairly sensitive method for detecting low parasitaemia caused by *T. evansi* (Boid et al., 1985; Jain et al., 2000; Olaho-Mukani et al., 1993a, 1996a; Pegram and Scott, 1976). It can detect 1.25 *T. evansi* organisms per 4 ml of blood from an infected animal (Reid et al., 2001), but is time consuming, expensive and the use of large numbers of laboratory animals is questionable from an ethical point of view, and therefore inappropriate for use in large surveys.

Trypanosome specific antibody detection tests such as immunofluorescent antibody test (IFAT), the Card Agglutination Test for trypanosomiasis (CATT/*T. evansi* (a simple, quick test), and enzyme-linked immunosorbent antibody assays (ELISA) have been developed for serodiagnosis of *T. evansi* infections (Boid et al., 1980; Luckins et al., 1979; Songa and Hamers, 1988; Rae et al., 1989; Nantulya 1994; Pathak et al., 1997; Gutierrez et al., 2000). A problem with specific antibody detection tests is that the antibodies against *T. evansi* remain detectable in the infected animals long after successful treatment i.e. one cannot distinguish current infections from those cured (Luckins, 1988; Olaho-Mukani et al., 1996a). Persistence of antibodies following sterilising treatments has been demonstrated to be between 1-4 months (Ferenc et al., 1990) and one month for the trypanosoma antigens to disappear from the blood circulation (Waitumbi and Nantulya, 1993). Therefore immunoassays to detect circulating trypanosomal antigens for early diagnosis in infected animals were developed (Rae and Luckins, 1984; Nantulya et al., 1989a,b; Olaho-Mukani et al., 1992; Pathak et al., 1997). Antigen tests such as the card latex agglutination test (Suratex®), a `pen-side` diagnostic test and Ag-ELISA have shown high correlation with surra in camels (Jain et al., 2000;

Nantulya et al., 1989b; Olaho-Mukani et al., 1996a; Pathak et al., 1997; Singh et al., 1994a-c; Waitumbi and Nantulya, 1993). Suratex® and Ag-ELISA can detect the parasites in subclinically infected camels (Olaho-Mukani et al., 1996b). The latter researchers showed that the Suratex® had a specificity of 100% and a fairly high sensitivity of 93.2- 94.6%. A cross-sectional study of 2227 camels in the eastern and central parts of Kenya showed similar results i.e. 100% specificity of both CATT/*T. evansi* and Suratex®, but lower sensitivities; for the former 68.6% and the latter 58.8% (Ngaira et al., 2003). All the available antibody detection tests are based on the native VSG (variable surface glucogen) of the predominant variable antigen type (VAT) RoTat 1.2 of *T. evansi*. A complication is that these tests may be negative in animals infected with *T. evansi* type B, which occurs in Kenya as Type B, does not express RoTat 1.2 VAT (Ngaira et al., 2003, 2004, 2005). A promising rapid, sensitive and specific test, detecting *T. evansi* Type B based on the Loop-mediated Isothermal Amplification (LAMP) of DNA employing real time PCR, has been developed (Njiru et al., 2010). The analytical sensitivity of this test is approximately 0.1 tryp/ml.

An antibody detection ELISA using a recombinant extracellular domain of invariant surface glycoprotein 75 (ELISA/ISG75), and

thus independent of antigenic variation, was published recently as a reference test for surra in camels (Tran et al., 2009) and showed promising results when compared with ELISA/*T. evansi*, CATT/*T. evansi* and immune trypanolysis assay (TL). The ELISA/ISG75 showed a sensitivity of 94.6% and a specificity of 100% when tested against a panel of 184 camel sera. The polymerase chain reaction (PCR) proved to be a useful tool for detecting *T. evansi* in infected camels (Singh et al., 2004b). DNA fingerprint studies by PCR-based amplification revealed intra-species differences in *T. evansi* (Watanapokasin et al., 1998). In Kenya PCR and Procyclic Transformation test (PTT) have been used successfully to characterize *Trypanosoma* spp. infections in camels (Masiga and Nyang'ao, 2001). It can differentiate between *T. evansi* and *T. brucei* (Olaho-Mukani et al., 1993b). Masiga and Nyang'ao (2001) detected *T. evansi* in 76 percent of the isolates tested confirming it to be the most important species causing trypanosomosis in camels in Kenya. They demonstrated that *T. brucei* was present in single infections and in mixed infections with *T. brucei* and *T. congolense*.

Trypanosoma brucei is morphologically indistinguishable from *T. evansi*. This finding may complicate treatment if the species of

the trypanosomes are not diagnosed i.e. if the drug used only cures the camel against one of the species (Masiga and Nyang'ao, 2001; Maina et al., 2003). In camels that had travelled through tsetse areas infested with *Glossina pallidipes*, *G. longipennis* and *G. brevipalpis* the PCR detected *T. congolense* that is known to be more fatal to camels than the *T. brucei* subgroup. Molecular tools are essential in understanding the epidemiology of *T. evansi* in camels and are essential in the study of the biggest threat to *T. evansi* control in camels, the emerging drug resistance.

Several other important fields of Surra in camels have been addressed by Kenyan researchers; as clinical pathology (Njiru et al., 2000), pathology, and pathogenesis (Njiru et al., 1997; Ouma et al., 1998a,b) and treatment and control including drug-testing and drug-resistance in particular (Maina et al., 1996, 1998, 2003; Olaho-Mukani et al., 1995; Olaho-Mukani et al., 1996b; Wesongah et al., 1997). Research interest has also been shown in the vectors involved in *T. evansi* transmission and the threat of other livestock and wildlife acting as reservoirs of the pathogen (Ngeranwa and Kilalo, 1994). Outside the scientific community most of the listed new tests are not available for routine diagnostic use

in Kenya. The KARI-KASAL² funded project, 'development of improved strategies for control of camel trypanosomosis in ASAL' carried out a study on *T. evansi* in camels in North Kenya. One main finding was inappropriate use of trypanocide drugs as a very common cause for treatment failure and also increasing the risk of emerging trypanocide resistance in *T. evansi* (Mdachi et al. 2011).

Calfhood infections - coccidia and other gastrointestinal pathogens

Bremaud (1969) assessed camel calf mortality in northern Kenya to be between 30 - 50%, but this is probably an overestimate (Hülsebusch, 1999). Kaufmann (1998, 2003) reported long-term camel calf mortalities (<1 year of age) in northern Kenya in different pastoral communities to be 20-30%. A calf mortality of 25% is regarded as typical for livestock in marginal areas of sub-Saharan Africa (Kaufmann, 2003). Diarrhoea was found to be one of the three most common signs of disease causing mortality in camel calves of Gabra, Rendille and Somali groups (Kaufmann, 1998). High calf mortality has often been cited as a

contributory cause to the low reproductive performance of camels in Kenya and elsewhere (Bremaud, 1969; Schwartz et al., 1983; Baumann and Zessin, 1992). Restricting milk intake of newborn calves, a common practice among some pastoral societies in Kenya (Hülsebusch, 1999), is thought to prevent such calves from getting enough or any colostrum during their first 36 hours of life (h.p.n.) and would explain the lower performance, disease and eventually deaths of camel calves. Hülsebusch (1999) studied the immunoglobulin-G (IgG) status of camel calves from birth to six months of age in camel herds in northern Kenya in relation to bodyweight, disease occurrence and mortality. He found no correlation between either serum IgG status of the camel calves and observed occurrence of skin disease or diarrhoea. In most calves the peak IgG concentrations were seen between 18-30 h.p.n. In 19% of cases the peak IgGs were attained later, between 30-65 h.p.n. Complete failure of transfer of maternal antibodies was observed in only three out of the 68 calves sampled. The start of calf IgG production in these three calves was seen at 14 days p.n. (Hülsebusch, 1999).

The coccidian parasite *Isospora orlovi* was identified as causing severe outbreaks of diarrhoea in young camel calves (aged 10-35 days) in Dubai (Kinne et

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al., 2002). *Isospora* sp. was found during a small field study of diarrhoeal disease in camel calves in northern Kenya (Younan et al., 2002) in five calves excreting *Isospora* sp. between 18 and 32 days of age. Four of the five calves excreting *Isospora* sp. exhibited diarrhoea and one of them died following a period of diarrhoea.

A longitudinal investigation of 159 suckling camel calves from birth to 12 weeks of age on 8 different camel herds of commercial private ranches in northern Kenya and a point prevalence study of 91 calves belonging to 42 pastoral herds in northern Kenya were sampled at convenience over a period of 18 months. The investigation confirmed the presence of *Isospora* sp. in Kenyan camel calves and the species as *I. orlovi* (Bornstein et al., 2008). Over 30% and 41% of the calves of the ranch herds and the pastoral herds respectively, exhibited diarrhoea. The *Isospora* parasites were only found in the calves <4 (ranches) and <8 (pastoral) weeks of age, respectively and only in calves with diarrhoea or following periods of scouring (Bornstein et al., 2008).

This study was part of a larger epidemiological study by Gluecks (2007), who investigated also the prevalence of bacterial intestinal pathogens in camel calves up to 12 weeks of age in Northern Kenya. The number of animals investigated in

the larger study comprised fewer animals (in the point prevalence study 157 calves in 8 ranch herds, and 72 calves in pastoral herds in 14 different locations were sampled). In the study 68.0% of the calves were healthy, 23.2% diseased exhibiting diarrhoea, 6.6% convalescent from diarrhoea and 2.2% were found dead. There was a peak amongst the calves suffering from diarrhoea during the second and third week of age in both management systems. In about one fourth of the calves suffering from diarrhoea *Isospora* sp. was found, and 92% of all the calves shedding *Isospora* sp. suffered from diarrhoea.

The same study also showed that *Salmonella* spp. and *Klebsiella pneumoniae* (Gluecks et al., 2006; Gluecks, 2007) were

common pathogens in camel calves between birth and 12 weeks of age. *Klebsiella pneumoniae* was isolated in 26.9% of calves sampled (n=229) and *Salmonella* spp. in 19.1%. Isolations of *K. pneumoniae* and *Salmonella* spp. were particularly high during the first three weeks of age. One third (n=3) of deaths in camel calves examined (n=9, exhibiting various post mortem signs) during the longitudinal study were related to septicaemic *K. pneumoniae* infections and another third to septicaemic *Salmonella* spp. infections (Gluecks, 2007; Younan et al., 2013). This study in Kenya is the first to describe commensal presence of *K. pneumoniae* in healthy camel

calves and *K. pneumoniae* involvement in camel calf septicaemia. Eighteen different *K. pneumoniae* capsular types were identified among the 62 isolates, including the highly pathogenic *K. pneumoniae* capsular type 2 isolated from a post-mortem case. *Klebsiella pneumoniae* isolates showed 100% resistance to Penicillin and Amoxicillin, 78% resistance to Sulphonamide-TMP and 61% resistance to Tetracycline, indicating very limited treatment choices.

This Kenyan study by Gluecks (2007) confirms reports from other camel keeping regions on the major importance of *Salmonella* spp. in diarrhoea and septicaemia of young camel calves. The prevalence of *Salmonella* spp. was high from the beginning of the second week of age and then slowly decreased (Gluecks, 2007). Among the 144 *Salmonella* spp. (including serotypes) isolations, eighteen different *Salmonella* spp. were found. The only *Salmonella* sp. associated commonly with calf disease and present in camel calves of both management systems was *S. typhimurium*. Seventyfive percent of camel calves that were found positive for *S. typhimurium* were suffering from diarrhoea. *Salmonella typhimurium* and *S. adelaide* were the only two serotypes found in both, ranch and pastoralist management systems. *Salmonella bovis morbificans*, *S. butantan* and *S. irumu* were isolated from post

mortem cases of ranch camel calves found dead. *Salmonella bovis morbificans* was the most common serotype in ranch camel calves but was not found in pastoralist camel calves. This may reflect the fact that camels on ranches are kept in close contact with cattle, a situation not typically found in all camel populations (Gluecks, 2007).

Echerichia coli was present in all camel calves throughout all age groups without significant variation of prevalence and was not a significant pathogen in nine investigated post mortem cases (Gluecks, 2007). There was no statistically significant relationship between diarrhoea status and detection of *eae*, *astA*, *hlyEHEC* and *stx* in the *E. coli* isolates. Only few *E. coli* with virulence-associated genes (14%) were isolated from diarrhoeic camel calves and none from dead calves (Gluecks, 2007). This comprehensive investigation of virulence-associated genes in *E. coli* isolates from camel calves indicates that pathogenic *E. coli* may not play such a significant role in the diarrhoea complex of camel calves of the studied age group as has been commonly thought (Gluecks, 2007). Earlier investigations ranking *E. coli* as a major pathogen of neonatal camel calves (Schwartz and Dioli, 1992; Bornstein et. al., 2000; Wernery, 2002; Abbas and Omer, 2005; Berrada et al., 2000) did not analyse virulence-associated genes of

their *E. coli* isolates. Conclusions based on isolations of *E. coli* only must be regarded with caution if virulence-associated genes are not tested.

Peri-articular abscesses located around the elbow (33.3%), tarsus (29.2%), carpus (25.0%), knee (8.3%) and fetlock (4.2%) joints have been described as a calf disease in North Kenya (Younan et al., 2007). Pure *Streptococcus agalactiae* infections accounted for 82% of investigated cases (n=49) in suckling calves while 4% involved mixed infection of *Streptococcus agalactiae* and *Streptococcus equi zooepidemicus* and only 2% were caused by single infection with mucoid *Streptococcus equi zooepidemicus*.

Gastro-intestinal helminths

Gastro-intestinal (G-I) helminthosis is arguably the greatest disease problem of all grazing livestock (Perry and Randolph, 1999). The greatest losses associated with nematode parasite infections are sub-clinical and economic assessments show that financial costs of internal parasitism in e.g. small stock are enormous (Preston and Allonby, 1979; McLeod, 1995).

In camels, G-I parasite infections are considered to be prevalent and to cause significant disease (Rutagwenda, 1982, 1984; Chabra and Gupta, 2006). Gastro-

intestinal helminthosis was claimed to be the third most important production constraint in camels in Kenya (Rutagwenda, 1982). It was also claimed that a significant part of the high mortalities (>30 %) of camel calves up to one year of age are due to G-I parasitic infections (Kaufmann, 1998). In camels in Kenya, G-I parasites are prevalent, as they are in sheep and goats, which are commonly grazing and browsing with camels. Many helminth species may cross-infect ruminants and camels including the most pathogenic nematodes, *Haemonchus* spp. (Jacquet et al., 1996). *Haemonchus contortus* is a common cause of acute disease with high mortality in small ruminants. This parasite may infect camels as well, causing significant disease. *Haemonchus longistipes*, which is a specific parasite of camels is thought to be the major nematode pathogen of camels and is often found in mixed infections with other nematode parasites.

Bekele (2002) in north-east Ethiopia and others have shown significantly different prevalence rates of G-I nematodes among different age groups of dromedary camels. The prevalence was lowest (59.6%) in the youngest group and increased with age being the highest for the oldest age group of >13 years (83.9%). Bekele (2002) also showed that adult female camels have a significantly higher worm burden

than adult male camels. The above studies need to be repeated elsewhere. If the findings are a general pattern among camels it will have relevance for milk producing camels considering that the age at first parturition in traditionally kept camels in Kenya is estimated to be on average 58 months (Wilson, 1995). Parturition intervals were 18 to 19 months and the average lifetime production of progeny was 3.5 camel calves per dam on commercial ranches in Kenya (Evans et al., 1995).

Studies of G-I helminthosis in Kenyan camels are relatively few (Wilson et al., 1980; Rutagwenda, 1982, 1983; Field, 1985; Olaho-Mukani and Kimani, 1999; Chemuliti et al., 2003). Experiences and knowledge drawn from disease surveys in the late 1970s and early 1980s in northern Kenya (Wilson et al., 1980; Rutagwenda, 1982, 1984; Field, 1985) were never disseminated extensively to the appropriate stakeholders. It is not known whether the economic benefits of parasitic control performed on cattle (Gross et al., 1999) apply to camels as well. However, there are reports that treatment against G-I nematodiosis in camels improves health and increases weight gains between 6.8 and 11% (Graber et al., 1967; Simpkin, 1983) and it was shown that veterinary inputs resulted in increased milk yield and lengthening of the lactation by three months (Simpkin, 1983).

Efficacy studies of a few different anthelmintics on camels in Kenya have been published (Rutagwenda and Munyua, 1983; Mukhwana and Mitema, 1997).

Adulteration of anthelmintics is a common practice in Kenya (Monteiro et al., 1998). Illiteracy and/or unfamiliarity with synthetic anthelmintics, resulting in incorrect dosage and wrong usage of anthelmintics are also a problem (Danø and Bøgh, 1999). Moreover, these drugs are relatively expensive and often unavailable to livestock owners in rural areas including ASAL. Widespread incorrect usage of antihelmintics may promote the development of resistance in G-I nematodes.

Hydatidosis

Echinococcosis/hydatid disease, caused by the larva of the cestode *Echinococcus granulosus*, is one of the most important helminth zoonoses globally (Thompson, 2008). It continues to exert an unacceptable burden on the health of people, on livestock, and on wildlife in many regions, among them Kenya. In certain areas of Kenya all livestock; goats, sheep, cattle, camels and donkeys harbour the parasite (Macpherson, 1981 cited McManus and Macpherson, 1984). The disease is e.g. found in the Turkana district, which was recognized as having the highest human prevalence, 10-15%

Figure 4. A dromedary camel with long standing sarcoptic mange.

of hydatid disease (French et al., 1982). A prevalence study was conducted in livestock at slaughter in northern Turkana. In total 5752 goats, 558 sheep, 381 cattle and 70 camels were investigated. *Echinococcus granulosus* metacestodes (hydatides) were found in 19.4% of the cattle, 3.6% of the sheep, 4.5% of the goats and 61.4% of the camels (Njoroge et al., 2002). The lifecycle of *E. granulosus* includes the intermediate host, which may be humans, domestic and wild animals and the final host, which are canids, dogs are particularly involved in the pastoral cycle. There are several different strains (genotypes) of *E. granulosus*, including a sheep/dog strain (G1) that is separate from the camel/dog strain (G6), which seems to be restricted to the Turkana region (Wachira et al., 1993). An earlier observation that humans appeared to be refractory to infections by the camel/dog strain

(Wachira et al., 1993) was later refuted when it was found that most cases of cystic echinococcosis (CE) in humans were caused by the sheep strain (G1) and by the camel strain (G6) (Magambo et al., 2006). In materials from the cysts of 59 humans with CE in the Turkana district of Kenya sampled during a hydatid control programme in 1993-94, 17% were identified as G6 (Casulli et al., 2010).

Skin diseases due to arthropods, bacteria, fungi and virus

Skin diseases of different aetiologies are often seen in camels. The most common skin condition apart from dermatophytosis (ringworm), camelpox and contagious ecthyma (ORF) is sarcoptic mange (Fig 4), caused by the burrowing mite, *Sarcoptes scabiei* (Fig 5).

Figure 5. The burrowing mite, *Sarcoptes scabiei* (x1560).

Sarcoptic mange is considered to be one of the most common diseases of camels (*C. dromedarius*) worldwide (Lodha, 1966; Higgins, 1984; Pegram and Higgins, 1992; Bornstein, 1992a) and in Kenya (Dioli and Schwartz, 1992; Bornstein, 1995, 2002a,b). *Sarcoptes scabiei* is a parasite of more than 100 host species including humans (Schillinger, 1987; Bornstein et al., 2001b). Different strains of the mite vary in their capacity to colonize and proliferate in different host species. The cardinal symptom is the pruritus

(Fig 6) that can become very intense and is soon followed by excoriations, papule formation, alopecia and reduced feeding time. Commonly recognized chronic signs are crusting, thickening (hyperkeratosis), discolouring and fissuring of the skin. The infection spreads rapidly within a herd and is responsible for loss of condition and production (Nayel and Abu-Samra, 1986). There are no scientific studies specifically on production losses in camels caused by sarcoptic mange, but it has been observed that milk production in infected camels falls rapidly (Dioli and Stimmelmayer, 1992). When mange is suspected the diagnosis can be confirmed by the demonstration of *S. scabiei* mites in skin scrapings. However, the sensitivity of this method is low. This was exemplified in natural and in an experimental *S. scabiei* infection of dromedary camels in Kenya even when applying multiple deep skin scrapings (Bornstein et al., 2001a, 2002b).

Figure 6. A herd of dromedaries in Kenya exhibiting the cardinal signs of sarcoptic mange – pruritus.

Only in 7% of 40 examined naturally infected camels in Kenya exhibiting typical symptoms of sarcoptic mange, could *S. scabiei* be found (Bornstein et al., 2001a, 2002b).

The development of an indirect ELISA demonstrating specific antibodies to *S. scabiei* markedly improved the diagnosis of sarcoptic mange in dogs and in swine and has increased the probability of achieving a correct diagnosis in these species from 30-50% to 95-98% (Bornstein et al., 1996, Bornstein and Wallgren, 1997). A similar ELISA developed for camels (Bornstein et al., 1997) demonstrated the presence of specific antibodies to *S. scabiei* in two-thirds of camels in a naturally infected herd in Kenya (Bornstein et al., 2002b). This indirect antibody-ELISA (Bornstein et al., 1997) is a promising candidate that could be developed as a useful diagnostic and seroepidemiological tool in the future.

Dermatophilus congolensis is a common pathogen in the humid tropics, producing mobile spores (zoospores) and infecting cattle, small ruminants, and horses (less frequently pigs, dogs, cats and humans), particularly during the wet season (Zaria, 1993). Transmission occurs through direct contact or by vectors, ticks and flies (Gitao et al., 1990; Gitao, 1992). *Dermatophilus congolensis* infections were found in camels in Kenya both on a

commercial ranch and in a pastoral herd. (Gitao et al., 1990; Gitao, 1992). During the same outbreak of dermatophilosis in the camels on the commercial ranch (Gitao, 1992) about 10 sheep were found with the same disease. Gitao et al (1998) found in the neighbouring country of Sudan widespread outbreaks of *D. congolensis* in many camel herds.

Dermatophilus congolensis and several other infectious pathogens have been described in connection with infectious skin necrosis of camels (Wernery, 2002). Observations by Edelsten and Pegram (1974) in Somalia and by Younan and Bornstein (2007) in Kenya indicate that various bacterial pathogens isolated from infectious skin necrosis lesions appear to be opportunistic invaders and that the underlying cause for this skin condition is most likely a nutritional deficiency, a view also held by Peck (1939) in Somaliland.

Camelpox is caused by *Orthopoxvirus cameli* and is regarded as one of the most common viral diseases of camels in East Africa and worldwide, except on the Australian continent (Kaaden, 2002). Camelpox has been isolated from infected camels in Kenya and characterized (Davies et al., 1975; Munz et al., 1986a; Gitao et al., 1996). Davies et al. (1985) showed high prevalence of antibodies to camelpox virus using the serum

neutralization test in five of six investigated pastoralist and ranch camel herds in Kenya, although no clinical pox was present. Animals recovered from pox show long lasting immunity, perhaps life long (Dioli and Stimmelmayer, 1992; Kaaden, 2002). Gitao (1997) reported camelpox outbreaks in two different areas of Kenya (Fig 7). Epidemics occur relatively regularly and occur mainly during the rainy seasons; outbreaks during the dry seasons appear to be mild and usually of the localized form (Munz, 1992). Severe secondary infections are common in camelpox, and may be localized to the skin (pyodermas), or generalized as septicaemia eventually leading to death (Dioli and Simmelmayer, 1992; Kaaden, 2002). Morbidity is high. Mortality is usually low, but can reach 28% in generalized forms of the disease (Jezek et al., 1983) and 40-50% in calves (Dioli and Stimmelmayer, 1992). Severe secondary infections affecting the respiratory tract were common in camel calves and weaners in West Pokot/Kenya suffering from camelpox (Younan, 2003 direct communication). In the wake of camelpox infection the spread of contagious mastitis increased significantly in lactating camels (Younan et al., 2001). Vaccines have been developed and tested. One called Ducapox[®] has been used in the UAE since 1994

(Kaaden, 2002), but is not registered and not available in Kenya.

Figure 7a. A dromedary with generalized camelpox lesions on the head.

Figure 7b. A dromedary with generalized camelpox lesions seen on the back.

Contagious ecthyma (ORF) is another common virus infection seen particularly in camel calves up to three years of age (Kaaden, 2002). The causative *Parapox* virus is thought to be identical to *Parapoxvirus ovis*, which infect sheep worldwide (Kaaden, 2002). Generalized skin lesions as in camelpox can occur (Dioli and Stimmelmayer, 1992) but more commonly the lesions are concentrated around the mouth and nostrils. They are very irritating and may lead to impaired suckling and grazing/browsing followed by severe loss of condition. The incidence of endemic contagious ecthyma is higher than that of camelpox. Morbidity can reach 100%, but is as low as 0% with the mild form (Gitao, 1994). Munz et al (1986b) in the first report of the disease in Kenyan camels, described an outbreak in a herd of 450 animals. Papules were seen on the lips occasional spreading to the nasal and oral mucosa. Pustules developed soon followed by encrustations. Scabs soon became dark in color and after 6-10 weeks dropped off. Many infected camels developed oedema of the eyelids, lips and nares. Gitao (1994) examined 600 camels during an outbreak in the Turkana district in Kenya. He saw swollen and oedematous cervical and mandibular lymph nodes in many of the calves. Under the scabs he observed yellowish exudates.

Ticks

Camels are commonly infested by large numbers of ticks. The main genera infesting the camels in Kenya are *Hyalomma*, *Rhipicephalus* and *Amblyomma*. The large majority of ticks are three-host species with the exception of the one-host tick *H. detritum scupense* and the two-host tick *H. marginatum rufipes*. Dolan et al. (1983) found 13 different tick species on camels on four different herds representing different eco-zones. Two of the herds were pastoral and two were ranches. In three of the herds tick control was applied by the use of acaricides. The latter herds carried fewer ticks compared to the former. Adult camels had heavier loads of ticks than the immature camels, which in turn carried more ticks than the calves. There were no significant differences according to sex. The most common tick species were *Hyalomma rufipes*, *H. dromedarii* and *Rhipicephalus pulchellus*. The relative proportions of the different tick species varied with the environment (Dolan et al., 1983). In the more arid traditional camel breeding areas of northern Kenya *Hyalomma* spp. were dominant, while in the perennial grasslands of the ranches and at higher altitudes *R. pulchellos* was the most common tick. Different tick species have different predilection sites: nostrils, ears, perineal and inguinal areas, udders. *Hyalomma dromedarii* are

found e.g. in the nostrils, whereas *H. rufipes* is found all over the body. The heavy tick burden found in one of the herds investigated was thought to have contributed to the slower growth rate and higher calf mortality in the herd (Dolan et al., 1983). Dioli et al. (2001) investigated ixodid ticks that infested camels in three herds in two separate areas of Kenya and in one area of Southern Ethiopia. Species composition, attachment sites, sex ratio and seasonal incidence were recorded. The species observed were *Rhipicephalus appendiculatus*, *R. evertsi evertsi*, *R. praetextatus* (or *R. muhsamae*), *R. pulchellus*, *R. pravus*, *Hyalomma dromedarii*, *H. marginatum rufipes*, *H. truncatum*, *Amblyomma gemma*, *A. lepidum* and *A. variegatum*.

The main effect of tick infestation in the dromedary camel, depending on the load, is a more or less pronounced anemia and loss of appetite followed by reduced growth rate and productivity, and higher calf mortality. At the site of tick attachment small lesions occur which easily become infected with purulent bacteria causing small abscesses. Tick bite on teats may act as a reservoir for mastitis pathogens (Younan et al., 2000a).

Tick paralysis in camels is a syndrome that appears to be rare. It has been reported in camel calves in Kenya caused by *Hyalomma truncatum* (Crees, 1985; Gitao, 2006)

and in Sudan (Musa and Osman, 1990) and is apparently caused by adult *Hyalomma* spp. and/or adults or nymphs of *Rhipicephalus* spp. Several other significant research studies on ticks in Kenya have been published (Branagan, 1973; Walker, 1974).

Mastitis - infections of the mammary gland

An important and quite recently acknowledged disease syndrome in camels, inflammation of the mammary glands (mastitis) has recently attracted considerable attention in Kenya. During the 1980s and early 1990s there were several reports on mastitis (both acute, chronic and subclinical) in camels (review see Abdurahman, 1996). Several bacteria species were found. The most common were *Staphylococci* and *Streptococci* spp. Acute mastitis and loss of teats was reported to occur in one third of Gabbra and Somali camels in northern Kenya (Kaufmann, 1998). Younan et al. (2001) found intramammary infections due to *Streptococcus agalactiae* and *Staphylococcus aureus* in lactating camels in six repeatedly sampled herds (331 udders sampled repeatedly, 1305 quarter milk samples examined, 53 non-infected camel udders monitored) in northern Kenya at a prevalence of 12% and 11%, respectively. There was a higher percentage of multiple quarter

infections for *S. agalactiae* infected udders (44%) compared to udders infected with *S. aureus* (23%). Repeat sampling over 10-12 months confirmed persistent subclinical infections for both pathogens. The California mastitis test (CMT) showed sensitivity of 77% for *S. agalactiae* and 68% for *S. aureus* and a specificity of 91% for both pathogens (Younan et al., 2001, Fig 8). Fifty percent of the isolates of *S. agalactiae* were resistant to tetracycline. Single sampling in another study in Kenya found subclinical mastitis due to *S. agalactiae* (Lancefield type B) to be present in six out of nine camel herds from different management systems (Younan et al., 2000b). In addition, *S. agalactiae* was isolated from camels with skin and joint infections, secondary respiratory infections and puerperal infections in the herds with subclinical mastitis, indicating a more complex epidemiology for *S.*

agalactiae in camels than in cattle (Younan et al., 2000b; Younan and Bornstein, 2007).

Over a period of 11 months a total of 435 milk samples were collected from lactating camels kept under ranch conditions in Northern Kenya and examined for presence of mastitis pathogens (Matofari et al., 2005). The most prevalent mastitis pathogens were group *D streptococci* (30%), coagulase negative *Staphylococcus* spp. (20.1%), *Staphylococcus aureus* (16%), Lancefield group *C streptococci* (*S. dysgalactiae*, 3.6%) and Lancefield group *B streptococci* (*S. agalactiae*, 1.5%). The 'infectious mastitis' streptococci, *S. dysgalactiae* and *S. agalactiae* (Lancefield group B and C), had a greater association with sub-clinical mastitis than mastitis streptococci originating from the environment. Average milk yield from quarters infected with

'infectious mastitis' streptococci was 38% lower as compared to quarters infected by environmental streptococci. While 84 of 196 camel milk samples collected at production and collection sites were found to contain *Salmonella* species, no *Salmonella* spp. were isolated from camel milk sampled at markets (Matofari et al., 2007).

A very limited parenteral treatment study of intramammary *S. agalactiae* infections in lactating camels showed that a three-day intramuscular course of benzylpenicillin procain dihydrostreptomycin or penethamate hydroiodide (a diethylaminoethyl ester of benzylpenicillin) at dose rates recommended for parenteral mastitis treatment in cattle was only successful if *S. agalactiae* infection of the gland had occurred recently (Younan, 2002).

Streptococci as opportunistic pathogens in camels

The role of Lancefield group B and C streptococci as commensals and common opportunistic pathogens in East African camels was addressed (Younan et al., 2006b; Younan and Bornstein, 2007). In the nasopharynx of Kenyan camels *S. agalactiae* was found in up to 100% and on the vaginal mucosa in up to 50% of clinically healthy carriers sampled per herd, respectively (Younan and Bornstein, 2007).

Streptococcus agalactiae was also isolated from non-abscessed lymph nodes of healthy camels. A healthy carrier state in Kenyan camels was also demonstrated for *S. equi zooepidemicus* in the nasopharynx, on vaginal mucosa and on the rectal mucosa and for *S. dysgalactiae equisimilis* in the nasopharynx, albeit at lower percentages (Younan and Bornstein, 2007). A total of 143 Lancefield Group C streptococci (*S. equi zooepidemicus* and *S. dysgalactiae equisimilis*) and Lancefield Group B streptococci (*S. agalactiae*) were isolated from diseased camels in Kenya and in Somalia. These camels showed clinical infections of the respiratory tract, tick bite lesions, abscessed lymph nodes, abscesses and other purulent skin lesions, peri-arthritis abscesses and arthritis, puerperal infection and gingivitis (Younan et al., 2000a; Younan and Bornstein, 2007; Younan et al., 2007). Using molecular diagnostic tools Younan et al. (2005) demonstrated that mucoid *S. equi zooepidemicus* isolated from camels with acute respiratory infections in Somalia and non-mucoid isolates from healthy camels in Kenya belonged to different sub-populations. Camel strains of *S. agalactiae* were shown to differ from human and bovine strains and thus represent a genetically distinct population (Fischer et al., 2013).