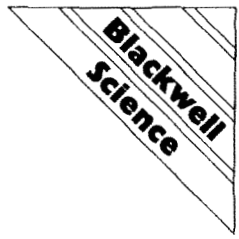
Ulrich Wernery · Oskar-Rüger Kaaden

Infectious Diseases in Camelids

„Dedicated to the fond memory of
Lt. Gen. Hamoodah Bin Ali,
from Central Veterinary Research Laboratory,
Priv.-Doz. Dr. Dr. habil. Ulrich Wernery"



Ulrich Wernery
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2nd, revised and enlarged edition

With 179 figures and 62 tables

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His Highness General Sheikh Mohammed Bin Rashid Al Maktoum, Defense Minister of the United Arab Emirates, with his best racing camels

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Foreword

The first edition of *Infectious Diseases of Camelids* was a significant contribution to the scientific literature of camel medicine. Clinicians, scientists, pathologists and camel owners all over the world used the book. The information was current, reflecting the extensive experience obtained at the Central Veterinary Research Laboratory (CVRL) and from world literature. The CVRL is one of the premier diagnostic laboratories in the world, with the staff devoting their efforts towards the diagnosis of disease in camels, horses and falcons in the Middle East. The CVRL has a professional staff of microbiologists, pathologists, molecular biologists and parasitologists working together to further the scientific knowledge necessary for the proper husbandry of camels.

The authors are pre-eminently qualified to write on this subject, having devoted much time, effort and expertise to studying camel infectious and parasitic diseases. The second edition continues the excellence of the first edition and adds significantly more information. The etiology of heretofore-questionable diagnoses has been clarified. More specific diagnostic procedures have been studied for sensitivity and specificity in camels.

Two new contributing authors have been invited to expand the areas of diagnostic pathology (Dr. J. Kinne) and parasitology (Dr. S. Bornstein). Publications dealing with the details of camel pathology are few and with this edition a valuable service has been rendered to diagnosticians and camel owners all over the world. The husbandry of camels will be improved as a result of more basic knowledge about diseases and disease processes in camels.

An important addition is the new chapter on parasites. Information on many of these parasitic diseases is now in concise, usable form.

It is significant that superbly skilled scientists have been given an opportunity to investigate and conduct research on camel diseases in the United Arab Emirates. His Highness General Sheikh Mohammed Bin Rashid Al Maktoum deserves the thanks of camel owners all over the world for having the foresight to establish the Central Veterinary Research Laboratory. Following more than a decade of investigation and collection of data on camelid diseases, the CVRL accumulated the expertise and knowledge to publish this book. His Highness' continued support of ongoing investigations on camel health is a reflection of his intense interest and support of the athletic camel. Camel owners, trainers, veterinarians and scientists from many disciplines are deeply appreciative of His Highness' benevolence.

The second edition has been completely updated, particularly in the areas of pathology, parasitology and mycology. The book is divided into bacterial, viral, fungal and parasitic diseases, with each chapter containing information on etiology, epidemiology, clinical signs, pathology, diagnosis, treatment and prevention. Treatment and control has been given special emphasis in this second edition.

Congratulations to the authors for their dedication and willingness to share their experiences with colleagues around the world.

Murray E. Fowler, DVM
Professor Emeritus, Zoological Medicine
University of California, Davis, USA

Preface

After working for a short period of time with dromedaries in Somalia some years ago, I now have the privilege of dedicating much of my time to this animal species in an optimal environment. The Central Veterinary Research Laboratory in Dubai was founded in 1985 and one of the major tasks of this institute was research on infectious diseases of camelids. Before 1970, very little was known about infectious diseases of camels. However, during the last two decades there has been a tremendous increase in the number of scientific papers in the world literature. It is now known that infectious diseases cause 50% of fatalities in New World camelids and 65% in Old World camelids. Pneumonia, peritonitis and diseases of the intestinal tract are the main ailments in NWC, whereas infectious diseases of the alimentary tract are the main causes of fatalities in OWC.

Most species of the camel family are domesticated and are used as beasts of burden, as "ships of the desert", and provide man with high quality fiber, meat and milk. OWC can produce a considerable volume of milk with excellent nutritional value in areas of the world where the traditional milk animals, the cow, the sheep and the goat, have difficulty surviving, not to speak of producing milk. It is therefore inconceivable that such a favorable animal species is so seldom used as a farm animal. Many people still believe that the camel is of low economic value and is synonymous with underdevelopment.

Only recently has the camel family been considered to aid man in many different respects. Understanding and utilizing this special gift could lead to the development of camel farms in famine areas and a reduction in human starvation.

This book is written as a gesture of appreciation from four European camel researchers for all that this animal family has meant to us.

Autumn 2001

U. Wernery, Dubai
O.-R. Kaaden, Munich
J. Kinne, Dubai
S. Bornstein, Uppsala

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The authors are deeply indebted to His Highness General Sheikh Mohammed Bin Rashid Al Maktoum, Minister of Defense of the United Arab Emirates, whose generosity helped realize the publication of the second edition of this book.

Sincere thanks are given to the owners of the Bin Hamoodah Group of Companies for their generous contribution to financing the publication of the second edition of this book and their interest in safeguarding camel breeding and racing traditions in the UAE.

The authors gratefully acknowledge the cooperation, help and advice from Dr. Ali Ridha, the Administrative Director of the Central Veterinary Research Laboratory. Dr. Ridha has taken a keen and critical interest in all of the authors' scientific work and has been our mentor during many years in a new culture.

Very special efforts have been contributed by the CVR Laboratory staff in Dubai: Dr. J. Sasse, Mrs. R. Wernery, Mr. O. Mathai, Mrs. R. Zachariah, Mrs. S. Joseph, Mrs. S. Korah, Mrs. L. George, Mr. Y. Abubakr, Mr. A. K. Nizarudeen, Mr. F. Joseph, Mr. A. Ali, Mr. Y. Ali, Mr. A. Siddique and Mr. N. Muthuvattil without whose help we could never have completed this work. With great enthusiasm and invaluable assistance, they helped to introduce new laboratory techniques and cared for our experimental animals.

We warmly thank the veterinarians and nutritionist who work for the ruling family of Dubai, Dr. A. M. Billah, Dr. J. Akbar, Dr. A. Ul-Haq, Dr. G. Munawar, Dr. M. Ali, Dr. A. Ali, Dr. H. Tesfamariam and Mr. J. Wensvoort, for their support. Their contributions and submission of specimens have made it possible for this laboratory to discover new facts regarding camel diseases.

The authors are particularly grateful to Mrs. S. Robinson, Mr. R. Babu and Mr. N. Chaudhry for their care and patience in typing the manuscript and to Mr. D. Wernery who introduced me to the world of computers and who had the painstaking job of typing most of the tables.

Many thanks go to the staff of the Camel Reproduction Laboratory in Nakhlee, Dr. J. A. Skidmore and Mr. M. Billah, for their support and to Dr. B. N. Kumar, who works for the Bin Hamoodah Group of Companies.

Many other people supported and helped us with this project, but we owe a particular debt of gratitude to Dr. E. Zabegina from Moscow and Dr. Zhao Xing-Xu from China, who introduced us to many excellent camel scientists in the former Soviet Union and China. We are also extremely grateful to Prof. M. E. Fowler from the USA, Prof. R. Gothe and Prof. M. Rommel from Germany for their valuable contributions.

Finally, I must thank my family, especially my wife Renate, for her invaluable assistance and advice as well as for her understanding of my absence from many social events.

Last, but not least, the authors are particularly thankful to the publisher, especially to Dr. A. Müller from Blackwell Wissenschafts-Verlag for his continuing support and the excellent design of the second edition of this book.

U. Wernery
O.-R. Kaaden
J. Kinne
S. Bornstein

Table of Contents

Abbreviations

AGID	Agar gel immunodiffusion test
CFT	Complement fixation test
CVRL	Central Veterinary Research Laboratory
ELISA	Enzyme-linked immunosorbent assay
ELMI	Electron microscopy
FAT	Fluorescent antibody test
NWC	New World camelids
OWC	Old World camelids
UAE	United Arab Emirates
SNT	Serum neutralizing antibody test

Introduction

Camelids have served the needs of people for thousands of years and have provided them with food, fiber and fuel. In many parts of the world they have also served as beasts of burden. They secured trade and communication throughout wide arid and semiarid expanses. To the Bedouin of the Arabian Peninsula and North and East Africa, the dromedary was, and still is in some parts, vital for survival in a most inhospitable environment. Bactrian camels inhabit the high deserts of Asia where they survive –40°C temperatures. For hundreds of years they have carried goods along the Old Silk Route to China. A few wild Bactrians still roam the steppes of the Gobi desert in Mongolia and China. In South America, the vicuña and guanaco remain wild species, while the llama and the alpaca are domesticated. They have adapted well to high altitude survival. In many countries camelids have now adapted to contained management, and in the last few years there has been a renaissance in both Old and New World camelids.

Until recently, scientific interest in camels and the majority of research projects involving camels have been concentrated in countries actively involved with the care and maintenance of the camel as a domesticated animal. A frequent opinion encountered in those countries not involved with camel husbandry is that the camel is an anachronism, an animal of the past and without a future (Wilson, 1984). It is therefore not surprising that many publications on camels appear in journals that are difficult to obtain or in lesser-known languages. There was obviously an urgent need for a comprehensive compilation and evaluation of the published literature for all those involved with camels. An important step in this direction was the publication of the bibliography *Sur le dromadaire et le chameau* by Saint-Martin et al. (1990), in which approximately 5500 pre-1990 publications regarding camels are catalogued by author and subject matter. Additional bibliographies about the camel can be found under Farid (1981), Mukasa-Mugerwa (1981), and Wilson et al. (1990).

Prior to 1987, approximately 1000 New World camelid veterinary references were published. During the period from 1987 to 1996 one thousand four hundred new references appeared in the world literature (Wernery et al., 1999).

The camelid family has become the focus of increasing study in the last few years. This has become apparent not only through the increase in scientific publications by, for example, Wilson (1984), Yagil (1985), Higgins (1986), Bitter (1986), Gründel (1988), Doose (1990), Saltin and Roose (1994), Wernery and Kaaden (1995), Manfield and Tinson (1996), Tibary and Anouassi (1997), Gauly (1997), Faye (1997), Wilson (1998), Fowler (1998), Beil (1999), Wernery et al. (1999), and Gahlot (2000), and the edition of a camel journal (Journal of Camel Practice and Research, editor Dr. T. K. Gahlot), but also through the increase in joint research projects between European universities and institutions in arid countries. This growing general interest in camelids also became evident when 300 camel experts from 30 countries took part in the First International Camel Conference in Dubai, United Arab Emirates, in February 1992 (Allen et al., 1992). Further international meetings and conferences took place in 1996 in Eilat, Israel, in 1997 in Al-Ain, UAE, and in 1999 in South and North America and in Morocco. Proceedings are available from most of these conferences.

As an important source of milk, meat and wool as well as transportation and labor, the camel should play a more important role than is currently the case in a world where food and energy reserves are dwindling (El-Gayoum, 1986). This is especially true as the camel is, due to its physiological attributes, the most suitable domestic mammal for uses in climatic extremes (Yagil, 1985; Wilson, 1989; George, 1992; Wernery, 1992).

For a long time it has been incorrectly assumed that one and two-humped camels derive from a sole wild species, i. e. the two-humped wild camel – *Camelus ferus*. There were two main reasons for this belief. Firstly, both the one and two-humped camels pass through a two-hump embryonic stage. Secondly, the crossbreeds between dromedaries and Bactrians are fertile. However, the latest osteological investigations on post-cranial skeletons of dromedaries *(Camelus dromedarius)* and Bactrian camels *(Camelus bactrianus)* have shown that they are in fact derived from two different species (Peters, 1997).

The tylopods originated in North America 50–60 million years ago (Tertiary period) at which time they branched into eight different families (Zeuner, 1963). They were at that time the size of hares. Six of the eight families died out in the middle Miocene. Then, five million years ago, the ancestors of the OWC migrated to northeast Asia across the isthmus today known as the Bering Strait. Today's OWC evolved from these early camels, branching out westwards. They were most widely spread during the Pleistocene era (ending two million years ago) when they reached as far as Eastern Europe, North and East Africa and eastern Asia (Koehler, 1981; Koehler-Rollefson, 1988). After some time, they died out in some of these regions. When the OWC migrated eastwards after crossing the Bering Strait, the ancestors of the humpless NWC migrated south over the newly formed isthmus between the half continents of North and South America (Sielmann, 1982). They populated South America where the different types are known today as llama (*Lama glama* – domesticated), vicuña (*Lama vicugna* – wild), guanaco (*Lama guanicoe* – wild) and alpaca (*Lama pacos* – domesticated) (Fig. 1). The

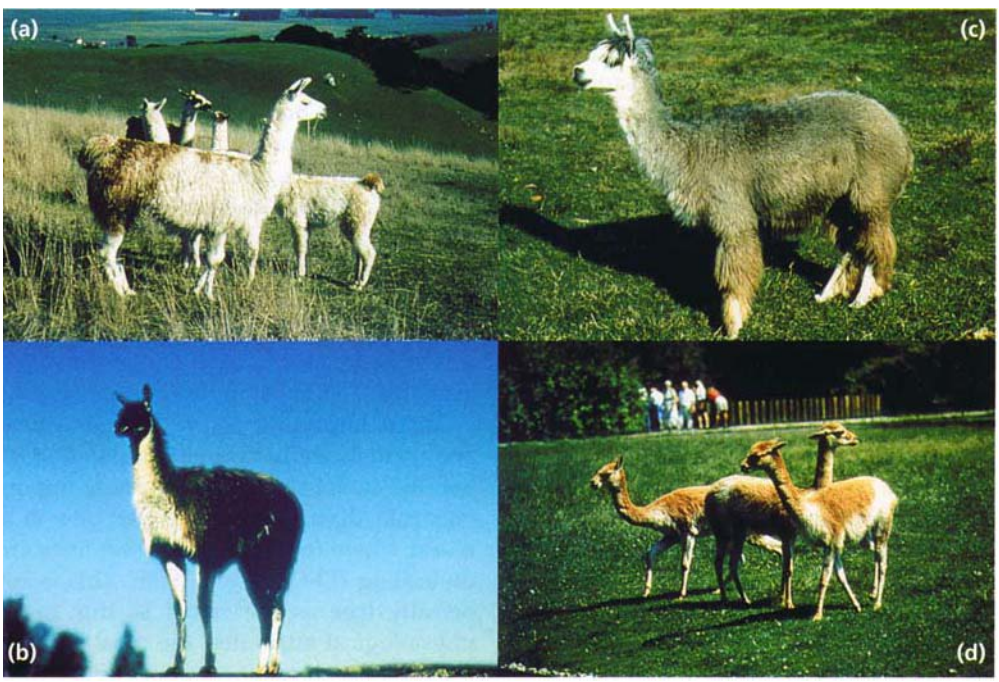

Figure 1 The four different South American camelids (courtesy of Prof. M. E. Fowler, USA) **(a)** Llama, **(b)** Alpaca, **(c)** Guanaco, **(d)** Vicuña

Table 1 Classification of camelids and other artiodactylids (Fowler, 1998)

Class	**Mammalia**	
Order	**Artiodactyla**	
Suborder	**Suiformes**	Hippopotamuses, swine, peccaries
Suborder	**Tylopoda**	Camelids
	Old World	*Camelus dromedarius* – dromedary camel *Camelus bactrianus* – Bactrian camel
	New World	*Lama glama* – llama *Lama pacos* – alpaca *Lama guanicoe* – guanaco *Vicugna vicugna* – vicuña *V. vicugna mensalis* (Peruvian) *V. vicugna vicugna* (Argentinean)
Suborder	**Ruminantia**	Cattle, sheep, goats, water buffalo, giraffe, deer, antelope, bison

OWC and NWC belong to the *Camelidae* (camel-like) family under the suborder *Tylopoda* (Table 1).

In North America, all camel species died out 10,000 years ago, the last being the genus Camelops, which was most probably hunted into extinction by the indigenous Indians. In South America today, between 7 and 8 million small camels have been counted (Peru, Bolivia) (Table 2). Llamas and alpacas have been domesticated there for 7,000 years and were among the first recorded domesticated animals, an achievement of high Indian culture.

Of the OWC, the two remaining species domesticated today are the one-humped camel or dromedary *(Camelus dromedarius)*, and the two-humped camel *(Camelus bactrianus)*, with the exception of a small, wild population of camels in China and Mongo-

Table 2 Estimated population of South American camelids (Carpio, 1991; Torres, 1992)

Country	**Llamas**	**Alpacas**	**Guanacos**	**Vicuñas**
Argentina	75,000	2,000	550,000	23,000
Bolivia	2,500,000	300,000	?	12,000
Chile	85,000	5,000	20,000	28,000
Peru	900,000	3,020,000	1,400	98,000
Australia	< 5,000	> 5,000	A few in zoos	0
Canada	> 6,000	> 2,000	< 100 in zoos	> 10
Europe	< 2,000	< 1,000	< 100 in zoos	< 100 in zoos
United States	> 110,000	> 9,500	145, mostly in zoos	0
In ISIS registry in zoos*	343	303	397	100
Total	**3,683,343**	**3,344,803**	**572,142**	**161,210**
Grand Total				**7,761,498**

* ISIS = International Species Inventory System

Figure 2a, b (a) The Bactrian camel, rutting male (courtesy of Dr. Zhao Xing-Xu, China) and **(b)** a wild Bactrian camel *(Camelus bactrianus ferus)* with a newborn calf (courtesy of J. Hare, The Wild Camel Protection Foundation, School Farm Benenden Kent TN174EN, UK)

lia (Fig. 2b). However, until today it has been impossible to establish whether the remaining populations of Bactrian camels in these regions are feral or genuinely wild camels. The dromedary's role in North and East Africa, Arabia and the Near East is mainly one of transportation of goods and man. The Bactrian camels fulfill a similar role in Mongolia, Western Siberia, Transcaspian, Asia Minor, Iran and Afghanistan. The extent of the OWC habitat and worldwide population is shown in Fig. 3 and Table 3.

Table 3 Old World camel population (Higgins, 1986; Bhattacharya, 1988; Wilson et al., 1990; Wernery, 1997)

Africa	Camel Population	Asia	Camel Population
Algeria	150,000	Afghanistan	270,000
Chad	446,000	India	1,150,000
Djibouti	60,000	Iran	27,000
Egypt	90,000	Iraq	250,000
Ethiopia	1,000,000	Israel	11,000
Kenya	610,000	Jordan	14,000
Libya	135,000	Kuwait	5,000
Mali	173,000	Mongolia	580,000
Mauritania	800,000	Oman	6,000
Morocco	230,000	Pakistan	880,000
Niger	410,000	Qatar	10,000
Nigeria	18,000	Saudi Arabia	780,000
Senegal	6,000	Syria	7,000
Somalia	6,000,000	Turkey	12,000
Sudan	2,600,000	United Arab Emirates	120,000
Tunisia	173,000	Yemen	210,000
Upper Volta	6,000	IPS*	200,000
Western Sahara	92,000	China	600,000
		Australia	120,000
		Canary Islands	4,000
Total	**12,999,000**	**Total**	**5,256,000**
	Grand Total		**18,255,000**

* Independent states of the Soviet Union

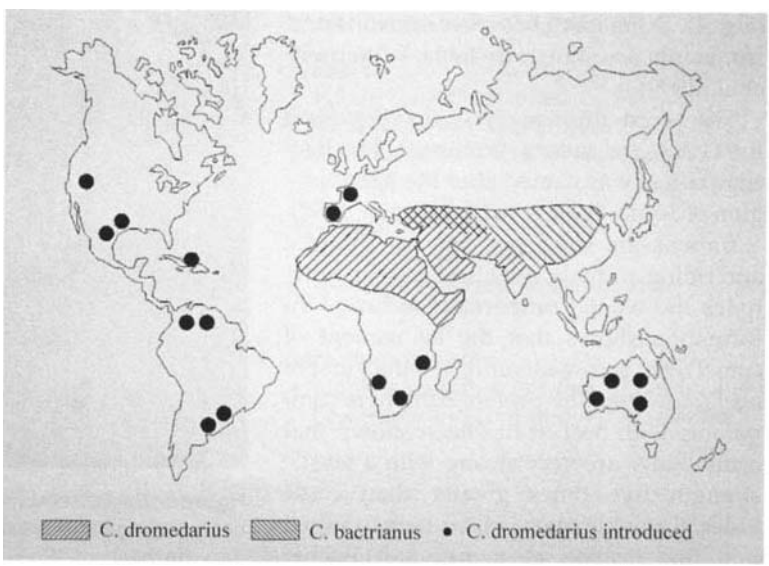

Figure 3 Distribution of *C. dromedarius* and *C. bactrianus*

OWC have adapted marvelously to life in either hot or cold environments and NWC to life in high altitudes. Sophisticated mechanisms have evolved that guarantee survival of this unique animal family under extreme conditions.

Camels regurgitate and re-chew their food, thus ruminating. However, in strict taxonomic terms, they are not recognized as belonging to the Ruminantia. Their three forestomachs are called compartments

Figure 4 The forestomach system of *Tylopoda*

(Fig. 4). Differences between camelids and ruminants are shown in Table 4 (Wernery et al., 1999).

The word dromedary is derived from the Greek and means "running". The Bactrian camel was named after the Bactria region of South-West Asia (Allen et al., 1992).

Camels are used not only as draught and riding animals, but also for meat, milk, hides and wool. Comparative technical information shows that the fat content of camel meat is considerably less than that of beef. However, the protein content is comparable with beef. It has been shown that camel hides are very strong with a tensile strength five times greater than cattle hides. Camel leather is now being crafted into fine fashion garments, soft leather wallets, handbags and purses. Wool is an important dromedary by-product in many camel-producing countries. The average wool clip is 3.28 kg for males and 2.10 kg for females. The Bedouins produce carpets and tents from camel wool. Camel wool is one of the world's most expensive natural animal fibers. It is similar to cashmere in both fiber diameter and texture. Of the OWC, the Bactrian camel produces superior wool to the dromedary (Anonymous, 1995). Male Bactrians can produce 10–16 kg of the magnificent fiber, but unfortunately there is very little interest in the camel wool industry. However, there is an increasing demand for NWC fiber since it is known that the vicuña produces the finest wool of all animals. The interest in its fiber has saved this magnificent animal from extinction. It produces only 200 grams of wool per year. This is one of the reasons why scientists have been involved in cross-

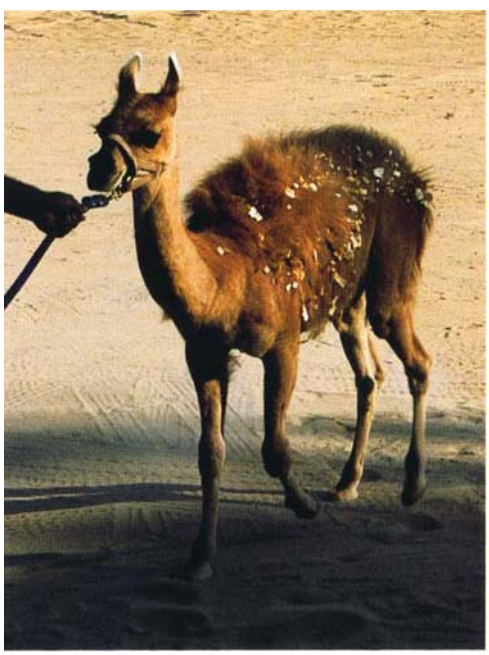

Figure 5 Crossbreed (male, 10 months old) between a guanaco (mother) and a dromedary (father)

Table 4 Differences between camelids and ruminants

Camelids	Ruminants
Evolutionary pathways diverged 40 million years ago	**Evolutionary** pathways diverged 40 million years ago
Blood • red blood cells elliptical and small (6.5 μ) • predominant white blood cell is neutrophil	**Blood** • red blood cells round and larger (10 μ) • predominant white blood cell is lymphocyte
Foot • has toenails and soft pad • second and third phalanges are horizontal	**Foot** • has hooves and sole • second and third phalanges are nearly vertical
Digestive System • foregut fermenter, with regurgitation, re-chewing and re-swallowing • stomach – 3 compartments, resistant to bloat • compartment 1 has a stratified squamous epithelium • 2 glandular sacs in C1, act as "reserve water tanks"	**Digestive System** • same (parallel evolution) • stomach – 4, susceptible to bloat • papillated epithelium • no glandular sacs
Reproduction • induced ovulator • no estrous cycle • follicular wave cycle • copulation in prone position • placenta diffusa • epidermal membrane surrounding fetus • cartilaginous projection on tip of penis • ejaculation prolonged	**Reproduction** • spontaneous ovulation • estrous cycle • no follicular wave cycle • copulation in standing position • placenta cotyledonary • no epidermal membrane on fetus • no cartilaginous projection on tip of penis • ejaculation short and intense
Urinary • kidney smooth and elliptical • suburethral diverticulum in female at external urethral orifice • dorsal urethral recess	**Urinary** • kidney smooth or lobed • no suburethral diverticulum • dorsal urethral recess in some species
Parasites • unique lice and coccidia • share some gastrointestinal nematodes with cattle, sheep and goats	**Parasites** • unique lice and coccidia • share gastrointestinal nematodes
Infectious diseases • minimally susceptible to tuberculosis • bovine brucellosis is rare • mild susceptibility to foot-and-mouth disease • rare clinical disease with other bovine and ovine viral diseases	**Infectious diseases** • highly susceptible to tuberculosis, bovine brucellosis and foot-and-mouth disease

breeding NWC with OWC. The first successful hybrid was produced in the United Arab Emirates (UAE) between a male dromedary and a female guanaco (Fig. 5).

Although there is evidence of the Bactrian camels' ancestors discovered at pre-historic sites in Kazakhstan and Mongolia, little is known about the dromedary's ancestry. An ancestor of the dromedary camel, the "giant" camel, is known zoologically as *Camelus thomasi* (named after the French paleontologist Thomas). *Camelus thomasi* is now considered a possible ancestor of the domestic one-humped camel (Peters, 1998). These camels are presumed to have existed in a wild state during the last ice age in North Africa and in the Negev Desert, where they probably died out some 12,000 to 20,000 years ago during extremely cold temperatures coupled with drought. However, no skeletal remains or rock paintings of camels in the Sahara mountains support this theory. Evidence of wild camels was only found once in South West Asia, at the beginning of the Holocene era. The remains were found at Sihi, a village in Yemen, and were dated at 7000 BC.

It is widely believed that the dromedary was domesticated 4,500 years ago, whereas the wild dromedary population died out 1000 BC. Exactly when the wild camel became domesticated is uncertain, but it is believed to have begun on the Arabian Peninsula (Wensvoort, 1991). Bones excavated at trading settlements in Jericho, Shar-I-Sokhta and Umm Al Nar (near the city of Abu Dhabi) prove that domestication began at that time. It was written in the Bible that around 1100 BC the Median Bedouin tribes used dromedaries to occupy Palestine. In 1000 BC large dromedary caravans brought incense from Oman and Yemen to the Mediterranean, which made both countries indescribably rich. Archaeologists are still trying to locate the fabled city of Ubar (Shisr) that was supposedly situated in Dhofar, the southernmost province of Oman. This city was the center of

Figure 6 Routes of the incense trade

the incense trade, from where the camel caravans made their way through Marib, Medina and Petra towards Gaza and the Mediterranean. The other incense routes through the great Arabian deserts towards Gerrha on the Arabian Gulf could only be traversed with the help of the camel (Fig. 6).

Camel breeding may have increased because of the lucrative incense trade. These heavily laden "ships of the desert" took about 50–70 days to cross the deadly stretch of land between Marib and Petra. The caravanserai reached its zenith during the reign of the Nabateen. Terracotta finds from Petra are richly decorated with dromedaries. With the advent of Christianity the incense trade began to decline, and Arabia Felix reverted to the deserted Empty Quarter. After the caravans vanished, only the Bedouins continued to utilize the dromedary.

When trade began with Arabia, dromedary numbers increased in Africa. It is presumed that between 1500–2000 BC, dromedaries spread into Africa from the Arabian Peninsula via the Horn of Africa. Beyond Somalia, the country with the highest proportion of dromedaries per person, the "ship of the desert" spread north and westwards. However, it was not introduced into Tunisia and the Atlas countries before Hellenistic times.

Dromedaries were not only introduced into countries with temperate climates such as Europe, South America and the Caribbean, but also into Australia and southern Africa, which have hot climates. An estimated 10,000–12,000 camels imported into Australia between 1860 and 1907 were used as draught and riding animals by people pioneering the dry interior (Viswanathan, 1991). The camels introduced into Australia were almost exclusively dromedaries, because they are highly suited to the Australian desert climate. Most of the camels were released in the mid-1920s, when motor vehicles began operating in the central areas of Australia. In the semi-arid deserts of Australia they established free-ranging herds, which nowadays number approximately 200,000 animals. These feral camels are scattered throughout the arid interior of Australia with an estimated 50% in Western Australia, 25% in the Northern Territory, and 25% in western Queensland and northern South Australia. In the late 1960s, there was renewed interest in camels, and by 1970, Australia had two camel tourist businesses with camel races being held around Australia (Anonymous, 1995). Several races were held in Sydney in August 1998 (with the support of the UAE) in preparation for the Olympic Games in 2000.

Dromedaries were also brought into southern Africa, mainly Namibia, around 1890. They were used by the German *Schutztruppe* in Namibia until the end of World War I for three reasons. Firstly, only dromedaries could survive in the Namibian and Kalahari deserts; secondly, oxen were eradicated by rinderpest and foot-and-mouth disease; and thirdly, horses were severely decimated by the devastating African horse sickness virus. In 1906, Lorenz Hagenbeck shipped 2,000 Sudanese camels to the small outpost of Swakopmund in Namibia. After the Versailles Peace Treaty (1919), the English police force then took possession of all remaining camels in Namibia. However, as in Australia, after motorized transport became popular, the camels were abandoned and it is believed that as a result of being eaten by lions and bushmen, they disappeared in southern Africa in the late 1960s (Massmann, 1981).

Dromedaries were also used in the United States after the Mexican war of the 1840s, on mail express routes across the newly acquired arid regions, but they were later eradicated.

In Europe, camel societies have emerged during the last two decades and animals have been used to attract tourists. In August 1997, camel races were held at Berlin's famous horse race course *Hoppegarten* in

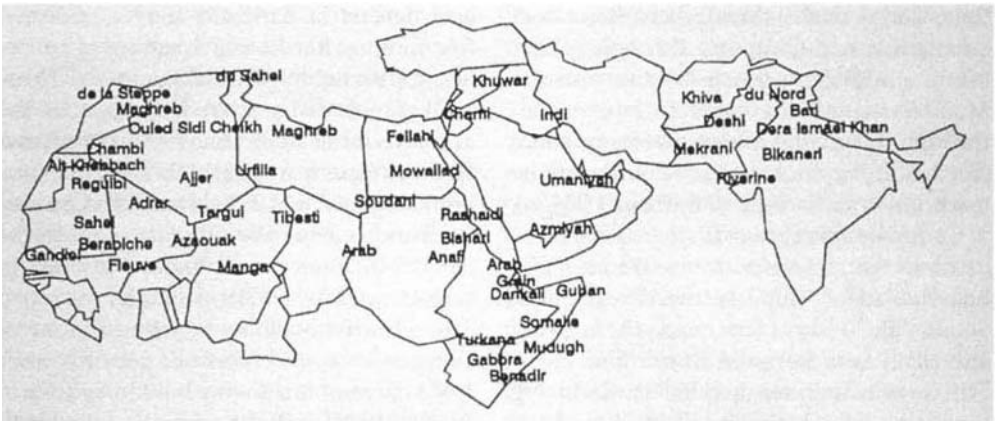

Figure 7 Geographical distribution of dromedary breeds (after Wilson, 1998)

front of 60,000 spectators. However, this was not the first camel race in Europe, as was then claimed. The first races took place in Cologne-Weidenpesch in 1969 with Moroccan camels (Leue, 1969).

Since the 1980s, the dromedary has again become popular, not only with scientists, but also in the countries where it is used for riding and transport. Its milk, skin and meat are all utilized and, additionally, it has become a tourist attraction. The future of the dromedary species is assured despite the competition of modern transport and other domesticated animals, and it offers no threat to domesticated animals or any endangered wildlife.

Scientists have recently intensified their study of the dromedary and are debating whether there are different dromedary "breeds". Until now, the dromedary has been classified in the following ways – by naming them after the tribes who rear them, or whether they are riding or transport camels, by their color, geographical background (Fig. 7), physical characteristics or their use for milk, meat, or racing (Wilson, 1998). This categorization has given rise to the classification of dromedaries under 48 "breeds" in 9 regions and sub-regions, under 3 main groups and 8 subgroups. The confusion is compounded because of the crossbreeding of Bactrian and dromedaries in Russia, Turkey, Afghanistan and Syria. As the second generation of these crossbreeds (Tulu) are generally weak and susceptible to diseases and the fourth generation is infertile, the breeders have to start all over again to achieve a good crossbreed (Fig. 8).

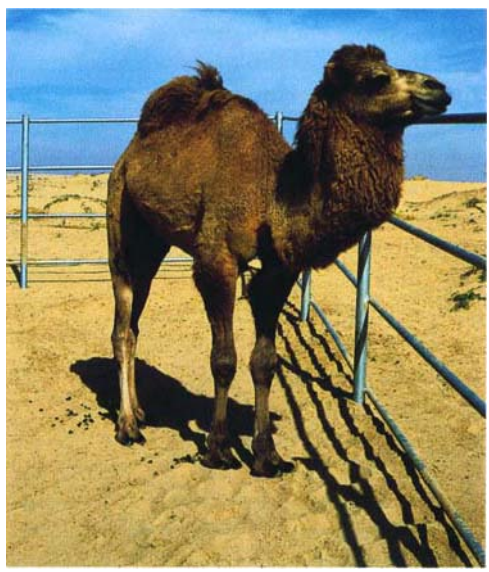

Figure 8 Crossbreed (Tulu) (female, 2 years old) between a Bactrian male and a female dromedary

In its great genetic diversity, the dromedary raises many questions which are not easily explained.

Thousands of years before the Pyramids were built, the Bedouins and their dromedary herds wandered through the great Arabian deserts and lived undisturbed throughout the successive reigns of the Pharaohs, Sumerians, Assyrians, Phoenicians, Greeks, Romans and Turks. The tribes could only survive in the desert thanks to the dromedary, which the Bedouins call *Ata Allah* (God's Gift). Only through its indispensable patience and perseverance did it enable survival in the perpetual sands. Over this period, the desert played a big part in the evolution of the dromedary as the only domesticated animal to survive in such extreme conditions. Not only does the dromedary produce milk, meat and wool, but it is also used as transport over thousands of kilometers. Not only the "ship of the desert's" ability to survive in the hottest climates, but its natural resistance to such deadly animal diseases as rinderpest and African horse sicknes makes it indispensable to its owner.

In an effort to find new grazing areas, it was often necessary for the Bedouin tribes to cross enemy territory. This sometimes resulted in feuds and skirmishes. Obviously, the tribe with the quickest and most nimble dromedaries had the most chance of surviving. Sir Wilfred Thesiger, in his book *Arabian Sands*, described disputes that still occurred until some 30 years ago. However, not only were the dromedaries vital during tribal conflicts, but the Bedouins also used them for racing during social occasions, such as weddings or births. The quickest dromedaries were selected to run over short courses.

In the Arabian Desert, it was the Bedouin who managed to breed the precious Arabian horse, the Saluki dog and the dromedary. In its perseverance, intelligence and beauty, the Arabian dromedary, bred over hundreds of years in one of the hottest climates on earth, is comparable with the

Table 5 Dromedary population on the Arabian Peninsula

Kuwait	5,000
Oman	6,000
Qatar	10,000
Saudi Arabia	780,000
United Arab Emirates	120,000
Yemen	200,000
Total	**1,121,000**

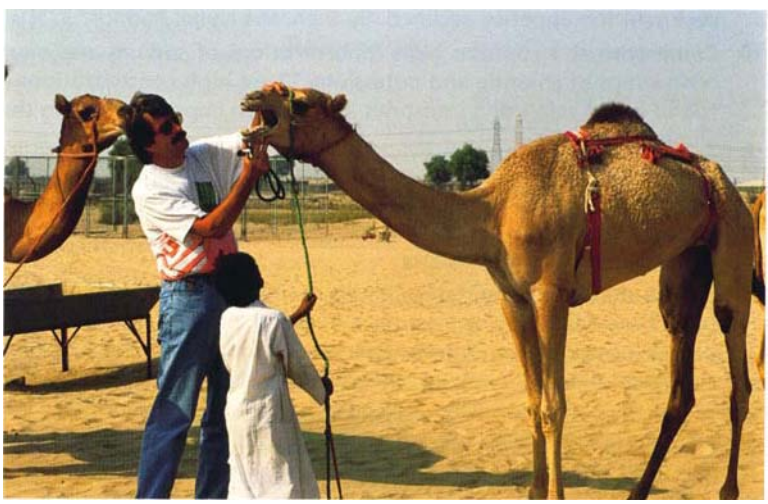

Figure 9 One of the authors examines a valuable female asil dromedary

Table 6 The main physiological particulars of dromedaries

1. Cattle lose 20–40 liters of fluid a day via their feces. Camels lose 1.3 liters of fecal water. This is one of the primary ways of combating water deprivation in the desert.
2. Thermoregulation in the camel is greatly affected by the availability of drinking water. The camel has a great dehydration tolerance; it can lose one third of its body weight in water without suffering any ill effects.
3. A dehydrated camel reacts to changes in external temperature. In the morning when the desert is cold, the camel's body temperature is low: 34.0°C. In the late afternoon the body temperature can reach 42°C. The camel adapts its body temperature to the outside temperature, preventing it from sweating. A rise in body temperature saves a camel a lot of water that would otherwise be used to dissipate the heat load. High blood temperature would do permanent damage to the brain and retina cells of the eyes. However, camels are able to cool the brain and eyes through extraction of water from exhaled air. The water vapor from the exhaled air stays in the long nose and cools the carotid rete, a network of small blood vessels supplying the brain and eyes.
4. Goats kept in an open yard with no shade are unable to survive more than 3 days without water; Barki sheep also die after 3 days of dehydration, but camels can survive 20 to 30 days without drinking water.
5. A 600 kg camel can replenish its entire water deficit of 200 liters in 3 minutes. Camel erythrocytes are extremely resistant to hypotonicity. Bedouin goats kept 4 days without water die of hemolysis after replenishing a 40% water loss in 8 minutes.
7. In camels, water rapidly enters the bloodstream after drinking. After 4 hours water is apparently equilibrated throughout most of the body. No other animal has such a rapid entry of water into the blood.
8. The hump is an accumulation of fat for the time when energy is needed. It indirectly aids in cooling the body as the accumulation of fat leaves the subcutis of the body fat free, allowing easy dissipation of heat.
9. In a dehydrated camel, alimentary tract water is its body's sole source of water because this water is continuously absorbed from the intestines. Camels hold 75% of their weight in fluids, and as long as the camel continues to eat, water will be present in the stomach. Camels withstand more than 3 weeks without drinking water and still continue to eat normally, because their stomachs still contain relatively large quantities of fluid. In a trial, a camel was dehydrated for 51 days and was only fed on dry grass. At the end of the experiment the appetite declined. By then, the camel had lost 37% of its body weight.
10. Compartment 1 contains high concentrations of sodium and bicarbonate and low concentrations of chloride and potassium. These high concentrations of electrolytes are also found in the saliva and intestines and play an important role in the camel's utilization of alimentary water.
11. Camel kidneys have long loops of Henle, and urine production is greatly decreased in the dehydrated camel. Salt is also well handled by the camel kidneys. Camels can drink seawater without showing any side effects. Camel can excrete urine with a salt concentration almost twice that of seawater.
12. Dehydrated camels can "store" sugar in their blood in order not to lose water through the urine (sugar is highly hygroscopic). In a trial, blood sugar rose as high as 1300 mg% without the appearance of glucose in urine. As soon as drinking water was made available, an enormous diuresis followed and blood glucose returned to normal.
13. A dehydrated camel is able to continue lactation.
14. Camels mate in crouched position. They are induced ovulators with a relatively short mating period. Their gestation period is 13 months.

Arabian horse. There is no other dromedary that compares with the Arabian. Through breeding, it has become an agile, fast, long-legged, slender, brown racing dromedary with fine limbs and a long head (Fig. 9). Although no studbooks exist, the Bedouin are extremely careful to keep the bloodlines pure. In the 30 years since the oil boom began, camel racing has gone through a fundamental change.

In the last few years, the worldwide camel population has risen from 17.5 million (Wilson et al., 1990) to 18.3 million (Table 3). The camel population has decreased in only a few countries, such as Libya and the Gulf States, where oil has brought nomadism to a virtual standstill (Wilson, 1984). However, in recent years, an opposite trend has been observed in the UAE where the dromedary is experiencing a renaissance resulting in a revival of the old Bedouin tradition of camel racing. What was earlier seen as playful competition and a pleasant pastime between the Bedouin has become a scientifically founded racing discipline following the oil boom of the 1960s. Based on this development, more than 100,000 racing camels are kept in the UAE. In the cooler months between September and May, competitions are held on 20 racetracks throughout the Emirates. Based on the age of the animal, the dromedary competes at distances between 3 and 10 kilometers. A dromedary can cover the 10 km course in 17 to 18 minutes (Wernery, 1992).

Due to a number of specific anatomical and physiological characteristics, the dromedary can survive and perform tasks in the extreme climate of the desert that can be utilized by man (Schmidt-Nielsen, 1964) (Table 6).

A further advantage is the low susceptibility of the camelids to disease (Fazil and Hofmann, 1981). This is especially true of viral diseases, although bacterial ailments play a larger role. Both the camelids' resistance to a number of pathogenic microorganisms, as extensively examined by scientists in the Institute for Horticulture and Animal Hygiene in Goettingen (El-Gayoum, 1986; Margan, 1987), and the previous lack of interest in the camel family in general, may have been decisive in the dearth of publications on infectious diseases of camelids. The second edition of this book will attempt to close this gap by surveying and compiling the published literature regarding bacterial, viral and fungal diseases as well as pathology and parasitology in the camelids as completely as possible. The majority of the literature encompasses the one-humped *Camelus dromedarius* as the available literature on the two-humped *Camelus bactrianus* is unfortunately very difficult to obtain. As the exchange of scientific research with countries where the Bactrian camel lives is now improving, it is hoped that more comprehensive data will soon become accessible. New scientific findings of NWC are also included.

In addition to a compilation of the known literature, results of the authors' personal research conducted since 1987 on a camel population of 30,000 racing dromedaries (including breeding animals) in the UAE, in conjunction with various research institutes abroad, will also be presented.

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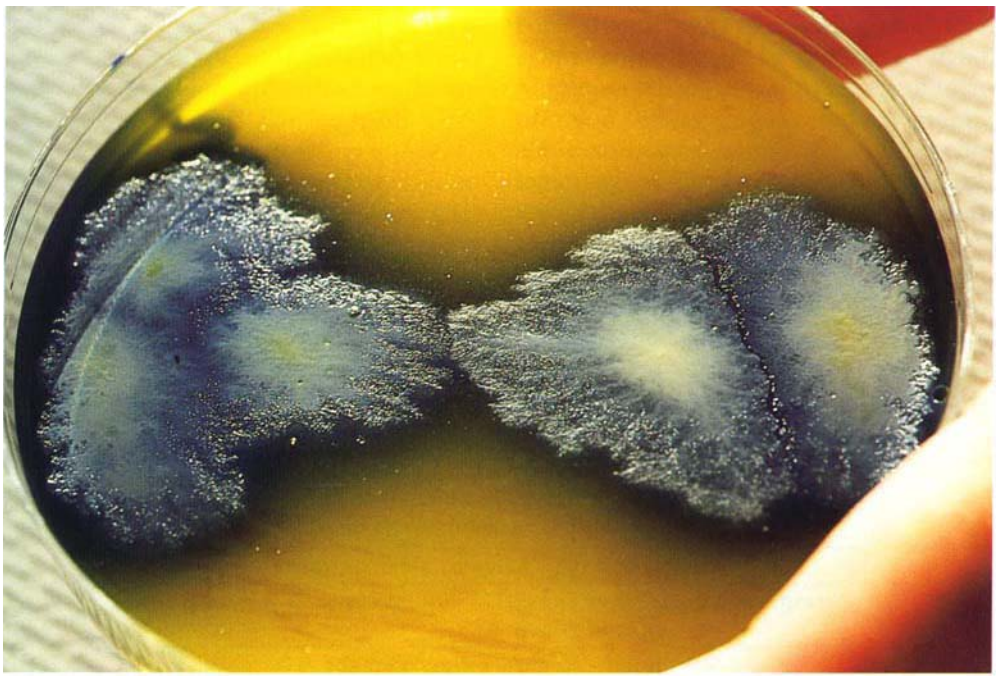
1 Bacterial Diseases



1.1 General Survey

1.1.1 Anaerobic Infections

Clostridial diseases are a constant threat to livestock in many parts of the world. Clostridia are all potent producers of exotoxins upon which their pathogenicity depends. Clostridial organisms are commonly present in soils and the intestinal tract of animals, including man, and cause disease only in special circumstances. The ubiquitous character of clostridial bacteria makes eradication of clostridiosis virtually impossible and necessitates control by prophylaxis. Both NWC and OWC may suffer from some of the clostridial diseases (Wernery and Kaaden, 1995; Fowler, 1998).

Etiology Clostridial diseases are caused by bacteria of the genus *Clostridium*. Clostridium bacteria are large, Gram-positive, anaerobic, endospore-producing rods. The spores bulge the mother cell. *C. perfringens* possesses a capsule in animal tissue and is non-motile. Clostridia are oxidase-negative and catalase-negative and the anaerobic requirements vary among the species. Most of the pathogenic species produce one or more exotoxins of varying potency. The vegetative organism is capable of forming spores that are able to survive long periods of time in the soil. Contaminated soil can contain up to 10^5 *Clostridium perfringens* spores per gram of soil (Seifert, 1992).

Epidemiology and Clinical Signs The older classical etiological classification of anaerobic infections that ascribes particular clinical signs to a specific clostridial agent can no longer be considered valid. Modern methods of infectious agent identification utilizing gas chromatography allow an exact determination of the etiological agent. These methods have also allowed the division of the epidemic anaerobic complex into three groups:
- gas edema complex,
- enterotoxemia complex,
- intoxication complex.

This new development is summarized in Table 7.

Table 7 Etiologic differentiation of the most important clostridial infections and intoxications in domestic animals modified after Seifert (1992)

Enterotoxemia complex – per os – enteral	
C. perfringens*, type A–F; *C. sordellii*; *C. difficile	Errors in husbandry, overgrazing, overcrowding
Gas edema complex – per os – parenteral	
***C. chauvoei*; *C. haemolyticum*; *C. histolyticum*; *C. novyi*, type A–C; *C. perfringens*, type A–F; *C. septicum*; *C. sordellii*; *C. chicamensis*; Madagascar wild strains 217, 335, 735; Mexico wild strains (809 and others)**	Changes in intestinal permeability, skin and mucosal lesions, periods of drought, hard lignin-containing feed, lack of food, overcrowding
Intoxication complex – per os	
***C. botulinum*, type A–F, *C. perfringens*, type A–F**	Errors in husbandry, overcrowding, mineral deficiency, P-deficiency, errors in nutrition
Intoxication complex – parenteral	
C. tetani	deep, anaerobic wounds

Diseases caused by clostridia are often difficult to identify in the tropics due to indigenous ecological influences, making diagnosis a challenge. *C. perfringens* types A, B and C, *C. novyi*, *C. chauvoei* and *C. septicum* have all been isolated from camelids.

Gas Edema Complex

The causative agents of the gas edema complex, which according to Seifert (1992) include the following diseases:

- black-quarter (blackleg),
- malignant edema,
- bacillary hemoglobinuria,
- infectious necrotizing hepatitis

are seldom isolated from camelids. As most of the available literature is outdated, it is possible that these disorders were falsely diagnosed in the past due to the prevailing incomplete, traditional analytical methods used. Current techniques have identified the following causative agents of the gas edema complex (Seifert, 1992):

- *C. chauvoei*, *C. septicum*, *C. chicamensis*, wild strains that have been exactingly characterized (335 and 735 Madagascar, 805 Mexico);
- *C. histolyticum*, *C. sordellii*, *C. novyi* type A–C, *C. haemolyticum*;
- *C. perfringens* type A–F and wild strains (217 Madagascar).

C. chauvoei infections in dromedaries have been reported as possibly occurring in North and East Africa, as well as in Chad and India (Gatt Rutter and Mack, 1963), but these reports are contradictory. With the exception of Cross (1919), Curasson (1947) believes that many previous authors have confused black-quarter with true anthrax caused by *Bacillus anthracis*. The progression of both disorders is similar, beginning with subcutaneous swellings on the shoulders that lead to the animal's death within 2 to 3 days. Hutyra et al. (1946) reported that camels were not susceptible to gas edema; however, Cross (1919) was able to elicit the disorder experimentally in three dromedaries through intramuscular injection of *C. chauvoei*. The type of swelling should allow the differentiation between gas edema and anthrax. Recent publications regarding gas edema in camels are not known.

Blackleg has been produced experimentally in alpacas, but there is one report of natural infection in a female llama that died suddenly. The causative agent was *C. novyi* (Anonymous, 1998). It is believed that OWC and NWC are more resistant to blackleg infections than bovines.

Malignant edema is an economically important disease in alpacas in Peru and has also been associated with rattlesnake bites in llamas in Colorado (Moro Sommo, 1956; Fowler, 1998). The disease in lamoids is caused by *C. septicum* with two types of syndromes: the typical wound infection and edema and the acute systemic disease, which may kill animals instantly.

The other two diseases of the gas edema complex, bacillary hemoglobinuria and infectious necrotizing hepatitis, have not been reported in *Camelidae*.

Enterotoxemia Complex

All types of *C. perfringens* as well as *C. sordellii* and *C. spiroforme* can cause the enterotoxemia complex. *C. perfringens*, most frequently type A (Bisping and Amtsberg, 1988), is also found in the intestines of healthy animals so that cultural evidence of *C. perfringens* has little disease-predictive value. Enterotoxemia caused by *C. perfringens* is found all over the world and is also found in all types of domestic animals. According to Seifert (1992), factors predisposing to disease include dietary errors, climatic influences, change of pasture, transportation, and weighing of animals.

Acute and subacute enterotoxemia as well as hemorrhagic enteritis due to *C. per-*

fringens, types A, C and D have been described in camels by Moebuu et al. (1966), Ipatenko (1974), Chauhan et al. (1985) and Gameel et al. (1986). Fowler (1998) has reported enterotoxemia due to *C. perfringens* types A, C and D in NWC.

Extensive studies of *C. perfringens* type A outbreaks in racing dromedaries in the UAE have been performed by Wernery et al. (1991), Seifert et al. (1992), Wernery et al. (1992b) and Wernery and Kaaden (1995). Peracute and acute enterotoxemia in breeding and racing dromedaries as well as severe myocardial degeneration and "pulpy kidney" in dromedary calves are known to occur. For all three age groups of dromedaries, predisposing etiological factors were proven to be responsible for the outbreaks.

In a herd of 90 breeding animals, 71% of the dromedaries were found to have an acute *Trypanosoma evansi* infection. Trypanosomosis is known to be able to cause immune suppression in domesticated animals (Losos, 1986), and may have been the predisposing factor for the peracute *C. perfringens* outbreak in this group, since nutritional errors and environmental influences had been excluded. The camels affected exhibited the following clinical signs:

- perspiration
- muscle tremor
- ataxia
- aggression
- hyperexcitability
- seizures

Affected animals died within one hour after the onset of clinical signs.

The pathological changes found in autopsied animals were mild. They included:
- petechiae in the thoracic musculature,
- petechiae in the cerebellum and brainstem,
- petechiae in the pharyngeal mucosa,
- subpleural (Fig. 10) and subepicardial petechiae,
- petechiae in the mucosa of the third compartment (Fig. 11) and the stomach,
- hydropericardium with fibrinous exudate,
- dark kidneys, with adherence of the capsule to the parenchyma (Fig. 12).

In another incident, salmonella paved the way for the outbreak of *C. perfringens* type A in racing dromedaries. The animals developed intractable diarrhea and died after 4 days. In those animals autopsied, even more severe pathological changes were

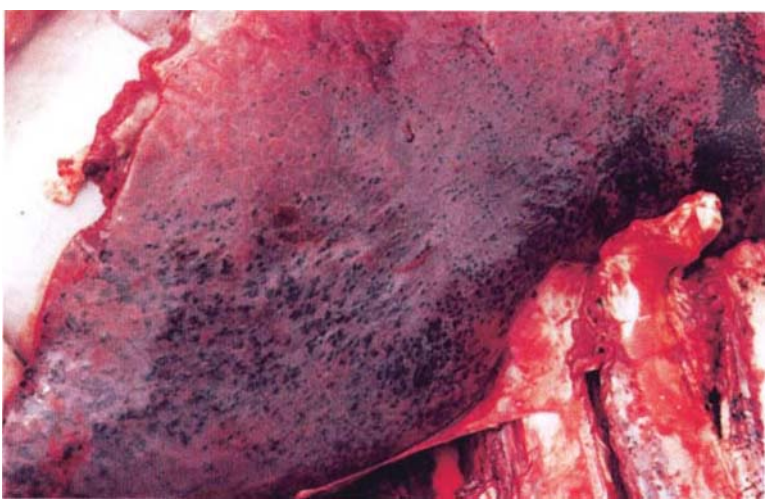


Figure 10 *C. perfringens* enterotoxemia: subpleural hemorrhages

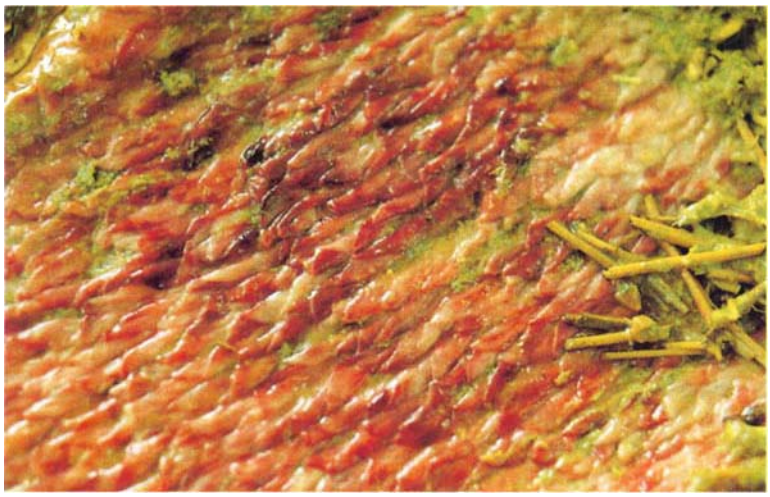


Figure 11 *C. perfringens* enterotoxemia: petechial hemorrhages in compartment 3

found in the same organs as listed above. These included severe hemorrhagic colitis, hydropericardium with fibrinous exudate and ecchymotic changes in compartment 3 and the stomach.

A further important etiological factor in the outbreak of enterotoxemia in racing dromedaries is a nutritional error prior to competition. Most likely due to ignorance, dromedaries are fed large amounts of uncrushed barley, cow milk, honey and alfalfa. At autopsy, large amounts of undigested milk or barley (Figs. 13 and 14) are found in their abomasum. The stress of racing is also certain to play an important role in the development of peracute enterotoxemia.

The dromedary calf exhibits distinctive features when affected by the enterotoxemia complex. The target organs for *C. perfringens* type A toxins in young dromedaries are the heart and kidneys. Wernery et al. (1992b) have reported severe myocardial degeneration (Figs. 15 and 16) and

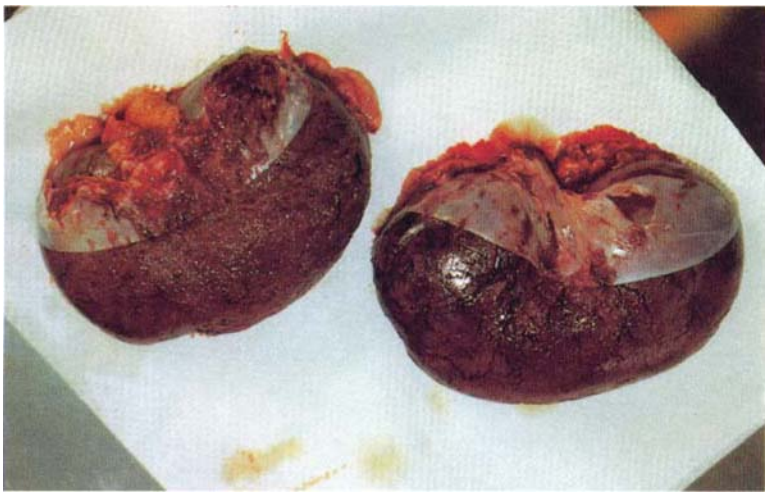


Figure 12 *C. perfringens* enterotoxemia: kidney capsule adherent to parenchyma

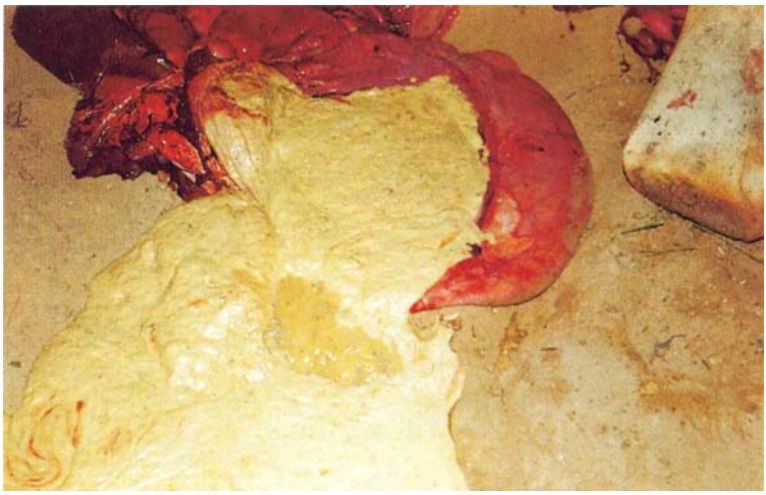


Figure 13 *C. perfringens* enterotoxemia: undigested milk in the gastric system of a racing dromedary

"pulpy kidney" (Fig. 17) in 4–6-week-old dromedary calves.

Degeneration, calcification and necrosis of the myocardium in 3 to 5-week-old camel calves in Saudi Arabia that died due to *C. perfringens* type D enterotoxemia have also been described by El-Sanousi and Gameel (1993). A predisposing factor for this disorder appears to be weaning. Between 4 and 6 weeks of age, the young calves begin to take nourishment other than milk. Autopsy findings in dromedary calves revealed, in addition to curdled milk and small amounts of roughage, increasing amounts of sand in the developing compartments (Fig. 18).

Examination of soil samples from these herds found up to 10^4 *C. perfringens* vegetative cells per gram of soil. Although the paddocks were cleaned daily, the sand where the breeding camels had been kept for years was heavily contaminated with vegetative cells and spores from clostridia. This situation represents a continuous risk

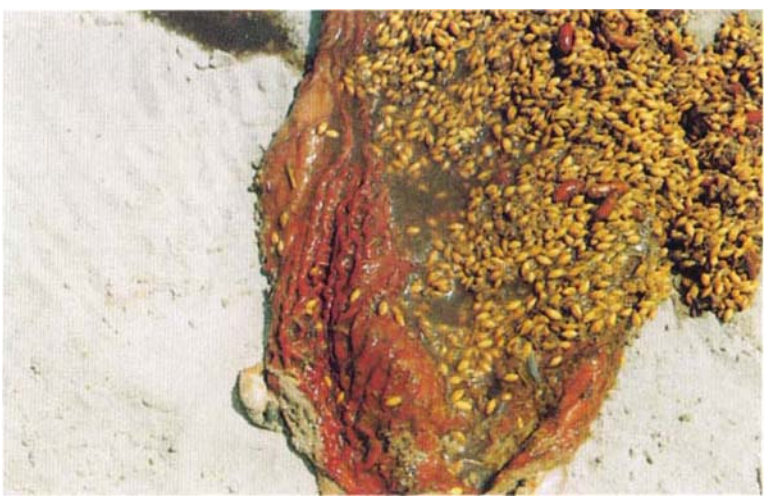


Figure 14 *C. perfringens* enterotoxemia: undigested barley in the gastric system of a racing dromedary

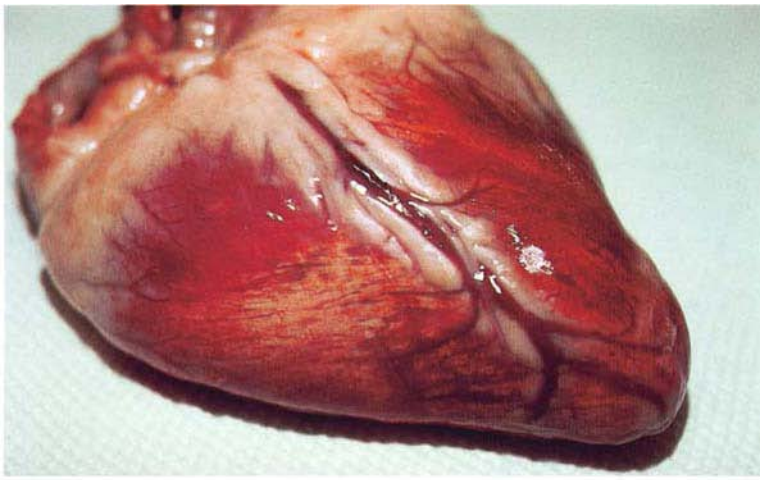


Figure 15 *C. perfringens* enterotoxemia in a young dromedary: severe myocardial degeneration

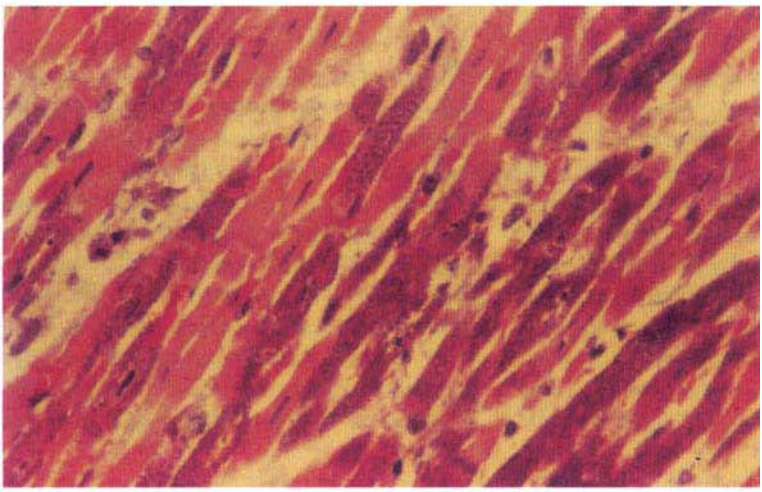


Figure 16 *C. perfringens* enterotoxemia in a young dromedary: hyaline degeneration of heart muscle

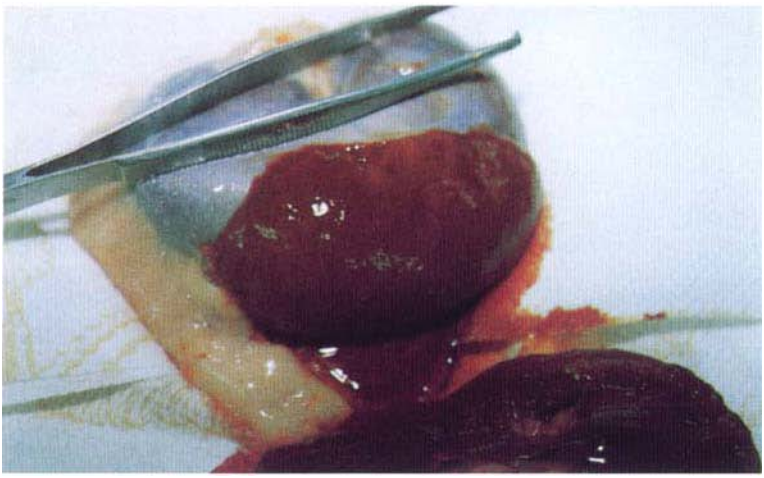


Figure 17 *C. perfringens* enterotoxemia in a young dromedary: "pulpy kidney"

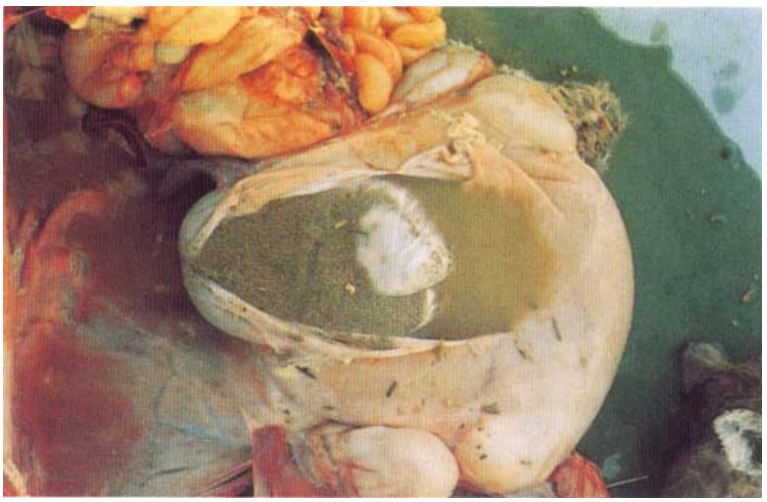


Figure 18 *C. perfringens* enterotoxemia in a young dromedary: sand in the compartments

of infection for the maturing young dromedaries. Knowledge of this epidemiological connection has increasingly led dromedary owners to relocate their breeding herds more often or to replace the contaminated sand with fresh sand.

To investigate the reasons for the young dromedaries ingesting sand, blood samples were taken and examined for the minerals calcium, magnesium, iron and phosphorus. Dromedary milk samples were obtained and analyzed using the same method. The results are summarized in Table 8.

The tests revealed no evidence of a deficiency of these minerals, so it was assumed that the dromedary calves ingested the sand more out of curiosity than due to an as yet undetected nutritional deficiency. Mineral licks were also placed in all the dromedary enclosures.

An additional important aspect in the development of clostridiosis in *Camelidae* is the amount of serum immunoglobulin in the young animals (see also 2.1.7 neonatal diarrhea). *Camelidae* have an epitheliochorial placenta, so that the calf, as in the foal, receives its passive protection against dis-

Table 8 Magnesium, phosphorus, calcium and iron values in sera of dromedary calves and milk from breeding dromedaries from herds where sand eating occurs

	Sera			Milk	
	*Reference values mg/dL	*Herd 1* 22 samples	*Herd 2* 26 samples	**Reference values g/kg	***5 samples
Magnesium	1.8–2.2	1.9	2.0	0.083	0.078
Phosphorus	3.2–6.5	10.7	9.8	0.95	0.82
Calcium	9.5–11.5	10.3	10.2	1.64	1.32
Iron	80–130	89	83		

* Normal values are for adult dromedaries (Samples were examined in a Dimension Autoanalyzer, Dupont)

** Whabi et al. (1987)

*** Examined by the J.A. Comloquoy, Dubai Aluminum Plant

ease through the intestinal reabsorption of immunoglobulins from the colostrum after birth. Although the newborn calf is immunocompetent at birth, the endogenous antibody production is not sufficient to produce a protective immunoglobulin level within the first month of life. The globulin fraction is naturally low at birth. Even after ingestion of colostrum, the globulin level declines after the seventh day and reaches the lowest level between the 20th and 30th day post partum. The highest losses due to *C. perfringens* enterotoxemia occur during this time.

Fowler (1998) made similar observations in NWC. He determined that the globulin content of NWC serum is very low at birth (< 5.2 mg/mL), increased following ingestion of colostrum to 5.5–6.2 mg/mL within 4–5 days yet reached its lowest level 3 to 4 weeks post partum. *C. perfringens* type A is a very serious disease in alpaca crias in South America and it is named *"Mal de Alpacas"* (Rath, 1950; Moro Sommo, 1963; Ramirez and Huaman, 1980–1981; Ramirez et al., 1983a and b; Huaman et al., 1981; Ellis et al., 1990; Fowler, 1996). The animal mortality rates vary between 10 and 70% and even on carefully managed farms may approach 50%. The disease occurs in crias between 8 and 35 days of age with sudden death or a short disease period during which the crias are recumbent, showing nervous system disorders. The pathological changes in alpacas are very similar to the lesions seen in OWC with petechiae in different organs, hyperemia, excess serosanguinous pericardial fluid and lesions in the intestinal tract.

Type C and D enterotoxemias are more common in lamoids than they are in OWC.

Diagnosis Specimens, including intestinal fluid, should be taken from freshly (less than 4 hours) dead animals, as clostridia are rapid post mortem invaders. Toxins are very labile and therefore small intestinal contents should be frozen as soon as possible until processed. The laboratory diagnosis of "clostridial enterotoxemia" is made by identifying clostridial toxins in the duodenum of recently expired animals. The intestinal contents are removed immediately post mortem and deep frozen. The next day the material is thawed, sterile filtered and tested for pathogenicity in mice. One milliliter of the sterile intestinal contents is injected intravenously into the tail vein of laboratory mice. In the presence of clostridial toxin, the mice expire within 2 to 8 hours, exhibiting seizures and the characteristic opisthotonus.

The colorimetric tetrazolium cleavage test (MTT) has widely replaced the mouse lethal test and is regularly used for the detection of clostridial toxins from intestinal fluids. It also has the advantage that the fluid can be diluted and a titer estimated. The higher the titer, the more toxin is present in the gut (Fig. 19).

In suspected enterotoxemia, the presence of large numbers of Gram-positive rods from mucosal scrapings from the small intestine of fresh dead animals is presumptive evidence of clostridial enterotoxemia (Fig. 20).

Fluorescent antibody (FA) technique is also routinely used for disease with *C. chauvoei, C. septicum, C. novyi* and *C. sordellii.*

Cultivation of *C. perfringens* from organs of dead dromedaries is performed on Sahidi-Furgeson-Perfringens (SFP) agar and Zeissler agar under anaerobic conditions with the gas generating kit. In *C. perfringens* outbreaks in the UAE, three different *C. perfringens* type A strains were identified using chromatography (Heitefuss et al., 1990; Heitefuss, 1991). These strains are now included in a local vaccine to protect dromedaries from clostridiosis.

Treatment and Control Treatment of sick dromedaries with a bovine *C. perfringens* hyperimmune serum is very rewarding. Many valuable racing camels were saved by the intravenous application of 100 mL of antiserum. This procedure can be repeat-

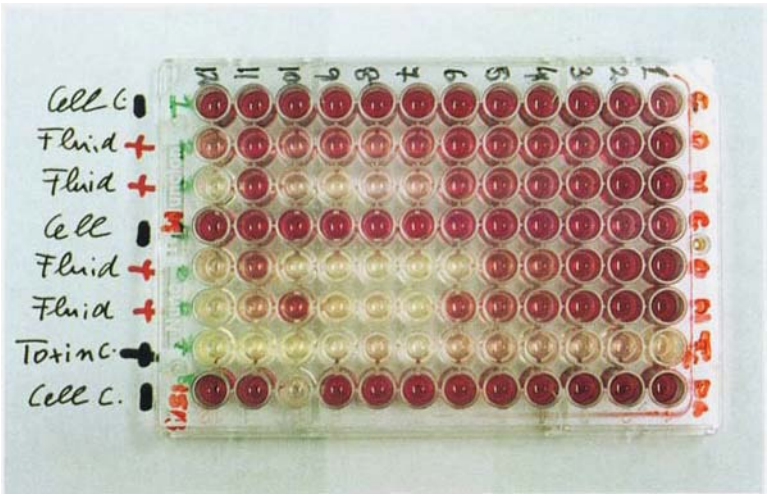


Figure 19 MTT results on vero cells indicating toxin in the intestinal fluids of dromedaries with clostridial enterotoxemia

ed without any side effects. For the prevention of this important disease, sanitation, feeding and general husbandry practices should be optimal. In endangered herds, chlortetracyclines at a rate of 25 mg/kg feed should be added to the feed.

Toxoid vaccines are commonly used to prevent enterotoxemia outbreaks in cattle, sheep and llamas. The vaccine should be administered to the dam, since neonates are unable to produce enough antibodies. The dam should be vaccinated 2 months before parturition and a booster administered 1 month prior to delivery.

Isolation and identification of clostridial strains are necessary to confirm the diagnosis and to develop a specific clostridial vaccine. For camels in the UAE, this toxoid vaccine was produced at the Institute for Applied Biotechnology of the Tropics (IBT) in Goettingen, as it is known that locally derived strains give optimal protection. This vaccine prevented further cases of *C. perfringens* enterotoxemia in adult dromedaries

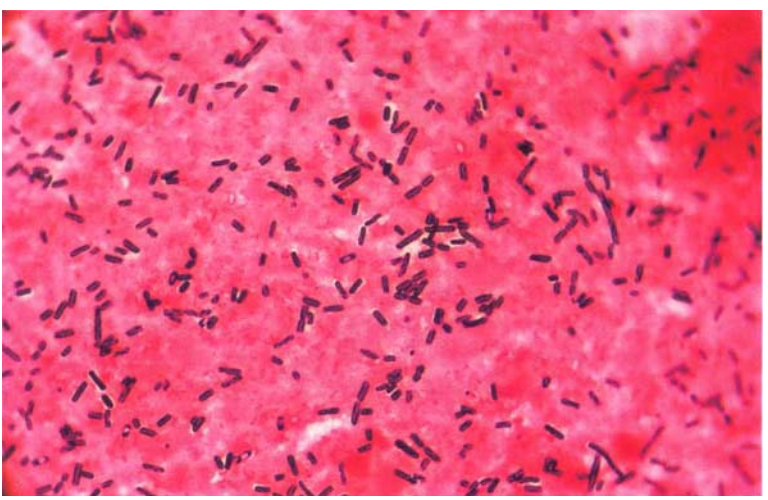


Figure 20 *C. perfringens*: increased number of Gram-positive rods in a mucosal scraping from the small intestine of a dromedary with clostridial enterotoxemia

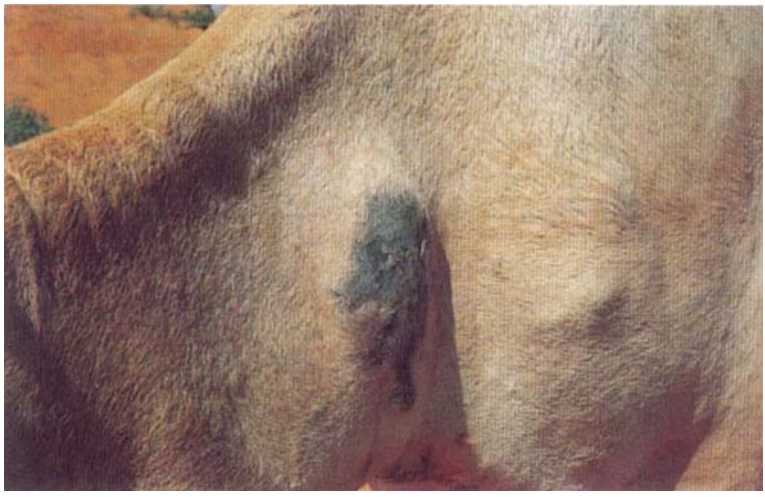


Figure 21 *C. perfringens* enterotoxemia: local reaction following subcutaneous vaccination with a Montanide adjuvant cell toxoid vaccine

Table 9 Development of antibodies, examined with the HIT, directed against a locally specific *C. perfringens* (type A) toxoid vaccine in dromedary calves and their mothers before and after two consecutive maternal protective vaccinations

Dromedary cows	Prior to vaccination	Weeks after vaccination 2	6	12	24
1	Neg.	1:64	1:64	1:32	1:64
2	1:2	1:32	1:16	1:16	1:16
3	1:4	1:64	1:64	1:64	1:64
4	1:2	1:32	1:32	1:32	1:32
5	1:4	1:128	1:64	1:64	1:64
6	1:2	1:32	1:32	1:32	1:32
7	Neg.	1:16	1:16	1:16	1:16
8	1:2	1:64	1:64	1:64	1:64
9	1:2	1:32	1:64	1:32	1:32
10	1:4	1:64	1:32	1:64	1:64
Dromedary calves	**Prior to colostrum ingestion**	**After colostrum ingestion**			
1	Neg.	1:32	1:32	1:16	1:8
2	Neg.	1:16	1:16	1:16	1:4
3	Neg.	1:64	1:32	1:16	1:4
4	Neg.	1:32	1:32	1:32	1:4
5	Neg.	1:64	1:64	1:32	1:2
6	Neg.	1:32	1:16	1:16	1:4
7	Neg.	1:32	1:16	1:8	1:8
8	Neg.	1:16	1:8	1:8	1:2
9	Neg.	1:16	1:16	1:8	1:2
10	Neg.	1:32	1:32	1:4	1:2

(Seifert et al., 1992) and reduced losses in young animals. After subcutaneous application of the Montanide adjuvant cell toxoid vaccine, 30% of the vaccinated dromedaries developed local allergic swellings (Fig. 21) (Seifert et al., 1992). Camels appear to be particularly sensitive to oil-based vaccines. Since then, an aluminum hydroxide vaccine has been used that is well tolerated both intramuscularly and subcutaneously.

The hemolysis inhibition test (HIT) (Schaper, 1991) was used to detect the production of antibodies in dromedaries following vaccination with the clostridia toxoid vaccine (Seifert, 1992) produced in Goettingen in the bioreactor. The results are shown in Table 9.

These results show that dams that were vaccinated twice with the clostridia toxoid vaccine prior to delivery developed a much higher antibody titer. The maternal protection that the young dromedaries then received by ingesting the colostrum of the vaccinated mothers lasted at least six months.

1.1.2 Botulism

Clostridium botulinum is responsible for botulism in man and animals. The toxin is absorbed from the intestinal tract and is transported via the bloodstream to the peripheral nerve cells resulting in flaccid paralysis. Death is caused by circulatory failure and respiratory paralysis. It is believed that camelids are susceptible to *C. botulinum* (Fowler, 1998). However, only a few clinical cases have been described in OWC (Wernery and Kaaden, 1995).

Etiology and Clinical Signs *C. botulinum* is a straight Gram-positive rod which produces subterminal spores at a pH near or above neutrality. The spores are resistant to heat and are only killed at 121°C for 15 minutes while the toxins of *C. botulinum* are destroyed at 100°C for 15 minutes. Eight different neurotoxins are produced by this strict anaerobe and even small traces of oxygen will inhibit growth.

Epidemiology Botulism is a classical epidemic of arid and semi-arid pastureland in the tropics and is distinguished by a characteristic paralysis. The disease is found primarily in cattle and is associated with a lack of phosphorus in the soil (Seifert, 1992). If there is a lack of minerals in their pasture grass, the animals attempt to cover this deficit by ingesting phosphorus-containing substances of animal origin. Cadavers serve as the source of the intoxication. In 1990, a devastating outbreak of botulism occurred on two feedlots in Queensland, Australia where over 5500 bulls died (Jones, 1991). Chicken scraps in the feed caused the outbreak.

Devastating losses due to botulism have also been observed in waterfowl. In 1983, 40,000 waterfowl died of botulism in the marshes west of Hamburg (Westphal, 1991). Wernery and Haydn-Evans (1992) have reported cases of botulism in seagulls, ducks, herons and flamingos in the UAE.

C. botulinum is usually found in the soil and mud, where the organisms can survive for many years. Eight types and subtypes of *C. botulinum* have been identified serologically by their toxin pattern. Their distribution is shown in Table 10.

The *C. botulinum* toxins are synthesized intracellularly in the last stage of the logarithmic growth phase and are first released through lysis of the bacterial cell. Today it is known that the bacterial cell alone is only capable of producing toxin C2, whereas at least the toxins C1 and D can only be produced in the presence of bacteriophages (Westphal, 1991). The knowledge of the relationship between *C. botulinum* and its bacteriophages is a decisive criterion in understanding botulism. By introducing phages, it is possible to transform a non-toxigenic *C. botulinum* strain into a toxigenic strain. If, for example, a neutral type of *C. botulinum* strain is infected with a C1-

Table 10 Types of *Clostridium botulinum* toxin and their distribution (Bisping and Amtsberg, 1988)

Type	Toxin	Distribution	Source of Intoxication	Susceptibility
A	A	Western USA, Ukraine	Feed, meat, fish, wounds	Man, waterfowl, mink
B	B	Central and Eastern USA, Northern and Central Europe	Meat and meat products	Man, cattle, horse, waterfowl
C	C_α, C_1, C_2, D	North and South America, South Africa, Australia, Europe	Lucilia larvae, plants, mud	Waterfowl
	C_α, C_2	Australia, South Africa, Europe	Spoiled food, cadavers	Cattle, horse, mink
D	D, C_1, C_2	South Africa, former USSR	Cadavers	Cattle
E	E	Northern Europe, former USSR, Canada, Alaska, Japan	Fish and fish products	Man
F	F	Scotland, USA, Denmark, former USSR	Liver pâté, fish	Man
G	G	Argentina	–	–

Tox phage, the strain will then produce the C1 toxin and will also become a type C strain. Infection with a D-Tox phage transforms the same neutral strain into a type D strain (conversion). It is even possible to infect a phageless neutral type *C. botulinum* strain with a phage of the closely related *C. novyi* and to convert the strain into a *C. novyi* strain (Westphal, 1991). All together, between the different types and strains of *C. botulinum* and its specific bacteriophages, a confusing, complex variety of new combinations are possible. The conventional differentiation between the types can no longer be upheld.

Reports of botulism in camels are rare. Provost et al. (1975) reported a catastrophic outbreak of type C botulism in dromedaries in Chad. Upon inspection of the herd of 150 animals, 45 were already dead and 40 severely ill. The sick animals had difficulty in standing, developed hindquarter paresis, and collapsed and died within a few hours. It was presumed that the well water was contaminated by a cadaver, which was the source of the toxin.

The danger of a botulism outbreak in racing dromedaries in the UAE is slight. In general, the animals are superbly cared for. Additionally, the feed is well balanced without animal additives, the camels are watered from deep wells and lick stones and mineral additives are readily available.

Diagnosis Botulism is often difficult to diagnose. A presumptive diagnosis is based on history, clinical signs and identification of toxin in serum of moribund or recently dead animals or feed. It is also possible to isolate *C. botulinum* in suspect foodstuffs. One milliliter of serum from diseased animals is inoculated intraperitoneally into mice. If toxin is present, the characteristic "wasp waist" appearance in the mice will be seen within a few hours to 3 days. Unfortunately, the mouse test is not very sensitive when large animals like camels are tested, as the concentration of toxin in the serum

or ruminal fluid is generally so low that toxin cannot be detected. The diagnosis then relies on the history and clinical signs. The toxicity of feed samples may be determined by test feeding the sample to specifically immunized laboratory animals or sheep.

Other methods for detection of botulinum toxin include immunodiffusion, complement fixation test and ELISA, but these tests are not commercially available and, except for the CFT, the sensitivity does not exceed that of the mouse bioassay.

Treatment and Prevention There is no specific treatment for diseased animals suffering from botulism, apart from the administration of hyperimmune serum specific to the toxin type involved. As the type of *C. botulinum* responsible for the disease in animals is generally not known until some time has relapsed, it is possible to mix antisera before administration. The antiserum is given intravenously. It is expensive, but may save very valuable camelids. Cattle and horses are treated with 5 mL of each type of antiserum and it is presumed that 5 mL should also be given to diseased OWC and 3 mL to NWC intravenously. The treatment may be repeated within 24 hours. In addition to this treatment, good nursing is essential when treating camelids suffering from botulism.
Prevention of botulism includes: vaccination, correction of phosphorus deficiency and removal of the source of intoxication. Vaccines are commercially available, sometimes as a combined vaccine for botulism and black-quarter. Camelids should be vaccinated in endangered areas. The initial vaccination should be followed by a second 5 weeks later and annually thereafter.

1.1.3 Anthrax

Bacillus anthracis causes anthrax in man and animals. Throughout the world there is a single uniform antigenic type, even though there are differences between local specific strains. Under natural conditions, the animals most frequently affected are the cow, sheep, goat, buffalo, horse, reindeer, elephant and mink. Birds (with the exception of the ostrich) and reptiles have a low susceptibility and are seldom affected (Bisping and Amtsberg, 1988). Pigs are not immune to anthrax, though they are generally afflicted with a subacute or chronic course of the disease following a primary lesion in the pharynx. Anthrax occurs throughout the world and is especially a problem where high concentrations of animals occur. This is the case, for example, at watering holes, animal markets and salt licks.

Anthrax is an acute, septicemic disease, which can affect camelids (Davis et al., 1981; Wernery and Kaaden, 1995; Fowler, 1998).

Etiology *B. anthracis* is an aerobic sporulating bacterium, which is a Gram-positive, non-motile, cylindrical rod. Inside the host it forms a capsule, which can be demonstrated by special stains. In organ smears the bacilli lie either singly or in short chains forming a so-called bamboo-stick form. Spores develop only in the presence of oxygen at temperatures above 12°C. *B. anthracis* grows on ordinary solid media and no hemolysis is produced on blood agar. Under low magnification the colonies give the appearance of a Medusa-like head or a woman's curly hair.

Epidemiology and Clinical Signs Anthrax is a peracute disease characterized by septicemia and sudden death. The anthrax endospores can survive for years in the soil. Masses of vegetative bacilli are discharged from the body in the final stages of the disease and sporulate in and on the ground at temperatures of 20–32°C (Seifert, 1992). Soil can be contaminated for years by buried cadavers, which then

serve as sources of infection, especially when the grazing animals bite off the pasture grass at ground level during periods of food scarcity. Inhaled contaminated dust can also lead to pulmonary anthrax. Fazil (1977) believes that anthrax is the most frequent bacterial disease of camels in Kenya with acute, peracute, and apoplectic forms.

Anthrax is greatly feared by nomadic camel breeders. They have given the disease many different names and are aware of its dangers. Anthrax is one of the most important zoonoses of the tropical regions and always occurs through a *B. anthracis* infection of an animal. The agent can enter the human host cutaneously, enterally, or via an airborne route. Punskii and Zheglova (1958) reported an outbreak of cutaneous anthrax in 37 Asians who came in contact with meat from a dromedary that had been infected with the disease. Mustafa (1987) believes that, along with trypanosomosis and mange, anthrax is one of the most loss-inducing diseases in dromedaries. An acute or peracute form of anthrax can be found in dromedaries that leads to sudden death without any previous clinical signs. Epidemics of anthrax tend to occur in association with marked climatic or ecological changes, such as heavy rainfall, flooding or drought.

A leaflet was prepared on anthrax in dromedaries by the Syrian German Technical Cooperation, in which the clinical signs and the pathological lesions are described (Tabbaa, 1997). A camel herd of 100 dromedaries from the steppe of Syria contracted the disease after drinking from a pond which was temporarily flooded with rainwater. The dromedaries affected exhibited difficult breathing, trembling and pronounced swelling of the throat, the base of the neck and the groin region. Before death camels became recumbent, excreting dark, foamy blood from the body orifices (Fig. 22).

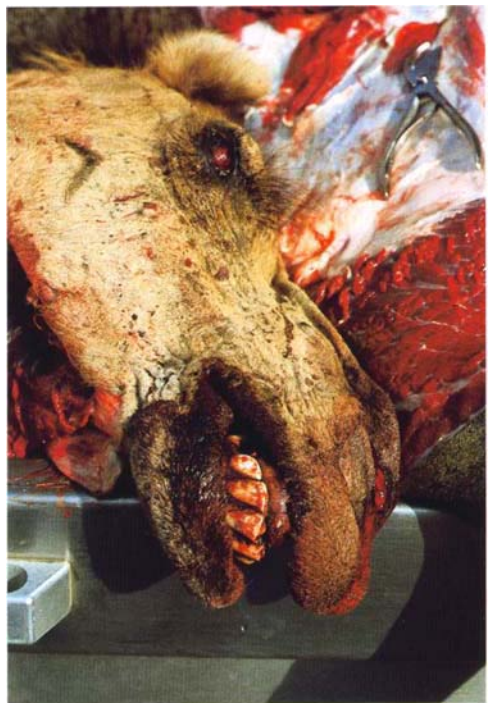


Figure 22 Unclotted blood protrudes from the nose of a dromedary with anthrax

More than 10 dromedaries died from anthrax infection. The disease ceased when town water was supplied, the remaining animals treated with antibiotics and the herd vaccinated.

The infection normally occurs via the alimentary tract due to ingestion of contaminated feed or pond water (Boue, 1962). Curasson (1947) has postulated that *Tabanidae* can induce cutaneous anthrax in dromedaries and that *B. anthracis* carried by nasal bots *(Cephalopina titillator)* can enter the body through injured mucous membranes. Barakat et al. (1976) reported an anthrax outbreak in Egypt during which 123 dromedaries died within 4 days, 9 apoplectically. Similar cases of sudden death due to anthrax have been described by Curasson (1947) and Gatt Rutter and Mack (1963). Barakat et al. (1976) are of the opinion that an outbreak of anthrax in

a dromedary herd was due to migrating birds. The outbreak was controlled by strict hygienic measures, administration of procaine penicillin and 50 mL of anthrax antiserum per dromedary over 5 days.

The clinical signs of anthrax in dromedaries are similar to those in the cow (Gatt Rutter and Mack, 1963): fever up to 42°C, extravasation of tar-like blood from the body orifices, diarrhea, colic, bloat and severe cardiovascular and pulmonary disturbances. Some dromedaries develop painful swellings on the throat and neck.

In NWC the clinical signs described for anthrax resemble those seen in OWC. Sudden death without any signs may occur as well as subcutaneous swellings on various parts of the body. Bloody discharge may exude from all body orifices and lamoids may die after 1 to 3 days (Fowler, 1998).

Pathology The principal lesions in septicemic anthrax in animals are hemorrhages, edema and necrosis. In dromedaries, there is evidence of rapid post mortem decomposition (Tabbaa, 1997) of the carcass with oozing of bloodstained fluid from nose, mouth and anus. Dark-red, poorly clotted blood, petechiae and ecchymoses are observed throughout the carcass. An enlarged pulpy spleen, which is the most characteristic feature at necropsy in ruminants, has also been described in camelids (Manefield and Tinson, 1996). There is no rigor mortis and the blood fails to clod. Splenomegaly with black tarry pulp, generalized congestion and lung edema were also observed by Boue (1962) and Richard (1975).

Diagnosis *B. anthracis* is easily cultured from blood and tissues. However, if anthrax is suspected one should avoid a necropsy to exclude contamination of the soil with spores. A small quantity of blood is sufficient for the diagnosis. A smear or a culture as well as a fluorescent antibody test (FAT) will confirm the diagnosis. In advanced autolysis, when no anthrax bacilli are demonstrable, the thermo-precipitation of Ascoli can be applied. For the cultivation of *B. anthracis* in laboratory animals, white mice are the animals of choice. They are subcutaneously infected and will die within 2 to 4 days. A gelatinous edema develops at the injection site.

Prevention and Control To prevent sporulation of *B. anthracis*, carcasses should not be opened. They should be incinerated with the contaminated bedding. After contact, equipment must be properly disinfected. The following disinfectant solutions can be used:

- 10% hot caustic soda solution,
- 4% formaldehyde solution,
- 7% hydrogen peroxide,
- 2% glutaraldehyde,
- calcium hypochloride with 5% active chlorine.

B. anthracis is susceptible to many antibiotics, including penicillin and tetracyclines.

Pasteur developed the first effective *B. anthracis* vaccine. It was replaced by the live, avirulent, spore vaccine developed by Sterne. This vaccine has been used worldwide with great economic value to the livestock industry and to wildlife. A single inoculation provides effective immunity for 9 months, but annual booster vaccinations are recommended. Anthrax can be a serious danger to camelids and it is therefore recommended to vaccinate *Camelidae* in endangered areas. However, anthrax vaccines should be carefully used in camelids and the dose adjusted to the weight of the animal, since bacteria-induced anthrax has been reported in young llamas (Cartwright et al., 1987). OWC should be given the dose of cattle and NWC should receive the dose that is recommended for sheep. A half sheep dose is recommended for NWC weaners (Fowler, 1998).

1.1.4 Endotoxicosis (Endotoxemia)

The large number of Gram-negative bacteria constituting the normal flora of the gastrointestinal tract provides a potential pool of endotoxin for the animal. This is especially true for ruminants and *Camelidae*, when the compartments' flora is destroyed by the decline of rumen pH. Impairment of rumen fermentation caused by highly digestible diets leads to inappetence and lactic acidosis. Ruminants and *Camelidae* with acute lactic acidosis often manifest clinical signs of endotoxemia or endotoxin shock, because ruminal Gram-negative bacteria are destroyed in large quantities. Lactic acid is apparently not the toxic factor, since huge quantities of endotoxins have been detected in cell-free ruminal fluid of acidotic animals. The endotoxin of alimentary origin is not the cause of lactic acidosis syndrome, but the result of it. The cause of lactic acidosis in dromedaries is the feeding of highly digestible diets to a desert animal, whose forestomachs are adapted to poor-quality feed. The new feeding practice has gained huge momentum, since camel races on the Arabian Peninsula have become extremely competitive.

Intensive investigations over the last decade now seem to have solved the mystery surrounding a disease of racing camels known as "*Bacillus cereus* intoxication", "hemorrhagic diathesis" or "hemorrhagic disease" (Wernery and Kaaden, 1995).

Etiology Endotoxins are lipopolysaccharides, which are found in the outer cell wall of Gram-negative bacteria and are released during periods of rapid growth or death of organisms. Structurally, endotoxins are composed of three parts:

- Lipid A: buried in the cell wall, it mediates most of the toxic effects of endotoxin.
- O Region: gives antigenic specificity and is highly variable between bacterial species.
- Core Region: acts as the link between the inner (lipid A) and outer (O) regions.

Endotoxins are extremely toxic and may be lethal at a concentration of 10^{-9} g/mL. They are chemically very stable and boiling does not destroy them. The toxins are also not significantly altered by acids or enzymes present in abdominal fluids. Small amounts of endotoxins are regularly produced in the gastrointestinal tract. They are absorbed through the intestinal mucosa into the circulation and are detoxified in the liver. However, if hepatic efficiency is reduced or the amount of toxins is too large, toxemia is produced, with severe consequences. Widespread vascular endothelial and subsequent tissue damage can be expected. Due to the vascular endothelial damage, endotoxin activates the clotting cascade and causes disseminated intravascular coagulation (DIC).

Clinical Signs and Pathology For numerous years a disease has been rife among racing dromedaries in the UAE that due to its clinical and pathological presentation has been called "hemorrhagic diathesis" or "hemorrhagic disease" (HD). The disease occurs primarily in racing dromedaries, of which 80% are between 2 and 4 years old or even younger. The disease affects individual camels, but also groups of up to 10 animals and more in a herd can fall sick. Cases have been diagnosed at all times of the year, but the highest incidence occurs during the summer months' high temperatures and high humidity. It is believed that not only the extreme climate aggravates outbreaks of this disease, but also the start of training sessions ahead of the new race season and a change of diet from a more high fiber to a high carbohydrate and protein diet.

The initial stage (24–48 h) of the disease is characterized by a dramatic decrease in the total number of leukocytes (WBC), fever as high as 41°C, inappetence, depression and dullness. Three to 4 days after the onset of the first clinical signs, the WBC counts increases (Table 11).

Table 11 Blood parameters and serum enzymes of 10 dromedaries with endotoxicosis (blood was taken 1 to 2 days and 3 to 4 days after the onset of the disease)

Parameters	Units	Reference Values**	1 to 2 days					3 to 4 days				
White Blood Cells	$x10^3/L$	6.0–13.5	2.5	1.6	2.6	0.8	2.9	19.3	24.8	18.0	17.3	26.6
Neutrophils	%	50–60	70	x	66	x	65	80	78	82	86	77
Lymphocytes	%	30–45	23	x	27	x	28	12	16	13	12	20
Monocytes	%	2–8	6	x	6	x	6	8	6	5	2	3
Eosinophils	%	0–6	0	x	1	x	1	0	0	0	0	0
Basophils	%	0–2	1	x	0	x	0	0	0	0	0	0
Erythrocytes	$x10^6/L$	7.5–12.0	7.9	8.4	8.0	9.0	9.5	8.0	7.8	8.4	9.9	8.6
Hemoglobin	g/dL	12.0–15.0	11.1	11.3	11.1	12.2	12.0	10.4	12.0	10.9	12.1	10.8
Platelets	$x10^3/L$	350–450	168	142	236	116	182	271	372	298	201	291
Creatine Kinase (CK)	IU/L	40–120	46	81	93	70	62	320	438	594	362	612
Glutamate-oxalacetate-transaminase (AST, GOT)	IU/L	60–120	120	104	83	97	110	490	119	257	421	401
Lactate-dehydrogenase (LDH)	IU/L	400–775	590	390	220	142	350	1812	675	730	1557	1210
Glucose	mg/dL	70–110	46	70	65	44	48	86	92	99	106	107
Blood Urea Nitrogen (BUN)	mg/dL	3–21	19	21	23	25	21	75	195	60	44	146
Creatinine (Crea)	mg/dL	0–2.2	2.0	2.2	2.0	2.0	1.8	4.5	9.3	4.2	3.7	9.6
Fibrinogen	mg%	250–400	98	102	72	93	106	180	201	305	298	172
Prothrombin time (PT)	Sec	17.6 ± 1.6	28.2	22.4	27.0	24.8	26.3	19.2	17.4	18.9	20.2	21.6
Partial thromboplastine time (PTT)	Sec	46.9 ± 13	82.4	60.2	62.0	54.6	70.3	50.1	48.0	47.6	53.1	60.0

x Differential count due to toxic changes not possible
** Wernery et al. (1999)

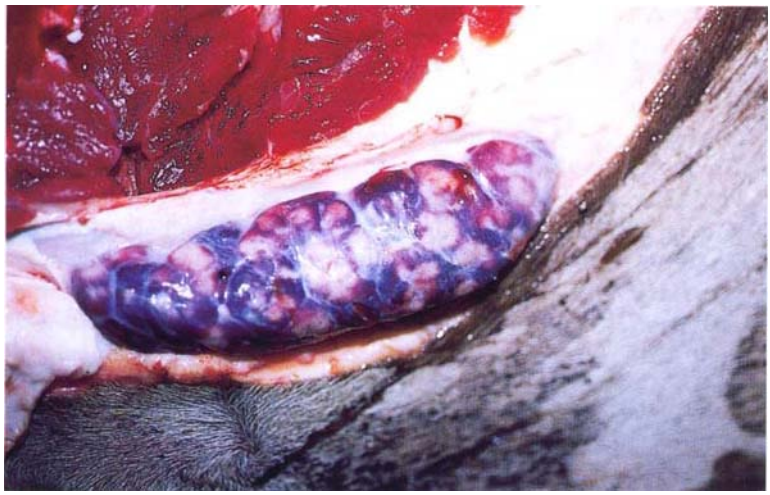


Figure 23 Swollen and hemorrhagic inguinal lymph node

Some animals develop a cough and swelling of the throat accompanied by a marked uni- or bilateral enlargement of the body lymph nodes (Fig. 23). Mucous membranes are often injected. Additionally, complete atonia of compartment 1, abdominal pain and regurgitation have been observed. Rectal examination of affected dromedaries reveals normally formed balls of stool that are covered in fresh or tar-like blood. Only very few camels develop diarrhea (Manefield and Tinson, 1996).

Affected dromedaries die between the 3rd and 7th day. Two or 3 days before death, the animals become recumbent. Some dromedaries develop central nervous system disturbances, lacrimation and hypersalivation. The development of nervous signs is a feature of terminal cases. The disease is the most serious ailment in racing camels and has been reported from all countries of the Arabian Peninsula where camel racing is performed. It is unknown in other camel-rearing countries.

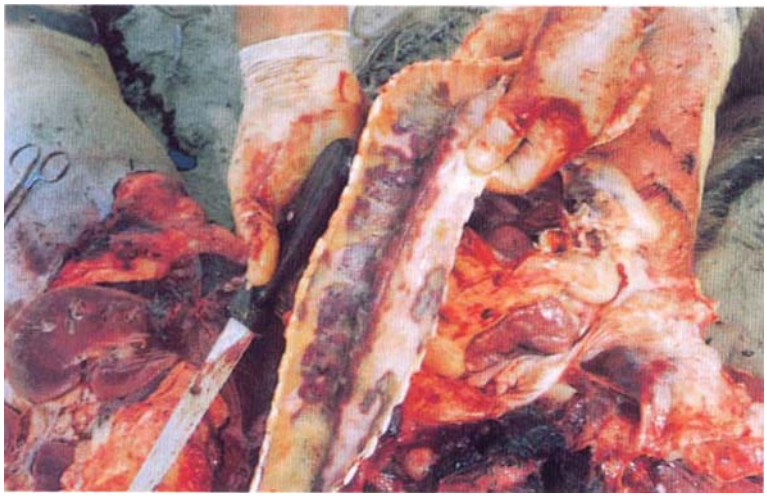


Figure 24 Tracheal ulcers caused by endotoxemia

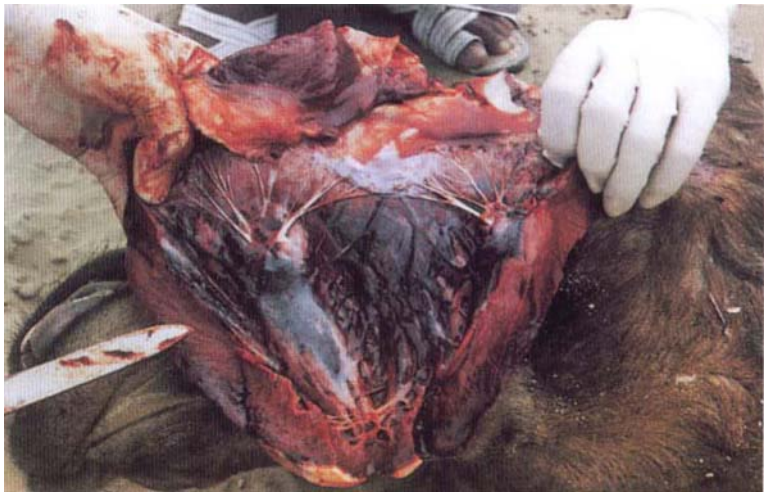


Figure 25 Subendocardial hemorrhage caused by endotoxemia

Over a 15-year span more than 200 racing dromedaries that died of endotoxicosis were autopsied. During necropsy the most striking changes are severe hemorrhages and bleeding into organs and the intestinal tract. Ecchymotic hemorrhages of varying severity are seen in the following organs:

- pharynx and trachea (some dromedaries develop ulcerations in the trachea) (Fig. 24);
- epicardium and subendocardium (Fig. 25);
- abomasum (ulcers are always found on the top of the folds of the fundus, some with blood clots attached to the ulcers, Figs. 26 and 27);
- intestinal tract, primarily in the ascending colon (the intestines are frequently filled with fresh or tar-like blood, Fig. 28);
- renal pelvis (mostly petechiae, Fig. 29).

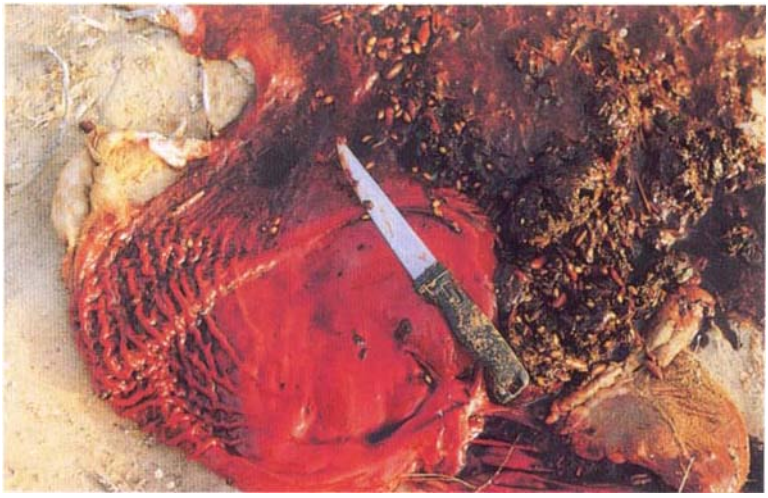


Figure 26 Hemorrhage in the abomasum caused by endotoxemia

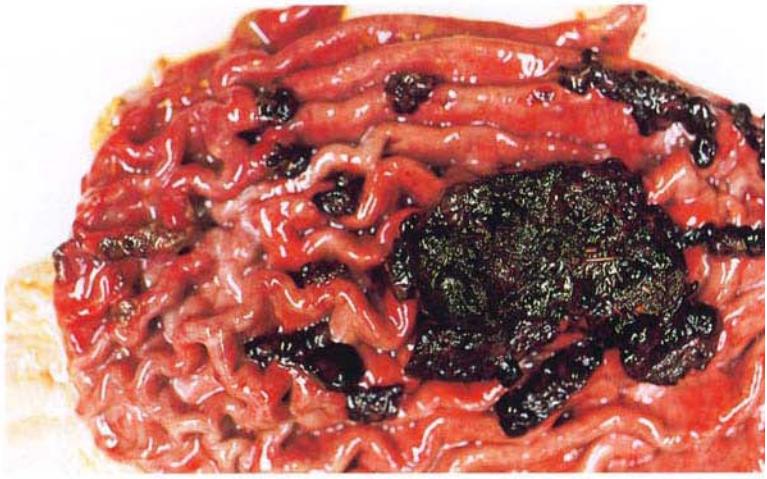


Figure 27 Ulcers in the abomasum, some with attached blood clots caused by endotoxemia

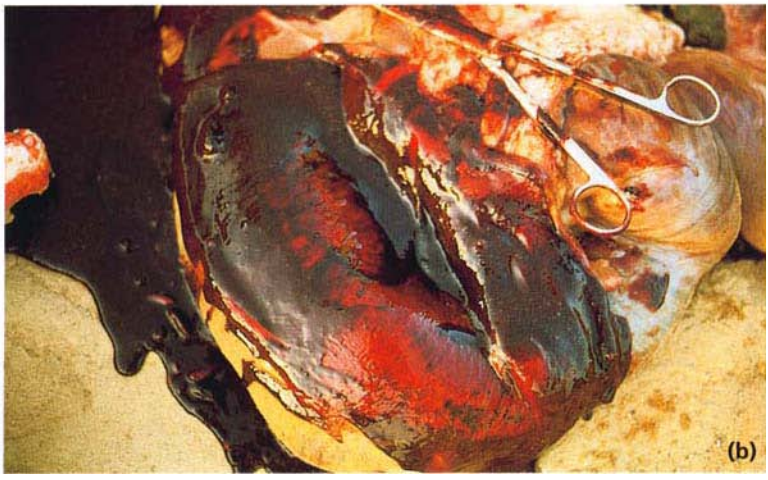


Figure 28a, b Ecchymosis in the ascending colon and small intestine caused by endotoxemia

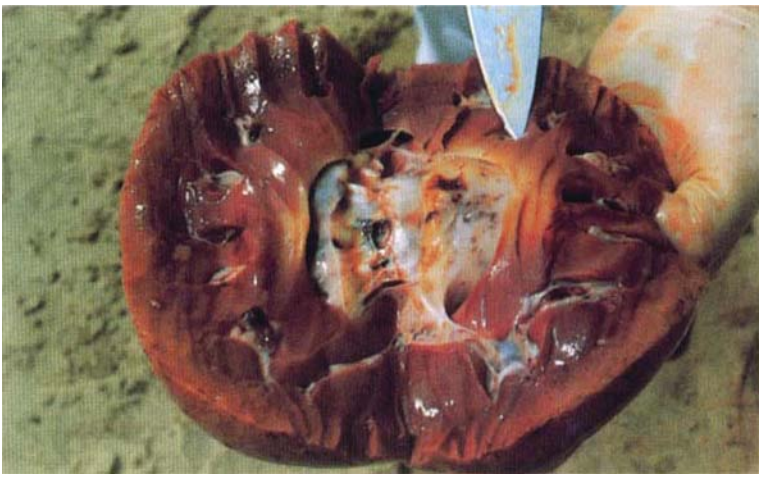


Figure 29 Petechiae in the renal pelvis caused by endotoxemia

All lymph nodes are enlarged, hemorrhagic often with necrotic centers (Fig. 30). The lungs are congested and exhibit subpleural and interstitial hemorrhages (Fig. 31).

All of the animals exhibit ruminal acidosis; the pH values are between 4 and 6. Smears from the ruminal fluid of necropsied racing dromedaries show a Gram-positive bacterial flora (Fig. 32) and there are no protozoa in the fluid of C1.

Histopathological examination demonstrates an intermediate to severe loss of lymphocytes in the lymphatic tissues, including the spleen and tonsils. Hemorrhages, necroses and karyorrhexis are primarily seen in the follicular centers and are very prominent in the Peyer's patches and in the mesenteric lymph nodes (Fig. 33). The changes point to viral involvement, but extensive studies including animal experiments yield no indication of viral diseases.

Severe hemorrhages are also observed in the abomasum, intestinal tract and the subepicardial as well as subendocardial layers of the heart. Pronounced necroses are regularly seen in the epithelium of the convoluted and straight renal tubules. In

Figure 30 Enlarged, hemorrhagic prescapular lymph nodes with necrotic centers caused by endotoxemia

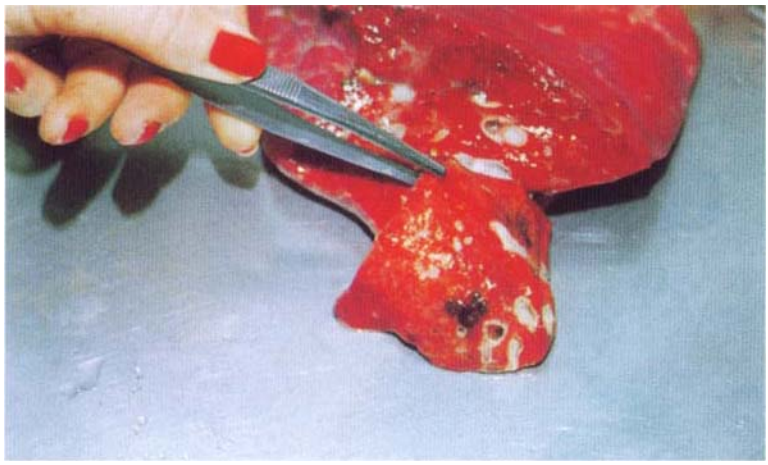


Figure 31 Interstitial hemorrhages in the lung caused by endotoxemia

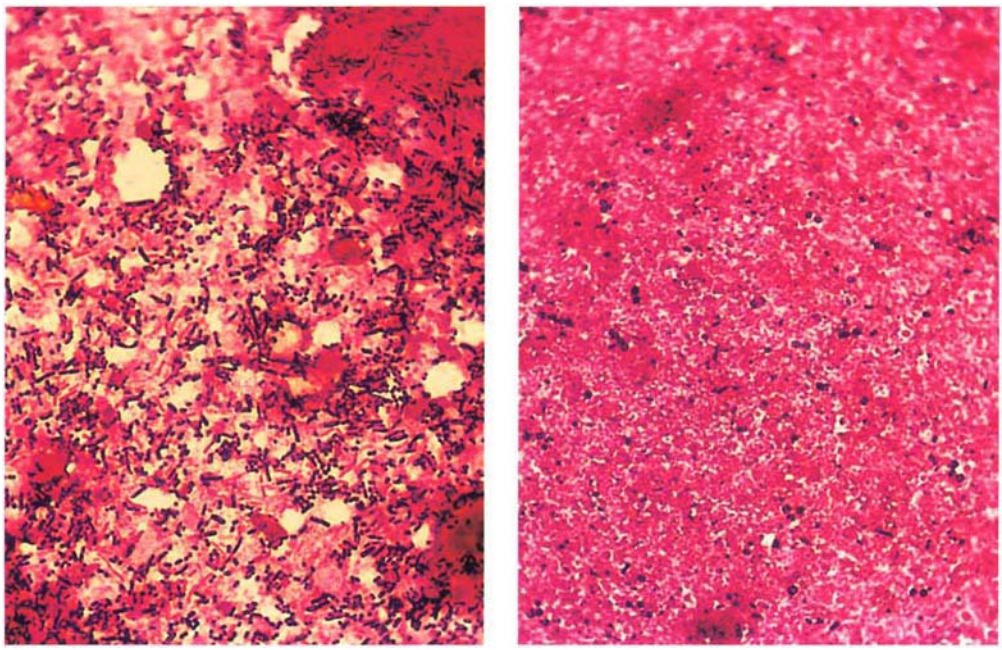


Figure 32 Gram-positive bacterial flora of compartment 1 of a camel with endotoxemia (left) and Gram-negative flora of a healthy camel (right)

numerous glomeruli, the Bowman's space is dilated and filled with protein material. The Bowman's capsule is often thickened due to deposits of PAS-positive material (Fig. 34). Some of the glomerular capillaries contain microthrombi (shock bodies). In dromedaries which survive longer, segmental necrosis of capillary loops is observed (fibrinoid necrosis). PAS-positive cylinders block the lumen of some distal tubuli showing tubulonephrosis. The livers of the animals autopsied exhibit a pan-

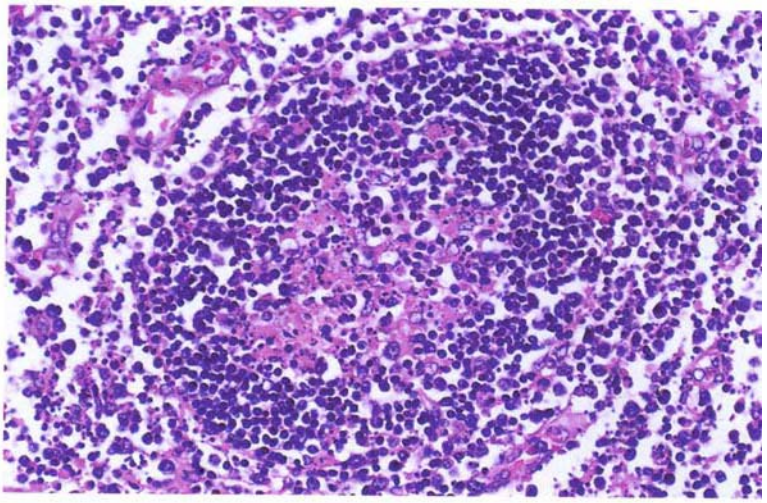


Figure 33 Necrosis and karyorrhexis in follicular centers of a mesenteric lymph node of a racing camel with endotoxemia

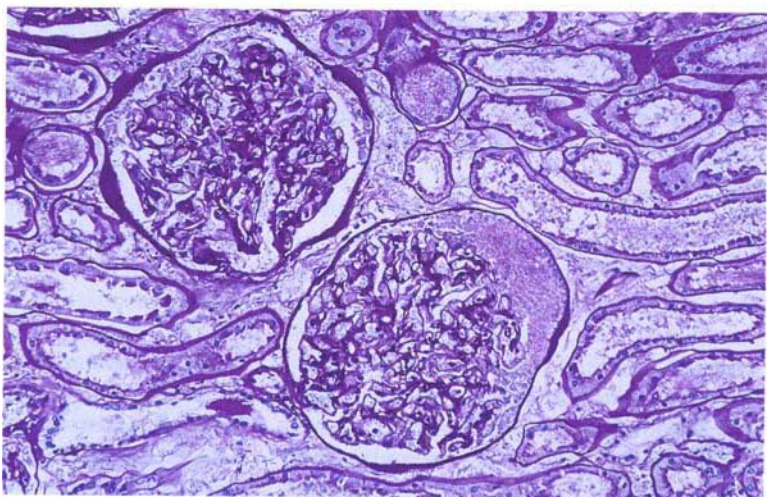


Figure 34 Dilated Bowman's space of a dromedary kidney: note the thickened Bowman's capsule due to deposits of PAS-positive material

lobular fatty degeneration as well as necrobiosis in centrolobular areas (Fig. 35). Hyperemia is regularly seen in the brain and both a perivascular and a meningeal edema may be observed. Strikingly, no inflammatory response is observed in any organs, most probably due to the toxin-induced destruction of follicles in the lymphoid tissues and the destruction of the circulating white blood cells.

Chemical analysis of the livers, kidneys and contents of the compartment 1 are negative for cumarine and its derivatives as well as organophosphates. The endotoxins have also a direct impact on the leukopoietic system causing aplasia and destruction, which is demonstrated in lymph nodes, tonsils, spleens and other lymphoid tissues. It also has a direct toxic effect on the circulating leukocytes, which are often not identifiable due to their toxic changes. The agranulocytosis induced by the lipopolysaccharides produces severe immunosuppression in diseased camels, predispos-

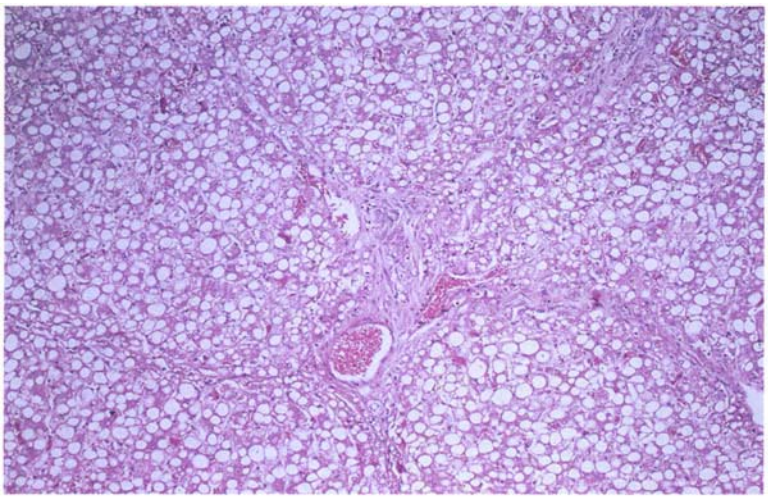


Figure 35 Severe panlobular fatty liver degeneration with necrobiosis in centrolobular areas of a racing camel with endotoxemia

ing them to secondary bacteriemias. Masses of different bacteria are regularly isolated from all organs: *E. coli, Pseudomonas aeruginosa, Proteus* spp., *Klebsiella pneumoniae, Staphylococcus aureus, Staphylococcus* spp. and *Streptococcus* spp. These facultative and opportunistic microorganisms multiply rapidly in all pre-damaged organs, producing further local toxins. Anthrax bacilli are never identified.

Bacillus cereus was formerly made responsible for this disease because toxic strains had been isolated from organs and feed of camels that had died from endotoxicosis. The disease was also reproduced by intravenous infusion of cell-free toxin of a toxic *B. cereus* strain (Wernery et al., 1992a; Walz, 1993; Wernery, 1994; Nothelfer and Wernery, 1995; Wernery and Kaaden, 1995). It is now known that the systemic effects of endotoxicosis can be experimentally demonstrated by the intravenous injection of purified toxin of many Gram-negative bacteria (Krogh, 1960; Huber et al., 1979; Nagaraja and Bartley, 1979).

Clinical Pathology Changes in total and differential leukocyte counts are typical of endotoxemia. There is a dramatic drop of leukocytes due to a decrease in neutrophils and lymphocytes (see Table 11). Leukopenia generally persists for 1 to 2 days and is reversed with an overshooting reaction (Tables 11 and 12) after the third day.

In many severe cases with less than 1.0×10^3/L of WBC, it is not possible to perform a differential count due to toxic changes in the white blood cells (Fig. 36a) (Wernery et al., 1999). These changes include pyknotic nuclei, vacuolation of the cytoplasm and false staining. The lowest WBC count is usually recorded on the day of presentation and is pathognomonic for this ailment. A rise towards a normal count occurs as the disease progresses. A correct hematological result is essential, because very early diagnosis and treatment is the key to recovery.

A sharp rise in serum enzymes is observed in the final stages of the disease, indicating internal organ damage. As can be seen in Table 11, some of the values are greater than 20 times the normal level. Creatinine and blood urea nitrogen (BUN) are always greatly elevated, indicating renal damage that is also seen histologically.

Camels develop a forestomach atony and forestomach acidosis. Normal forestomach fluid pH in camelids is higher than 6.5 but

Table 12 Blood parameters and serum enzymes of a dromedary that survived endotoxemia

Parameters	Units	Reference Values*	Days 1	2	3	6	11	25
White Blood Cells	$x10^3/L$	6.0–13.5	1.0	1.5	5.2	22.4	20.8	9.9
Neutrophils	%	50–60	82	84	92	84	78	61
Lymphocytes	%	30–45	12	10	7	10	16	32
Monocytes	%	2–8	4	5	1	4	5	4
Eosinophils	%	0–6	2	0	0	2	1	3
Basophils	%	0–2.0	0	1	0	0	0	0
Erythrocytes	$x10^6/L$	7.5–12.0	8.3	9.1	7.6	8.3	7.2	7.6
Hemoglobin	g/dL	12.0–15.0	12.2	13.5	12.2	11.5	10.1	11.1
Platelets	$x10^3/L$	350–450	176	140	193	251	301	483
Creatine Kinase (CK)	IU/L	40–120	67	112	721	882	324	140
Glutamate-oxalacetate-transaminase (AST, GOT)	IU/L	60–120	196	380	475	680	232	160
Lactate-dehydrogenase (LDH)	IU/L	400–775	338	790	819	1083	660	379
Glucose	mg/dL	70–110	46	38	44	70	78	108
Blood Urea Nitrogen (BUN)	mg/dL	3–21	13	23	51	58	34	22
Creatinine (Crea)	mg/dL	0–2.2	1.7	3.1	3.8	4.1	2.6	1.6
Fibrinogen	mg%	250–400	92	103	112	210	370	391

* Wernery et al. (1999)

most HD cases were presented with pH between 4 and 6. Protozoa are not detected during microscopic examination of gastric fluid and Gram-stains of the fluid reveal a population of predominantly Gram-positive bacteria. Furthermore, the fluid is sour-smelling and yellow and always contains undigested pieces of barley. These changes have also been described in acidotic NWC (Cebra et al., 1996).

Consistent gross lesions in all dromedaries are hemorrhages in different organs and severe bleeding into the intestines, especially in the colon (see Fig. 28a). The colon of camels has an extremely effective absorbing capability, which explains why bleeding in this part of the intestine is very intensive. It is also believed that huge quantities of toxins are already absorbed through compartments 1 and 2, as these are lined with non-papillated, smooth stratified squamous epithelium in camelids. It is also known that camels in general have a very rapid entry of fluids into the bloodstream. This anatomical aspect makes them very vulnerable to endotoxemia. The camel cannot detoxify the cell-free endotoxins produced in the forestomachs due to their extreme stability and due to the pre-damaged liver. Furthermore, not only do bacterial endotoxins accumulate, but metabolic toxins also are produced as a result of impaired metabolism in the compartments caused by ruminal and intestinal impaction, which is always observed with endotoxicosis in racing camels. The intestinal motility ceases due to lactic acidosis. Diarrhea is seldom observed in camelid endo-

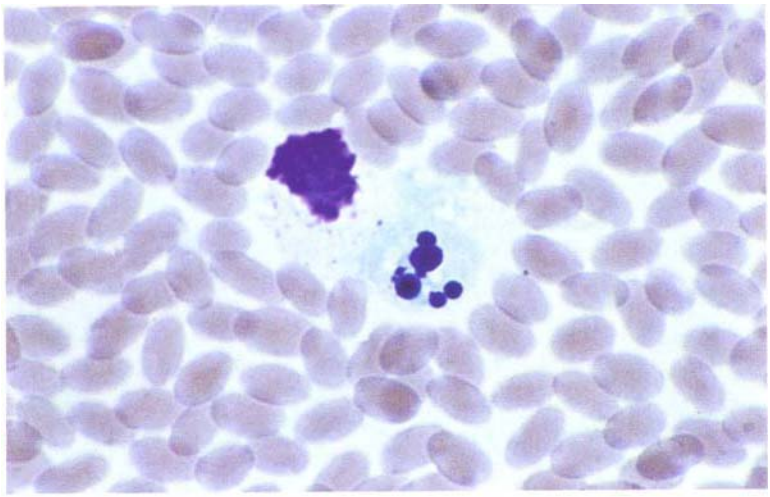


Figure 36a Endotoxicosis of a racing camel with 1.0 × 10^3/L leukocytes with two unidentifiable WBCs with severe vacuolated cytoplasm and pycnotic nuclei

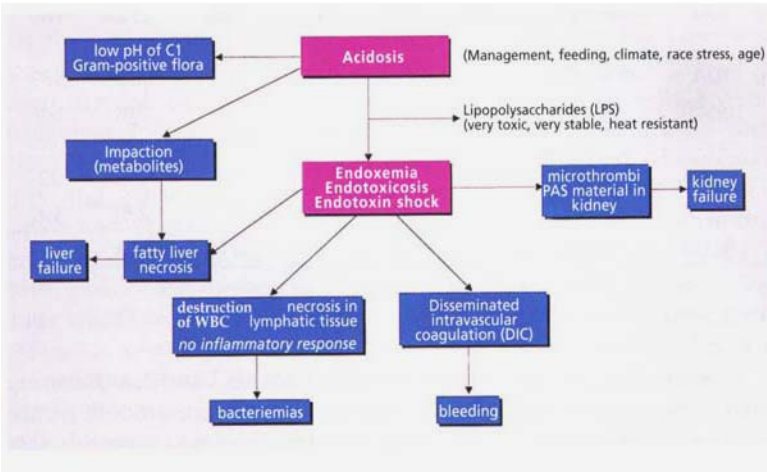


Figure 36b The pathogenesis of camelid endotoxemia

toxicosis, most probably due to the special structure of the cells in the rectum, which absorb most of the fluid accumulated in the rectal feces. Fresh or tar-like blood may be passed through the rectum depending on where the intestinal bleeding occurs. Tar-like blood in the abomasum is caused by the coagulation of oozed blood caused by gastric acid effects, whereas the tar-like blood in the colon (melena) is fermented oozed blood from the small intestines.

Lipopolysaccharides trigger disseminated intravascular coagulation (DIC). DIC is characterized by a decrease in fibrinogen content, the presence of soluble fibrin and fibrinogen degradation products and severe deficiency of coagulation factors which inhibit thrombin activity, fibrin polymerization and platelet aggregation. Partial thromboplastin time (PTT) and prothrombin time (PT) are prolonged (Table 11). DIC is a most serious consequence of endotoxemia. Fibrin not only further elevates blood viscosity, but may also clog the glomeruli of the kidneys. Both findings are often observed in dromedaries suffering from endotoxemia. Most blood samples of the early stages of the disease possess very

little serum after centrifugation. The supernatant above the blood clot is composed mainly of fibrin. On histology, protein deposits are seen in the glomeruli (see Fig. 34). This leads to renal failure, which is indicated by the elevated levels of BUN and creatinine. The effects of the decrease of coagulation factors are often visible when a blood sample is drawn from the jugular vein of affected camels. After the needle is withdrawn from the vein, the puncture hole continues to bleed. Further evidence of this situation is the low platelet count in all camels with the disorder. The pathogenesis of endotoxemia in camels is summarized in Fig. 36b.

Not every acidotic camel develops an endotoxicosis. A field trial with 2 camels, in which ruminal acidosis was artificially induced by feeding a high carbohydrate diet, (Wernery and Wensvoort, 1992) did not yield clinical signs similar to endotoxicosis. It is not clear which mechanism ultimately triggers the disease. Camel owners wonder that some animals develop endotoxemia and some do not, although they receive the same feed.

A connection between mycotoxins and hemorrhagic diathesis has long been known (Blood and Radostits, 1990). Racing dromedaries in the UAE are given fodder of superb quality, so that fungal contamination of the fodder can usually be ruled out. The lack of organ mycosis in the autopsied camels is a strong indication that the hemorrhagic diathesis is not caused by mycotoxins. In breeding herds of dromedaries where animal management occasionally does not meet the standards seen in racing dromedary herds, yearly losses due to mycotoxins are seen in the rainy season (Gareis and Wernery, 1992; Gareis and Wernery, 1994). The mycotoxic disease exhibits a course similar to endotoxemia with agranulocytosis and intestinal hemorrhages, but a gray, foul-smelling diarrhea is also always present. These clinical signs were reproduced in five young dromedaries by feeding them hay that was highly contaminated with fungi.

A second group of researchers from the neighboring emirate of Abu Dhabi, who have intensively studied hemorrhagic diathesis in dromedaries, were able to isolate *Aspergillus fumigatus* from nearly every organ of autopsied camels, as well as detecting aflatoxin in some sera (EL-Khouly et al., 1992). The authors added that it was not possible to determine whether these findings were due to a secondary infection with the fungi or were the primary cause of HD. The application of thiabendazole as an antifungal agent had no affect on the outcome of the disease.

Treatment and Control :: Therapeutic success of endotoxemia of camels depends primarily on early diagnosis and treatment. The earlier the diagnosis is made, the greater are the chances of survival. Since so much knowledge has been accumulated over the last decade on this disease, camel owners nowadays inform practitioners when the first signs of disease are observed. Endotoxemia is a very severe and complex ailment and extremely difficult to treat. With early treatment, even cases with WBC counts of less than 1.0×10^3/L are curable. However, despite the best treatment, fatalities can be expected. The prognosis is poor once the condition has reached an advanced stage.

The therapy for endotoxemia should include these main treatments:

1. Binding of endotoxins and their removal from the system
2. Administration of antacids to reverse the lactic acidosis
3. Fluid therapy
4. Control of inflammatory response
5. Prevention of the development of gastric ulcers
6. Supportive therapy to increase the detoxifying capacity of the liver
7. Broad spectrum antimicrobial administration

8. Activation of the coagulation system
9. Prevention of cerebral corticonecrosis (CCN).

To avoid endotoxicosis, special attention is to be paid to feeding. It is still common practice in the UAE to feed racing camels with cow milk, dates, excessive barley and fresh alfalfa. A better balance between high energetic diet and roughage has to be achieved. If carbohydrate overload occurs, a laxative such as liquid paraffin should be given by gastric tube to reduce the gastrointestinal transit time and to avoid any impaction and bacterial proliferation and endotoxin absorption. A more stringent laxative may be used in severe impaction with magnesium sulfate at a dose of 500 to 1000 g per animal. It is believed that charcoal also administered through a gastric tube, at a dose of 500 g daily (for a 400 kg racing camel), may reduce the cell-free endotoxin by adsorption.

Polymyxin B is an antimicrobial drug with a good affinity for lipid, a portion of endotoxin. This drug is toxic in horses with severe side effects of renal damage, but should be tried in camels suffering from endotoxemia due to its known endotoxin-binding capacity. The suggested dose rate in equines is 6000 IU/kg or 2.5 mg/kg polymyxin B sulfate, diluted in up to 5 liters of saline and given by slow i.v. infusion.

It has been demonstrated in a number of studies in equines that the administration of an antiserum directed against the core region of the endotoxin reduces mortality. The recommended dose in horses is 1 to 2 mL/kg body weight (Gaffin, 1987). It is available from Veterinary Dynamics as Hypermune-J® and from Immvac, Columbia, MO 65201 as Endoserum®. Ideally, the treatment with these sera should start in peracute cases. Stegantox 60® (Schering-Plough Animal Health) is a freeze-dried, purified endotoxin-specific IgG and, in the presence of complement, the product is also bactericidal against many Gram-negative microorganisms. The content of one 60 mg vial provides a single dose for an animal of 200 kg body weight. This product has already been used in camels suffering from endotoxemia.

In endotoxemia, oxygen-free radicals lead to tissue damage and inflammation. Dimethyl sulfoxide (DMSO) should be given as a 10 to 20% solution with isotonic fluids or water through gastric tube. In horses a dose of 250 mg to 1 g/kg every 12 hours is recommended.

Non-steroidal anti-inflammatory drugs are very important in the control of the inflammatory response, which always follows the endotoxin shock.

Phenylbutazone, ketoprofen or flunixin should be used to block the formation of inflammatory mediators. Finadyne has been shown to be efficient in horses since it possesses an anti-endotoxic, analgesic, anti-inflammatory and anti-pyretic effect, but it is toxic for camels.

Correction of the circulating fluid deficits is an important procedure to save camels from endotoxic death. Great quantities of fluids are lost by internal bleeding due to the impairment of capillary wall integrity. Furthermore, to correct acidosis, a rigorous fluid replacement therapy should immediately take place. A 5% sodium bicarbonate solution should be given i.v. at a dose of 5 l/camel in the very early stages of the illness followed by an i.v. infusion of 60 l/camel of a 1.3% sodium bicarbonate solution in saline with dextrose. In addition to this therapy, antacids should be administered twice daily through a gastric tube including 500 g/camel of magnesium hydroxide dissolved in warm water. This treatment has to be repeated daily for several days. In severe cases of ruminal lactic acidosis and endotoxemia involving very valuable camels, a rumenotomy should be considered. Compartment 1 should be emptied and washed out with a siphon and the compartment ingesta replaced by ingesta from healthy ruminants such as sheep and goats.

For the prevention of gastric ulcers, the substituted benzimidazole, omeprazole, should be orally given at a dose of 0.7 mg/kg body weight. This drug prevents the secretion of gastric acid through blocking the H+ and K+ ATPase.

In endotoxemia, hepatic function is also compromised. Because of the role the liver plays in the storage, activation and synthesis of many vitamins and, because of its detoxifying capability, multiple vitamins (including K) and liver stimulants should be administered.

Consumption coagulopathy may be stopped by the administration of heparin, but this treatment has not been tried in camelids. To help prevention of polioencephalomalacia thiamine hydrochloride at a dose of 2.5 to 10 mg/kg should be given i.v. or i.m.

Endovac-Bov®, a vaccine against *E. coli* mastitis, has also been tried against endotoxicosis in camelids. The vaccine enhances both the T- and B-lymphocytes, and in combination with the mutant Re-17 bacterin it seems to protect against other endotoxin-mediated diseases. Its efficacy has not been proven in camelids.

Hundreds of valuable racing camels have succumbed to endotoxemia on the Arabian Peninsula. Since much is now known about the pathogenesis of this devastating disease, research should be directed into the prevention and prophylaxis of endotoxemia. It has been shown that even modest grain feeding can cause severe acidosis with fatal consequences (Cebra et al., 1996). The practices of feeding cow milk, dates, honey, excessive uncrushed barley and alfalfa should be carefully considered as well as the prophylactic administration of probiotics, antisera to endotoxins and paramunity inducer like Baypamun®. Furthermore, training of very young racing camels should be avoided in order to reduce stress.

1.1.5 Pasteurellosis

Pasteurella species have a worldwide distribution with a wide host spectrum. Most pasteurella organisms are commensals on mucous membranes of the upper respiratory and intestinal tracts of animals. *Pasteurella multocida* has been isolated from the respiratory tract of healthy NWC and no reports exist that pasteurella causes disease in NWC (Fowler, 1998), except one from Fowler and Gillespie (1985) of a llama with osteitis of the ear. *Pasteurella* sp. was isolated from a slight exudation of the left external ear. OWC seem to be less susceptible to pasteurella than ruminants (Awad et al., 1976b) and few scientists have observed hemorrhagic septicemia (HS) caused by *P. multocida* in dromedaries (Hassan and Mustafa, 1985).

Etiology The *Pasteurella* are small, Gram-negative rods or coccobacilli. They are non-motile, non-sporing and facultative anaerobes. They are oxidase-positive and catalase-positive. *Pasteurellae* grow best on media enriched with serum or blood. The mechanism of disease production by *Pasteurella* is not fully understood, but it is known that endotoxins are particularly important in septicemic cases such as HS. It is not known if camelids harbor their own *Pasteurella* species. Some scientists believe that camelids are not susceptible to bovine *Pasteurella* species.

Types or serotypes of *P. multocida* have been identified based on differences in capsular substances (polysaccharides). These polysaccharides have been designated A, B, C, D and F. Somatic types (lipopolysaccharides) have also been identified and given numbers. A *P. multocida* serotype is identified by its serotype followed by its somatic type. For example: *P. multocida* E: 978 or B: 925, the first one being the cause of HS in Africa, the second in Southeast Asia. *P. haemolytica* has analogous capsular types, which are identified by numbers.

Epidemiology and Clinical Signs *Pasteurella* species can be found associated with numerous animal diseases, and although they are responsible for a few primary diseases, their main role is as the causative agents of secondary disease. The nomenclature of diseases caused by pasteurella organisms is non-uniform and confusing. Blood and Radostits (1990), Seifert (1992), De Alwis (1992), as well as Smith (1994, personal communication) differentiate the following diseases:

- hemorrhagic septicemia (HS) in cattle and buffalo caused by *Pasteurella (P.) multocida*, serotype B (1) and E;
- pasteurellosis in cattle (shipping fever) accompanied by bronchopneumonia, caused by *P. multocida*, serotype A (2) and *Pasteurella (Mannheimia) haemolytica* (A1 and A2);
- pasteurellosis in sheep and goats (enzootic pneumonia) caused by *P. haemolytica*, type A 2;
- "fowl cholera", a septicemic disease of chickens and waterfowl, caused by *P. multocida*;
- *P. anatipestifer* infection in ducks, geese, pheasants and quails.

Stress is thought to be of great importance in the initiation of pasteurellosis in large animals, i.e. in HS of cattle and buffalo and in transit fever (shipping fever) of cattle. Viruses occur along with the *Pasteurellae* in transit fever pneumonias (e.g. parainfluenza 3, bovine herpes virus 1, mucosal disease virus, bovine respiratory syncytial virus). The form of stress varies but often appears to be linked with overexertion and fatigue such as caused by working (e.g. use of Asian buffaloes for plowing at the beginning of the rainy season), trekking (hence the belief that "change in pasture" is a cause), transportation in mechanical vehicles with associated fear and prolonged muscle tension. The belief is that – once an index case occurs (in a stressed animal) – the organism undergoes a temporary increase in virulence, allowing it to pass to other less stressed individuals, especially if the group is housed in a crowded corral e.g. at night, or closely confined in a truck.

Different authors have documented *Pasteurella* infections in camels. However, there are discrepancies regarding the clinical presentation and the pathogenesis to a distinct species of *Pasteurella*. Mistaking outbreaks of *Pasteurella* with other diseases presenting similar clinical signs such as anthrax and salmonellosis (Donatien and Boue, 1944; Fazil and Hofmann, 1981; Mustafa, 1987) has led to uncertainty in defining this disease in camels. According to the WHO/FAO/ OIE (1961), HS occurs in Bactrians in the former Soviet Union, in dromedaries from Algeria, Sudan and Somalia, seasonally in Mauritania and is suspected to exist in Chad and the Sahara. Leese (1927) isolated *Pasteurella*-like organisms from exudates of 2 camels in India that exhibited acute pleurisy, pericarditis and peritonitis.

According to Higgins (1986), *P. multocida* exhibits three different clinical courses in camels: acute, peracute and an abdominal form. The latter is differentiated by diarrhea that is frequently mixed with blood. Chauhan et al. (1986) reported that HS in camels is a highly contagious disease caused by *P. multocida*. The disease spreads through contact and contaminated feed and water. Clinical signs are associated with fever, nasal discharge, lacrimation, dyspnea, congestion of mucous membranes, swelling of throat and neck, and pneumonia. Schwartz and Dioli (1992) suggest that the acute form is identical with HS. However, various authors believe the camel to be generally very resistant to HS (Leese, 1918; Cross, 1919; Gatt Rutter and Mack, 1963) and not susceptible to bovine pasteurellosis.

P. multocida outbreaks in camels have been reported in various African countries, Russia, India and Iran (Table 13). Schwartz

and Dioli (1992) have characterized HS as being associated with pyrexia (up to 40°C), tachycardia and tachypnea, anorexia and extremely painful swellings on the neck. The mandibular and cervical lymph nodes are swollen and, in nearly all cases, a hemorrhagic enteritis occurs with tar-like feces. A further symptom of this disease is the occurrence of chocolate colored urine. Outbreaks of HS are mainly seen in the rainy season and in areas that are regularly flooded. The disease occurs primarily in adult camels but can be seen in all age groups. The morbidity is low, but the mortality can reach 80% (Schwartz and Dioli, 1992). Momin et al. (1987) reported an outbreak of pasteurellosis in India in which 11 out of 14 dromedaries died. The animals developed high fever, cervical edema with acute respiratory problems and sudden death. Bipolar organisms were seen in blood smears that resembled *P. multocida*. No laboratory confirmation has been performed on any of these cases and it is believed that the disease could have been confused with anthrax, since the clinical signs and lesions described resemble anthrax.

Pasteurella multocida, serotype B, was isolated by Hassan and Mustafa (1985) from the organs and bone marrow of Sudanese dromedaries that died during an HS outbreak. The authors were able to prove that this strain causes HS in cattle calves. The application of a bouillon culture to rabbits led to their death within 24 hours. However, the disease was not reproduced in dromedaries, but a bacterin vaccine used for cattle and sheep controlled the outbreak in the camels.

As in other animals, *Pasteurella* are also symbionts in camels. They are found on mucous membranes (mainly in the upper respiratory tract) assuming pathogenicity when the host's resistance is lowered due to a disturbance of the host-parasite balance as in mange, trypanosomosis or heat stress (Higgins, 1986). Different authors have reported on the clinical course following experimental infection of dromedaries with cultures of *Pasteurella*. Fayed (1973) isolated 6 *P. multocida* strains from 100 nasal swabs of healthy dromedaries in Egypt. All of the isolates were pathogenic for mice and rabbits. However, two dromedaries that were infected intranasally with these strains recovered following a brief period of illness. Cross (1919) also inoculated two dromedaries with a bovine "HS culture" and observed no systemic disease other than minor local swelling.

Awad et al. (1976a and b) reported inappetence, fever, hypersalivation, rapid pulse and respiration in dromedaries following intramuscular and nasal application of *P. multocida*, type 1. *Pasteurella* was re-isolated from the saliva, but not from the blood. The dromedaries infected by this route all recovered after 5 days.

In addition to the septicemic form of *Pasteurella* infections, different authors have described other clinical presentations. Donatien and Larrieu (1922) observed pneumonia, generalized myositis and diarrhea as well as exudative pericarditis and peritonitis (Donatien, 1921). Richard (1975) believes that abortions occur more frequently in conjunction with *Pasteurella* infections. None of these reports provide any information regarding the isolation or identification of the causative agent.

In a small field trial (Wernery et al. 1994, unpublished) two *P. multocida* strains (type B: 925; type E: 978), which are both highly virulent in cattle and buffalo (Smith, 1994, personal communication), were sprayed into the nostrils (5 mL of nutrient broth containing 10^6 CFU/mL) of two healthy 9-month-old camels. These camels developed no signs of illness. Furthermore 5 mL of the same strains containing 10^6 CFU/mL were given intratracheally into four healthy 8-month-old camels (Fig. 37).

Two out of the four camels developed an increase in body temperature to 39.2°C, a slight rise in white blood cell count (WBC)

Figure 37
5 mL containing 10^6 CFU/mL of *P. multocida* type E is injected into the trachea of an 8-month-old dromedary

and one out of the two also showed a slight mucopurulent nasal discharge from which no *P. multocida* was isolated. After 3 days the body temperature and the WBC had reached normal values, and no nasal discharge was detected. Tesfaye (1996) and Bekele (1999) reported a respiratory disease that has caused 29.6% morbidity and 6.4% mortality in the Somalian region of Ethiopia. *P. haemolytica* was isolated from the lungs, thoracic fluid and whole blood from diseased and dead animals that showed fever, depression, loss of appetite and severe nasal discharge. Necropsied dromedaries revealed hydrothorax, pneumonia, emphysema, hydropericardium and fibrinous pericarditis. Early treatment with oxytetracyclines resulted in the recovery of many diseased camels. The authors believe that a morbillivirus may have been the initiator of this outbreak. It was not clear from the authors' report whether this outbreak had any connection with the one reported by Yigezu et al. (1997) (see under chapter 1.3.2 Pneumonia). However, this is the first recent report that dromedaries can suffer from pasteurellosis.

A comprehensive scientific study is necessary to clarify the disease complex "Pasteurellosis in camelids". Diseases with similar clinical pictures such as anthrax, salmonellosis and endotoxemia mentioned above would be less likely to be confused with pasteurellosis. Although *Pasteurella* infections (*P. multocida* and *P. haemolytica*) are widespread among sheep, goats and cattle in the Emirates and dromedaries live in close association with the smaller ruminants, the authors have not had evidence of or encountered one case of HS among 30,000 racing dromedaries during a period of 15 years. As previously mentioned, this may be due to the excellent management and the superb feed given to dromedary herds in the UAE. It is unlikely that pasteurellosis is an important disease in OWC (Manefield and Tinson, 1996).

Serological studies by various authors have identified the presence of antibodies to *P. multocida*, serotypes A, B, D, E and *P. haemolytica*, type I (Table 13). The sera were obtained from healthy dromedaries, further proof that many dromedaries are host to the organism without any ill effects.

Diagnosis The aforementioned demonstrates that most of the reports about pasteurellosis in camels are confusing and often contradictory. A diagnosis can only be

Table 13 Occurrence of *Pasteurella* infections in camels in various countries

Country	Year	Author	Disease/Isolate
Mauritania	1985	Kane	
	1987	Kane	*P. multocida* E antibodies
India	1927	Leese	Swelling in the neck region
	1968	Ramachandran et al.	
	1987	Dahl	
	1987	Momin et al.	Septicemia, *P. multocida*
Chad	1967	Maurice et al.	Serology: 427 sera, 80% positive:
	1968	Perreau and Maurice	*P. multocida* A, B, E, D and *P. haemolytica*
	1971	Perreau	
Egypt	1976a, b	Awad et al.	*P. multocida* I (experimental infection) Inappetence, fever, hypersalivation
	1973	Fayed	*P. multocida* from healthy dromedaries
Sudan	1985	Hassan and Mustafa	*P. multocida* B, HS
French North Africa	1921	Donatien	HS confused with anthrax, salmonellosis ? Mortality 50%
	1922	Donatien and Larrieu	Fever, inappetence, myositis, pneumonia, diarrhea
Iran	1936	Delpy	*Pasteurella* isolated
	1969	Goret	
	1943	Ono	Hemorrhagic enteritis
Ethiopia	1975	Richard	Serology: 161 sera, 65% positive: *P. multocida*, A, B, D, E
Tunisia	1975	Burgemeister et al.	No reaction in 52 sera
Russia	1965	Oinakhbaev	
	1973	Sotnikov	*P. multocida*

made on the epidemiology, clinical signs, pathology and the isolation of *Pasteurella* organisms from blood, liver, spleen, kidney and lymph nodes. Specimens of the bone marrow in cases that have been dead for some time should be submitted. Intraperitoneal inoculation of mice is sometimes necessary to recover *Pasteurellae* from clinical samples that contain large numbers of other bacteria. Specific identification of the organism as to species and serotype is essential to establish if *Pasteurella* bacteria unique to camelids exist. Serotyping should be done in reference laboratories.

Treatment and Control The acute nature of pasteurellosis limits the efficacy of antimicrobial therapy of sick animals. However, an outbreak may be controlled by the early administration of sulfonamides, penicillin or oxytetracyclines to healthy camelids that only show a febrile reaction.

Large-scale vaccinations of cattle and sheep against pasteurellosis are practiced in Asia and Africa and dromedaries are also vaccinated against HS in the Emirates. There has also been considerable success in Asia by the immunization of buffaloes with alum-precipitated or oil-adjuvant vaccines. Vaccination with bacterin and alum (Alum potassium sulfate) *Pasteurella* vaccines to control outbreaks of HS in dromedaries was reported by Hassan and Mustafa (1985) and Momin et al. (1987).

Mohamed and Rahamtalla (1998) used an indirect hemagglutination (IHAT) and a mouse protection test (MPT) to assess the antibody response in dromedaries vaccinated with HS type B plain bacterin, alum precipitated vaccine and the combination of the two vaccines. The authors could show that sera from camels vaccinated with vaccines containing type B *P. multocida* antigen seroconverted and protected mice against challenge with *P. multocida* type B. However, no challenge experiments were performed in vaccinated and unvaccinated dromedaries.

1.1.6 Camel Plague

In previous centuries, *Yersinia pestis* produced pandemics which killed millions of people. It is said that the "Black Death" killed 40 million Europeans before 1400 AC, cutting Europe's population by one third. Nowadays plague is still endemic in many countries of Africa, in the former Soviet Union, Indonesia, India, Vietnam, and in some parts of North and South America where natural foci exist. The recent outbreak in humans in Zambia was linked to heavy rain and flooding, causing rats to invade higher grounds. *Y. pestis* is mainly transmitted by fleas from tolerant rodents. Cats are also susceptible to rabbit *Y. pestis* and can therefore pose a health hazard to humans in endemic areas. There are two forms of plague. In bubonic plague, the bacteria reach the regional lymph nodes, which become inflamed, soft and may suppurate (buboes). Dissemination via the blood stream may lead to pneumonia and meningitis. The pneumonic plague is an airborne infection and droplets may allow aerosol infection between humans. This form of plague is fatal. Plague has been reported to occur in OWC and both Bactrians and dromedaries play an important role in the transmission to humans (Sotnikov, 1973).

Etiology *Y. pestis* is a short, oval coccobacillus with rounded ends, occurring singly or in pairs when directly stained from tissue or exudate. In fluid culture, the bacilli tend to form chains. *Y. pestis* is Gram-negative, non-motile, non-sporing and capsulated. In smears from tissues stained with methylene blue, the bacilli show characteristic bipolar staining. *Y. pestis* grows on nutrient, blood and McConkey agars. Great care must be taken during necropsy of an animal that might be infected with plague.

Epidemiology and Clinical Signs The camel's role in the epidemiology of plague has been known for hundreds of years (Curasson, 1947; Fedorov, 1960). Wu et al. (1936) and Pollitzer (1954) have reviewed past reports of camel plague and determined that many scientists are skeptical about the earlier reports of plague outbreaks in camels. Fedorov (1960) considered that *Yersinia pestis* infections play an important role as anthropozoonoses as well as zooanthroponoses, even up to the present. Sotnikov (1973) reported outbreaks of plague in camels in Mongolia, China, India, Iran, Iraq, Africa and Russia. Plague outbreaks among Bactrian camels have been known in Russia since 1911 and various plague outbreaks in man were due to contact with Bactrian camels. One such outbreak in Russia affected numerous people following the consumption of infected camel meat (Kowalevsky, 1912). The last reported outbreak of plague in Russia occurred in 1926 (Strogov, 1959).

Plague as a zoonosis has played a role in the past, not only in Russia, but also in Mauritania and Libya where outbreaks of plague involving men and dromedaries have been recently reported by Alonso (1971) and Christie et al. (1980). *Yersinia pestis* was isolated from buboes in dromedaries. Bubonic plague, described by Sacquepee and Garcin (1913) as occurring among dromedaries of French North

Africa, not only affected the lymph nodes, but also caused abscesses disseminated over the entire body. *Y. pestis* was isolated from these lesions as well as from pleural effusions. In addition to a cutaneous manifestation, septicemic and pulmonary forms also occur in the camel (Lobanov, 1959 and 1967). The incubation time in camels is 1 to 6 days followed by death within 20 days. Martynchenko (1967), Alonso (1971) and Klein et al. (1975) described the clinical presentation of camel plague in dromedaries in Turkmenistan, Algeria and Mauritania. The authors also proved that the flea is the main vector of disease transmission among camels. Ticks of the genus *Hyalomma* and *Ornithodoros* are also able to transmit the disease mechanically (Fedorov, 1960).

Treatment and Control Prevention involves eliminating contact with infected rodents, cats and rabbits and their fleas. Before necropsy of a plague-suspected camel is carried out, the entire carcass should be sprayed with insecticides to destroy any ectoparasites.

Streptomycin and tetracyclines in combination are effective and, based on human cases, should be administered for at least 5 days. Sotnikov (1973) used a freeze-dried anti-plague vaccine for the immunization of camels; their immunity lasted for 6 months. A genetically modified vaccine against bubonic plaque has recently been developed in Britain, mainly to protect armed forces operating in countries where plaque occurs naturally and where *Y. pestis* may be used in biological warfare.

1.1.7 Leptospirosis

Leptospirosis occurs worldwide and there are reports of leptospirosis in OWC as well as NWC (Wernery and Kaaden, 1995; Fowler, 1998). Leptospires are present in tubules of mammalian kidneys and are excreted in urine, often for several months. Streams and ponds can be the source of infection as well as aerosols of urine in cowsheds and milk from infected cows.

Etiology The order *Spirochaetales* includes the families *Spirochaetaceae* and *Leptospiraceae* with the following genera, which are of significance to animals and humans:

- Spirochaetaceae:
  Serpulina (Brachyspira)
  Treponema
  Borrelia
- Leptospiraceae:
  Leptospira

Leptospira are spirochetal organisms divided into serotypes based on their antigenic structure. Within the genus *Leptospirae*, only the species *L. interrogans* is of medical importance. All of the pathogenic leptospirae are included under this designation. Due to a varying antigenic structure, *L. interrogans* consists of 19 serogroups and approximately 180 serotypes (serovars).

Leptospires can be demonstrated in urine, body fluids and tissues by dark field microscopy and fluorescent antibody technique (FAT). Leptospires grow in special media like Stuart or Korthof broths.

Epidemiology *Leptospira* are found ubiquitously around the world. All domesticated animals, wild game, rodents in particular, as well as man are susceptible to infection. In some animals, chronic renal involvement serves as a reservoir for the organism (Bisping and Amtsberg, 1988). Direct or indirect infection of man or animal is possible from these reservoirs via contact with infected urine or ingestion of urine-contaminated food or water. A broad spectrum of manifestations, from inappetence to more severe clinical signs, can be expected. According to Seifert (1992), rodents and dogs serve as the most important epidemiological reservoirs in intensive cattle husbandry in the tropics. Man,

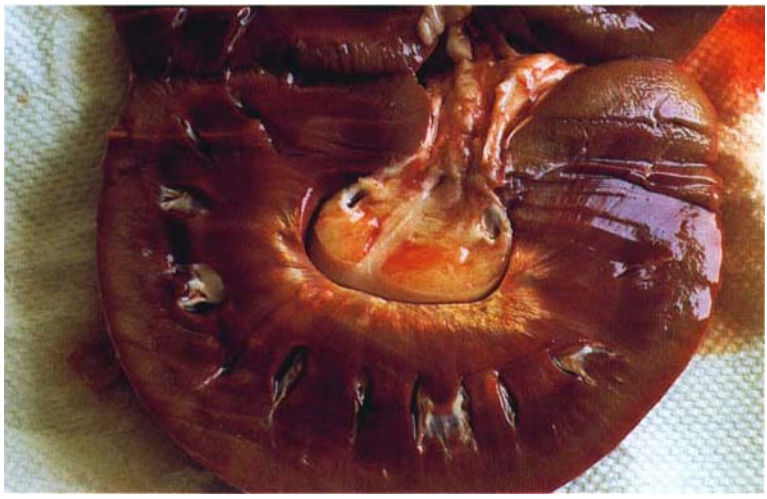


Figure 38 Dromedary suffering from hematuria: calcification of the renal papilla

living in close contact with his animals, can also be affected.

Wilson (1984) and Higgins (1986) considered leptospirosis as being insignificant in OWC. The clinical presentation of leptospirosis in OWC has not yet been described and there is some doubt as to whether the camel is even susceptible to the disease. Rafyi and Maghami (1959) as well as Higgins (1986) suspected that hematuria may occasionally be caused by *Leptospira*. Wilson (1984) also observed hematuria in dromedaries without finding a cause. Bloodied urine occurs in both genders of racing camels in the UAE, but is not associated with leptospiral infection (Wernery and Wernery, 1990). Serological examinations of and cultural isolation of Leptospirae from 50 dromedaries with hematuria were negative. Hematuria has mainly been observed in the Emirates among racing dromedaries, but rarely in breeding stock. Intensive microbiological examination and serum biochemistry (cre-

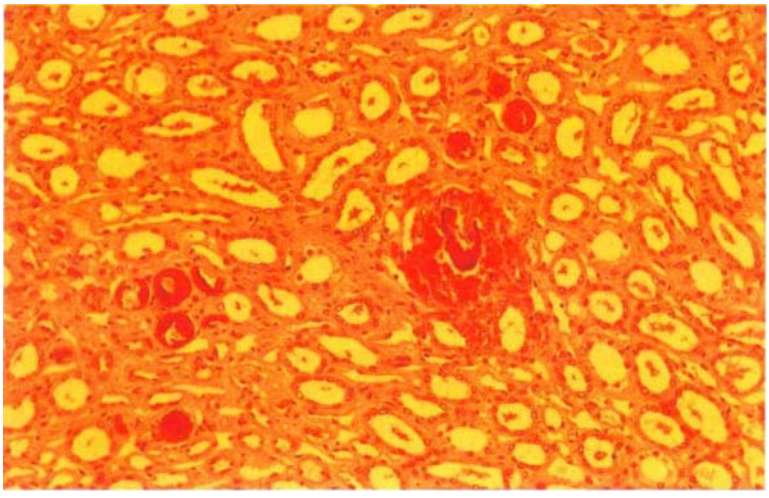


Figure 39 Histological preparation (HE stain) of a kidney from a dromedary with hematuria: spot-like paratubular calcification with hemorrhage

atinine and urea) yielded no indication of renal infection or renal insufficiency as the cause of the hematuria. Also, examination of the urine did not disclose an increased precipitation of crystals or casts. The affected dromedaries exhibited no signs of kidney-related pain or systemic disease. During intensive research of hematuria in dromedaries, one animal with hematuria was euthanized and the urinary organs examined. This revealed massive calcification of the distal renal tubules surrounded by hemorrhages (Figs. 38 and 39) as well as focal glomerulonephritis.

The etiology of this disseminated renal calcification has not as yet been elucidated. It has been surmised that these deposits result from the higher mineral content of the feed given to the racing dromedaries. This would explain why hematuria has rarely been known to occur in breeding stock. Breeding animals are given a less well-balanced diet, consisting mostly of hay. Further studies should clarify possible connections in the etiology of the hematuria.

Krepkogorskaya (1956) is the only author to have isolated *Leptospira* from camel organs. The following species were identified: *L. kazachstanica* I, II and *L. vitulina*. No clinical signs of disease were described. All other studies report agglutinating antibodies specific for different leptospiral serovars (Table 14). These studies originated in various African countries, Afghanistan, Iran, India, Russia, Mongolia and the UAE.

Maronpot and Barsoum (1972) found leptospiral antibodies in 34% of the dromedaries examined in Egypt. The authors are of the opinion that subclinical leptospirosis in dromedaries is a worldwide phenomena and therefore may pose a health risk for man.

Wernery and Wernery (1990) found serological reactions to *Leptospira* in 2.5% of breeding stock and 5.6% of racing dromedaries. The authors have not observed clinical leptospirosis in any of 30,000 dromedaries over a period of 15 years.

Leptospirosis has been described in alpacas (Ludena and Vargus, 1982) and in a 3-month-old guanaco at the Detroit Zoological Park (Hodgin et al., 1984), but in general the studies of leptospirosis in lamoids are not clearly defined. It is believed that clinical signs and pathology changes are similar to those in other species. Leptospires gain entry into the organism through mucous membranes or damaged skin. They localize and proliferate in parenchymatous organs after hematogenous spread. In the kidneys, the organisms propagate in the lumen of the proximal convoluted tubules. Here the leptospires persist for long periods. Some strains produce hemoglobinuria (red water). Gross lesions include icterus of the mucous membranes and of the fat, and in histology there may be interstitial and tubular nephritis.

Diagnosis In suspected leptospirosis serology, dark-field examination of urine and FA technique of smears or cryostat sections from organs as well as cultivation and laboratory animal inoculation are used for diagnosis. However, leptospires may only be isolated during the short acute stage of the disease. Serology titers of over 1:100 using the microscope agglutination test are considered positive. This test, which uses live leptospires as antigen, is highly sensitive and serovar-specific. Since it is difficult to interpret a disease from a single sample, sera from acute and convalescent cases should be collected.

Treatment Clinically ill camelids can be treated successfully with 25 mg/kg body weight of dihydrostreptomycin administered intramuscularly for 5 days. A wide range of bacterin vaccines are available for farm animals, but since leptospirosis is of less importance in *Camelidae*, vaccination with the appropriate strain is only recommended in endemic areas.

Table 14 Leptospirosis in camels, prevalence and serotypes

Country	Year	Author	Prevalence %	Serotypes
Afghanistan	1972	Sebek et al.		*L. grippotyphosa*
	1974	Sebek	0.8	
	1978	Sebek et al.		
Sudan	1974	Shigidi	0.0	–
Ethiopia	1975	Moch et al.	15.4	*L. grippotyphosa* *L. pyrogenes* *L. butembo* *L. borincana*
Egypt	1964	Brownlow and Dedeaux		
	1972	Maronpot and Barsoum	34.0	*L. pyrogenes* *L. tarassovi* *L. autumnalis* *L. butembo* *L. javanica*
	1976	Hatem Ahmed	9.2	*L. pyrogenes* *L. tarassovi* *L. butembo*
Iran	1959	Rafyi and Maghami	20.0	*L. icterohaemorrhagiae*
Somalia	1960	Farina and Sobrero	16.2	*L. icterohaemorrhagiae* *L. canicola* *L. grippotyphosa* *L. ballum*
	1982	Arush	0.0	–
	1986	Hayles		
India	1986	Mathur et al.	51.4	*L. canicola* *L. icterohaemorrhagiae* *L. ballum* *L. pomona* *L. wolfei* *L. autumnalis*
Tunisia	1975	Burgemeister et al.	48.0	*L. icterohaemorrhagiae* *L. pomona* *L. bataviae*
	1989	Gallo et al.	0.0	–
Russia	1956	Krepkogorskaya		*L. kazachstanica I* *L. kazachstanica II* *L. vitulina* (serologically and in culture)
Mongolia	1974	Sebek		
	1988	Sosa et al.		Zoo camels
UAE	1990	Wernery and Wernery	2.5	Breeding dromedaries
			5.6	Racing dromedaries
	1994	Afzal and Sakkir	4.1	*L. interrogans*

1.1.8 Rickettsial Diseases

Rickettsiae are tiny obligate intracellular Gram-negative bacteria. They are important parasites of arthropods and replicate in the gut cells. Rickettsiosis has been described in NWC (Barlough et al., 1997), but there are no reports that the disease occurs in OWC (Wernery and Kaaden, 1995).

Etiology *Rickettsiae* are often erroneously called large viruses. However, they are true bacteria. They possess both DNA and RNA, multiply by binary fission, have their own metabolism and are sensitive to some antibiotics. *Rickettsiae* are rods and coccobacilli, non-motile and aerobic. They stain poorly with basic aniline dyes, which are used in Gram stain, but they stain well with Romanowsky stain or Giemsa stain. Most of the *Rickettsiae* require living cells for their multiplication. They may be cultured in tissue cultures or embryonated chicken eggs.

With the exception of *Coxiella burnetii*, *Rickettsiae* are typical causative agents of vector epidemics, since mammals can only be infected with the help of insect intermediates. *C. burnetii* can be transmitted either by ticks or by inhaling contaminated dust. A systemic classification of *Rickettsiae* important to veterinary medicine is presented in Table 15 and their diseases in Table 16.

Table 15 Taxonomic classification of *Rickettsiae* (modified from Bisping and Amtsberg, 1988) and their vectors

Classification		Vector
Order 1:	*Rickettsiales*	Tick
Family 1:	*Rickettsiaceae*	Louse
Tribe 1:	*Rickettsiae*	Flea
Genera:	*Rickettsia*	Mite
	Rochalimaea	*Ixodidae*
	Coxiella	*Heteroptera*
Tribe 2:	*Ehrlichieae*	Tick
Genera:	*Ehrlichia*	
	Cowdria	
	Neorickettsia	
Tribe 3:	*Wohlbachieae*	
Genera:	*Wohlbachia*	
	Rickettsiella	
Family 2:	*Bartonellaceae*	
Genera:	*Bartonella*	
	Grahamella	
Family 3:	*Anaplasmataceae*	Tick
Genera:	*Anaplasma*	Horsefly
	Paranaplasma	Mosquito
	Aegyptianella	Louse
	Haemobartonella	Flea
	Eperythrozoon	
Order 2:	*Chlamydiales*	see 1.4.3
Family:	*Chlamydiaceae*	
Genus 1:	*Chlamydia*	
Genus 2:	*Chlamydophila*	

Epidemiology and Clinical Signs Q-Fever, petechial fever, tick fever, Heartwater and anaplasmosis can cause great losses in cattle and small ruminants in the tropics (Blowey and Weaver, 1991; Seifert, 1992). Although various authors have identified antibodies to *Coxiella burnetii*, *Rickettsia prowazekii*, *R. rickettsii*, *R. mooseri*, *R. conorii*, *Anaplasma* and *Cowdria*, there have been no reports of disease or losses in the camel due to this group of organisms. A few authors (Beer, 1987; Legel, 1990) mention that camel may suffer from cowdriosis, but there was no original case report traced by the authors. Reiss-Gutfreund (1955), who isolated *R. prowazekii* from ticks *(Hyalomma rufipes)* on dromedaries in Ethiopia, found no clinical signs of disease in the animals. This observation was confirmed by Ormsbee et al. (1971), who did not succeed in re-isolating *R. prowazekii* from the blood of young dromedaries that had been artificially infected. The authors are of the opinion that dromedaries do not play any role in the epidemiological cycle of classical epidemic typhus.

Infections with *Anaplasma* in dromedaries appear to be subclinical. Reports from Somalia (Monteverde, 1937; Anonymous, 1939 and 1960) regarding cases of *Anaplasma marginale* in healthy dromedaries support this observation. Kornienko-Koneva

Table 16 *Rickettsiae* of veterinary importance and their diseases

Family	Genus	Species	Cell parasitism	Disease
Rickettsiaceae	*Coxiella*	*burnetii*	in cell vacuoles of the reticulohistiocytic system	Q-Fever
	Ehrlichia (Cytoecetes)	*equi*	Granulocytes	Ehrlichiosis
		phagocytophila		
	Ehrlichia	*canis*	mononuclear cells	
	Cowdria	*ruminantium*	cytoplasm of the vascular endothelium	Heartwater
Anaplasmataceae	*Anaplasma*	*marginale*	marginal (in erythrocytes)	Anaplasmosis
		centrale	central (in erythrocytes)	
		ovis	marginal	
	Aegyptianella	*pullorum*	in erythrocytes	Aegyptianellosis
	Haemobartonella	*felis*	on erythrocytes (in folds)	Hemobartonellosis
	Eperythrozoon	*wenyoni*	on erythrocytes	Eperythrozoonosis
		ovis	on erythrocytes	
		suis	on erythrocytes	

(1955) was successful in transmitting *Anaplasma*-contaminated camel blood to cattle. However, the two-humped camel may be susceptible to natural infection of *A. marginale*. Ristic and Kreier (1974), Ristic (1977) and Ajayi et al. (1984) found antibodies to *A. marginale* in 10.7% (3/28) Nigerian camel sera using 3 different serological tests.

C. burnetii is the organism responsible for Q-fever, a zooanthroponosis. The role of rodents and domesticated animals as hosts or reservoirs for infection in man has long been established. The dromedary is no exception. Numerous authors (for example Maurice and Gidel, 1968; Mathur and Bhargava, 1979) have indicated the danger of rickettsial disease in humans due to close contact with dromedaries. The greatest danger is most likely from the consumption of raw camel milk.

Different authors have identified antibodies to various rickettsial species in the camel. A summary appears in Table 17.

Eperythrozoonosis has frequently been identified in young llamas (McLaughlin et al., 1990; Semrad, 1994). Juvenile llamas, from weaning to several years old, have been found to have apparent immunodeficiency disorders. Such llamas have a history of weight loss and stunted growth and develop acute or recurrent infectious conditions. Affected llamas usually die or are euthanized because of the grave prognosis. In these cases, infections with uncommon pathogens or opportunistic microorganisms are often detected. During necropsy, severe fibrinous polyserositis involving the thoracic and abdominal organs, moderate diffuse non-suppurative interstitial pneumonia, splenic hyperplasia, necrotizing enteritis, widespread vascular thrombosis and anemic infarcts in the liver are observed. *Eperythrozoon*-like organisms resembling *Eperythrozoon suis* have frequently been diagnosed in these immunodeficient llamas. There is an indication that this *Rick-*

Table 17 Literature survey regarding rickettsial antibodies in OWC

Species	Author	Year	Country	Prevalence
C. burnetii	Blanc et al.	1948	Morocco	22.2
	Giroud et al.	1954	Chad	2.0
	Rafyi and Maghani	1954	Iran	
	Veeraghavan and Sukumaran	1954	India	
	Kalra and Taneja	1954	India	
	Elyan and Dawood	1955	Egypt	13.9
	Brown	1956	Kenya	20.0
	El-Nasri	1962	Sudan	0.0
	Imamov	1964	Kazakhstan	4.8
	Maurice et al.	1967	Chad	13.6
	Sabban et al.	1968	Egypt	4.8
	Bares	1968	Chad	
	Maurice and Gidel	1968	Central Africa	
	Pathak and Tanwani	1969	India	11.9
	Choudhury et al.	1971	India	23.8–26.9
	Harbi and Awad El Karim	1972	Sudan	12.2–12.8
	Kulshreshtha et al.	1974	India	17.3
	Burgemeister et al.	1975	Tunisia	15.8
	Gosh et al.	1976	India	5.6
	Schmatz et al.	1978	Egypt	
	Mathur and Bhargava	1979	India	6.7–7.7
	Addo	1980	Nigeria	12.0
	Harrag	1986	Tunisia	
	Abbas et al.	1987	Sudan	14.5
	Djegham	1988	Tunisia	3.06
	Gallo et al.	1989	Tunisia	0.0
R. prowazekii	Reiss-Gutfreund	1955	Ethiopia	Ticks
	Imam and Labib	1963	Egypt	44.1
	Maurice et al.	1967	Chad	1.8
	Bares	1968	Chad	11.6
	Reiss-Gutfreund	1970	Mongolia	Experimental
	Ormsbee et al.	1971	Egypt	Experimental
R. mooseri	Imam and Labib	1963	Egypt	26.0
	Maurice et al.	1967	Chad	11.6
	Reiss-Gutfreund	1970	Mongolia	Experimental
R. rickettsii	Bares	1968	Chad	1.8
	Schmatz et al.	1978	Egypt	3.7
R. conorii	Maurice et al.	1967	Chad	1.0
Anaplasma	Monteverde	1937	Somalia	40.0 (direct)
	Anonymous	1939		
	Anonymous	1960		
	Ristic and Kreier	1974		
	Ristic	1977		
	Anonymous	1981	Somalia	4.4 (direct)
	Ajayi et al.	1984	Nigeria	10.7
Cowdria ruminantium	Karrar et al.	1963		
	Karrar	1968	Sudan	

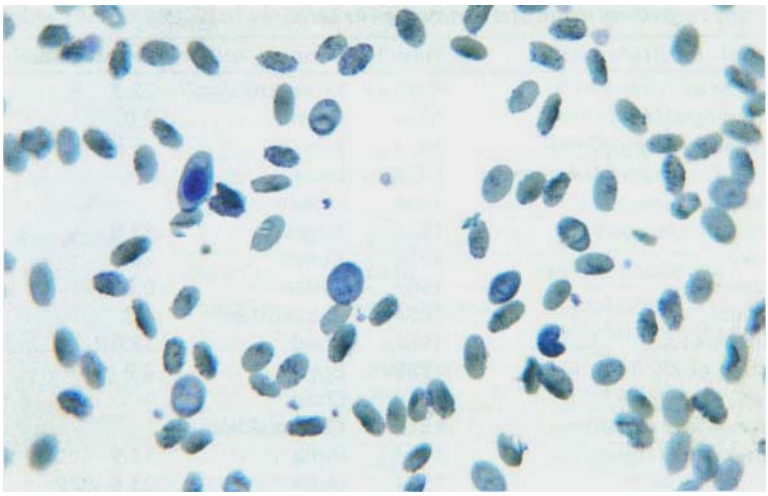


Figure 40 Eperythrozoonosis in a young llama suffering from immunodeficiency disorder (Giemsa stain)

ettsia is responsible for the anemia which often accompanies this ailment. *Eperythrozoon*-like parasites are attached to the surface of red blood cells of the affected llamas and are often found in clusters, usually towards the edge of the cell (Fig. 40) (Wernery et al., 1999).

Barlough et al. (1997) reported the identification of an *Ehrlichia* in a llama suffering from granulocytic ehrlichiosis. This *Ehrlichia* strain was sequenced showing close relationship to members of *Ehrlichia phagocytophila*. The same *Ehrlichia* was also found in llama-associated *Ixodes pacifus* ticks collected from the same llama farm. Clinical signs were non-specific and included lethargy, slight ataxia and anorexia. The llama showed a mild lymphopenia, monocytosis and eosinophilia. Cytoplasmatic inclusion bodies of *Ehrlichia* were detected in neutrophils, and the diagnosis "granulocytic ehrlichiosis" was made. The llama became recumbent, but after treatment with oxytetracyclines recovered fully.

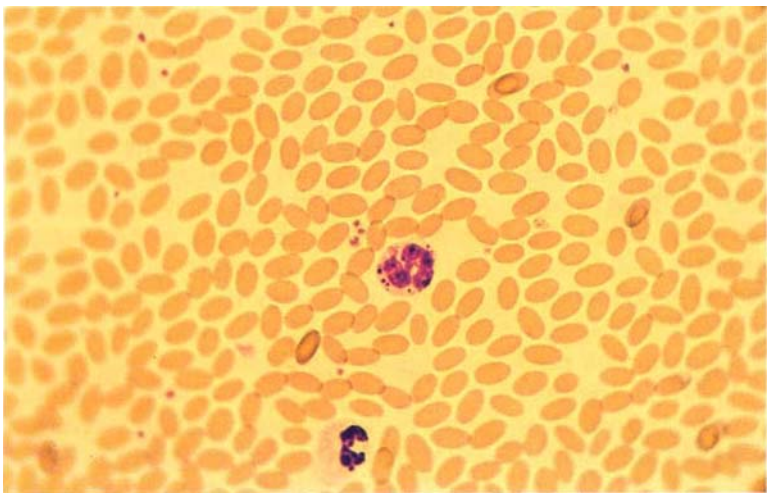


Figure 41 Eosinophilic inclusion bodies in the cytoplasm of a neutrophil of a guanaco with rickettsiosis (Giemsa stain)

Cytoplasmatic inclusion bodies were also observed in neutrophils of a guanaco in the UAE (Wernery et al., 1999) (Fig. 41).

This guanaco was also lethargic and anorexic and revealed a monocytosis and eosinophilia.

Diagnosis For the laboratory diagnosis of rickettsiosis, unclotted blood and affected tissue including brain (Heartwater) should be dispatched to the laboratory. As *Rickettsiae* are poorly stained by Gram, Giemsa, Romanowsky, Giminez, Machiavello or Leishman stains as well as FA staining are used for both blood and tissue smears. *Rickettsiae* do not grow on agars and it is therefore necessary to cultivate them in embryonated hen's eggs or tissue culture. Penicillin and streptomycin should be added to the test sample to suppress contaminants. Some embryos might die 6 days after infection and the remainder should be examined after 12 days. Demonstration of *Rickettsiae* by animal inoculation is also advisable, especially when few bacteria are expected in the sample. The animal of choice is the guinea pig. Furthermore, a serological diagnosis can be made for some rickettsial diseases like Q-fever on paired serum samples using the CFT, microagglutination or ELISA. Soliman et al. (1992) detected antibodies to *C. burnetii* in 66% of Egyptian dromedaries with the competitive enzyme immunoassay (CEIA). The laboratory diagnosis of important rickettsial diseases is summarized in Table 18.

Table 18 Laboratory diagnosis of important rickettsial diseases (after Quinn et al., 1994)

Disease	Laboratory diagnosis	Appearance of agent in Giemsa-stained smears
Q fever *(Coxiella burnetii)*	Fluorescent antibody (FA) or Giemsa-stained smears from ruminant placentas. Paired serum samples for serology (CFT, ELISA or microagglutination). Antibody rise 2–3 weeks post infection	Small purple-red cocci (0.2–4 µm) or short rods within cells. Similar in appearance to *Chlamydia psittaci* when stained with the Giemsa stain
Canine ehrlichiosis *(Ehrlichia canis)*	Giemsa-stained blood smears, best at about the 15th day post infection. Indirect FA test on serum for antibody	Purple-staining cells (0.5 µm diameter) or inclusions (morulae) up to 4.0 µm diameter in monocytes or lymphocytes
Equine ehrlichiosis *(Ehrlichia equi)*	Giemsa-stained blood or buffy coat smears. Inclusions can be seen 48 hours after onset of disease. Indirect FA test on serum for antibody	As for *E. canis* but cells or inclusions present in granulocytes, especially neutrophils
Potomac horse fever *(Ehrlichia risticii)*	FA or Giemsa-stained blood smears. ELISA or indirect FA for antibodies in serum	Purplish-staining agent in monocytes. Inclusions similar to other *Ehrlichia* spp.

Table 18 *(cont.)*

Disease	Laboratory diagnosis	Appearance of agent in Giemsa-stained smears
Tick-borne fever *(Ehrlichia phagocytophila)*	FA or Giemsa-stained blood smears	Purplish inclusions varying from 0.7–3.0 µm in neutrophils, eosinophils, basophils and monocytes
Heartwater *(Cowdria ruminantium)*	FA or Giemsa-stained smears from brain tissue (cerebral cortex). Inoculation of mice or susceptible cattle	Purple-staining cocci (0.2–0.5 µm) or short bacillary forms in cytoplasm of vascular endothelial cells of capillaries in the brain
Salmon poisoning *(Neorickettsia helminthoeca)*	Clinical signs and the finding of fluke eggs *(Nanophyetus salmincola)* in feces. Demonstration of the agent in lymph node aspirates	Purplish morulae in cytoplasm of macrophages with individual cocci (0.3–0.4 µm) scattered within the cells
Anaplasmosis *(Anaplasma marginale)*	Giemsa, acridine orange and FA staining of blood smears. Serology: indirect FA, CFT and card agglutination	Reddish-violet pleomorphic forms (0.2–0.4 µm diameter) within erythrocytes and near the periphery. Up to 50% of red cells may be parasitized
Avian aegyptianellosis *(Aegyptianella pullorum)*	Giemsa-stained blood smears. Inoculation of susceptible birds by parenteral routes or skin scarification with infected blood	Great variety of violet-reddish forms: oval, round and ring (0.3–3.9 µm diameter), and also larger inclusions in erythrocytes
Feline infectious anemia *(Haemobartonella felis)*	Giemsa or FA-stained blood or tissue smears. Check Giemsa-stained smears daily for 1 week as the presence of the agent on red cells is inconsistent	Deep purple, small coccoid or rod-shaped (0.2 µm diameter) organisms on erythrocytes. A few ring-forms occasionally seen
Ovine eperythrozoonosis *(Eperythrozoon ovis)*	Giemsa-stained blood smears. With acridine orange staining, there is bright orange fluorescence	Pale purple organisms in disc- or ring-forms (0.5–1.0 µm diameter). Rod-forms are most common at the margin of the erythrocytes
Porcine eperythrozoonosis *(Eperythrozoon suis)*	Giemsa or FA-stained blood smears. Serology: indirect FA or CFT	Bluish-violet cocci or ring-forms (up to 2.5 µm diameter) on erythrocytes. Largest species in the genus
Camelid rickettsiosis *(Anaplasma, Eperythrozoon, Ehrlichia)*	Giemsa, acridine orange blood smears. Serology: CFT	Reddish-violet-purple cocci in RBCs or WBCs

Treatment and Control Tetracyclines and chloramphenicol are the drugs of choice. For acute cases they should be administered for 2 weeks. Long-term medication of feed is sometimes necessary to eliminate carrier animals as in an *A. marginale* infection. Prevention is enhanced by controlling ectoparasites, since it is known that camel ticks spread many diseases, some of which are extremely dangerous to other livestock and humans.

1.1.9 Rhodococcus equi in New World Camelids

Corynebacteriae are small pleomorphic Gram-positive rods or cocci. They are pyogenic bacteria causing a variety of suppurative conditions in many animal species.

Rhodococcus equi is primarily an equine pathogen, but seems to play an important role in NWC (Leite et al., 1975; Elissalde and Renshaw, 1980). The authors described multiple caseous abscesses in the lungs, liver and spleen of llamas from North and South America from which *R. equi* was isolated (Fig. 42). A *R. equi*-associated necrotizing lymphadenitis in a llama was also reported (Hong and Donahue, 1995).

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Figure 42
Rhodococcus equi abscess in a llama liver (courtesy of Prof. M. E. Fowler, USA)

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1.2 Digestive System

1.2.1 Salmonellosis

The incidence of salmonellosis in humans has increased in recent years and animals have been incriminated as the principal reservoir. *Salmonella* infections occur worldwide in all animals and are the focus of intensive scientific study. A widespread distribution of known serotypes has occurred, induced in part by the global animal and food trade. Infections occur due to ingestion of feed or water contaminated with *Salmonella* as well as by direct contact with the contaminated excreta of carriers.

Etiology The genus *Salmonella* comprises a single species that has been divided into more than 2000 serotypes (serovars). The genus *Salmonella* is classified in the family *Enterobacteriaceae*, whose members are Gram-negative coccobacilli. With the exception of *S. gallinarumpullorum*, all *Salmonellae* are motile with peritrichous flagella. Salmonellosis in livestock is caused by the infection with both host-specific and non-host-specific *Salmonella* serovars. The disease is characterized by one or more of 3 major syndromes: septicemia, acute and chronic enteritis.

Epidemiology and Clinical Signs Numerous authors have reported salmonellosis and *Salmonella* infections in camels in different parts of the world. Reports of *Salmonella* infections have appeared from Sudan (Curasson, 1918), Palestine (Olitzki and Ellenbogen, 1943), French North Africa (Donatien and Boue, 1944), USA (Bruner and Moran, 1949) and more recently from Somalia (Cheyne et al., 1977), Ethiopia (Pegram and Tareke, 1981), Egypt (Refai et al., 1984; Yassien, 1985; Osman, 1995) and the UAE (Wernery, 1992). A literature summary appears in Table 19.

In camels, *Salmonella* can cause enteritis, septicemia and abortion. Chronic salmonellosis is characterized by diarrhea, weight loss and death within a few weeks (Fazil and Hofmann, 1981). Pegram and Tareke (1981) reported that salmonellosis in Ethiopia is the most important disease in young dromedaries, leading to losses of up to 20% in some parts of the country. In recent investigations of camel calf deaths, several scientists have reported severe *Salmonella* enteritis in association with *E. coli*, *Eimeria cameli*, rota- and coronaviruses. Faye (1997) reported 68.3% deaths in young dromedaries in Niger caused by a mixture of enteric pathogens: *Salmonella*, rotavirus, coronavirus and *E. coli* and *Eimeria cameli*. The author believes that *S. typhimurium*, *S. enteritidis*, *S. kentucky* and *S. saint-paul* are the most important serovars in camels. The disease manifests itself in hemorrhagic diarrhea with dehydration and death. Berrada et al. (1998) examined 27 fecal samples from diarrheic dromedary calves aged between 1 and 10 weeks raised in 9 herds in the Moroccan Sahara. From 14.8% of the diseased calves, 5 different *Salmonella* strains were isolated. Salih et al. (1998 a and b), who examined 106 diarrheic camel calves cultured *Salmonellae* from 14 (13%) of them. *S. typhi* was the most prominent strain. The highest incidence of salmonellosis was during the month of October. *Salmonellae* were also isolated from 8 healthy dromedary calves and from 1 diseased calf from the UAE (Nation et al., 1996) as well as from 9.5% (4/42) diarrheic camel calves from Sudan (Mohamed et al., 1998). Some *Salmonella* strains can cause an unusually wide range of clinical syndromes including ischemic necrosis of the tips of the ears, tail or limbs. Nothelfer et al. (1995) reported a case of ear tip necrosis. The dried-off ear parts could easily be re-

Table 19 Summary of literature regarding salmonellosis and *Salmonella* infections in camelids

Author	Year	Country	Number of serotypes	Disease
Kowalevsky	1912	Russia	not typed	enteritis
Curasson	1918	Sudan	not typed	enteritis
Olitzki	1942	Palestine	1	none
Olitzki and Ellenbogen	1943	Palestine	1	enteritis
Donatien and Boue	1944	French North Africa	not typed	abortion enteritis septicemia
Sandiford	1944	Egypt	1	enteritis
Bruner and Moran	1949	USA	2	enteritis
Floyd	1955	Egypt	3	none
Zaki	1956	Egypt	1	none
Farrag and El-Afify	1956	Egypt	1	none
Hamada et al.	1963	Egypt	2	none
Kamel and Lotfi	1963	Egypt	7	none
Malik et al.	1967	India	6	none
Ramadan and Sadek	1971	Egypt	8	none
Ambwani and Jaktar	1973	India	5	none
Cheyne et al.	1977	Somalia	1	enteritis
Andreani et al.	1978	Somalia	1	None
El-Monla	1978	Egypt	7	None
Sayed	1979	Egypt	5	None
Pegram and Tareke	1981	Ethiopia	2	septicemia
Elias	1982	Egypt		enteritis
El Nawawi et al.	1982	Egypt	5	None
Refai et al.	1984	Egypt	11	None
Yassien	1985	Egypt	5	None
Selim	1990	Egypt		None
Pegram	1992	Ethiopia	6	enteritis
Wernery	1992	UAE	28	None
Anderson et al.	1995	USA	llama	septicemia
Nation et al.	1996	UAE	3	diarrhea
Faye	1997	Niger	4	diarrhea septicemia
Berrada et al.	1998	Morocco	5	diarrhea
Salih et al.	1998a 1998b	Sudan	1	diarrhea
Mohamed et al.	1998	Sudan	4	diarrhea

moved by hand leaving a clear and slightly bleeding surface (Fig. 43).

This is evidence that endotoxin damages the endothelium of blood vessels leading to a localized disseminated intravascular coagulation that causes terminal ischemia.

Selim (1990) compared two groups of dromedaries in Egypt. They found that 3% of healthy dromedaries showing no sign of diarrhea were *Salmonella* carriers, compared to 17% of dromedaries with enteritis.

Salmonellae have been isolated from the feces of healthy camels in India (Malik et al., 1967; Ambwani and Jaktar, 1973) and in the UAE (Wernery, 1992), as well as from

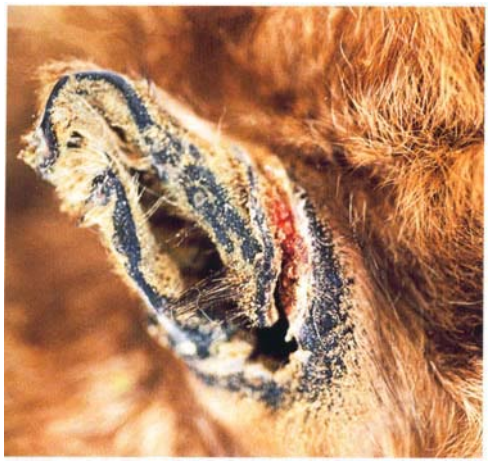

Figure 43 After removal of the dried-off tip of the ear, a clean, slightly bleeding surface is visible

the lymph nodes and intestines of slaughtered dromedaries in Egypt (Zaki, 1956; Hamada et al., 1963; El-Nawawi et al., 1982; Refai et al., 1984; Yassien, 1985).

As seen in Table 20, different *Salmonella* serotypes were isolated in various countries. However, the *Salmonella* spp. isolated from diseased and healthy camels were identical. This variation in pathogenicity is a consequence of individual resistance, either of the individual animal or of the breed of animals. Disease resistance is due to a combination of a genetically determined insensitivity towards certain pathogenic microorganisms (Mayr, 1991), the age, immunological status, stamina and condition of the animal. Additionally, the infective dose and stressors play an important role in outbreaks of salmonellosis in older animals. It is generally accepted that this disease can be promoted by transportation, malnutrition, birth, over-stocking, surgery and medication (Blood and Radostits, 1990).

Wernery et al. (1991) reported a *Clostridium perfringens*, type A outbreak among racing dromedaries in the UAE. The authors indicated that a concurrent infection with *S. saint-paul* and *S. cerro* were the predisposing factors for the loss of several dromedaries. They identified *Salmonellae* in all of the organs of the dead animals, which presumably served as harbingers of the deadly *C. perfringens* enterotoxemia. Enteropathogenic *E. coli* infections in piglets appear to play a role similar to that of *Salmonellae* as described above. Sinkovics (1972) observed an increased *C. perfringens* activity in the small intestine of piglets when they were infected with enteropathogenic *E. coli*.

Salmonellosis has increased in importance as a zoonosis in the last few years. Preventive measures must also take into account that inadequate treatment can lead to unapparent subclinical cases or carriers that may then persist in the stock. These chronic carriers are not only a threat for the remaining animals, but also present a human health hazard through contact with contaminated animal products. This is especially true in several African countries such as Egypt, Sudan and Somalia where meat from dromedaries is consumed. Food poisoning due to dromedary meat has been reported by Sandiford et al. (1943); Sandiford (1944); Ramadan and Sadek (1971) and El-Nawawi et al. (1982). In the UAE, Wernery and Makarem (1996) identified a large number of identical *Salmonella* serotypes in the stool of people afflicted with salmonellosis and in the fecal samples of dromedaries. In Egypt, Kamel and Lotfi (1963) examined intestinal lymph nodes and fecal samples from 915 slaughtered dromedaries for the presence of *Salmonella*. They isolated *Salmonella* species from 3.1% of the animals examined (*S. typhimurium* (15×), *S. saint-paul* (6×), *S. reading* (3×), *S. dublin* (2×), *S. eastborne* (1×), *S. enteritidis* (1×), *S. bovis-morbificaus* (1×)). The authors believe that their study proved that the dromedary is an important reservoir for *Salmonellae* and could therefore represent a health hazard for man.

Table 20 *Salmonella* serotypes isolated from camel specimens from various countries

Nr.	*Salmonella* Serotypes	Country
1	*S. typhimurium*	Palestine, Egypt, UAE, USA, Nigeria, USA (llama)
2	*S. dublin*	Egypt
3	*S. kentucky*	Palestine, UAE, Nigeria
4	*S. saint-paul*	Egypt, Ethiopia, UAE, Nigeria
5	*S. derby*	USA, UAE
6	*S. cholerae-suis*	Egypt, Somalia
7	*S. limete*	India
8	*S. cerro*	India, UAE
9	*S. anatum*	India, UAE, Egypt
10	*S. typhi and paratyphi*	Egypt, India, Sudan
11	*S. frintrop*	India, UAE
12	*S. muenchen*	India, UAE, Egypt
13	*S. reading*	Egypt, UAE
14	*S. give*	India, Ethiopia
15	*S. eastborne*	Ethiopia, Egypt
16	*S. bovis-morbificans*	Egypt, UAE
17	*S. münster*	Egypt, UAE
18	*S. bredeney*	Somalia
19	*S. chester*	Ethiopia, Egypt
20	*S. glostrup,*	Egypt
to	*S. enteritidis,*	Nigeria, Morocco
37	*S. uganda, S. newport, S. kottbus,*	
	S. brandenburg,	
	S. shubra, S. sandiego,	
	S. heidelberg,	
	S. newlands,	
	S. brazzaville,	
	S. goettingen,	
	S. lokstedt, S. israel,	
	S. newbrunswik,	
	S. santiago,	
	S. thompson,	
	S. tshiongwe	
38	*S. hindmarsh,*	UAE
to	*S. nchanga,*	
69	*S. mbandaka,*	
	S. oranienburg,	
	S. meleagridis,	
	S. havana,	
	S. infantis,	
	S. senftenberg,	
	S. chailey,	
	S. livingstone,	
	S. amsterdam,	
	S. agona,	
	S. tarshyne,	
	S. johannisburg	
	S. tennesse	Morocco
	S. talahassie	Morocco
	S. tananarive	Morocco
	S. altona	UAE (n.p.)
	S. newport	UAE (n.p.)
69	*S. blockley*	UAE (n.p.)

n.p. = not published

Pathology Special toxins of *Salmonellae* are responsible for the systemic and enteric forms of salmonellosis. These virulence factors include:
- lipopolysaccharides (LPS),
- endotoxins,
- enterotoxins,
- cytotoxin,
- plasmids.

The usual route of infection is oral. The bacteria penetrate into the lamina propria and production of cytotoxins and enterotoxins contribute to gut damage causing enteritis. Acute enteritis is the common form in camel calves and in adult camelids when predisposing factors like clostridiosis, coccidiosis or candidiasis exist. The feces have a putrid odor and contain mucus and sometimes blood. A severe hemorrhagic enteritis may develop (Fig. 44).

Chronic enteritis is a common form in adult camelids. There is persistent diarrhea, with intermittent fever, emaciation and poor response to treatment. From the lamina propria of the intestines, *Salmonellae* may be transported into the vascular system, causing septicemia. During septicemia, *Salmonellae* may localize in the brain, meninges, pregnant uterus and distal aspects of limbs, ears and tails. The organisms also frequently localize in the gallbladder and mesenteric lymph nodes, and survivors intermittently shed the bacteria in the feces. *Salmonella* septicemia is a usual syndrome in newborns with outbreaks occurring for up to 6 months. This illness is acute with fever and depression. Death occurs within 48 hours. At necropsy there are petechiae in all organs and often pneumonia. A factor predisposing to *Salmonella* septicemia seems to be mineral deficiency. Anderson et al. (1995) reported septicemic salmonellosis in a llama caused by *S. cholerasuis*. The disease was characterized by fibrinopurulent pericarditis, pleuritis and peritonitis. The llama had been in contact with pigs. The second case was a premature llama infected with *S. typhimurium*. The animal revealed hydropericardium, hydrothorax, pulmonary congestion and hemorrhages of the mucous membranes.

Figure 44 Hemorrhagic enteritis caused by *S. typhimurium* in a young dromedary

Diagnosis *Salmonellae* have simple nutrient requirements and growth *in vitro* is therefore possible on many different media. However, selective procedures are used for the isolation of *Salmonella* from specimens that contain a mixed flora. Colonies characteristic for *Salmonellae* can be easily serotyped. Serotyping is based on the O (somatic) and H (flagellar) antigens.

Prevention and Control The reservoir for *Salmonellae* is the intestinal tract of warm-blooded and cold-blooded animals and the majority of infected animals become subclinical excretors. Transmission of *Salmonella* is usually by the fecal-oral route, but infection via the mucous membranes of the conjunctivae or upper respiratory tract as well as through the skin occurs. It is therefore important that every ef-

fort be undertaken to prevent introduction of carrier animals into a herd. It should also be ensured that feed supplies are free of *Salmonellae*. Certain procedures should be followed in a *Salmonella* outbreak on a camel farm:

1. Carrier animals should be identified, isolated and treated vigorously. Treated camels must be re-examined several times before there can be confidence that they are not carriers.
2. Feed and water supplies must be protected from fecal contamination (beware of pigeons and rodents).
3. Movement of animals around the farm should be restricted.
4. All persons should be aware of the health hazards of working with infected camels.
5. The use of vaccines should be considered.

Supportive therapy and good nursing are important especially in camel calves with enteritis. This includes oral or parenteral rehydration, correction of electrolyte imbalances and stabilization of the acid-base equilibrium. It is also very important to avoid factors that lead to mineral deficiencies in the dams and their offspring. Nonsteroidal anti-inflammatory drugs, such as ketoprofen, may be of benefit since it is known that many camel calves subsequently also suffer from endotoxic shock. In these cases, the treatment protocol described under the heading Endotoxicosis (1.1.4) should be followed.

Antimicrobial drug treatment of salmonellosis is controversial because it may create a carrier state in camelids and antibiotic resistant strains of *Salmonellae*. The drug should be administered parenterally, since oral treatment of an animal species that ruminates can create severe disturbances of the gastrointestinal flora. Antimicrobial drugs generally recommended for parenteral use in salmonellosis are ampicillin, amoxycillin and trimethoprim-sulfonamide combinations. Baytril® is also a very effective drug, but the drug of choice should be based upon culture and sensitivity. Treatment must start immediately and should be continued daily for up to 6 days.

Some commercial live, avirulent vaccines are now available. However, autogenous *Salmonella* vaccines (see chapter 4) are of greater value because they include the *Salmonella* strains involved in the outbreak. These vaccines should be used in problem herds and should be administered twice before parturition in order to provide protection against salmonellosis for the newborns. The colostral immunity will last approximately 6 to 8 weeks. The autogenous vaccines are killed vaccines, which should be administered either intramuscularly or subcutaneously at a dosage of 5 to 8 mL depending on the weight of the animal. It is of no use to vaccinate newborn camelids because their immune system is still immature. Camelid calves in problem herds whose dams have not been vaccinated should receive up to 50 mL of the herd-specific vaccine orally for 14 days.

1.2.2 Colibacillosis

While *Escherichia coli* is the cause of various diseases of great economic magnitude, especially in young animals, it also constitutes a large part of the normal commensally aerobic intestinal flora. Willinger (1981), Quinn et al. (1994), Wernery and Kaaden (1995) and Fowler (1998) ascribed the following diseases to *E. coli*:

- colibacillosis (white scours) in bovine calves less than 1 week old;
- colisepticemia in bovine calves less than 1 week old;
- joint ill in bovine calves surviving a colisepticemia;
- neonatal diarrhea (colibacillosis), colisepticemia in piglets less than 1 week old;
- colibacillosis in piglets about 2 weeks after weaning;

- *E. coli* enterotoxemia in weaned pigs;
- mastitis in cattle and other animals (ewes);
- MMA-complex (metritis-mastitis-agalactia syndrome) in sows;
- colibacillosis and colisepticemia in neonatal lambs, "watery mouth" in neonatal lambs;
- colibacillosis and colisepticemia in camelids 2 to 4 weeks old;
- dysentery in rabbits;
- diarrhea and pyometra in dogs;
- septicemia, coli-granulomatosis in fowl;
- inflammation of the urogenital and respiratory tracts;
- wound infections and healing impairment.

Colibacillosis and colisepticemia have been reported in OWC and NWC (Wernery and Kaaden, 1995; Fowler, 1998).

Etiology *Escherichia coli* is a straight Gram-negative motile rod that belongs like *Salmonella* to the *Enterobacteriaceae* family. Enteric infection caused by *E. coli* can be due to at least 5 different varieties of bacteria operating through different mechanisms:

1. Enterotoxigenic *E. coli* (ETEC) with their fimbrial adhesions K88, K99 and others – these strains cause the majority of neonatal colibacillosis.
2. Enteropathogenic *E. coli* (EPEC) do not produce enterotoxins, but can cause diarrhea.
3. Enteroinvasive *E. coli* (EIEC) invade enterocytes and produce virulence factors – they are responsible for colisepticemia and release endotoxins when dying.
4. Attaching and effacing *E. coli* (AEEC) produce verotoxins and destroy the microvilli – they produce enteric diseases.
5. Enterohemorrhagic *E. coli* (EHEC) cause hemorrhagic colitis and have been associated with the hemolytic-uremic syndrome in children.

All five pathogens share the general properties of demonstrating specific interactions with the intestinal mucosa, of elaborating various toxins, and of possessing plasmid-encoded virulence factors.

E. coli possess different antigens (K = capsular, O = cell wall or somatic, H = flagellar, F = fimbrial), which can be used to serotype strains. A plasmid, of which there may be more than one in an *E. coli* cell, may code for several virulence factors including antibiotic resistance.

Epidemiology and Clinical Signs *E. coli* is a natural inhabitant of some parts of the intestines of all mammals and is excreted in feces. The presence of *E. coli* in water and food samples is taken as evidence of fecal contamination.

E. coli infections in young animals are mostly due to errors in husbandry and, in calves, are frequently associated with *Salmonella, Clostridia, Pasteurella,* corona- and rotaviruses as well as *Cryptosporidia* infections. Different serotypes can cause different clinical signs. Enterotoxigenic *E. coli* strains produce enterotoxins, which cause enteritis with dehydration. Some strains, especially pathogenic ones, can cause hemolysis (Bisping and Amtsberg, 1988). Pathogenic *E. coli* strains possess a variety of virulence factors upon which their pathogenicity depends. These factors include endotoxin, protein adhesions, α- and β-hemolysins, capsular polysaccharides, heat-labile and heat-stable enterotoxins and verotoxin. A considerable economic loss to the camel industry results from colibacillosis or colisepticemia in young camelids. These losses can reach 40%.

E. coli infections in dromedary calves have been described by various authors. Schwartz and Dioli (1992) reported a morbidity of 30% in neonatal dromedary calves in East Africa. Without immediate veterinary intervention, mortality can reach 100%. The authors believe that the unsanitary conditions in the breeding herds along with

contaminated water and inadequate feeding of colostrum cause the disease. The calves suffer from dysentery, abdominal pain, anorexia and dehydration. Death occurs within a few days. Chauhan et al. (1986) reported colibacillosis in two dromedary calves presented with diarrhea, fever, general malaise and anorexia. The authors isolated *E. coli* serotype 083 from the fecal samples of the afflicted animals. Rombol (1942) also described *E. coli* infections in newborn dromedaries with severe diarrhea. Mohamed et al. (1998) who examined 42 one to 3-month-old dromedary calves in Sudan found 40% (12/42) infected with pathogenic *E. coli*, of which 5 were identified as EIEC, 2 as EPEC and 1 as VT2 pathotypes. Salih et al. (1997 and 1998 a) isolated 69 (66%) *E. coli* (K88, F41) out of 106 diarrheic camel calves with different adhesion factors.

E. coli infections in dromedary calves occur regularly every year in certain breeding herds in the UAE. Clinical signs have not as yet been seen in neonates, only in animals between 2 and 4 weeks old. Severe losses have occurred in certain breeding herds. As in cases of clostridial enterotoxemia in young dromedaries, the *E. coli* dysentery appears to be associated with the initial consumption of solid food and sand. The affected animals develop a yellowish watery diarrhea. The hind legs and tail are covered with dried feces (Fig. 45) and the eyes are sunken deep in their orbital cavities due to the resulting dehydration. Hemolytic *E. coli* are isolated from the gastrointestinal tract and most of the organs.

A rare case of *E. coli* enterotoxemia due to a hemolytic *E. coli*, serotype 0139 reportedly occurred in dromedaries in Bahrain (Ibrahim et al., 1998). Sporadic cases were observed in adult breeding camels at different camel centers. The incidence of the disease was more than 50% and the mortality rate approached 90%. The camels showed severe swelling of the abdomen and a distention of the abdominal cavity, which was filled with 100 to 150 liters of fluid. There was also edema of eyelids, throat, ears and forehead. Some dromedaries developed CNS signs. The hemolytic *E. coli* was isolated from intestinal contents and from the abdominal fluid.

Strauss (1991) and Fowler (1998) have remarked that *E. coli* infections in young NWC are an important ailment. Colibacillosis develops mainly in undernourished crias. Neonatal colisepticemia is a serious disease in NWC in the USA followed by

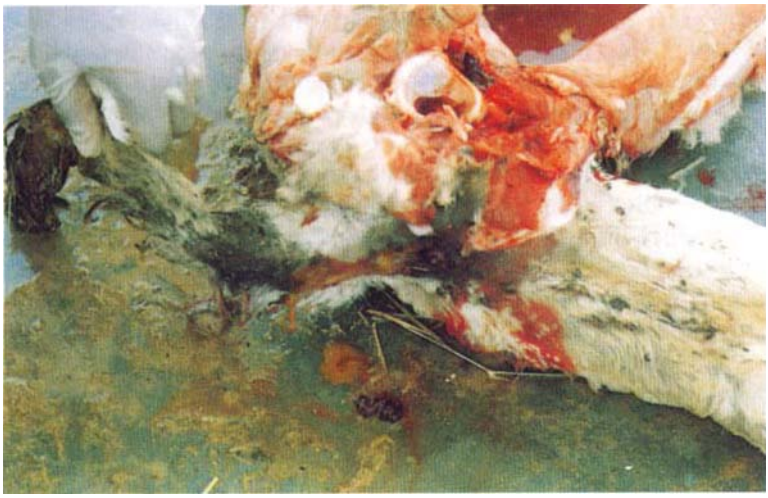

Figure 45 Colibacillosis in a 3-week-old dromedary calf with yellowish diarrhea

metritis, mastitis and abscess formation. The affected animals suffer from profuse diarrhea, weight loss, abdominal distention and debility. Haenichen and Wiesner (1995) also described colisepticemia in alpacas with severe meningitis.

Pathology Colibacillosis and colisepticemia in camelids produce anorexia, weakness, fever and yellowish diarrhea (Chauhan et al., 1987). The disease occurs in calves up to 6 months of age. Colisepticemia often develops in animals suffering from enteric colibacillosis, but may also occur without any evidence of enteric involvement. In both enteric colibacillosis and colisepticemia lesions are non-specific. Camelids that are affected by enteric colibacillosis are dehydrated and their hindquarters and tails are soiled with feces caused by diarrhea. At necropsy there is congestion of the small intestine with catarrhal enteritis, the gut contents are gray to yellowish and the mesenteric lymph nodes are edematous. In colisepticemia, a generalized congestion, petechiae in the serous membranes and edema of the meninges are observed.

Young dromedaries develop a fever of between 40°C and 41°C. Death follows within 2 to 3 days. On autopsy, there is an extreme pallor of the entire cadaver (Fig. 46), inflammation of the intestinal mucosa and, regularly varying amounts of sand in the compartments, especially in compartment 1 (Fig. 47). The contents of the gastrointestinal tract are gray colored with a pungent foul odor. In severe cases of colisepticemia, a fibrin exudate covers the abdominal organs (Fig. 48).

Diagnosis The clinical characteristics of colibacillosis are similar to those manifested in infections caused by rotavirus, coronavirus, *Salmonellae* and *Coccidia*. The diagnosis therefore depends on microbiological examinations. Specimens for bacteriology should consist of sections of intestinal tract, mesenteric lymph nodes and pieces of different organs. All tissue samples should be collected aseptically soon after death. *E. coli* has no special nutritional requirements and grows on many different agars. However, selective media are generally employed to differentiate them from other *Enterobacteriaceae*. Individual serotypes are found more frequently in certain species and in certain disease conditions. In camelids, serotyping of cultured *E. coli* strains has just begun (Jin, 1985). Method-

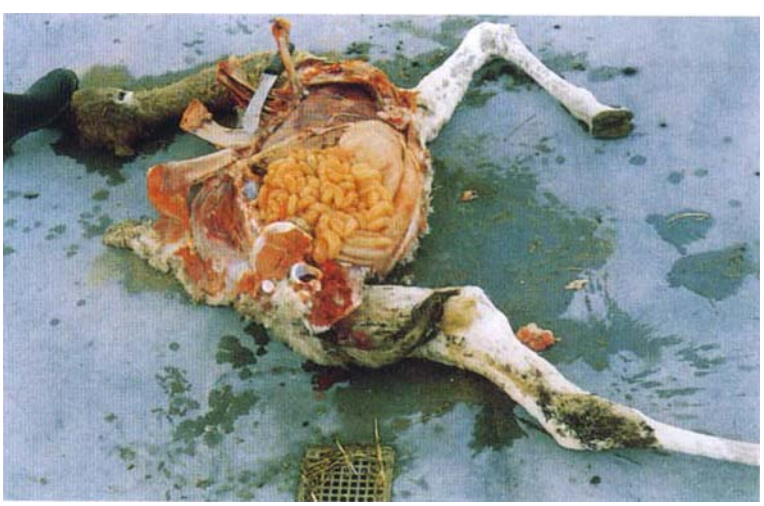

Figure 46 Extreme pallor in a young dromedary with colisepticemia

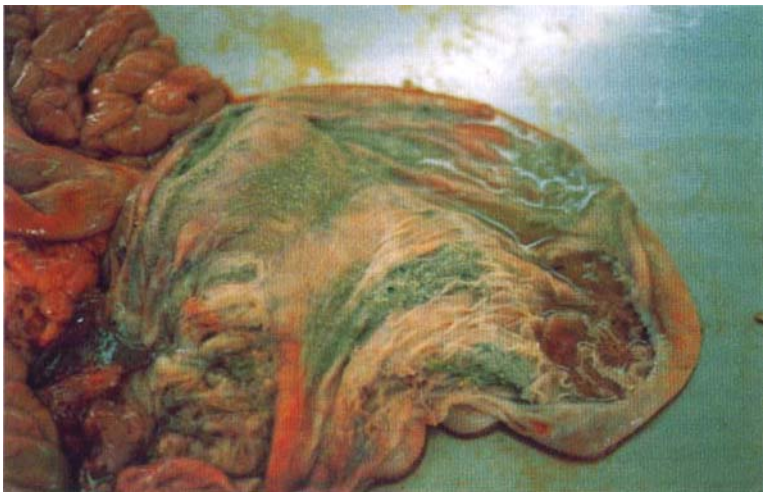

Figure 47 Sand in compartment 1 in a young dromedary with *E. coli* dysentery

ological procedures about the agglutination for the identification of various *E. coli* antigens are provided by the commercial companies which produce the antisera.

Treatment and Control Camelids with diarrhea caused by *E. coli* should follow the same regimen of treatment as mentioned under the chapter salmonellosis. Oral or parenteral electrolytes must be administered to restore fluid balance because death usually results from dehydration. It is also recommended to restrict milk intake. However, initial ingestion of colostrum should occur within the first hours of life to maximize the absorption of immunoglobulins. Colostrum banks should be established for emergency cases. Before antibiotic therapy, resistance testing should be performed on the *E. coli* strain causing the outbreak, but it should be kept in mind that many *E. coli* isolates possess multiple resistance to antibiotics. Colisepticemia must be treated with injectible antimicrobials like Baytril®,

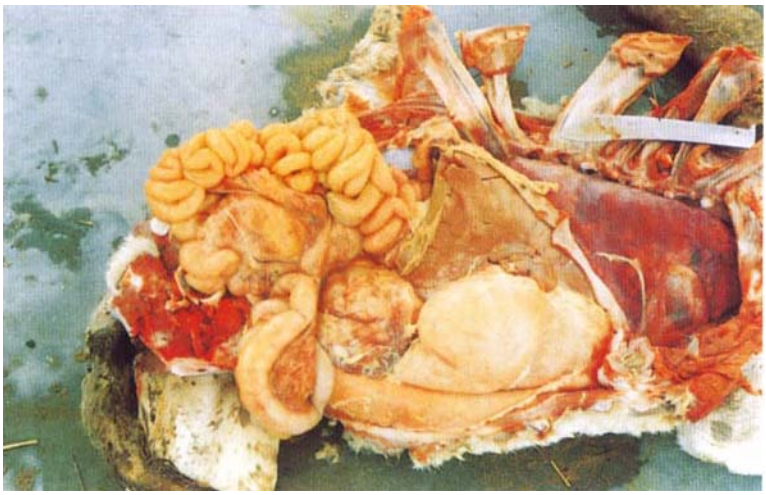

Figure 48 Colisepticemia in a young dromedary with fibrin exudate covering the internal organs

trimethoprim/sulfonamide, kanamycin or colistin (Manefield and Tinson, 1996).

In order to reduce losses among young dromedaries, maternal vaccination with herd-specific *E. coli* vaccines should be administered annually or oral vaccinations in young camelids should be tried.

Due to intensive animal keeping, for example in animal parks and zoos, considerable losses can arise due to *E. coli*. For this reason, Strauss (1991) applied a protective maternal vaccination (Colivac®) twice to all pregnant camels about 8 and then 4 weeks prior to the calculated date of parturition. After that, losses in young animals were greatly reduced.

1.2.3 Paratuberculosis (Johne's Disease)

This disease is characterized by persistent and progressive diarrhea, weight loss, debilitation and eventually death. The disease produces a chronic, contagious enteritis and affects cattle, sheep, goats, farmed deer and other domestic and wild ruminants. It has also been reported to occur in OWC (Wernery and Kaaden, 1995) and in NWC (Appleby and Head, 1954; Schwarte, 1956; Belknap et al., 1994; Ridge et al., 1995).

Paratuberculosis occurs worldwide. In tropical areas with intensive dairy farming, paratuberculosis presents a serious economic problem. *Mycobacterium avium* spp. *paratuberculosis* is excreted in the feces of infected animals and so can then be ingested with contaminated food or water. The bacteria spread to the intestinal mucosa or mesenteric lymph nodes where they can cause chronic inflammation. *M. avium* spp. *paratuberculosis* is also able to cross the placenta to the fetus.

Etiology *Mycobacterium avium* spp. *paratuberculosis* is a non-motile, non-sporing, aerobic and oxidative bacterium which does not take up dyes of the Gram stain because the cell wall is rich in lipids and mycolic acid. *M. avium* spp. *paratuberculosis* is acid-fast and the best stain is Ziehl-Neelsen. The disease can be diagnosed by the demonstration of the bacteria and by serological and allergic tests.

Epidemiology and Clinical Signs *M. avium* spp. *paratuberculosis* is shed in feces and the organisms are found within macrophages of the intestinal mucosa and adjacent lymph nodes. A cell-mediated immune response appears to be involved in the pathogenesis of this disease. Not all infected animals become clinical cases, but they remain excretors of *M. avium* spp. *paratuberculosis*. Following oral infection, *M. avium* spp. *paratuberculosis* enters the lymphatics through the tonsils and the intestinal mucosa. Peyer's patches take up the microorganisms from the intestinal lumen and transport them through the intestinal mucosa. The incubation period is generally 18 to 24 months.

Paratuberculosis is one of the most important and widespread diseases of the Bactrian camel in the former Soviet bloc, as reported by Ivanov and Skalinskii (1957), Ovdienko et al. (1985), Fassi-Fehri (1987) and Buchnev et al. (1987). The disease has also been diagnosed in Bactrian camels from Mongolia suffering from severe diarrhea (Guake et al., 1964) and has been known in Turkmenistan since 1949 (Strogov, 1957). Strogov (1957) and Buchnev et al. (1987) reported that paratuberculosis is more prevalent in young Bactrians between weaning and 4 years of age. The authors believe that older camels may recover from the disease. Recovery of adult infected camels takes place slowly over a period of 6 months. The annual incidence of infection in Bactrian camels in Turkmenistan between 1946 and 1952 was between 0.3 and 1.5% (Strogov, 1957).

Paratuberculosis is also seen in dromedaries, but is less prevalent than in Bactri-

an camels due to the conditions in which the dromedaries are kept. Of 105 dromedaries in India, Chauhan et al. (1986) found only four that had paratuberculosis (3.8%). These dromedaries suffered from intractable diarrhea and acid-fast bacilli were identified in one animal upon biopsy of the rectal epithelium. All four dromedaries had a positive skin test following the intradermal application of "Johnin". Paratuberculosis was also found in one female dromedary in an American zoo (Amand, 1974). The dromedary was weak and emaciated and passed blood streaked, mucoid, loose stools. The camel showed a marked hypoproteinemia (total protein: 2.7g/dL, normal is 5.7–7.5 g/dL). Clumps of acid-fast rods were detected in fecal samples. However, intradermal tests with Johnin and tuberculin were negative. Gameel et al. (1994) stated that dromedaries can contract paratuberculosis from cattle.

In NWC, the clinical signs of paratuberculosis vary, with some animals developing severe diarrhea, weakness and emaciation. Death follows after 6 to 10 days in these cases. Some lamoids develop weakness and weight loss as well as terminal diarrhea over a 3-month period and other llamas lose weight and become debilitated without developing any diarrhea. All clinically affected lamoids develop hypoproteinemia, which may be used as a diagnostic tool as in sheep (Scott et al., 1995).

Pathology Pathological lesions of paratuberculosis were described in Bactrians by Strogov (1957), Ivanov and Skalinskii (1975) and Guake et al. (1964), in dromedaries by Amand (1974) and Radwan et al. (1991) and in NWC by Belknap et al. (1994) and Ridge et al. (1995).

Russian authors are of the opinion that paratuberculosis causes more pathological changes in Bactrian camels than in cattle. Lesions have been observed in the ileum, cecum and colon, although additionally inflammation of the liver, spleen and lymph nodes has also been reported. Infected animals die within 4 to 6 weeks after the initial occurrence of diarrhea. At necropsy, Amand (1974) described severe intestinal thickening and enlargement of the regional mesocolic lymph nodes. Histologically the lesions are characterized by a marked accumulation of macrophages in the mucosal layer that were laden with acid-fast bacilli (Fig. 49).

Radwan et al. (1991) were the first to identify the disease in Saudi Arabia. Six-

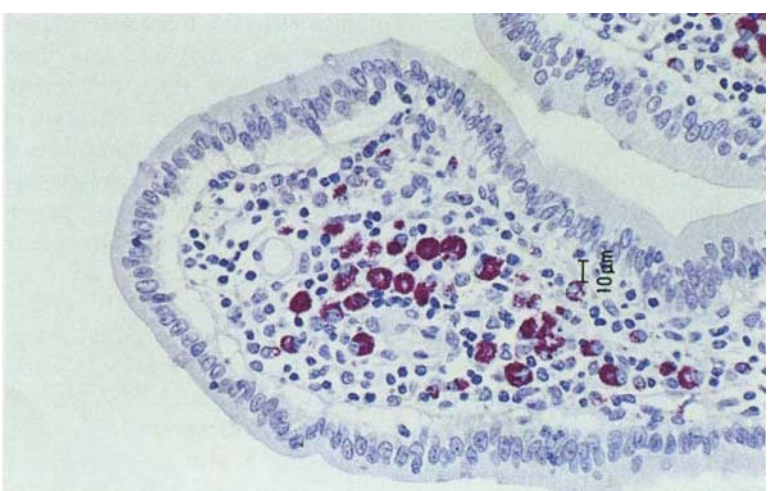

Figure 49 Acid-fast rods in macrophages of intestinal mucosa of a dromedary suffering from paratuberculosis

ty cases of paratuberculosis were found among three dromedary herds consisting of 3000 animals. The dromedaries were between 2 and 4 years old and suffered from severe weight loss and chronic intermittent diarrhea. The animals did not develop fever. In spite of antibiotic treatment, the animals died 1 to 4 months after the development of the initial clinical signs. A massive thickening of the ileal, cecal and colonic walls was seen upon autopsy. The intestinal lymph nodes were greatly enlarged. Acid-fast bacilli were found in the feces, intestines and the lymph nodes.

The lesions of paratuberculosis in lamoids are similar to those seen in OWC. The animals are emaciated, the Peyer's patches are prominent in the intestine (Fig. 50) and the mesenteric lymph nodes are edematous and enlarged.

Histological sections of the lymph nodes contain numerous colonies of acid-fast staining bacteria. In some animals the jejunum, the ileocecal junction and the proximal large intestines are thickened and there are sometimes granulomatous lesions in liver, lung and lymphatics of the peritoneal serosa, from which *M. avium* spp. *paratuberculosis* is isolated.

Diagnosis Paratuberculosis can be diagnosed by culture and allergic and serological tests. Bacteriological culturing of feces is the most sensitive and specific test for *M. avium* spp. *paratuberculosis*, but it can require up to 16 weeks to obtain the results. Biopsy specimens of intestinal mucosa and fecal smears stained by the ZN-stain usually yield characteristic clumps of *M. avium* spp. *paratuberculosis* organisms. However, examination of feces will detect only about 25% of subclinical excretors.

Intradermal testing with avian tuberculin or "Johnin" produced from *M. avium* spp. *paratuberculosis* yields unsatisfactory results (Higgins, 1986). In Russia, the intradermal injection of avian tuberculin produced a reaction in 40% of the Bactrian camels tested. However, no acid-fast bacilli were isolated from the fecal samples taken from 600 reactive animals. In another investigation, no changes typical of paratuberculosis were identified at post mortem despite a strong test reaction in 7 Bactrian camels (Khon, 1983a).

Dependable serological results in the detection of paratuberculosis have been obtained with the complement fixation test (Khon, 1983a and b; Seifert, 1992). Burgemeister et al. (1975) were able to detect an-

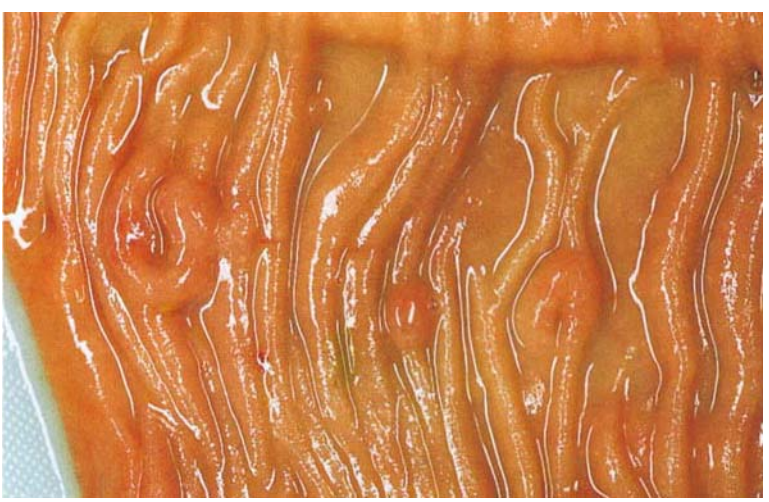

Figure 50 Prominent Peyer's patch of a dromedary with paratuberculosis

tibodies to *M. avium* spp. *paratuberculosis* in the sera of 11 of 52 (21.2%) dromedaries in Tunisia. Feldmann et al. (1981) were also able to diagnose the disease serologically in Kenya. However, serological tests for paratuberculosis on individual animals are often inconclusive, but they are of value when entire herds are screened. Serological tests include CFT, AGID and ELISA. Nothing is known about their specificity and sensitivity in camelids.

Sporadic cases of paratuberculosis have also been seen among racing dromedaries in the UAE. Five cases were diagnosed between 1987 and 1993. The animals suffered from intractable diarrhea. Acid-fast bacilli were found in all five fecal samples (Fig. 51). Complement-fixing antibodies (titers between 1:64 and 1:256) were found in the infected animals, confirming the diagnosis. All of the animals died in spite of antibiotic treatment within one year.

M. avium spp. *paratuberculosis* can be differentiated from other mycobacteria by its mycobactin-dependence in culture. Benzalkonium chloride (Zephiran) is used to decontaminate specimens and Herrold's egg yolk medium with mycobactin is often used as culture medium. The slants of Herrold's medium are incubated at 37°C and examined for growth, once a week for up to 16 weeks.

Treatment and Control No satisfactory treatment of paratuberculosis is known. Control requires good sanitation and management. Radwan et al. (1991) suggested methods of eradicating the disease in dromedaries. They include the following recommendations:

1. Clinically suspected camels should be isolated until the disease is confirmed. All infected camels should be slaughtered and carcasses properly disposed.
2. Where possible, camelid calves should be removed from their dams at birth and reared in a paratuberculosis-free environment.
3. Appropriate sanitary measures should be applied to prevent contamination of food, water and soil; and ponds and ditches should be fenced off.
4. Newly purchased camels should be examined for paratuberculosis.
5. Vaccination should be considered.

In many countries, vaccines are used in cattle, sheep and goats. The available vaccines are prepared from either a live or heat-killed strain of *M. avium* spp. *paratu-*

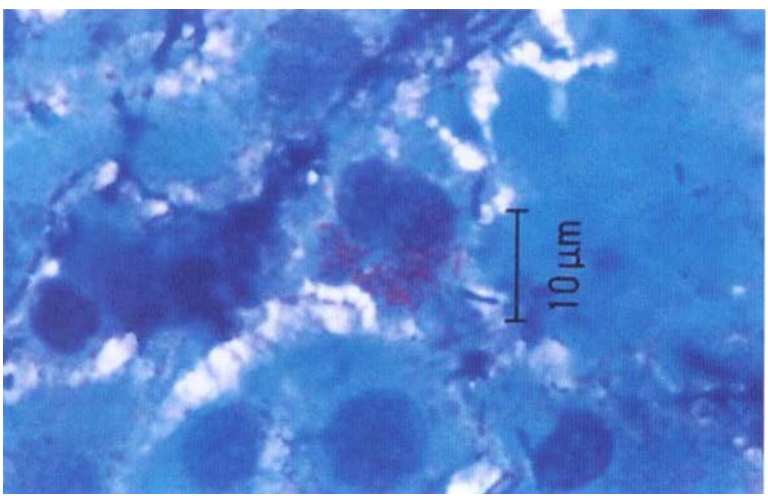

Figure 51 Acid-fast bacilli (Ziehl-Neelsen stain) in a stool sample of a dromedary with paratuberculosis

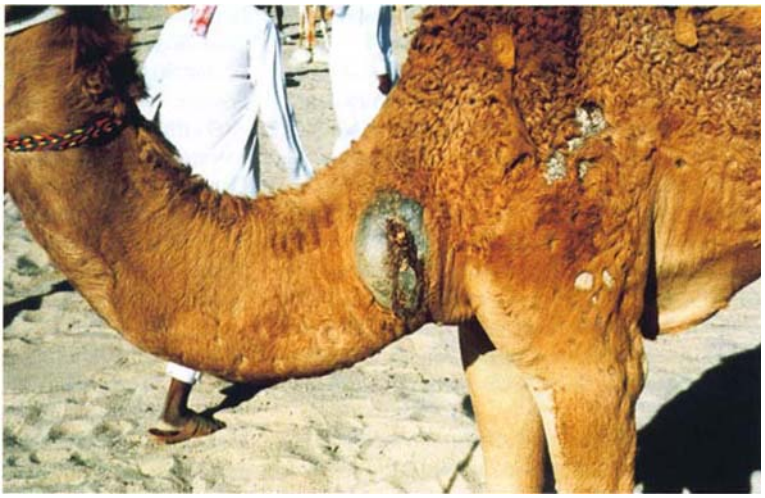

Figure 52 Severe abscess formation 4 months after vaccination against paratuberculosis in a dromedary (ringworm lesions are also seen, courtesy of Dr. Cheyne, Qatar)

berculosis and both vaccines seem to possess the same efficacy. Vaccination can be effective in reducing disease incidence, but does not eliminate infection. In general young animals less than one month of age are vaccinated. Camels that have been vaccinated may develop severe granulomas of several centimeters in diameter at the inoculation site (Fig. 52) causing camel owners to dislike this vaccine. However, camels with these local reactions have shown a detectable serological response, whereas camels without skin reactions were negative in the CFT (Cheyne, 1995). Accidental self-inoculation can result in severe reaction with synovitis and tendonitis.

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1.3 Respiratory System

In general, camels do not suffer from respiratory disease. However, when it occurs, it is usually initiated by predisposing factors such as a sudden change in weather, poor hygiene and inadequate management (Shah and Khan, 1935–1936; Mustafa, 1987) as well as underlying debilitating conditions (Wernery and Kaaden, 1995; Manefield and Tinson, 1996). Pneumonia has been observed in association with endotoxicosis, colibacillosis, enterotoxemia, leukemia, chronic skin infections and vitamin E/selenium deficiencies. These facts must be kept in mind when pneumonia is diagnosed in camelids. A number of bacterial species have been found in camels with respiratory disease. However, it is not known if these agents are responsible for the disease, except in tuberculosis.

1.3.1 Tuberculosis

Tuberculosis is a chronic contagious disease caused by mycobacteria, which affects many vertebrate animals and particularly manifests itself in lungs and lymph nodes. The lesions are granulomas known as tubercles. The lesions differ greatly according to the animal species infected and the species of mycobacteria involved. Tuberculosis in humans still remains one of the major global reportable diseases; it has caused more deaths in humans than all the wars together. The widespread outbreaks of *M. tuberculosis* are of considerable concern to public health officials, conservation agencies and veterinarians responsible for the health status of animals in zoos, animal parks and private herds. Many strains have become resistant to medication. The two most important members of the genus *Mycobacterium* are *M. tuberculosis* and *M. bovis*. Both have been isolated from OWC and NWC.

Etiology The genus *Mycobacterium (M.)* of the family *Mycobacteriaceae* are acid-fast rods of various lengths, non-motile and non-sporulating. The genus *Mycobacterium* contains multiple species (about 50) with different pathogenicity, the atypical mycobacteria being grouped by Runyon. The atypical mycobacteria are widespread in pastures, soil and water. Some of them may infect animals. The most important mycobacterial species causing disease in livestock are:

- *M. bovis* – occurs in many animal species including man;
- several serovars of the *M. avium* complex occur in poultry, wild birds, pigs, horses;
- *M. avium* spp. *paratuberculosis* (see chapter 1.2.3);
- *M. farcinogenes* (*Nocardia farcinica*? See chapter Dermatophilosis, 1.5.3) which causes bovine farcy.

M. tuberculosis affects humans, non-human primates, dogs, canaries, and psittacines and has also been isolated from camelids (Elmossalami et al., 1971; Osman, 1974).

Epidemiology and Clinical Signs A severe increase in tuberculosis (TB) has recently been observed in cattle in Britain. Badgers are considered to be a wildlife reservoir for TB and this increase in TB has been observed in cattle-keeping areas where badgers are common. *M. bovis* has recently been isolated in 2 llamas in south Wales and the strain is identical with isolates causing TB in cattle, deer and badgers.

There are different modes of spread of tuberculosis between camelid herds. One is the introduction of an infected animal into a non-infected herd (Bush et al., 1990). Gatt Rutter and Mack (1963) reported that in Egypt, tuberculosis did not occur in nomadic camels but in those belonging to

Table 21 Summary of literature regarding tuberculosis in OWC *(Mycobacterium tuberculosis = Humanus, Mycobacterium bovis = Bovinus)*

Country	Year	Author	Type
Egypt	1888	Littlewood	–
India	1905	Lingard	–
India	1908	Leese	–
India	1910	Leese	–
Sudan	1910	Archibald	–
Egypt	1912	Mason	–
Egypt	1917a, b	Mason	*Bovinus*
	1917	Cross	
Egypt	1918	Mason	
Germany	1928	Andree	*Bovinus*
Somalia	1942	Pellegrini	*Bovinus*
Egypt	1953	El-Afifi et al.	*Bovinus, Humanus*
Somalia	1957	Casati	*Bovinus*
Circus camel	1957	Panebianco	*Bovinus*
Russia	1962	Abramov	
Somalia	1962	Angrisani	*Humanus*
Circus camel	1962	Dekker and Van Der Schaaf	*Bovinus*
Russia	1963	Abramov	
Russia	1964a	Abramov	*Bovinus, Humanus*
Russia	1964b	Abramov	*Bovinus, Humanus*
India	1969	Damodaran and Ramakrishnan	
Egypt	1970	Abd El-Aziz	*Bovinus, Humanus*
Egypt	1971	Elmossalami et al.	*Bovinus, Humanus Atypical*
Russia	1971	Fedchenko	
Russia	1972	Akhundov et al.	
Russia	1972	Fedchenko	
Egypt	1974	Osman	*Bovinus, Humanus*
Russia	1975a	Donchenko et al.	*Bovinus*
Russia	1975b	Donchenko et al.	*Bovinus*
Russia	1975c	Donchenko et al.	
Russia	1976	Kibasov and Donchenko	
Russia	1978	Donchenko and Donchenko	
Ethiopia	1979	Richard	
Somalia	1982	Arush	
USA	1983	Kennedy and Bush	
Mauritania	1985	Chamoiseau et al.	
USA	1986	Bush et al.	
Somalia	1986	Hayles	
India	1986	Chauhan et al.	
Mauritania	1989	Diatchenko	
Pakistan	1993	Rana et al.	
USA	1977 (llamas)	Thoen et al.	*Bovinus*
UAE	1995	Wernery and Kaaden	
USA	1990 (Bactrian)	Bush et al.	*Bovinus*

farmers who kept them in close contact with cattle. The mode of transmission of tuberculosis is unknown in camelids, but it is presumed similar to that in cattle. In cattle it is mainly horizontal. It is believed that camelids suffering from pulmonary tuberculosis infect healthy animals via aerosols. The alimentary, congenital, venereal and cutaneous routes that may occur in cattle have not been described in camelids. Kogramanov et al. (1971) found that the *Ixodes* tick *Hyalomma asiaticum* can transmit *M. tuberculosis* to Bactrian camels.

In tropical developing countries, where tuberculosis has received little attention, substantial economic losses can occur, especially in cattle. Tuberculosis, as a zoonosis, also plays an important role among nomadic people where milk and milk products are consumed raw (Seifert, 1992). This is also true for camel milk. Donchenko et al. (1975b) isolated *M. bovis* strains from 46 pooled milk samples from 712 lactating camel cows in Russia. Tuberculin tests were performed in these herds whereby 9.1% were reactive. Other than unheated camel milk, circus and zoo camels with active disease also present a danger to man (Panebianco, 1957; Dekker and van der Schaaf, 1962).

In tropical animal husbandry there are two different routes of infection with tuberculosis:

1. Aerogenic transmission by inhalation of the organisms in contaminated droplets from infected animals, either directly or on dust particles. The primary lesion is in the lung.
2. Alimentary transmission by ingestion of food contaminated with infected feces, urine or milk. The primary lesion is in the intestinal lymph nodes.

Tuberculosis is rare among camels kept under nomadic conditions. The disease occurs more frequently when camels are kept in close quarters with other camels or in close contact with cattle, for example in Russia and Egypt (Mason, 1917 a and b and 1918; Elmossalami et al., 1971; Donchenko et al., 1975a and c). Most of the publications regarding tuberculosis originate from these countries (Table 21).

Tuberculosis is a disease that had already been diagnosed around the turn of the century in dromedaries in Egypt (Littlewood, 1888) and in India (Lingard, 1905; Leese, 1908). As can be seen in Table 21, *M. tuberculosis* (typus *Humanus*), *M. bovis* (typus *Bovinus*) and atypical mycobacteria (Table 22) have been isolated from dromedaries (Elmossalami et al., 1971; Osman, 1974).

Table 22 Atypical mycobacteria isolated from dromedaries in Egypt

M. kansasii	33.3%
M. aquae	16.7%
M. aquae var. ureolyticum	16.7%
M. fortuitum	16.7%
M. smegmatis	16.7%

Osman (1974) examined 120 lymph nodes that had been collected from slaughtered camels in 3 abattoirs in Egypt showing macroscopic lesions of tuberculosis. In 91 camel lymph nodes tubercle bacilli were found, of which 85 (93.4%) belonged to typus *Bovinus* and 6.6% to typus *Humanus*.

Natural and experimental infections of tuberculosis have also been reported in NWC (Moro Sommo, 1957; Castagnino Rosso et al., 1974; Cambre et al., 1981). The 4 major mycobacteria, *M. bovis, M. tuberculosis, M. avium* and *M. avium* spp. *paratuberculosis* have been isolated from NWC as well as some atypical mycobacteria *(M. kansasii, M. microti)*. There are only a few reports on tuberculosis in NWC. It is believed that llamas are not particularly susceptible to tuberculosis. There were several occasions in North America where cervids and llamas were kept together. Although many of the cervids developed tuberculosis, only two llamas in two herds

contracted the disease developing diffuse granulomas. *M. bovis* was cultured from eight llamas during a 5-year period at the Veterinary Service Laboratory in America (Thoen et al., 1977).

Llamas are being kept in increasing numbers in Europe as pets, show animals, pack animals for trekking, and for guarding sheep. Tuberculosis infections have been reported in llamas in South America and most infections occur when camelids live in close contact with other infected livestock or infected humans. *Mycobacterium tuberculosis* and *M. bovis* infections were diagnosed in guanacos in zoos and private herds in Germany (Haenichen and Wiesner, 1995). Bovine tuberculosis was described by Barlow et al. (1999) in a small llama herd near the border of England and Wales. A female llama that was in poor condition was necropsied and numerous caseous lesions were observed from which *M. bovis* was isolated. These included pleura, lungs and pericardial sac. The bronchomediastinal lymph nodes were enlarged and also showed caseous foci (Fig. 53).

The authors showed that the *M. bovis* isolate from the llama was the same type as that of isolates from cattle and badgers of this area.

Diagnosis The diagnosis of camelid tuberculosis in living animals faces many difficulties. None of the tests available can diagnose tuberculosis with certainty. Intradermal tuberculin testing, which is the classical diagnostic test, often gives non-specific reactions in camelids. Several papers report non-specific skin reactions in OWC and NWC.

The literature contains reports of dromedary camels with positive responses to intradermal tuberculin testing that range from 1.9% reactors in India (Chauhan et al., 1986) to 37% in Kenya (Paling et al., 1988). In a study of 874 Bactrian camels in Russia, there were 107 cases of tuberculosis resulting in a 12.2% incidence rate, but only 68% of the camels with tuberculosis had positive tuberculin reactions (Abramov, 1963). Tuberculin testing in Bactrians in the USA resulted in a number of false positive reactions. At necropsy no tubercles were observed and no mycobacteria isolated, although the lymphocyte stimulation test was also positive (Kennedy and Bush, 1978). Positive tuberculin test results with *M. avium* and *M. bovis* were seen in 10 to 20% of Australian dromedaries whereby no indicative lesions were found after the animals were slaughtered (Schillinger,

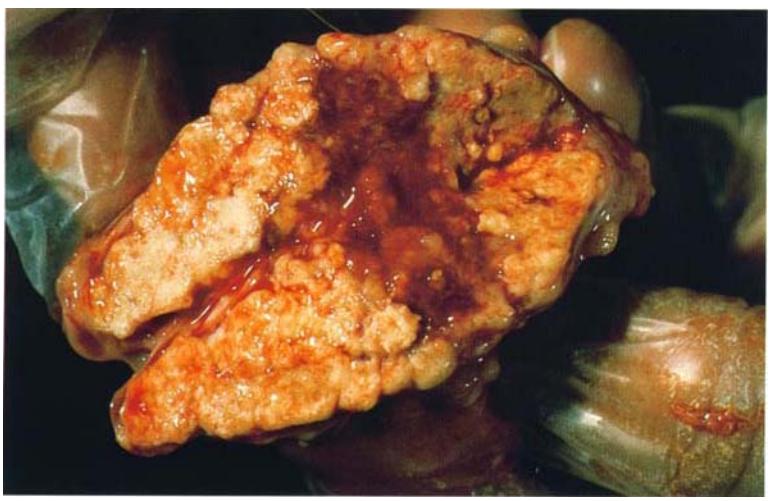

Figure 53 Caseous foci in bronchomediastinal lymph nodes in a llama (courtesy of Dr. A. M. Barlow, UK)

1987). Tuberculosis caused by *M. bovis* was diagnosed in 2 of 19 Bactrian camels in a herd of the National Zoological Park in Washington, USA (Bush et al., 1990). The tuberculin testing with old mammalian and avian tuberculin (MOT, AOT) as well as with avian purified protein derivative (APPD) and bovine purified protein derivative (BPPD) were discontinued in this herd, because many camels showed positive skin reactions, but no clinical disease or indication of infection was discovered on post mortem examination. The Bactrians were tuberculinized into the caudal tail fold and the cervical area. Several years later two Bactrians developed marked leucocytosis that persisted for 3 months despite broad-spectrum antibiotic therapy. These camels were euthanized and disseminated pyogranulomatous lesions were observed in various organs, including lung, mesentery, pancreas, liver, spleen, skin, trachea and many regional lymph nodes. *M. bovis* was isolated from these lesions. A cervical tuberculin test using MOT, AOT, BPPD and APPD had been performed before euthanization with negative results.

A program to control tuberculosis in camelids based only on intradermal tuberculin tests will face severe deficiencies. Other than the intradermal test, the ante mortem tests, such as lymphocyte transformation and ELISA tests, have also not been very reliable in undomesticated mammals because of false-negative and false-positive reactions. This is also true for tuberculosis testing in camelids. However, it is recommended that several tests be used to aid in the diagnosis of tuberculosis in camelids (Fowler, 1998).

The ante mortem diagnosis of tuberculosis in lamoids also presents a challenge. Simmons (1989) believes that one of the reasons for the non-specific reactions in llamas is that the skin of the neck of the llama is very thick and resilient, which makes an accurate measurement very difficult. In a small experiment, the author injected avian and mammalian tuberculin intradermally into different sites in 12 llamas. He suggests that the base of the pinna may be the most suitable location for tuberculin testing. In other experiments in North and South America, the axillary site was determined to be a sensitive site for the allergic tuberculin test. The Mexican study concluded that the axillary site was sensitive in lamoids, but the response was more diffuse and more difficult to interpret than the cervical area. One of the main reasons for false-positive or false-negative reactions is not the structural compounds of the camelids skin but the presence of atypical mycobacterial antigens that are common in camelids. Bush et al. (1990), who also unsuccessfully used the caudal tail fold for tuberculin testing in Bactrians, proved that 12 Bactrian camels showing a positive intradermal test possessed antibodies to atypical mycobacteria when tested with the ELISA and the fused rocket immunoelectrophoresis. The common antigens shared with *Nocardia* and *Corynebacteriae* further negatively affected the specificity of these tests.

It is obvious that OWC and NWC are susceptible to tuberculosis but this disease is very difficult to diagnose on clinical grounds. A definitive diagnosis of tuberculosis requires the culturing and specification of the organism. Considerable efforts have been undertaken in the development of serological tests for the diagnosis of tuberculosis, but they still remain inadequate for the clinical application of this disease.

Mycobacteria are slow-growing organisms that usually appear on culture media within 2 to 6 weeks. Cultural methods are as reliable as animal inoculation methods. Different agars like Loewenstein-Jensen or Ogawa media are used and some mycobacteria require enriched media for successful culturing. For the isolation, the infected tissue is minced and decontaminated by treatment with alkali or acid.

Pathology The studies by Mason (1912, 1917, 1918), on Egyptian dromedaries especially, have provided information on pathological changes found in the disease. The organs most frequently affected in the dromedary are the lungs, bronchial and mediastinal lymph nodes, pleura and liver. The trachea, kidney and spleen can also be affected. Miliary nodes on the surface of the lung and deep in the tissue have been observed. Tubercle bacilli have been isolated from these lesions that cause typical tuberculous lesions in the guinea pig and rabbit. Similar changes in the organs of dromedaries have been described in India by Leese (1918), in Somalia by Pellegrini (1942) and in Egypt by Elmossalami et al. (1971) and Osman (1974). The lesions primarily observed in the lymph nodes and lungs revealed a productive and proliferative response of fibrous tissue and few Langhan's giant cells. The disseminated form of tuberculosis is rarely observed and the alimentary form has not yet been reported in camelids.

Histopathological lesions are pyogranulomas with dense centers containing caseous remnants of neutrophils surrounded by epitheloid macrophages with few giant cells. Application of the Ziehl-Neelsen staining technique to these sections reveals few acid-fast bacilli.

Tuberculosis in dromedaries is rare in the Emirates. Only one case of pulmonary tuberculosis (Fig. 54) among 30,000 dromedaries has been seen within a 15-year observation period. Differentiation of the tubercle bacilli was not performed.

Treatment and Control In many countries tuberculosis is a reportable disease, and bovine tuberculosis has been eradicated due to large-scale campaigns. Positive animals must be slaughtered. Permission was sometimes granted to treat valuable zoo camelids with isoniazid at a dose of 2.4 mg/kg of pelleted feed, which was given ad libitum to Bactrian camels (Bush et al., 1990). However, most probably due to an overdose, several camels died, exhibiting severe leukopenia and thrombocytopenia. On infected properties, surfaces and utensils are disinfected with 3% formalin, 2% Lysol and 2.5% phenol.

A program to control tuberculosis in camelids based on intradermal tuberculin tests is not possible.

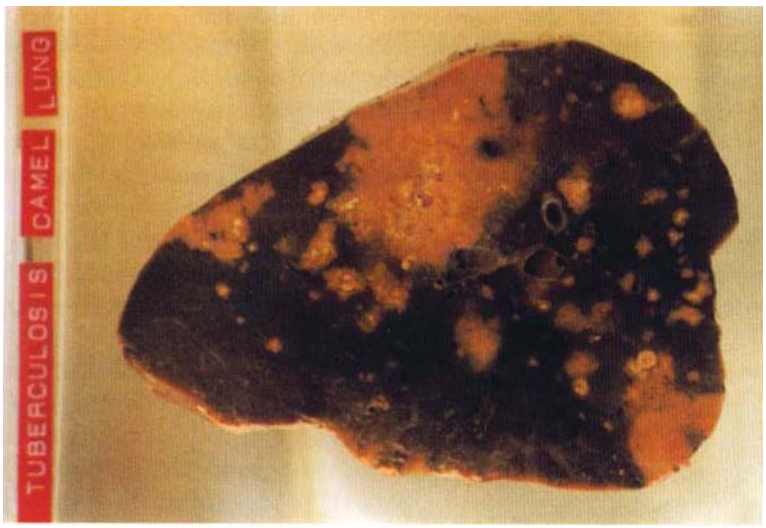

Figure 54 Pulmonary tuberculosis in a dromedary

1.3.2 Pneumonia

The most common respiratory disease is pneumonia, which is defined as an inflammation of the lungs. There are several systems for classifying the various types of pneumonia. One useful method is to classify according to the appearance or etiology of a particular pneumonia, which has been done for pneumonias in camelids (Table 23). Pneumonia can be caused by direct infection with viruses, bacteria, fungus or aspiration, as well as by toxins arriving hematogenously or by inhalation. In many pneumonias, a sudden alteration in the normal nasal bacterial flora with a dramatic increase in one or more species is the trigger for a lung infection. The bacteria are inhaled into the lungs in large numbers where they multiply after they have overwhelmed defense mechanisms. Also a viral respiratory infection may act as precursor to bacterial pneumonia. Handling, transport, mixing and overcrowding are also often considered predisposing factors.

Epidemiology, Clinical Signs and Pathology Various authors have reported changes in the inspected lungs of slaughtered dromedaries. Abdel Rahim et al. (1990) examined 204 slaughtered dromedaries in Libya and found pathoanatomical changes in the lungs due to hydatid cysts and pneumonia in half of them. Al Darraji and Wajid (1990) identified bacteria in 56% of the lungs of 220 slaughtered dromedaries in Iraq. The authors described seven different forms of pneumonia (Table 23).

Table 23 Types of pneumonia in camelids (except tuberculosis)

Type of pneumonia	Microorganisms isolated	Animal species	Cases	Authors	Year	Country
Granulomatous	*Streptothrix cameli* (*Actinomyces farcinicus*, *Nocardia farcinica*?) Pseudotuberculosis	Dromedary	2	Mason Leese	1919 1927	Egypt Sudan Egypt
	Histoplasma capsulatum	Dromedary	2	Chandel and Kher	1994	India
	Nocardia asteroides	Llama	1	Ching-Dong Chang et al.	1993	USA
	Aspergillus and *C. pyogenes*	Dromedary	1	Bhatia et al.	1983	India
Catarrhal (Acute, subacute, chronic)	*Diplococcus*	Bactrian	endemic	Semushkin	1968	Mongolia
	Diplococcus	Bactrian	endemic	Buchnev et al.	1987	Russia
	% *C. pyogenes* 20 Hem. *Streptococci* 12 Diphtheroids 16 *Str. viridans* 20 *S. enteritidis* 2 Coliforms 16 *Alcaligenes faecalis* 22 *Actinomyces pyogenes* 2	Dromedary (abattoir)	50	Farrag et al.	1953	Egypt

Table 23 *(cont.)*

Type of pneumonia	Microorganisms isolated	Animal species	Cases	Authors	Year	Country
Abscess	*Staphylococcus* sp.	Dromedary (abattoir)	79	Moallin and Zessin	1990	Somalia
	Ps. aeruginosa		1	Abdurahman	1987	Somalia
		Dromedary	1	Gautam et al.	1970	India
... with purulent bronchitis	Hem. *Streptococci Staphylococcus*	Dromedary (abattoir)	15	Vitovec and Vladic	1983	Somalia
Necrotic	*Burkholderia pseudomallei*	Dromedary	4	Bergin and Torenbeck	1991	Australia
			1	Wernery et al.	1997	UAE
Hemorrhagic	*Str. equi* spp. *equi*	Dromedary	Epizootic	Yigezu et al.	1997	Ethiopia
Unspecified (mixture of different types)	*St. epidermidis* *Micrococcus roseus* *Micrococcus luteus* *Str. pyogenes* *Str. pneumoniae* *St. aureus* *Micrococcus* sp. *Aerococcus viridans* *Klebsiella ozaenae* *Edwardsiella*	Dromedary (abattoir)	20	Elmossalami and Ghawi	1981	Egypt
	% *Corynebacterium* 21 *Staphylococcus* 30 *Streptococcus* 12 *E. coli* 5 *Pseudomonas* 8 *Proteus* 11 *Klebsiella* 6 *Bacillus* 5	Dromedary (abattoir)	63	Rana et al.	1993	Pakistan
Suppurative		Dromedary	6	Al Darraji and Wajid	1990	Iraq
	Klebsiella pneumoniae	Dromedary	2	Arora and Kalra	1973	India
Chronic non-suppurative		Dromedary	6	Al Darraji and Wajid	1990	India
Interstitial		Dromedary	83	Al Darraji and Wajid	1990	India
Lymphoid	like Maedi/Visna	Dromedary	6	Al Darraji and Wajid	1990	India
Chronic proliferative		Dromedary	7	Al Darraji and Wajid	1990	India
Fibrosis (Silicosis)	-	Dromedary (abattoir)	11	Abdurahman	1987	Somalia

Vitovec and Vladik (1983) found lung abscesses in 15 slaughtered Somali dromedaries from which they isolated hemolytic *Streptococci.* Etiologically, the pulmonary changes arose from a pyogenic bronchitis that tended to spread into the pulmonary parenchyma. Moallin and Zessin (1990) isolated *Staphylococcus* spp., *Pseudomonas aeruginosa* and *Citrobacter freundii* from the lungs of Somali dromedaries that were infiltrated with abscesses. Abdurahman (1987) found *Pseudomonas* spp., *E. coli, Diplococci, Staphylococcus* and other bacteria in the pathoanatomically altered lungs in 6 (3%) of 200 slaughtered Somali dromedaries, and Ghawi (1978) isolated *St. aureus* and *Klebsiella pneumoniae* from pneumonic camel lungs in Egypt. Farrag et al. (1953) diagnosed a large number of cases of pneumonia in slaughtered dromedaries in Cairo. The authors believed that predisposing factors led to the disease development in these cases. Dromedaries that are slaughtered in Cairo must first endure long periods without food on the trek to the slaughterhouse. Upon arrival they are kept in dirty and unkempt stalls. As a rule, 2 to 3 months pass before they are slaughtered. These stressful conditions presumably lead to the increased incidence of pneumonia among these dromedaries. In the histological examination of 50 lungs with pathoanatomical changes, the same authors identified 9 different bacterial species of acute and chronic pneumonia.

One hundred dromedary lungs from Lahore and Faisalabad abattoirs were examined histologically and bacteriologically. The correlation between the pathological findings and the organisms isolated is seen in Table 24.

Judging from slaughterhouse reports from various countries, pathoanatomical changes in the lungs of the camel appear to occur frequently. It is therefore surprising that reports of respiratory diseases in the camel are rather rare. Only a few scientists have reported cases of bacterial pneumonia or bronchopneumonia.

Table 24 Comparison between the bacterial flora and the pathological findings of 100 dromedary lungs

Microorganisms	Frequency	Pathological findings
Staphylococcus	7	Congestion
Corynebacterium	8	Congestion
Klebsiella	2	Congestion
Pseudomonas	5	Congestion
Staphylococcus	6	Hepatization
Streptococcus	7	Hepatization
Klebsiella	2	Hepatization
Corynebacterium	5	Hepatization
Escherichia	3	Hepatization
Staphylococcus	6	Bronchitis
Bacillus	2	Bronchitis
Proteus	4	Pneumonicosis
Bacillus	1	Pneumonicosis
Proteus	3	Hydatid cyst
Mycobacterium	2	Tubercle nodule

Buchnev et al. (1987) reported a septic pneumonia that they called "contagious cough". It manifested itself as an acute catarrhal inflammation of mucous membranes of the upper respiratory tract and lungs, high fever and general illness. The causative agent was an encapsulated diplococcus that was fatal for guinea pigs. According to Semushkin (1968), this disease was also known in Mongolia and was called "black lung" or "contagious cough". The disease was widespread but was not mentioned before 1920 (Amanzhulov et al., 1929). Oinakhbaev (1965) described a cough outbreak when 5000 camels were moved from Mongolia into Kazakhstan. It is believed that starvation, heavy work and prolonged and exhausting journeys were responsible for this respiratory disease. Such lowered resistance aggravated the illness. Once the disease became clinically evident, the disease lasted for 1 to 2 months with fever, enlargement of lymph nodes, sweating and depression. Cough-

ing became steadily worse with prolonged attacks and difficult breathing (Kuznetsov, 1962; Voikulesku, 1963). Diatchenko (1989) compiled a summary of the literature of the different bacterial and viral respiratory diseases in the dromedary.

Arora and Kalra (1973) described cases of chronic bronchopneumonia in Indian dromedaries. The authors reported that the disease occurred only during the colder months and affected almost exclusively adult animals. The morbidity reached 30%, yet only a few camels died. The animals exhibited a protracted course of the illness, during which time they were unfit for work thereby causing economic losses. *Klebsiella pneumoniae* and hemolytic *Diplococci* were isolated from the lungs of two dromedaries that died of bronchopneumonia.

Different authors have reported individual cases of pneumonia in the dromedary. Leese (1927) attributed some isolated cases of respiratory disease to pulmonary abscesses. Gautam et al. (1970) described a pulmonary abscess in a 10-year-old dromedary encompassing nearly the entire right lung. The authors failed to report the causative agent. Pathoanatomical changes in the lung of a young Sudanese dromedary with pseudoactinomycosis similar to tuberculosis were described by Mason (1919) and Hansen et al. (1987) described lung silicosis in dromedaries in Somalia. Kamel (1939) reported pneumococcal pneumonia in Egyptian dromedaries and Agab et al. (1993) isolated a pathogenic *Bacillus coagulans* from a camel lung with pneumonia.

Streptococcus species seem to play an important role in lung infections and other ailments, but they have also been isolated from the lungs of clinically healthy camelids (Shigidi, 1973; Mahmoud et al., 1988; Rana et al., 1993). A hemolytic *Pneumococcus* has been isolated from Bactrian camel lungs in the Gobi Desert (Oinakhbaev, 1965), *Str. viridans*, *Str. pneumoniae* and *Str. pyogenes* from dromedaries (Thabet, 1994), and a β-hemolytic *Streptococcus* also from dromedaries (Pal and Chandel, 1989). In Bahrain, Ibrahim et al. (1998) cultured *Str. zooepidemicus* serotype 2 from the nasal cavities of a dromedary. It had died from suffocation due to necrotic material completely blocking its nasal passages and frontal sinuses. *Str. zooepidemicus* was also isolated from a septic peritonitis of a male Australian dromedary (Heller et al., 1998).

Of great importance is the report by Yigezu et al. (1997) who cultured *Str. equi* spp. *equi* from a sick dromedary during an epizootic outbreak in Ethiopia. The disease was highly contagious with high morbidity and low mortality. The predominant clinical signs were fever, lacrimation, edema of the throat and supraorbital fossa, loss of appetite, cough, dyspnea and purulent nasal discharge. Upon necropsy the lungs revealed hemorrhages and thickened interlobular septae. This is the first report of the isolation of bacteria that causes equine strangles. It is assumed that donkeys were responsible for this outbreak.

Streptococcal infections are also common in NWC. Various streptococcal species have been isolated from NWC abscesses and septicemic enterococcus infections in adult llamas have also occurred (Burkhardt et al., 1993). "Alpaca fever", a septicemia caused by *Str. zooepidemicus*, has been described by Thedford and Johnson (1989). Stress is often a predisposing factor in the disease. Pneumonia is especially common in NWC neonates (Fowler, 1998) and several bacterial agents are involved. As in other animal species (also in NWC calves), septicemic animals often develop pneumonia. *E. coli* is the most common bacteria. *Actinomyces lamae* may also produce pneumonia with abscessation. A llama which was euthanized due to severe dyspnea and cyanosis revealed necrotic pneumonia from which *Nocardia asteroides* was isolated. Microscopically, the lung contained multiple small scattered pyo-

granulomas filled with cellular debris, macrophages and neutrophils surrounded by a few multinucleated giant cells. The visceral pleura of this llama was also altered, thickened by fibrinoserous material. It is believed that the prolonged antibiotic administration that was given to this llama increased the likelihood of infection by the opportunistic *Nocardia* bacteria.

Basic differences of opinion exist whether camels are susceptible to contagious bovine pleuropneumonia (CBPP) caused by *Mycoplasma mycoides*. Most opponents believe that the proponents have confused pulmonary changes due to *Pasteurella* with those due to *M. mycoides*. Walker (1921) was not able to elicit this pulmonary disease through the subcutaneous application of "virulent lymph". Samartsev and Arbuzov (1940), Hutyra et al. (1946), Curasson (1947) and Turner (1959) are of the opinion that camels are not susceptible to contagious bovine pleuropneumonia, though they have not provided any supporting scientific proof. However, those scientists who purport that camels are susceptible to this disease have not supplied any proof of their theory either. This group includes Vedernikoff (1902) and Kowalevsky (1912) who supposedly have often observed respiratory CBPP among Bactrian camels in Kazakhstan. This has also been reported by Davies (1946).

Bares (1968), who reported finding very low (non-specific?) antibody titers using complement fixation against *M. mycoides* in dromedary sera from Chad, is of the opinion that dromedaries are most likely not susceptible to CBPP, and that they play no role in the epizootiology of this disease. All earlier publications implicating *M. mycoides* as a cause of pulmonary changes in the camel should therefore be interpreted with reservation.

Paling et al. (1978) identified antibodies against contagious caprine pleuropneumonia (*Mycoplasma* strain F38) in 49% of the dromedary sera examined in Kenya. The significance of these results is unclear since the causative agent was not isolated.

Although it has not yet been possible to isolate *M. mycoides* from pulmonary pathoanatomical changes in camels, other *Mycoplasma* species have been cultivated from the respiratory tracts of healthy dromedaries. In Egypt, Refai (1992) was able to identify the following isolates from the anatomical sites given:

Mycoplasma arginini:	nose lung mediastinal lymph nodes
Acholeplasma laidlawii:	respiratory tract
Acholeplasma oculi:	nose

Of great significance are the reports by Bergin and Torenbeeck (1991) and Wernery et al. (1997), who were the first to diagnose melioidosis due to *Burkholderia pseudomallei* in dromedaries. Six dromedaries died of this disease in two different outbreaks in Queensland, Australia. Severe necrotic pneumonia was observed in all of the dead animals. The Australian authors are of the opinion that dromedaries living in damp climates appear especially susceptible to this disease. As melioidosis is widespread among the Aborigines in Australia (Asche, 1991) and deaths due to *Burkholderia pseudomallei* have occurred in humans there, the authors urge great care in the treatment of dromedaries with pneumonia. Choy et al. (2000) reported that several dromedaries and alpacas which were brought to the Northern Territories of Australia have died from melioidosis. Melioidosis was also diagnosed in a 7-year-old female dromedary from the UAE that showed signs of wasting disease and severe emaciation before it died (Wernery et al., 1997). Gross pathological lesions revealed granulomas in the uterus and the trachea (Fig. 55) and massive caseous necrosis of three quarters of the lungs (Fig. 56), the mediastinal lymph nodes (Fig. 57), diaphragm (Fig. 58), spleen, liver and kidneys.

Figure 55 Melioidosis lesions in the trachea of a dromedary

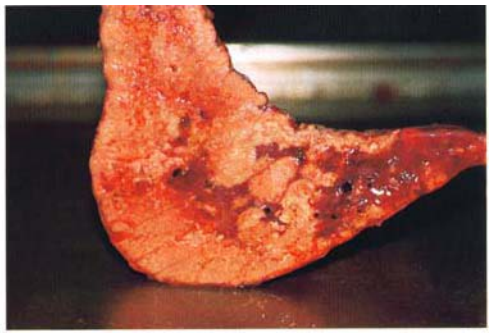

Figure 56 Melioidosis lesions in the lung of a dromedary

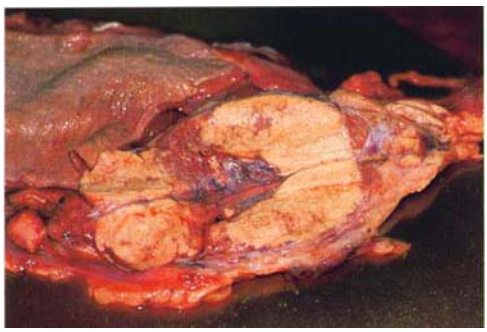

Figure 57 Melioidosis lesions in the mediastinal lymph nodes of a dromedary

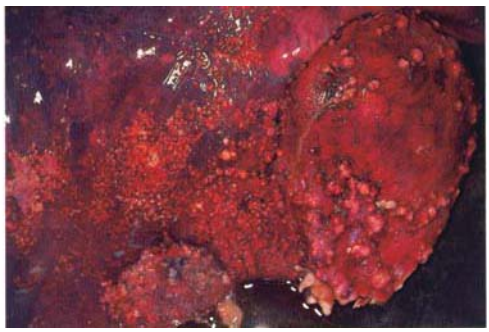

Figure 58 Nodular melioidosis lesions on the diaphragm of a dromedary

Histopathological investigations showed an acute necrotic caseous pneumonia (Fig. 59) and a necrotic lymphangitis.

The authors presume that this single case of melioidosis was caused by a very rainy season that occurred in 1997 in the UAE.

Camelids are also susceptible to *Burkholderia mallei* (Curasson, 1947), but there are no records of natural glanders in camelids. When dromedaries were inoculated with *B. mallei* the animals developed characteristic nodules and ulcers in the nasal wall and in various organs 11 to 15 days p.i. Transmission by contact to dromedaries, horses and giraffes is possible. Samartsev and Arbuzov (1940) consider this disease to be of no significance in camels. Mass malleinisation and clinical observation was carried out on 45,922 camels, but there was no evidence of the disease.

Shigidi (1973) examined nasal swabs and bronchial lymph nodes from 64 slaughtered Sudanese dromedaries, and Chauhan et al. (1987) investigated nasal swabs from 219 healthy Indian dromedaries. The results of the two studies are compared in Table 25.

Pneumonia in dromedaries in the UAE is rare. This is most certainly a result of the adequate management of the racing and breeding herds implemented in the Emirates. If pneumonia occurs, it is usually in conjunction with systemic disease and not as an independent illness. Pneumonia has been observed in dromedaries associated with the following diseases:

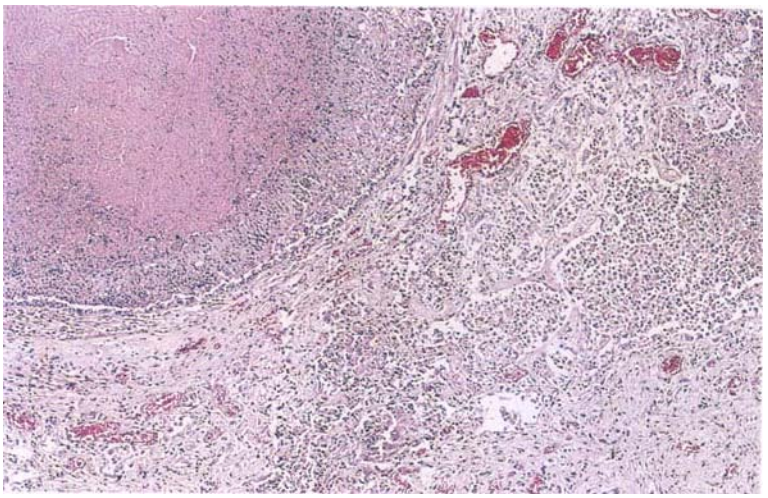

Figure 59 Lobularly chronic productive pneumonia in a dromedary caused by *Burkholderia pseudomallei*

1. colibacillosis,
2. omphalitis,
3. clostridial enterotoxemia,
4. selenium and vitamin E deficiency,
5. pyodermatitis,
6. hyaline membrane disease.

Hyaline membrane disease in premature dromedary calves deserves special mention. The disease has been observed in other animals (lamb, monkey) and in humans (Jones and Hunt, 1983), and is most probably related to a hypofunction and atelectasis of the lungs as well as perinatal asphyxia. During autopsy of the dromedary calves, the compactness of the lungs is readily apparent. Histologically, hyaline membranes in the alveoli (Fig. 60), arterial thrombi, desquamation of alveolar macrophages due to fibrin exudation and cell detritus are seen. This disease is regularly associated with pneumonia.

As stated above, pneumonia in adult dromedaries in the UAE, as in young animals, is observed only in conjunction with other diseases. Bronchopneumonia is regularly associated with leucosis in the dromedary (see 2.2.4) and with aspiration of oral medication given with a bottle. This type of aspiration pneumonia due to the improper application of medication is relatively frequent. In addition to a severe suppurative bronchopneumonia, the majority of these cases also develop pleuritis (Fig. 61).

Table 25 Bacteriological results of nasal swabs and bronchial lymph nodes from Sudanese and Indian dromedaries (Shigidi, 1973 and Chauhan et al., 1987)

Bacteria Isolated	Sudan n = 64 %	India n = 219 %
Aerobic bacteria	30.5	–
Coagulase-negative *Staphylococci*	26.2	2.4
Diphtheroids	15.9	13.7
Aspergillus spp.	8.7	–
Actinomyces pyogenes	5.4	10.9
α-hemolytic *Streptococci*	5.1	2.7
Streptomyces	4.1	–
Staphylococcus aureus	2.6	10.5
E. coli	1.0	24.7
Enterobacter spp.	0.5	–
Klebsiella pneumoniae	–	11.9
Rhodococcus equi	–	8.6
ß-hemolytic *Streptococci*	–	3.7
Hemolytic *Diplococci*	–	3.7
Arcanobacterium hemolyticum	–	0.9
Neisseria spp.	–	0.5

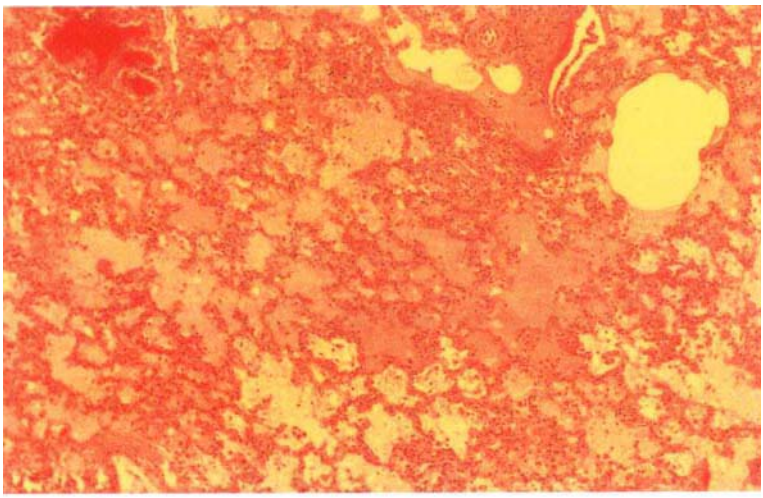

Figure 60 Hyaline membrane disease in a dromedary calf: hyaline membranes in the pulmonary alveoli (HE stain)

Figure 61 Aspiration pneumonia with severe pleuritis following improper oral application of medication

Therapy ⁂ Broad-spectrum antibiotic therapy in association with anti-inflammatory drugs is recommended as well as proper general nursing and supportive treatment. Antibiotics of choice are: Trimethoprim/Sulfadiazine, Procaine penicillin G, Gentamycin and Oxytetracycline. Anti-inflammatory drugs include: Flunixin meglumine and Dexamethasone.

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1.4 Urogenital System

1.4.1 Brucellosis

Through intensive health control measures, many industrialized countries have succeeded in eradicating brucellosis. In developing countries, however, brucellosis remains widespread in domesticated and wild animal populations and presents a great economic problem for tropical animal husbandry (Seifert, 1992). Brucellosis is also one of the most important zoonoses in the tropics. On tropical dairy farms the rate of infection can reach 80%. In areas of extensive animal husbandry in the Sahel, the rate of contamination has been estimated between 25 and 30% (Seifert, 1992). Domenech et al. (1982) estimated the yearly losses in stock due to brucellosis to be 6%.

Especially OWC are frequently infected with brucellosis, particularly when they are in contact with infected ruminants (Abo El-Hassan et al., 1991; Radwan et al., 1992; Barsoum et al., 1995; Al-Ani et al., 1998). Brucellosis in NWC is rare (Fowler, 1998). Humans are at risk through consumption of unheated milk (WHO/FAO, 1986; Kiel and Khan, 1987; Madkow, 1989; Radwan et al., 1995).

Etiology Brucellosis is a contagious disease caused by the bacteria of the genus *Brucella*. *Brucella* bacteria are Gram-negative coccobacilli, which are non-motile and non-spore-forming. Except for *B. ovis* and *B. abortus*, biotype 2, which require media enriched with serum or blood, the growth of other *Brucellae* is enhanced by enriched media, but they are also able to grow on nutrient agar. Extreme care must be exercised when working with *Brucella* organisms.

Epidemiology and Clinical Signs Brucellosis is characterized by abortion, and to a lesser extent by orchitis and infection of the accessory sex glands in males. The disease has a worldwide distribution and affects cattle, pigs, sheep, goats, camelids, dogs, and occasionally horses. In humans, the disease referred to as undulant fever or Malta fever is a serious public health problem.

The infection occurs via the mucous membranes or skin or by ingestion of contaminated foodstuffs, whereby the causative agent then enters via the upper gastrointestinal tract. Infections through the mucosa of the respiratory tract or the eyes are also possible. The spread of brucella during sexual activity plays a subordinate role.

Theoretically, all three known *Brucella* species can cause infection in camels (Higgins, 1986). However, it is surmised that *B. melitensis* is widespread in Africa and the Middle East and *B. abortus* is widespread in the former USSR. Solonitsyn (1949) reported mixed infections with various *Brucella* species in Bactrian camels in Russia.

As can be seen in Table 26, the majority of reports on brucellosis utilize serological methods of identification. A survey of the prevalence of brucellosis in Africa was published by Chukwu (1985). The incidence of brucellosis in camel populations appears to be related to breeding and husbandry practices (Richard, 1980). The infection rate in some regions of the former USSR, where Bactrian camels are kept on large farms, is 15% (Pal'gov and Zhulobovski, 1964). In countries with more extensive forms of husbandry, as in Chad or Ethiopia, the prevalence is 3.8% (Graber, 1968) and 5.5% (Richard, 1980) respectively.

Similar differences in the seroprevalence have been reported from Saudi Arabia by Radwan et al. (1992) and Ghoneim and

Table 26 Summary of literature regarding the occurrence of antibodies to *Brucellae* in OWC arranged by country

Country	Author	Year	Number of camels examined	Prevalence %	Serology			
					RBT	CFT	SAT	MRT
Egypt	Abou-Zaid	1998	422	10.4–12.3	x	x	x	
	Ahmed	1939	200	3.5				
	Ayoub et al.	1978	216	24.2			x	
	El-Nahas	1964	200	4.0	x			
	El-Sawally et al.	1996		2.3–14.0	x	x	x	
	Fayed et al.	1982	300	6.6	x	x	x	
	Hamada et al.	1963	175	10.3			x	
	Nada	1984	780	23.1	x	x		
	Nada	1990		5.3–7.9	x	x	x	
	Zagloul and Kamel	1985	37	8.1				
	Zaki	1948	200	14.0 m			x	
				26.0 f			x	
Sudan	Abbas et al.	1987	238	3.0			x	
	Abu Damir et al.	1984	740	4.9	x	x	x	
	Agab	1993	453	30.0	x			
				32.9 f	x			
				15.1 m	x			
	Agab	1998		2.9	x			
	Ali and Ghedi	1978	250	10.4			x	
	Bornstein and Musa	1987	102	5.9		x	x	
	Mustafa and Awad El-Karim	1971	310	1.8–5.8			x	
	Mustafa and Hassan	1971		1.75–5.75				
	Osman and Adlan	1987	137	8.0	x	x		
Somalia	Ahmed and Ibrahim	1980	802	8.0–11.0	x			
	Andreani et al.	1982	250	10.4			x	
	Anonymous	1981		5.0–7.8			x	
	Baumann et al.	1990		3.1			x	
	Baumann and Zessin	1992	1039	0.3–1.9		x	x	
	Bishof	1979	47	4.0		x		
	Bornstein	1984		8.5–11.5	x			
	Bornstein	1988		1.3		x	x	
	Bornstein et al.	1988	234	5.9		x	x	
	Elmi	1982	514	12.6			x	
Ethiopia	Domenech	1977	977	4.4			x	
	Richard	1980	762	5.5			x	
Kenya	Kagunya and Waiyaki	1978	174	4.6–10.3	x	x	x	
	Waghela et al.	1978	172	14.0	x	x	x	
	Wilson et al.	1982		6.0–38.0				
Chad	Bares	1968	543	5.3			x	
	Graber	1968	316	3.8			x	
Tunisia	Burgemeister et al.	1975	52 and 150 milk samples	3.9–5.8 0	x			x
Nigeria	Okoh	1979	232	1.0	x		x	

Table 26 *(cont.)*

Country	Author	Year	Number of camels examined	Prevalence %	Serology				
					RBT	CFT	SAT	MRT	*
Niger	Bornarel and Akakpo	1982	109	8.3	x	x			
	Saley	1983							
Russia	Pal'gov and Zhulobovsky	1964	500	15.0			x		
	Solonitsyn	1949	27				x		
Mongolia	Shumilov	1974	54,673	1.0–3.7		x	x		
Libya	Ben Faraj et al.	1990	666	3.75	x	x	x		
	Gameel et al.	1993	967	4.1	x	x	x		
India	Kulshrestha et al.	1975	315	1.8			x		
	Mathur and Bhargava	1979	210	3.8–5.2			x		
Iran	Zowghi and Ebadi	1988	953	8.0	x	x	x		
Iraq	Al-Ani et al.	1998	215	7.0–17.0	x		x		
	Jawad	1984	235	3.8	x		x		
Saudi Arabia	Radwan et al.	1983	116	2.8–3.5			x		
	Radwan et al.	1992	2630	8.0		x	x		
	Radwan et al.	1995	2536	8.0	x	x			
Kuwait	Al-Khalaf and El-Khaladi	1989	698 and 209	14.8	x	x	x		
			milk samples	8.0				x	
Oman	Harby and Ismaily	1995	550	3.6		x			
UAE	Afzal and Sakkir	1994	392 racing	0.76	x				
	Moustafa et al.	1998	7899	0.01	x	x	x		
	Wernery and Wernery	1990	196 breeding	2.0		x	x		
			348 racing	6.6		x	x		

RBT = rose bengal test
CFT = complement fixation test
SAT = serum agglutination test
MRT = milk ring test
m = male
f = female
* = see page 115

Amjad (1993). They reported a higher incidence of camel brucellosis in intensive farming than in free-grazing desert camels. According to the system of camel husbandry in Sudan, agropastoralists reported a higher prevalence of brucellosis (31.5%) in contrast with nomadists (21.4%) (Agab, 1993 and 1998).

Remarkably, studies by Wernery and Wernery (1990) in the UAE have shown that the incidence of brucellosis among racing dromedaries not yet certified for breeding is three times higher than in breeding stock (2% compared to 6%). The opposite situation had been expected. The authors surmise that this is due to inherent differences in feeding. In the Emirates, racing dromedaries are usually given non-pasteurized cow milk, which is not given to breeding stock. Various herds of cows were

shown to have an incidence of brucellosis of up to 40%.

The lower incidence of infection in the breeding camel cows when compared to the racing dromedaries in the UAE indicates a spontaneous recovery among currently non-reproductive dromedaries, as described by numerous authors (Ostrovidov, 1954 a and b; Gatt Rutter and Mack, 1963; Fazil and Hofmann; 1981). Interestingly, seropositive racing dromedaries exhibited no reduction in performance during the racing season. The hematology parameters as well as the enzyme activity remained within normal limits.

Moustafa et al. (1998) reported on a serological survey in dromedaries and a brucellosis eradication campaign in the eastern region of the UAE during a 5-year period. The highest prevalence was in 1991 with 5.8% reactors, whereas the lowest was in 1996 with 0.01%. Since no camels have been culled due to brucellosis, it is believed that the reduction in camel brucellosis was caused by the reduction in brucellosis in sheep and goats.

According to various researchers, brucellosis in breeding camelids occurs in all of the known forms, whereby abortion is its most obvious manifestation (Acosta et al., 1972; WHO/FAO, 1986; Fazil and Hofmann, 1981; Radwan et al., 1995; Agab et al., 1996). Infections may also cause stillborn calves, retained placenta and reduced milk yield as is common in cattle and sheep. Retained placentas have not been described in *Camelidae*. This may be a result of the difference in the placental attachment (Fowler, 1998). *B. abortus* and/or *B. melitensis* have been isolated from milk, vaginal swabs, aborted fetuses, lymph nodes and hygromas of infected camelids from different countries.

Although camels appear to be very susceptible to *Brucella* infection, isolation of *Brucella* organisms from camel samples has proved less successful. Only recently have attempts at isolation of *Brucella* from milk been successful. *Brucella abortus* biovars 1 and 3 were isolated from camels in Senegal (Verger et al., 1979). Radwan et al. (1992) were able to isolate *B. melitensis*, biovar 1 and 2 twenty six times from a total of 100 milk samples from seropositive Saudi Arabian dromedaries. This poses a severe health risk for man, since camel milk is not pasteurized. Sharing this conviction, Gameel et al. (1993) were also able to isolate *B. melitensis*, biovar 1 five times from the milk of Libyan dromedaries and four times from aborted fetuses and a vaginal swab. Zaki (1943) both inoculated guinea pigs with milk samples from seropositive dromedaries and cultured the milk samples *in vitro*. Both tests were negative. Al-Khalaf and Al-Khaladi (1989) examined cultures of 209 milk samples from Kuwaiti dromedaries. The samples were obtained from herds with an increased incidence of abortion. The results were negative. However, the authors were successful in isolating *B. abortus* from the gastric fluids of five aborted fetuses. Pal'gov (1950) was able to isolate *B. abortus* from Bactrian camels in Russia. In the herds examined, 2% of all animals aborted in the first half of the pregnancy. Fifteen percent of the herds were seropositive to brucellosis. Zowghi and Ebadi (1988) cultured 3500 lymph nodes from 300 slaughtered dromedaries from Iran for *Brucella* organisms. *B. melitensis*, biovar 1 and 3 were isolated from these lymph nodes in 1% (3/300) of the camels. The authors are of the opinion that the *B. melitensis* infections in the dromedaries originated from neighboring sheep and goat herds.

Radwan et al. (1995), who examined a large camel herd with 2536 dromedaries in Saudi Arabia from which a 12% abortion rate and a *Brucella* seroprevalence of 8% were reported, isolated *B. melitensis*, biovars 1, 2 and 3 from aborted camel fetuses. During their investigations, Malta fever was diagnosed in 30% of the camel handlers and milkers and the same *B. melitensis*

biovars were cultured from aborted sheep and goats sharing the same premises.

B. abortus, biovar 3 was recovered from 3 different specimens obtained from free-ranging camels in eastern Sudan (Agab et al., 1994; Agab et al., 1996). The *Brucella* organisms were isolated from a supramammary lymph node, a vaginal swab and an inguinal lymph node in dromedaries with histories of abortion, presence of hygromas or testicular lesions. It is worth mentioning that both isolates of *B. abortus* biovar 3 from Senegal and Sudan were the only oxidase-negative biovars reported in the literature. Ramadan et al. (1998) have recently recovered *B. melitensis* from a hygroma of an Indian camel. *B. melitensis* was isolated twice from milk samples of seropositive camels in the UAE (Moustafa et al., 1998).

Non-pregnant dromedaries artificially infected with *B. abortus* (wild strain, 6×10^6 bacteria) developed only mild clinical signs. Reduced appetite, slight lameness and bilateral lacrimation were observed. The bacteria were re-isolated 45 to 65 days later from the cranial and genital lymph nodes, which showed follicular hyperplasia of cortical and paracortical areas with active germinal centers, atrophy of medullary cords and sinusoidal congestion. A mild interstitial hepatitis was also observed (Abu Damir et al., 1984).

Brucellosis is not a major disease in NWC, but a severe *B. melitensis* outbreak occurred in a herd of alpacas in Peru. Nearly 30% of the 1449 alpacas tested had a positive plate agglutination titer. Over 25% of the alpaca handlers were seropositive to brucellosis and some developed Malta fever. It was felt that sheep were the source of infection in this alpaca herd (Acosta et al., 1972). In an experimental infection trial in llamas in the United States, it was found that llamas are susceptible to *B. abortus* and that they develop positive serological titers and histological lesions similar to those found in cattle, sheep and goats (Fowler, 1998).

Pathology Very little is known about the pathological changes caused by *Brucella* organisms in camelids. The predilection organs for these bacteria are the pregnant uterus, udder, testicle, accessory male sex glands, lymph nodes, joint capsules and bursae. Lesions may be found in these tissues. Nada and Ahmed (1993) described lesions in non-pregnant dromedaries. They found inflammation of the uterus lining with reddening, edema and necrotic foci in the uterus epithelium, as well as fibrosis of the endometrium and atrophy of the uterine glands. The authors also observed an increased number of ovariobursal adhesions and hydrobursae. The adhesions occurred between the *Bursa ovarica* and the ovary and in several cases also between the *Bursa ovarica* and the salpinges, causing a severe induration of the latter. Hydrobursitis was often observed in brucellosis-positive dromedaries causing an enlargement of the bursa, which was then filled with a clear amber-colored fluid. No lesions have been described so far in aborted camelids and in brucellosis-positive camelid males. A pregnant llama was infected by inoculating viable *B. abortus* bacteria into the conjunctival sac. Forty-three days p.i., the llama aborted an eight-month-old fetus. *B. abortus* was isolated from the placenta and all fetal specimens as well from the dam's mammary gland numerous lymph nodes. Histologically there was a moderate, multifocal, lymphocytic and histiocytic, subacute placentitis with marked loss of trophoblastic epithelial cells. The chorioallantoic stroma contained abundant necrotic and mineralized debris and the swollen capillaries were expanded by large numbers of Brucella organisms (Gidlewski et al., 2000).

Diagnosis Brucellosis is usually diagnosed in the laboratory by culture of blood, milk or tissue or detection of antibodies in sera. *Brucella* organisms can be recovered from the placenta, but more conveniently

in pure culture from the stomach and lungs of aborted fetuses.

However, difficulties may arise in the diagnosis of brucellosis. Abortion and reduced fertility in the camel frequently have other causes, such as salmonellosis, trypanosomosis, or infections with *Campylobacter* or *Trichomonas fetus* (Wernery and Amjad Ali, 1989; Wernery, 1991; Wernery and Wernery, 1992). An incorrect diagnosis of brucellosis may occur when based on serology alone. Sunaga et al. (1983) reported that five dromedaries imported into Japan had positive complement fixation (CFT) and slow agglutination reactions. The animals were immediately slaughtered. No brucella organisms were isolated; however, *Yersinia enterocolitica*, serotype 09 was identified. It is known that false-positive (unspecific) reactions with various other bacterial species can occur (Bisping and Amtsberg, 1988).

Many authors regard the CFT as being the most sensitive and specific test for brucellosis (Gatt Rutter and Mack, 1963; Pal'gov and Zhulobovski, 1964; Tserendash and Shumilov, 1970; Waghela et al., 1978). This is true for both acute and chronic infections. Shumilov (1974) determined that the CFT was four times more sensitive than the agglutination test. He tested Bactrians in Mongolia where brucellosis is widespread among camels. He examined two herds with the following results:

- Herd 1: 3751 head: CFT 4.3% and SAT 0.6%;
- Herd 2: 54,673 head: CFT 3.7% and SAT 1.0%.

In the serum agglutination test an end titer of 1:20 (40 IU) was regarded as suspicious according to different researchers (Arbusov, 1940; Pal'gov 1950; Zhulabovski and Pal'gov, 1954; Ghazi, 1996). Fayed et al., 1982; Salem et al., 1990; El-Sawaly et al., 1996 believe that the Serum or tube agglutination test detects a higher percentage of reactors to brucellosis than other assays due to its greater sensitivity to IgM than IgG.

In order to eliminate unspecific reactions in the serum agglutination test, Wernery and Wernery (1990) utilized a 5% solution of phenol sodium chloride.

In addition to this cross-reactivity with other bacteria that make the serological diagnosis of brucellosis more difficult, Zhulobovski and Pal'gov (1954) observed prozones in some sera of Bactrian camels in Russia and Nada (1984) in dromedaries from Egypt. The absence of a visual positive reaction in low dilutions has also been observed in 1.5% of all positive dromedary sera in the UAE. The Coombs test is necessary to verify the diagnosis of brucellosis in these cases.

In an attempt to overcome the difficulties in the serological diagnosis of brucellosis in camel sera using traditional methods, the authors recently utilized a commercial brucellosis ELISA for cattle with good results. The labeled second antibody was produced in cooperation with the Institute for Medical Microbiology in Munich, but nowadays anticamel IgG is commercially available. Other researchers have recently used ELISA for the detection of *Brucella* antibodies, not only in camel sera (Azwai et al., 1996; Abou-Zaid, 1998), but also in camel milk (Straten et al., 1997). The camel milk ELISA seems to be an important alternative to the conventional serodiagnosis of camelid brucellosis.

Several researchers have evaluated the different serological tests for the diagnosis of camel brucellosis (Abo El-Hassan et al., 1991; Nada et al., 1992; Ghoneim et al., 1993; Abou Zaid, 1998). It was concluded that the elimination of non-specific reactions to *Brucella* in camelid sera is essential for the correct diagnosis. It is also important to apply more than one test, of which the tube agglutination test (TAT) using 5% NaCl phenolized solution must be included for the serological diagnosis of camelid brucellosis. Atwa (1997) and Abou Zaid

(1998) found agreement between five different serological tests ranged between 80.6% and 95.6%.

Mohammed (1996) evaluated the rose bengal plate test (RBPT), the tube agglutination test (TAT), and the complement fixation test (CFT) for the diagnosis of brucellosis in camels. He found that the RBPT and the CFT demonstrated equal ability in detecting positive and negative sera as well as prozone reactions. However, for optimal sensitivity, the RBPT has to be used with serum-antigen at a 3:1 dilution. When using the CFT, the 1:10 diluted sera have to be inactivated at 54°C for 30 minutes and the cold fixation technique has to be applied. Using the TAT, the classical neutral pH antigen has to be replaced by a buffered (pH 3.5) antigen to achieve optimal results.

In llamas experimentally infected with *B. abortus*, the CFT, the standard test tube (STT) and the D-tec ELISA were less reliable for the detection of antibodies in comparison with the buffered acidified plate agglutination test (BAPAT), the card test, the standard plate test (SPT) and the rivanol test (Fowler, 1998).

Radwan et al. (1995) examined a large camel farm comprising 2536 dromedaries in Saudi Arabia for Brucella antibodies. The authors used a combination of two tests to identify seropositive dromedaries – the rose bengal test (RBT) and the standard United States of America buffered plate agglutination test. With these two methods, the authors successfully eradicated the disease from the farm that caused 12% abortions. The authors adopted these tests due to their sensitivity, simplicity and applicability in the field.

In contrast to cattle milk, camel milk cannot be used to detect lacteal brucellosis antibodies using the conventional milk ring test (MRT) because camel milk lacks the agglutinating substance required to cluster fat globules (Straten et al., 1997). The MRT results summarized in Table 26 should therefore be interpreted with great caution. Straten et al. (1997) established a MRT that can also be used to detect antibodies in camel milk. The researchers named this test a modified MRT because *Brucella*-negative cow milk is added to the camel milk, producing a typical colored creamy ring when antibodies to *Brucella* bacteria are present. Selective *Brucella* medium was found to be the optimal culture medium for the growth of *Brucella* organisms from fresh camel milk and camel tissue (Radwan et al., 1995). During intensive investigations, it was found that on a camel farm in Saudi Arabia 34% of all *Brucella*-seropositive milking dromedaries were *Brucella* shedders.

Treatment and Control For the eradication of brucellosis in animals, the "test and slaughter" and "vaccination" policy is recommended. This method should be implemented when the disease is serologically and bacteriologically confirmed. Seropositive animals should be slaughtered and the entire herd tested until all reactors are eliminated. In *Camelidae*, as in other animals, this will be achieved when two to three successive tests are negative. After this procedure, a vaccination program may then be implemented to protect the entire herd from re-infection. The greatest danger comes from replacement animals. Infected vaccinated animals remain a severe hazard to public health.

Radwan et al. (1995) treated 202 seropositive dromedaries with a combination of oxytetracycline (25 mg/kg body weight) every 2 days for 30 days and streptomycin (25 mg/kg body weight) every 2 days for 16 days. In addition to this parenteral treatment, milking camels received 10 mL oxytetracycline as intramammary infusions in each teat every 2 days for 8 days. This regimen of treatment was effective in eliminating the shedding of *Brucella* organisms through milk. All treated dromedaries also became negative within 16 months after treatment.

Both inactivated and attenuated *Brucella* vaccines have been used successfully in OWC. Dromedaries were vaccinated with *B. abortus* strain Buck 19 (Chichibabin, 1971) and with *B. melitensis* Rev 1 (Radwan et al., 1995). Young dromedaries received a full dose of the vaccine and adults a reduced dosage. Both groups developed *Brucella* antibodies after vaccination, which receded after 8 months in young stock and after 3 months in adult camels. After vaccination no further abortions were reported. Agab et al. (1995) vaccinated five dromedaries with a reduced dose (5×10^8 CFU in 2 mL) of *B. abortus* strain 19 (S 19) against brucellosis. All five camels seroconverted after one week and their antibodies declined 6 to 7 weeks later. The camels tested negative 14 weeks later.

1.4.2 Infections of the Uterus

In *Camelidae*, the reproductive biology presents some very important particularities not seen in other domesticated animal species. These special features were unknown for a long time and were discovered only during the last decade. Camelids have a unique ovarian cycle – they are induced ovulators, the fetuses possess an epidermal membrane and they exclusively develop left-horn pregnancies. Intensive research has been carried out over the last 10 years on the reproductive physiology of *Camelidae*. This was done on NWC by Fowler and Bravo (1998) and on dromedaries by two groups from the UAE (Skidmore, 1994; Tibari and Anouassi, 1997). A third group from the UAE investigated the causes of uterine infections (Wernery and Kaaden, 1995). A comprehensive compilation of scientific papers concerning the reproductive tract of OWC has recently been gathered by Beil (1999).

In the last decades there has been intensive research undertaken to clarify the causes of uterine infections in the horse and cow and to identify a causal relationship between the bacteria isolated in the uterus and endometritis. A great number of bacterial species have been isolated from the equine and bovine uterus; however, only a few of these microorganisms are primarily pathogenic. The majority of these bacteria are opportunistic. It is therefore important to view all bacteriological results together with the clinical presentation of the genital tract, such as uterine inflammation and discharge. Further relevant information can be obtained through endometrial smear preparations and uterine biopsies (Ricketts, 1989).

In general, *Camelidae* are very fertile animals. Bactrians and dromedaries produce fertile hybrids and NWC interbreed as well. With advanced technology it is even possible to enter a completely new field – the production of hybrids between OWC and NWC (Skidmore et al., 1999).

According to Wilson (1989), the dromedary birth rate under natural conditions is very low, although dromedaries are supposedly very fertile. The author estimates a number of reasons for the low birth rate. In a large field study encompassing many Asian and African countries, he determined that only three calves are born per breeding female. This is due to the late first pregnancy (at five years of age), the long gestation period of 13 months, the long interbirth interval (> 24 months) and the early slaughter of breeding stock.

The fertility rate of dromedaries in Saudi Arabia lies between 80 and 90% with only 1% permanent sterility (Arthur et al., 1985). Yagil (1985) reported similar figures. His experimental dromedaries attained a fertility rate of 100%. However, when kept under conditions of intensive husbandry, Mukasa-Mugerwa (1981) reported a dromedary fertility rate of only 50%, which could be improved up to 65% with corresponding improvements in management. Nutritional deficiencies, trypanosomosis, tuberculosis, ecto- and endoparasites as

well as recurrent endometritis can reduce fertility.

A variety of bacterial species have been isolated from the uterus of infertile camelids, but it is often unclear whether they play an important role in primary uterine infections.

Epidemiology and Pathology Uterine infections in *Camelidae*, as in other domesticated animal species, are the most commonly acquired reproductive failures resulting in infertility (Tibary and Anouassy, 1997). Only a few scientists have examined uterine bacterial infections in *Camelidae*, contrary to those found in horses and cattle. They primarily focus on dromedaries used for slaughter (no information on reproduction is available [Merkt et al., 1987]). More intensive studies of breeding dromedaries in the UAE have recently been performed by Wernery and Amjad Ali (1989), Wernery (1991) and Wernery and Wernery (1992). There were only a few reports on reproductive failure except for brucellosis in Bactrian camels. The UAE scientists were the first to isolate *Campylobacter fetus* and *Trichomonas fetus* from the uterus of sterile dromedaries suffering from endometritis. These findings could be of major clinical implication and may be associated with a particular form of endometrial lesions seen in many biopsy samples taken from dromedaries with endometritis. These lesions are characterized by the presence of lymphoid granulomatous infiltrations of varying size (Fig. 62).

Granulomas consisting of mononuclear cells have also been described in non-pregnant dromedaries by Nada and Ahmed (1993) and Tibary and Anouassi (1997).

Wernery (1991) was able to prove that the bacterial species isolated from the dromedary uterus are identical to those found in the mare and cow, with the exception of *Taylorella equigenitalis* and *Streptococcus zooepidemicus* which were not isolated. In order to evaluate the role of various microorganisms in the development of uterine infections in dromedaries, the scientists from Dubai suggest following the bacterial classification for horse and cattle formulated by Ricketts (1981) and Arthur et al. (1985) (Table 27).

In addition to the classical venereal microorganisms *Campylobacter fetus* and *Trichomonas fetus* that cause sterility in dromedaries through endometritis, *Actinomyces pyogenes* also appears to play an important

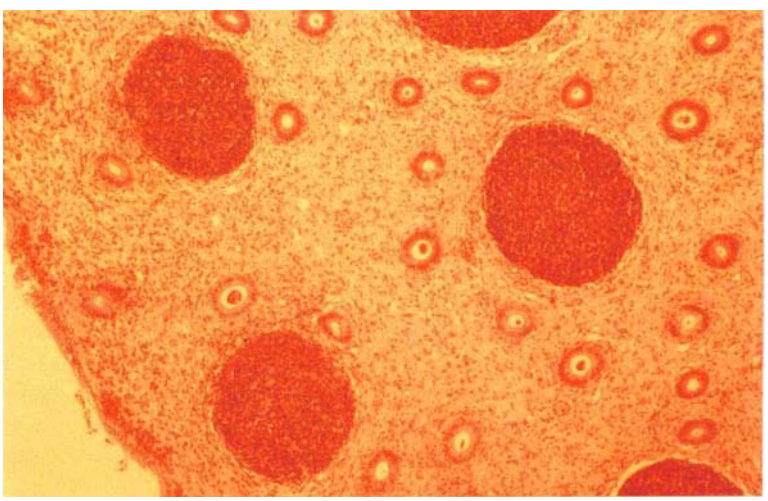

Figure 62 Lymphoid granulomas in the uterus of a barren dromedary (HE stain)

Table 27 Classification of bacteria and protozoa isolated and cultured from the equine and bovine genital tract juxtaposed with the microorganisms found in the genital tract of camels

Horse, Cattle	Camel
Venereal infections due to bacteria and protozoa	
1. *Taylorella equigenitalis*	–
2. *Klebsiella pneumoniae* (Capsule type 1,2,5)	–
3. *Pseudomonas aeruginosa*	Nawito (1973), Wernery and Amjad Ali (1989), Hassan (1990), Wernery (1991)
4. *Campylobacter fetus*	Wernery and Amjad Ali (1989)
5. *Trichomonas fetus*	Wernery (1991)
Non-specific bacteria in conjunction with endometritis	
1. *Streptococcus zooepidemicus*	Nawito (1973)?, Awad et al. (1978)?
2. *E. coli* (hemolytic)	Nawito (1973), Eidarous et al. (1983)
3. *Staphylococcus aureus*	Nawito (1973), Awad et al. (1978), Hegazy et al. (1979), Ali et al. (1987), Wernery and Amjad Ali (1989), Hassan (1990), Wernery (1991)
4. *Proteus* sp.	Hegazy et al. (1979), Ali et al. (1987)
5. *Klebsiella pneumoniae* (Capsule type 6,7,21,68)	Awad et al. (1978), Hegazy et al. (1979), Wernery and Amjad Ali (1989), Hassan (1990)
6. *Pseudomonas fluorescens*	–
7. *Pseudomonas aeruginosa* (non-venereal strains)	–
8. *Enterobacter aerogenes*	Hegazy et al. (1979), Eidarous et al. (1983)
Contaminants and commensals	
1. *Enterococcus faecalis*	Wernery and Amjad Ali (1989)
2. *Staphylococcus albus*	Nawito (1973), Wernery and Amjad Ali (1989), Wernery (1991)
3. *E. coli* (non-hemolytic)	Hegazy et al. (1979), Ali et al. (1987), Wernery and Amjad Ali (1989), Hassan (1990), Wernery (1991)
4. *Actinomyces* sp.	Zaki and Mousa (1965), Nawito (1973), Awad et al. (1978), Hegazy et al. (1979), Eidarous et al. (1983), Ali et al. (1987), Hassan (1990)
5. *Neisseria* sp.	–
6. *Anthracoid organisms*	Zaki and Mousa (1965), Eidarous et al. (1983), Wernery and Amjad Ali (1989), Wernery (1991)
7. *Clostridium sporogenes*	Wernery (1991)
8. *Bacteroides fragilis*	–
9. *Fusibacter* sp.	–

role in this disease (Nawito, 1973; Awad et al., 1978; Hegazy et al., 1979; Al-Ani et al., 1992).

Pal'gov (1950) observed abortions in Bactrians in Kazakhstan over a 3-year period. The camels aborted after a 5 to 6-month pregnancy. The aborted fetuses showed inflamed umbilical cords, hemorrhages of the epicardium and enlarged spleens and livers. Tuberculosis, brucellosis and glanders were excluded as the cause of abortion. *Streptococcus pyogenes* was isolated from the aborted fetuses and their membranes.

Table 28 Microorganisms isolated from 98 infertile dromedary cows with and without endometritis (Wernery and Wernery, 1992)

With endometritis	Without endometritis
Staphylococcus spp.	*Staphylococcus* spp.
Staphylococcus aureus	*Staphylococcus aureus*
Streptococcus spp.	*Streptococcus* spp.
Aerobic Bacilli	Aerobic Bacilli
Diplococcus	*Diplococcus*
E. coli	*E. coli*
C. sporogenes	*C. sporogenes*
Campylobacter fetus	
Pseudomonas aeruginosa	
Klebsiella ozaenae	
Salmonella spp.	
Serratia marcescens	

To what extent opportunistic microorganisms are involved in the etiology of infection in the dromedary uterus has not yet been determined. When the bacteria isolated from dromedaries with and without endometritis are compared (Table 28), the inherent difficulties in the interpretation of the bacteriological results become apparent.

In camelids, as in equines and bovines, successful diagnosis and treatment of infertility depends on the evaluation and interpretation of all results gained through vaginoscopy, uterine culture, uterine cytology and eventual biopsy.

Scientific interest has also turned to the NWC following the intensive study of OWC in the last few years. Powers et al. (1990) studied uterine infections in llamas. They examined 90 animals with fertility problems and discovered uterine infections in 45 (50%). In 27 of the barren llamas, of which 21 had a culture-positive uterus, the following bacterial species were cultured:

- *Actinomyces pyogenes* 7×
- *Bacillus* spp. 6×
- *Staphylococcus* spp. 6×
- *E. coli* 6×
- *Streptococcus* spp. 3×
- *Bacteroides* spp. 1×
- *Fusobacterium necropherum* 1×
- mixed culture 9×

The llama specimens were also classified on the basis of a grading system used for mare endometrial biopsies, the results of which are seen in Table 29.

Table 29 Uterus pathology of llamas compared with cultural growth

Grade of Uterus	Uterus Pathology	Growth	No Growth
I A	Normal	2	2
I B	Mild endometrial changes Few lymphocytes Minimal gland fibrosis	4	1
II A	Endometritis	15	3
II B	Endometritis		0
III A, B	Moderate to severe gland fibrosis	0	0

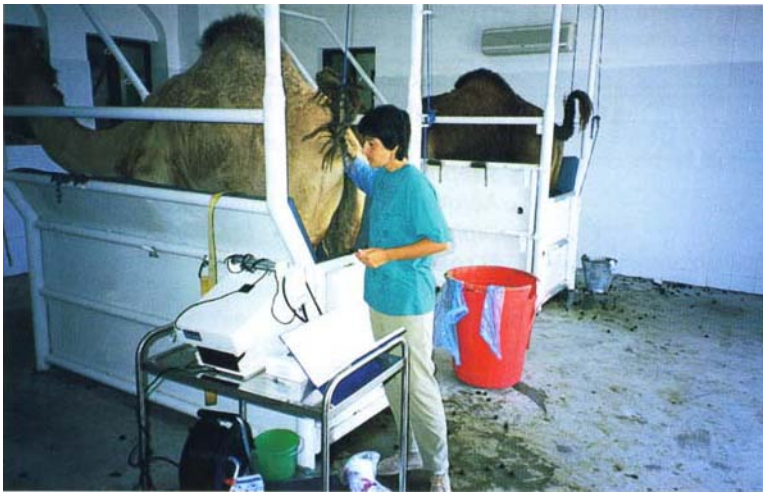

Figure 63 Uterine swabbing; the dromedary is swabbed in standing position with its tail fixed upwards preventing it from crouching

Diagnosis Evaluation of a female breeding camel should begin with a good history of her reproductive record. Uterine infection should be suspected in any animal that has a history of repeated breeding. Thorough examination of the reproductive tract of camelids requires restraint of the animal to avoid any injuries to the veterinarian or the animal. *Camelidae* can be restrained in lateral recumbency or sternal crouching position or placed into a rectal palpation chute (Fig. 63). The restraint in crouching position is the only possible way to examine the animal in the field. The vagina and the cervix orifice of the camel can be examined through a vaginal speculum and swab specimens can be obtained. In case of suspected endometritis swabs should be taken from the endometrium and cervix. For research purposes, Wernery (1991) followed the procedures used for the isolation of *Taylorella equigenitalis* in horses. Swabs of the endometrium, the clitoral fossa and the urethral orifice were taken.

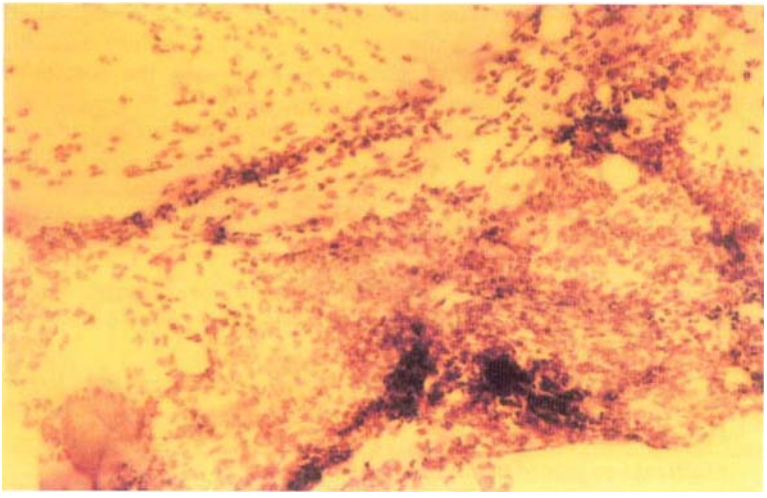

Figure 64 Uterine smear from a dromedary with endometritis due to *Campylobacter fetus*, stained on Testsimplets slides (Boehringer Mannheim, Germany)

The author also used cytological techniques to identify the presence of uterine infection (Fig. 64). However, no biopsies were taken to evaluate the extent of inflammation and duration of the endometrium.

Histological investigations of the uterus have been performed on camelids by various scientists in connection with the follicular waves (Fowler and Bravo, 1998; Beil, 1999). Since camelids do not cycle as most animals, a variable histological picture associated with stages of estrus cycle cannot be described.

Histological changes of the uterus in connection with an endometritis have been reported by various scientists (Nawito, 1973; Hegazy et al., 1979; Laila et al., 1987; Fetaih, 1991; Al-Ani et al., 1992) who found 4.53% (94/2075), 25.0% (24/96), 86% (67/78), 74.6% (97/130) and 4.0% (2/50) cases of endometritis in slaughtered camels from Egypt and Iraq. The results of these investigations exclusively stem from slaughtered camels with no reproductive history (Table 30).

So far no reports are available of uterine biopsies taken in connection with uterine cytology and uterine culture from living OWC. The results from slaughtered camels show that they can suffer from different forms of metritis, from which a variety of different bacterial species have been isolated (Table 31).

Abortion rates in *Camelidae* are low. In the dromedary, abortion rates may range between 2% and 18%. Various infectious agents have been associated with abortion in camelids, but generally very little is known. Several diseases have also been implicated in abortions in camelids, but their prevalence is unknown except for brucellosis. The following diseases are considered responsible for abortions in OWC and NWC:

- brucellosis,
- clostridiosis,
- camelpox,
- trypanosomosis,
- toxoplasmosis,
- leptospirosis.
- chlamydiosis.

Bacillus cereus has been recently isolated from the placenta and different organs of an aborted fetus of a dromedary (Wernery et al., 1996). The fetal membranes revealed severe hemorrhagic necrotizing placentitis and edema.

During recent years, veterinary journals have published several small reports dealing with abortions in NWC outside their native countries. In most of the cases, no confirmed diagnosis was made, although in many cases a thorough investigation was carried out including testing for leptospirosis, bovine viral diarrhea, enzootic abortion agent and toxoplasmosis. In a recent incident, four alpacas aborted and *Enterobacter cloacae* and *Klebsiella pneumoniae* were isolated from different tissues (SAC, 1999).

Treatment and Control Treating camelid uterine infections follows the same protocol as for equines and bovines. The treatment may be grouped into local application of drugs into the uterus, parenteral ad-

Table 30 The incidence of endometritis in slaughtered dromedaries

Author	Year	Country	Total	Endometritis	Percentage
Nawito	1973	Egypt	2075	94	4.53
Hegazy et al.	1979	Egypt	96	24	25.0
Laila et al.	1987	Egypt	130	97	74.6
Fetaih	1991	Egypt	78	67	86.0
Al-Ani et al.	1992	Iraq	50	2	4.0

Table 31 The frequency of bacterial isolates in different forms of metritis in dromedaries

Metritis	Authors	% of uteri with changes	Bacterial isolates	Number of bacteria isolated
Acute suppurative endometritis	Fetaih (1991) Egypt		Aerobic Bacilli	4
			Staphylococcus aureus	3
			β-hemolytic *Streptococcus*	3
			Streptococcus epidermidis	2
			Corynebacterium pyogenes	2
			E. coli	1
	Hegazy et al. (1979) Egypt	25 (24/96)		
Subacute suppurative endometritis	Fetaih (1991) Egypt		*Corynebacterium pyogenes*	5
			Streptococcus epidermidis	4
			Staphylococcus aureus	3
			Aerobic Bacilli	3
			Streptococcus spp.	3
			Proteus morgani	2
			non-hemolytic *Streptococcus*	2
			E. coli	1
			β-hemolytic *Streptococcus*	1
Catarrhal endometritis	Nawito (1973) Egypt	1.5 (31/2075)	*Staphylococcus aureus*	2
			Staphylococcus albus	2
			Streptococcus spp.	1
			E. coli	4
	Hegazy et al. (1979) Egypt	25 (24/96)		
Chronic catarrhal endometritis	Fetaih (1991) Egypt		Aerobic Bacilli	18
			Streptococcus epidermidis	16
			Staphylococcus aureus	6
			E. coli	6
			Proteus morgani	4
			Klebsiella pneumoniae	3
			Corynebacterium pyogenes	2
			Non-hemolytic *Streptococcus*	1
	Hegazy et al. (1979) Egypt	25 (24/96)		
Hemorrhagic endometritis	Nawito (1973) Egypt	0.24 (5/2075)	*E. coli*	2
			Staphylococcus albus	2
			E. coli / Micrococcus pyogenes	1 / 1
			Staphylococcus aureus	2
			β-hemolytic *Streptococcus*	2
Acute suppurative metritis	Fetaih (1991) Egypt		*E. coli*	1
			Proteus morgani	1
			Alcaligenes faecalis	1
Chronic non-suppurative metritis	Fetaih (1991) Egypt		*Bacillus* spp.	3
			Streptococcus epidermidis	3
			Staphylococcus aureus	3
	Al-Ani et al. (1992) Iraq	4.0 (2/50)	*Corynebacterium pyogenes*	2

Table 31 *(cont.)*

Metritis	Authors	% of uteri with changes	Bacterial isolates	Number of bacteria isolated
Pyometra	Nawito (1973) Egypt	1.9 (39/2075)	*Pseudomonas aeruginosa*	5
			Staphylococcus aureus	7
			Streptococcus spp.	10
			E. coli	5
			Staphylococcus albus	5
			β-hemolytic *Streptococcus*	6
	Laila et al. (1987) Egypt		*Streptococcus epidermidis*	83.3%
			Streptococcus pyogenes	66.6%
			Proteus	33.3%
Pyometra plus macerated fetuses	Nawito (1973) Egypt	0.72 (15/2075)	*Staphylococcus aureus*	5
			Staphylococcus albus	2
			β-hemolytic *Streptococcus*	4
			E. coli	3
	Laila et al. (1987)		*Streptococcus epidermidis*	
Chronic active metritis	Fetaih (1991) Egypt		*Staphylococcus aureus*	2
			Streptococcus epidermidis	2
			Streptococcus spp.	2
			non-hemolytic *Streptococcus*	2
			E. coli	1
			Aerobic Bacilli	1
			Pseudomonas aeruginosa	1
			Proteus morgani	1
			α-hemolytic *Streptococcus*	1
Necrotic endometritis	Hegazy et al. (1970) Egypt			
Hydrometritis	Laila et al. (1987) Egypt		*Corynebacterium*	
			E. coli	75%
			Sarcina	25%
Endometritis with abscessation	Nawito (1973) Egypt	0.05 (1/2075)	*Staphylococcus aureus*	1

ministration, or both. Local administration consists of uterine lavage or infusion with weak disinfectants and/or appropriate antibiotic solutions. Before applying any antibiotics, a sensitivity test should be performed on the isolated organisms. Antiseptic solutions such as Lotagen® at a dilution of 1 to 4 in physiological saline or phosphate buffer should be infused through an artificial insemination pipette. In NWC up to a 100 mL and in OWC up to 1000 mL should be infused. This procedure should be repeated daily for 3 to 5 days until the uterine culture is negative. In order to achieve an optimal distribution of the medicine, the uterus may be massaged per rectum. In severe pyometra cases, before infusion of any drug, the uterus should be massaged to reduce the volume of pus which must be drained through a catheter. Parental administration of antibiotics may accompany the infusion procedures in severe uterine infections. Even after specific treatment following sensitivity

Table 32 Treatment and the resulting reproductive success in dromedaries with bacterial endometritis

No.	Bacteria isolated	Treatment	Pregnancy
1	*E. coli*	Enrofloxacin[1]	no
2	*Diplococcus*	Furazolidone[2]	no
3	*Streptococcus*		
	Aerobic bacilli	Neomycin[3]	no
4	*E. coli*	Furazolidone	no
5	Aerobic bacilli	Chloramphenicol[4]	yes
6	*E. coli*	Furazolidone	yes
7	*E. coli*	Furazolidone	no
8	*E. coli*		
	Staphylococcus spp.	Neomycin	no
9	α-hemolytic *Streptococci*	Ampicillin[5]	yes
10	*E. coli*		
	α-hemolytic *Streptococci*	Furazolidone	yes
11	*E. coli*	Furazolidone	yes
12	*E. coli*	Furazolidone	no
13	*Pseudomonas aeruginosa*	Neomycin	
		Enrofloxacin	no
14	*E. coli*	Enrofloxacin	yes
15	*E. coli*	Neomycin	no
16	α-hemolytic *Streptococci*	Neomycin	no
17	*Staphylococcus* spp.	Ampicillin	no
18	*E. coli*	Enrofloxacin	no
19	*E. coli*		
	α-hemolytic *Streptococci*	Gentamicin[6]	no
20	*E. coli*		
	α-hemolytic *Streptococci*	Furazolidone	no

Suppliers: [1]Bayer, [2]Smith Kline Beecham, [3]Intervet U.K., [4]Antarres Vet. Products, [5]Bristol, [6]Farvet Lab., Holland

testing, treatment is not always successful. Powers et al. (1990) reported that 22 of the 36 llamas (67%) that were treated became pregnant. Wernery and Kumar (1994) had less success in treating endometritis in dromedaries. The authors attained a 30% fertility rate following antibiotic treatment of the uterus in dromedary cows that had been infertile for 2 to 5 years. The bacterial species isolated, the antibiotics used in the treatment and the success rate are summarized in Table 32.

Undiluted Lugol's iodine may be employed as the last resort for severe uterine infections. However, the value of this treatment is controversial.

1.4.3 Chlamydiosis

Chlamydiosis in livestock is caused by *Chlamydia psittaci* and is characterized by a variety of clinical syndromes. *C. psittaci* can affect the respiratory and the intestinal tracts, the nervous and reproductive system and the joints and eyes. While *C. psittaci* affects various animal species and humans, *C. trachomatis* is mainly limited to humans. *Chlamydia psittaci* is known to cause enzootic ovine abortion and epizootic bovine abortion (Beer, 1980). The role of this bacterium in OWC is unknown. However, it is known that *C. psittaci* causes disease in NWC (Schroeder et al., 1998; Goepner et al., 1999; Goepner, 1999).

Etiology *Chlamydiae* are classified in the order I *Rickettsiales*, order II *Chlamydiales*, family *Chlamydiaceae* and two genera; Genus 1: *Chlamydia* with species *trachomatis, suis* and *muridarum* and Genus 2: *Chlamydophila* with species *psittaci, pneumoniae, pecorum, abortus, caviae* and *felis*. Isolates of *C. psittaci* from cattle and sheep are grouped into two main antigenic groups: serovars 1 and 2. Serovar 1 causes abortions and genital and enteric infections, while serovar 2 causes polyarthritis, polyserositis, keratoconjunctivitis, interstitial pneumonia and meningoencephalomyelitis. *Chlamydiae* are intracellular bacteria, Gram-negative, non-motile and they possess a unique development cycle.

Epidemiology and Pathology *C. psittaci* occurs throughout the world. The bacteria are shed in feces and other body discharges from the genital and respiratory tracts. Transmission by arthropods is also possible. Different authors have identified antibodies to *Chlamydiae* in dromedaries. Giraud et al. (1954) discovered two positive camels out of nine in Chad; Burgemeister et al. (1975) found 7.7% dromedaries with a positive reaction in Tunisia; Schmatz et al. (1978) 11% in Egypt and Djegham (1988) 4.4% also in Tunisia. Wernery and Wernery (1990) were able to identify antibodies against *Chlamydia* in racing (15.0%) and breeding (24.0%) dromedaries in the UAE. The authors are of the opinion that this organism does not have any influence on pregnancy in the dromedary since no increase in the abortion rate was observed in the herds studied. They were also unable to identify any positive reactors using a *Chlamydia* ELISA (Abbott Laboratories) on the uterine swabs taken from 28 seropositive dromedaries.

Sheep are usually infected by vaginal discharge containing *Chlamydiae* and through contact with aborted fetuses. Some of these animals then develop a subclinical intestinal infection, whereby large numbers of organisms are excreted with the feces (Morgan et al., 1988). In the UAE, the source of infection of dromedaries seems to be close contact with sheep and goats, with an infection rate of up to 50%.

Chlamydia spp.-induced abortion in one alpaca, and a suspected chlamydial pneumonia were observed in one vicuña in Germany. Chlamydiosis was reported in alpacas from a zoological garden in Leipzig, Germany (Goepner et al., 1999). The disease was introduced by a male alpaca brought to the zoo. The outbreak was characterized by conjunctivitis, keratoconjunctivitis, iridocyclitis and uveitis (Fig. 65).

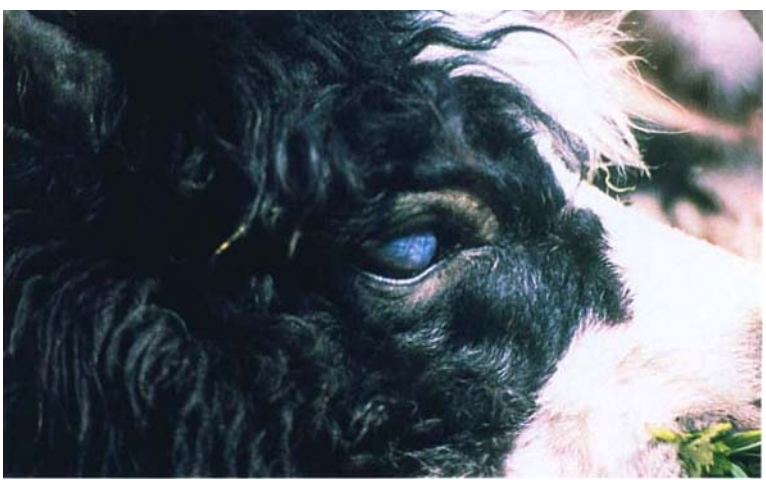

Figure 65 Keratoconjunctivitis in an alpaca with chlamydiosis (courtesy of Prof. Dr. K. Eulenberger, Germany)

Many stillborn crias were born and several young animals developed arthritis. Of the 53 crias born in this zoo over a period of 12 years, 32 died from chlamydiosis. Popovici et al. (1970) were the first to report on a *Bedsonia* (*Chlamydia*) outbreak in llamas in a zoo in Bucharest. Young llamas died of encephalomyelitis.

Diagnosis Cultivation of *Chlamydia* organisms is possible in mouse brain, embryonated hen's eggs and in tissue culture. Antigen ELISA, immunofluorescent and immunoperoxidase stainings are faster methods for the diagnosis of chlamydiosis. More recently molecular biological methods have been introduced. The complement fixation test was commonly used for the detection of antibodies to *C. psittaci* but is now being replaced by an antibody ELISA.

Treatment and Control Tetracyclines and chloramphenicol are the most effective drugs for the treatment of chlamydiosis because they inhibit the multiplication of *Chlamydiae*. However, Goepner et al. (1999) stated that treatment with antibiotics stopped the acute disease, but had no effect on chronic or arthritis cases. During the outbreak it was extremely important to separate any sick animal from the healthy herd. An inactivated vaccine for sheep was used to control the disease. The healthy alpacas were vaccinated twice within 3 weeks and thereafter every 6 months.

1.4.4 Urinary Retention in Young Dromedaries

Annually, in certain breeding herds in the UAE, recurrent urinary retention is seen in 2–4 week-old dromedaries. The calves affected no longer suckle, exhibit fever of up to 41°C and die within 4–6 days. Some of the affected animals also develop torticollis. Upon autopsy, urinary retention without urethral obstruction is seen (Fig. 66).

Urine-filled cysts of varying size are found in the kidneys caused by the urinary reflux (Fig. 67).

The histological examination of the brain in the young dromedaries suffering from torticollis demonstrated intracerebral hemorrhages and perivascular cellular infiltrates that were infected with microorganisms. Similar changes were seen in the meninges. These lesions could be readily seen macroscopically and indicated the presence of an infectious encephalitis and meningitis.

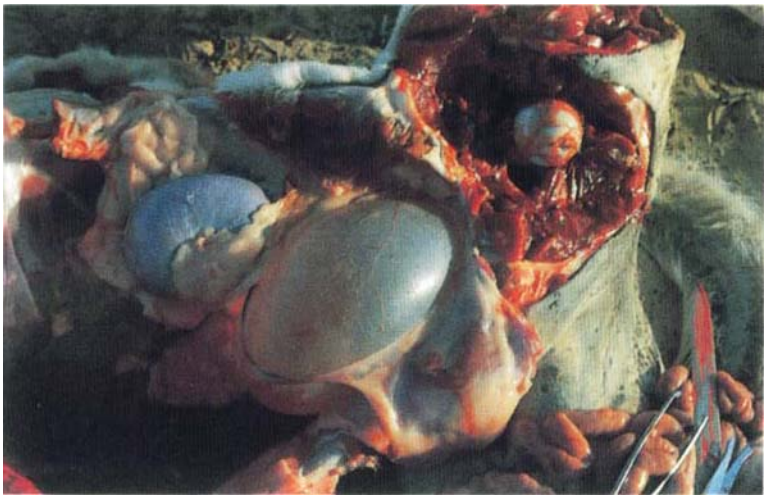

Figure 66 Urinary retention in a 2-week-old dromedary calf

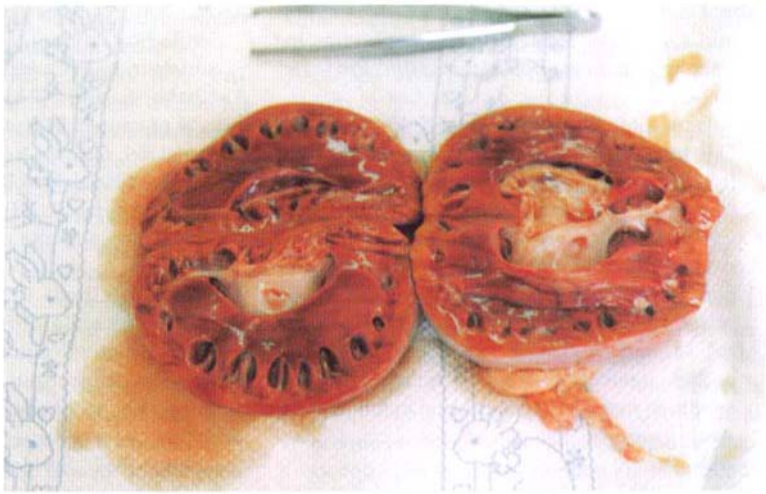

Figure 67 Renal cysts in a 2-week-old dromedary calf secondary to urinary retention

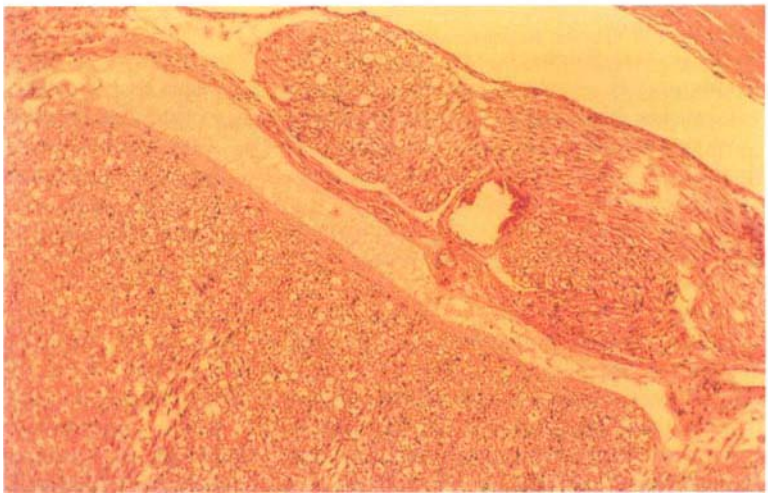

Figure 68 Severe demyelination of the cauda equina and afferent nerves in a 2-week-old dromedary

Pseudomonas putida was regularly isolated from these histopathological lesions. The cauda equina was examined in a number of dromedaries revealing a severe demyelination of the spinal cord and the afferent nerves (Fig. 68).

It is not yet clear whether a causative relationship exists between the urinary retention and CNS pathology. Further studies are required to identify the cause of this disease in young dromedaries. Presumably the infection is secondary to a deficiency syndrome (i.e. vitamin B, copper).

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1.5 Integument

The general opinion that wound healing in camels is slower than in other mammals is not true. Purohit and Chouhan (1992) determined that camel skin is well vascularized with good wound healing. However, there is no doubt that *Tylopodae* in general tend to develop abscesses (Strauss, 1991). The abscesses in the subdermis, superficial lymph nodes and musculature frequently observed in camels are most likely due to the animals' preference for the leaves and small branches of the thorny acacia. The long thorns (up to 5 cm) not only penetrate the skin and cause deep-seated infections, but can also injure the mucous membranes of the oral cavity. Frequently, abscesses of the cranial, cervical, thoracic and popliteal lymph nodes are seen without noticeable superficial injury. Such injuries are more frequent in free-grazing breeding and racing dromedaries than in racing dromedaries that are kept in the paddock the entire year. Eighty percent of Australian feral camels, which browse on sharp thorns and branches, are affected (Manefield and Tinson, 1996).

A multiplicity of skin diseases has been described and there are confusing reports regarding their presentation and etiology. Many reports do not mention whether the bacteriological samples were obtained from a closed or open abscess or from wounds. It is theoretically possible that parasitic cysts, for example due to *Onchocerca fasciata*, may be confused with abscesses (Bergin, 1986). The severe allergic reaction accompanied by swelling that many camels exhibit following the subcutaneous application of certain medications must also be considered (Schwartz and Dioli, 1992). As mentioned earlier, camels are very sensitive to oil-based vaccines (see Fig. 21).

Infectious skin diseases in camelids are caused by many different bacterial, viral and mycotic pathogens. The minor bacterial skin infections are caused by *Corynebacterium pyogenes, Streptococcus* spp., *Nocardia asteroides, Actinobacillus lignieresi* (Daneji et al., 1996), *E. coli*, and *Fusobacterium necropherum*. However, the following chapters particularly deal with skin diseases that are of economic importance in camelids. They include pseudotuberculosis, *Staphylococcus aureus* dermatitis and dermatophilosis.

1.5.1 Pseudotuberculosis (Caseous Lymphadenitis)

Pseudotuberculosis in sheep and goats occurs worldwide. It is a chronic disease caused by *Corynebacterium pseudotuberculosis (ovis)* (Behrens, 1987; Lloyd et al., 1990; Lindsay and Lloyd, 1991). It is characterized by abscessation of one or more lymph nodes. It sometimes also causes pneumonia, hepatitis, mastitis, arthritis, orchitis and subcutaneous abscesses. *C. pseudotuberculosis* also affects horses and produces an ulcerative lymphangitis in cattle. Pseudotuberculosis is widespread in OWC and the organism has also been isolated from abscesses in alpacas (Barsallo et al., 1984 a and b; Greenwood, 1991).

Etiology The French veterinarian Nocard first described *Corynebacterium pseudotuberculosis* in 1888. It is a short, irregular ovoid, Gram-positive rod almost resembling a coccus. In smears made from abscesses, the bacteria show a marked pleomorphism. For routine isolation, sheep or ox blood is used and the plates should be incubated at 37°C for at least 48 h. *C. pseudotuberculosis* colonies are small, white and dry and

can be surrounded by a narrow zone of hemolysis. At least two toxins are produced by the organism and may vary between strains.

Epidemiology Camel pseudotuberculosis has been observed in Iran (Esterabadi et al., 1975), Egypt (Caprano, 1934; McGrane and Higgins, 1985; El-Sergany et al., 1991; Refai, 1992), Ethiopia (Domenech et al., 1977; Hoste et al., 1985), Kenya (Bergin, 1986), Australia (Bergin, 1986), Saudi Arabia (Radwan et al., 1989), India (Purohit et al., 1985), Russia (Spesivtseva and Nosko, 1959; Sadykov and Dadabaev, 1976), China (Chen et al., 1984), UAE (Tarek and Abu-Bakr, 1990; Wernery and Kaaden, 1995) and East Africa (Dioli and Stimmelmayer, 1992). Serologically, two distinct strains have been identified – strain sheep/goat and strain horse/cattle. Only the first strain has been found in camels. The isolation of *C. pseudotuberculosis* from abscesses poses certain difficulties as the colonies resemble streptococcal colonies and are frequently overgrown by accompanying bacteria. For example, *C. pseudotuberculosis* was not isolated in 15% of infected goats showing typical lesions (Lindsay and Lloyd, 1991).

The infection is spread via ingestion, inhalation or directly through wounds in sheep and goats. *C. pseudotuberculosis* is a pyogenic, facultative intracellular bacterium. It penetrates the tissue and produces filterable toxins. At least two toxins, a toxic cell-wall lipid and a hemolysin, play essential roles in the development of caseous lymphadenitis. The toxic cell-wall lipid is associated with the virulence of the bacterium and the hemolysin causes hemorrhages, increased vascular permeability and enhanced bacterial invasion.

In contrast to pseudotuberculosis in sheep and goats, *C. pseudotuberculosis* is not always the only bacteria isolated from the abscesses in camels. Dominic et al. (1977) were able to isolate the following bacterial species in Ethiopian dromedaries:

- *Streptococcus* (Lancefield Group B) 57%
- *C. pseudotuberculosis* 37%
- *Staphylococcus* spp. 10%
- *C. pyogenes* 6.7%

Apart from *C. pseudotuberculosis*, Radwan et al. (1989) were also able to isolate *Staphylococcus aureus, C. renale, C. equi, Shigella* spp. and *E. coli* in 15% of 2500 dromedaries in Saudi Arabia. The authors also reported abscess formation in the musculature and subdermis over the neck, tail and joints. There was a generalized lymphadenopathy without abscess formation in the lymph nodes. The afflicted animals concurrently suffered a severe infestation of ticks *(Hyalomma)* from which the authors were able to isolate *C. pseudotuberculosis*. Guinea pigs that were injected intraperitoneally with cultures of *C. pseudotuberculosis* died 3 weeks later with multiple abscesses.

Hoste et al. (1985) believe that *Actinomyces pyogenes* is of similar importance in the pathogenesis of pseudotuberculosis as *C. pseudotuberculosis*. Spesivtseva and Nosko (1959) and Dalling et al. (1966) purport that *Histoplasma farciminosum* is responsible for an outbreak of pseudotuberculosis among Bactrian camels in the Soviet Union. The disease occurred in 1958 when camels were walked from Central Asia to several farms near Moscow. The lesions were observed in the pre-shoulder lymph nodes. *Mycelium* and *Cryptococcus*-like organisms were detected in the draining lymph nodes. *Cryptococci* were also observed in macrophages.

Ismail et al. (1985) reported a *C. pseudotuberculosis* outbreak in 21 dromedaries in 6 Egyptian villages that also affected cattle and buffalo. The primary manifestation was edema of the elbows, the chest and the external lymph nodes. The authors also reported ulceration of some of the lymph nodes. This was associated with a bloody exudate. *C. pseudotuberculosis* alone was isolated from the non-ulcerative lymph

nodes, though *C. pseudotuberculosis* and *Staphylococcus aureus* were isolated from the ulcerations.

Skin lesions caused by acacia thorns, ticks, contaminated injection needles and nodular worms may inadvertently result in damage to the skin and thus create portals of entry for *Corynebacteria*. The mucous membranes of the oral cavity might be damaged by acacia thorns and/or by dry and hard stems from desert plants. Following its entry through the skin or mucous membrane, *C. pseudotuberculosis* bacteria are then transported via the afferent lymphatics to the regional lymph nodes in which lesions may develop. Lymphogenous and hematogenous distribution of the infection from the primary site to internal organs and tissues may occur latently. Different scientists conclude that *C. pseudotuberculosis* may not always be the sole cause of lymphadenitis in camelids. However, there is some confusion whether the samples were obtained from closed or open abscesses. Stowe (1984) reported that in open abscesses, secondary infection with coccal organisms can be expected.

Abou-Zaid et al. (1994) detected lymphadenitis in 10.9% (37/339) dromedaries from Egypt. The affected adult camels revealed enlargement and abscess formation in the superficial lymph nodes. The lymph nodes released a thick, caseated creamy pus and/or calcified material. *C. pseudotuberculosis ovis* was isolated in pure culture from 62.1% cases and associated with *Staphylococcus aureus* and *Streptococcus* spp. from the rest.

Afzal et al. (1996) isolated pure cultures of *C. pseudotuberculosis* from 11 racing camels from the UAE suffering from lymphadenitis. Six of the camel isolates and a sheep strain used as control produced necrosis of rabbit skin and redness. In an experiment, one of each isolate (with and without dermonecrosis and the sheep strain) was inoculated into the base of the ear of experimental camels. Camels infected with the sheep strain and the dermonecrotic isolate produced lymph node swelling only, whereas the strain without dermonecrosis produced multiple abscesses in the experimental camels 40 days after infection. Re-infection of the experimentally infected dromedaries after they had recovered from the disease did not produce any lesions.

Clinical Signs and Pathology The incubation period of *C. pseudotuberculosis* abscesses ranges from 25 to 40 days in sheep and goats. After 40 days, Afzal et al. (1996) observed multiple abscess formation in camels experimentally infected with *C. pseudotuberculosis*. Extensive caseous necrosis in lymph nodes and other organs (especially lung) develop in sheep and goats. In comparison, pathological changes in the internal organs due to *C. pseudotuberculosis* are rare in camels (Radwan et al., 1989). The generalized cutaneous form is also seldom observed (Dalling et al., 1966; Eldisougi, 1984). Pathognomonic for the disease are cold, closed, painless abscesses up to the size of a lemon or orange in the external lymph nodes (Fig. 69), especially at the base of the neck and in the prescapular lymph nodes (Schwartz et al., 1982).

If opened, the abscess extrudes thick, yellow cream-like pus. Most abscesses are enveloped by well-developed connective tissue capsules. In most cases a concentrically lamellated (onion ring) pattern of the abscess develops in sheep and goats (Behrens, 1987; Nashed and Mahmoud, 1987). These pathological changes have never been described in camelids.

A few cases have been seen in dromedaries whereby the abscesses break through the ribs and the organism enters the lung, producing severe bronchopneumonia with pulmonary caverns (Fig. 70).

The microscopic lesions described by Nashed and Mahmoud (1987) consist of caseous necroses of the lymph nodes with a lymphoid and epitheloid reaction. Giant

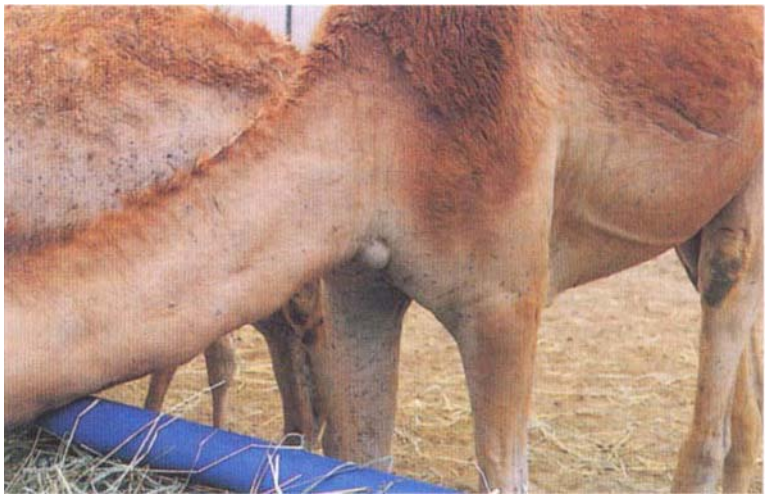

Figure 69 Pseudotuberculosis in a one-year-old dromedary

cells were not observed. Histopathological examinations of the affected lymph nodes by Abou-Zaid et al. (1994) revealed acute serous, acute suppurative and chronic suppurative lymphadenitis. Pseudotuberculosis occurs primarily in camels more than 3 years old (Schwartz and Dioli, 1992).

Treatment and Control Affected animals serve as reservoirs of infection. They should be separated from healthy ones. Ripe superficial abscesses should be lanced, providing strict aseptic procedures are employed. The infected material must be destroyed and contaminated equipment disinfected.

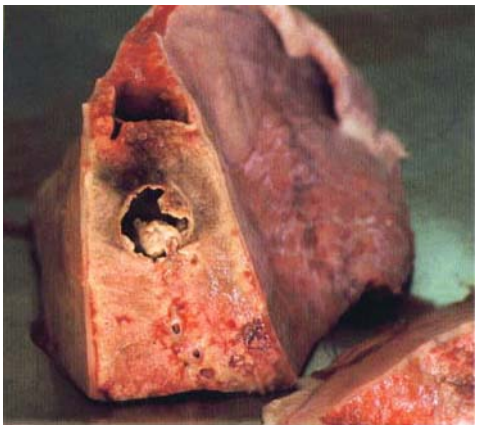

Figure 70 Pulmonary cavern caused by *C. pseudotuberculosis* in a dromedary

Corynebacteria are extremely sensitive to penicillin, tetracyclines and cephalosporines, yet the pus in the abscess prevents the medication from reaching the bacteria. Since erythromycin is more able to penetrate the tissues, Bergin (1986) suggests a combination of penicillin and erythromycin to treat pseudotuberculosis in camels. Another possibility of treating pseudotuberculosis is the intravenous injection of 20 mL dimethyl sulfoxide (DMSO) and 20 mL Baytril® for 12 days. The abscess will eventually subside with no relapse. Afzal et al. (1996) are of the opinion that their experiment indicates that a vaccine against lymphadenitis of camels might be developed based on a sheep strain of *C. pseudotuberculosis*. Several scientists have started research in the production of a vaccine against pseudotuberculosis (Han et al., 1983; Anonymous, 1995). The successful toxoid vaccine used in sheep and goat pseudotuberculosis is also intended for trials in camels (Bergin, 1986).

Pseudotuberculosis remains one of the most important bacterial diseases in camelids (Domenech et al., 1977; El-Sergamy

et al., 1991; Abou-Zaid et al., 1994) with an infection rate between 10% and 60%. The disease also occurs in dromedaries in the Emirates. For the reasons mentioned at the beginning of the chapter, the disease is seen much more frequently in breeding than in racing dromedaries. Since the affected lymph nodes seldom develop abscessation, pseudotuberculosis in this country is more of an aesthetic problem than a health problem. Staphylococcal dermatitis is of greater importance.

1.5.2 Staphylococcus aureus dermatitis

Staphylococcus aureus is a commensal bacterium of animals and humans that mainly occurs on the skin and the nasopharynx. It may also be present in the alimentary and genital tract. *St. aureus* is a potential pathogen and can cause a wide range of pyogenic conditions, the major one in livestock being mastitis in cattle, sheep and goats. It may infect the skin of different animal species under the following names:

- folliculitis and furunculosis in horses, goats, sheep, dogs;
- pyoderma in goats, piglets, cattle;
- facial or periorbital eczema in sheep;
- impetigo or subcorneal pustular dermatitis of piglets;
- dermatitis of the udder in goats.

It also produces systemic diseases like botryomycosis in equines, pyemia of lambs and polyarthritis in young animals. Pyoderma is one of the major infectious skin diseases in OWC. *St. aureus* has also been isolated from abscesses of an alpaca (Fowler, 1998) that was diagnosed with botryomycosis, a purulent granulomatous lesion.

Epidemiology and Pathology Difficult to treat medically, pyoderma in camels is a suppurative, chronic inflammation of the skin primarily caused by *Staphylococcus aureus* and occurring mainly in young dromedaries. The disease begins with a folliculitis, which frequently progresses to a furunculosis with individual or grouped 3–5 mm big abscesses. These have a small, easily removable scab that covers a small amount of pus. A crater is revealed when this pus is removed. The abscesses can become quite large and, when lanced, yield a whitish-green pus (Fig. 71). Larger abscesses are frequently encountered between the forelegs of the animal.

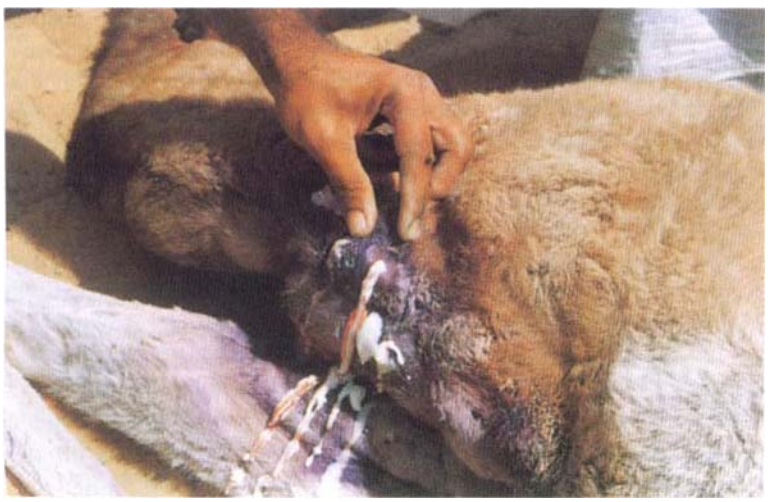

Figure 71 *Staphylococcus aureus* abscess in a 6-week-old dromedary

Bornstein (1995) also described similar lesions as lymphadenitis in camel calves less than 4 months old. These lesions consisted of several abscesses found at the base of the neck and between the front legs. These abscesses were warm and painful and often as big as an orange. The pus from the abscesses was yellow and creamy. Affected animals are disturbed, can lose condition or might succumb. Often several calves of a herd are affected. *Streptococcus* spp. and *Staphylococcus* spp. have been isolated from these lesions.

As in caseous lymphadenitis, the abscesses located between the front legs of the camel calves may rupture into the thoracic cavity, causing septicemia and/or severe bronchopneumonia with pericarditis and hydropericardium (Fig. 72).

An exudative eczema with pustules also colonized with *Staphylococcus aureus* can be present in addition to the furunculosis. The disease can be chronic and difficult to treat depending on, among other factors, the pathogenic qualities of the staphylococcal strain present. *Staphylococcus aureus* strains possess a multitude of virulence factors that can harm the host organism and protect themselves from the host's defenses (Schels, 1989).

Only a few reports of bacteriological studies of skin abscesses in camels exist. Ismail et al. (1990) isolated the following bacterial species from non-draining abscesses of the head, shoulder, chest, leg and abdomen:

1. *Staphylococcus aureus,*
2. *Actinomyces pyogenes,*
3. *C. pseudotuberculosis,*
4. *Streptococcus pyogenes,*
5. *E. coli,*
6. *Klebsiella* spp.,
7. *Proteus vulgaris,*
8. *Proteus mirabilis,*
9. *Pseudomonas aeruginosa,*
10. *Clostridium perfringens,*
11. *Fusobacterium necrophorum.*

The same bacterial species were isolated by El-Seedy et al. (1990) from wither fistulae in 93 pack camels in Egypt.

According to Buchnev et al. (1987), staphylococcal disease is widespread among Bactrian camels in Central Asia. Semushkin (1968) called the condition "contagious skin abscesses" which can affect 5 to 20% of the Bactrian camel population and induce a mortality of 15%. The etiology of this disease was largely unknown until Sadykov and Dadabaev (1976) identified the cause. The disease presents as a puru-

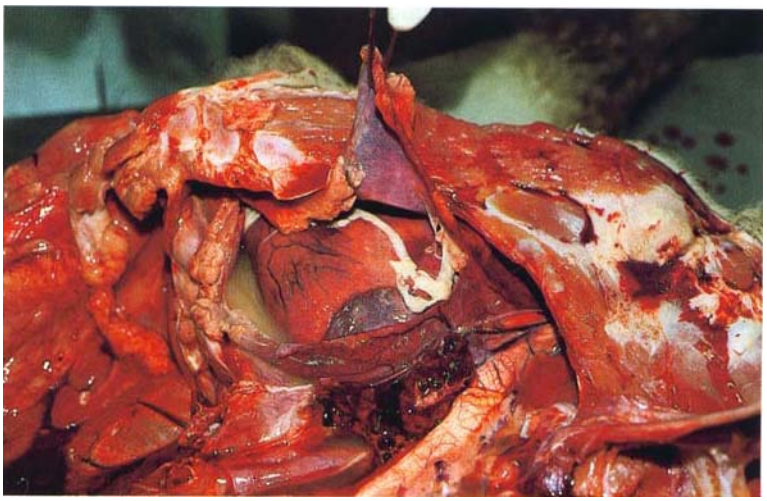

Figure 72 Pericarditis and hydropericardium caused by *St. aureus*

lent lymphangitis in Bactrian camels, affecting the superficial lymph nodes of the head, neck and shoulder. Lancing the abscess reveals thick, whitish pus. In some cases, abscesses containing 500 mL of pus have been reported. Pyogenic septicemia is a frequent complication and many Bactrian camels have died from the disease. Several staphylococcal strains have been isolated from Bactrians from different areas. In various tests, all strains possessed identical properties. The strain has been named *St. cameli*. Samartsev (1950) reported an infectious pustular dermatitis in camels in Kazakhstan, which was caused by *St. pyogenes citreus*. The pustules were 0.5 to 2.0 cm in diameter and disappeared after one month without treatment.

Domenech et al. (1977) have studied the pyogenic affections of the one-humped camel in Ethiopia. Their study showed two well-defined skin diseases: "mala" or lymphadenitis and "maha" or "doula" or cutaneous necrosis caused after ulceration of skin abscesses. *Staphylococcus aureus* and *Streptococcus* B have been isolated from these lesions.

Pyogenic dermatitis also plays an important role among young dromedaries in the Emirates. During the course of 15 years, bacteriological studies were performed on abscesses, wounds, ulcers and other skin lesions. The results are summarized in Table 33.

Treatment and Control In order to control the disease, affected animals should be isolated and treated. Since some *St. aureus* strains are very resistant to antibiotics, a sensitivity test should be performed on all isolated strains. Affected skin lesions should be cleaned daily with 5% Lotagen® solution, and ripe abscesses lanced and drained. In severe cases, parenteral antibiotic administration should be tried.

As can be seen from Table 33, *St. aureus* was isolated from 71% of the abscess specimens. *St. aureus* was found in small numbers on the skin of healthy dogs (Schels, 1989). However, the bacterial counts increase 50 to 100 fold in pathological skin lesions. Since pyoderma is difficult to treat with antibiotics, the authors have regularly produced auto-vaccines for the afflicted dromedaries. The auto-vaccines were developed for the individual animal or for a small group of animals from the same herd.

The production of an individual autovaccine is necessary as there are many different immunological and virulence factors present in *St. aureus* strains. This also prevents the industrial production of a vac-

Table 33 Bacterial species isolated from skin lesions from dromedaries in the UAE

Bacteria Isolated	Skin Lesions		
	Abscesses: Open and Closed	Wounds/ Ulcers	Others
Staphylococcus aureus	82	12	7
Staphylococcus spp.	7	6	25
Actinomyces pyogenes	5	1	0
C. pseudotuberculosis	3	0	0
Dermatophilus congolensis	0	0	4
Streptococcus spp.	4	3	3
Pseudomonas spp.	3	2	5
Proteus spp.	3	1	2
E. coli	4	2	2
aerobic bacteria	4	5	7
Total	**115**	**32**	**55**

cine. Dromedaries suffering from *St. aureus* dermatitis were given 5 to 8 mL of a formalin-inactivated vaccine subcutaneously. Sixty percent of the dromedaries vaccinated showed initial improvement within the first few days; the abscesses underwent exsiccation and reduced in size. Only a few animals required a booster injection after 14 days. All cases of *St. aureus* dermatitis were successfully treated in this manner. It was also possible to inoculate the unaffected animals prophylactically and so inhibit the spread of the disease. The remarkable success of the *St. aureus* vaccine is based on a general non-specific stimulation of the immune system, a paraimmunization, as well as a specific immunization against all of the antigenic exotoxins and other virulence factors of the dermatopathogenic strains of *St. aureus*. Phagocytosis resumes following neutralization of anti-phagocytosis virulence factors of the pathogenic *Staphylococci*. The major problem in the treatment of pyoderma is being able to adequately increasing the body's own defense mechanisms (Schels, 1989).

1.5.3 Dermatophilosis

The infection ascribed to *Dermatophilus congolensis* is a typical epidemic in the humid tropics. It is widespread in Africa, Australia and New Guinea. In the Americas, the in-

Table 34 Contagious skin necroses in the dromedary and their isolates

Author	Year	Country	Designation/Isolates
Cross	1917	India	*Streptococcus*
Curasson	1918	Africa	Cutaneous *streptothricosis*
	1920		*Actinomyces (Nocardia) cameli*
	1936		*Nocardia farcinica*
	1947		*Streptothricosis*
Mason	1919	India	Contagious skin necrosis
Leese	1927	India	Skin necrosis
Peck	1938a, b	Somalia	
	1939		Contagious skin necrosis, salt deficiency
Edelsten and Pegram	1974	Somalia	Contagious skin necrosis *Streptococcus agalactiae*
Domenech et al.	1977	Ethiopia	Skin necrosis, various bacterial species
Fazil and Hofmann	1981		Skin necrosis *Actinomyces cameli*
Schwartz et al.	1982	Kenya	Skin necrosis on hind legs, urine
Wardeh	1989	Mauritania	Contagious skin necrosis, *Streptothrix* spp.
Gitao et al.	1990	Kenya	*Dermatophilosis*
Gitao	1992		*D. congolensis*
Gitao	1993a		*Dermatophilosis*
Wernery and Ali	1990	UAE	*Dermatophilosis* *D. congolensis*
Joseph et al.	1998	UAE	*Dermatophilosis*
Gitao et al.	1998a, b	Saudi Arabia	*Dermatophilosis*

fection has been reported in Argentina, Canada and the USA and sporadic reports have appeared from Europe (Seifert, 1992). Dermatophilosis occurs primarily in cattle, small ruminants, equidae, humans and certain non-domesticated species such as the zebra and red deer. Dermatophilosis is transmitted to man by contact with infected animals (Bucek et al., 1992).

There are distinct genetically determined differences in resistance to the disease in cattle. Hybrid European cattle are extremely susceptible, African zebus less so and N'Dama cattle of West Africa only slightly (Seifert, 1992). The disease is known under different synonyms; streptothricosis, mycotic dermatitis, lumpy wool disease of sheep and strawberry foot-rot of sheep. Dermatophilosis also occurs in OWC and NWC, although there is only one published report dealing with cases in NWC (Thedford and Johnston, 1989).

Etiology and Epidemiology *Dermatophilus* belongs to the order *Actinomycetales*. The mycelial fungi are distinguished by their branching hyphae, subdivided by transverse and longitudinal septae (Gitao et al., 1990). The hyphae produce motile spores (zoospores) that are predominantly released during the rainy season and are transmitted either by direct contact or by vectors (ticks, flies). Supposedly the thorns of the acacia and grain awns are also able to transmit the spores (Wilson, 1984). The hyphae developing from the spores in the epidermis attack the hair sheath. This causes an exudative inflammatory reaction, resulting in a bulging of the slow-growing epidermis away from the corium, thereby allowing growth of a new layer of epidermal cells (Seifert, 1992). Drying of the serous exudate forms a crust that is a distinguishing characteristic of this disease. The crusts can be removed, revealing a wet reddish area that secretes a thick, blood-contaminated exudate (exudative dermatitis) (Losos, 1986).

Dermatophilosis in dromedaries has only recently been reported by Wernery and Ali (1990) in the UAE; by Gitao et al. (1998a, b) and Gitao (1992) in Sudan; by Samuel et al. (1998) in Ethiopia and by Bornstein (1995) in Kenya. The latter studied the morphological and biochemical properties of different strains. A review of the literature in 1976 by Abu Samra et al. found no mention of a natural infection with *Dermatophilus* in the camel, although various authors have reported streptothricosis-like organisms (Table 34).

A non-hemolytic *D. congolensis* strain was recently isolated from dromedaries' skin lesions in the UAE (Joseph et al., 1998). A similar strain was identified from scabs originating from limbs of dromedaries in the UAE suffering from skin necrosis (Fig. 73).

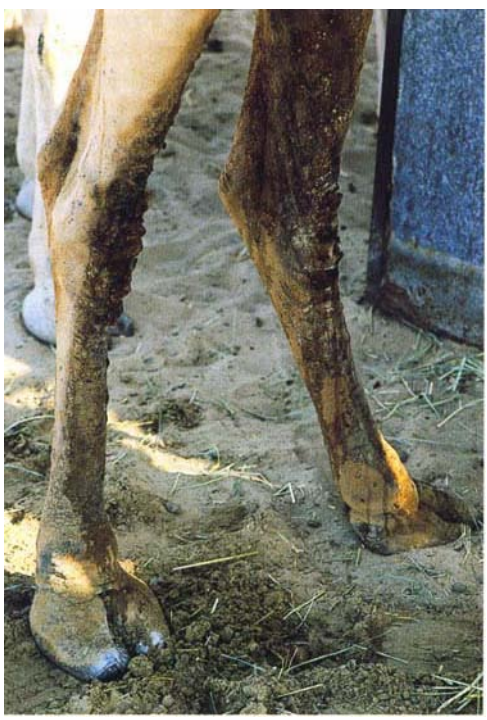

Figure 73 Skin necrosis on the hind leg of a dromedary from which *D. congolensis* was isolated

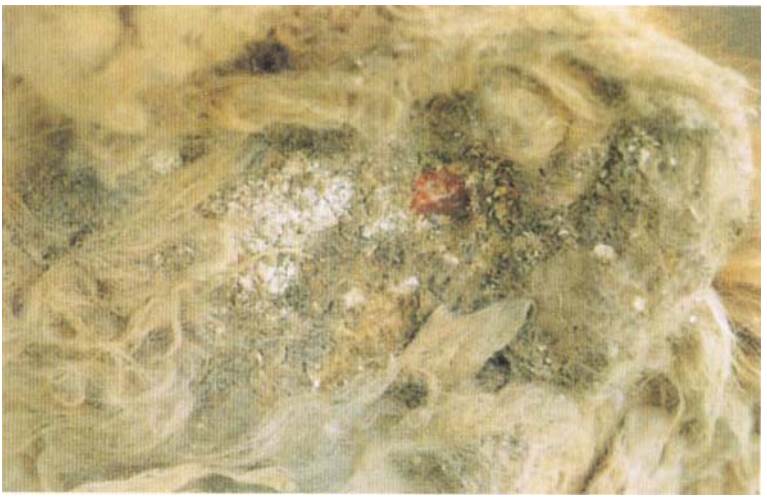

Figure 74 Dermatophilosis in a racing dromedary: the matted hair stands erect. These clinical signs are seen in areas with long hair

From these results it may be assumed that contagious skin necrosis and streptothricosis are identical to dermatophilosis. Abu Samra et al. (1976) was able to prove that the dromedary is susceptible to an experimental infection with *D. congolensis*.

Clinical Signs and Pathology The different manifestations of dermatophilosis in the horse are dependent on the length of the hair and the place of infection (Pascoe, 1990). Dermatophilosis is divided into a winter and summer type. Similar differences in the development of the skin lesions in horses have been described in camels by Gitao et al. (1990) who differentiated between an early or acute form and a chronic form of dermatophilosis. The different forms of the disease have also been seen by the present authors in dromedaries in the UAE. As in the horse, there are distinct differences between infections involving short or long hair. Long hairs in the vicinity of the exudate become matted yielding the characteristic "paint-brush" affect. The matted hair tufts can be easily detached leaving a wettish pink, hyperemic wound surface (Fig. 74). These areas become covered with a suppurative exudate in cases of severe infection. High humidity and the behavior of the female dromedaries during urination leading to chronic wetness of the hindquarters have been implicated in the etiology of skin necroses (Schwartz et al., 1982).

Dermatophilosis of short-haired areas occurring on almost all areas of the body was described by Wernery and Ali (1990). The lesions ranged from nodules to thickened, raised areas covered with thick scabs. Upon removal of the scabs, a raw area with a serosanguinous exudate is exposed (Figs. 75 and 76).

D. congolensis has produced severe cases of wool rot in llamas. Heavy wool cover over the back in high moisture climates predisposes lamoids to this disease. Lesions consist mostly of crusting, particularly over the dorsum of the back (Thedford and Johnson, 1989).

The histological lesions of dermatophilosis were described by Gitao et al. (1998a and b). Congestion and edema of the dermis, degeneration, necrosis and hyperkeratosis of the cells in the epidermis characterize the typical lesions. There is accumulation of exudate on the surface of the skin and infiltration of neutrophils in the dermis and epidermis. *D. congolensis* showing

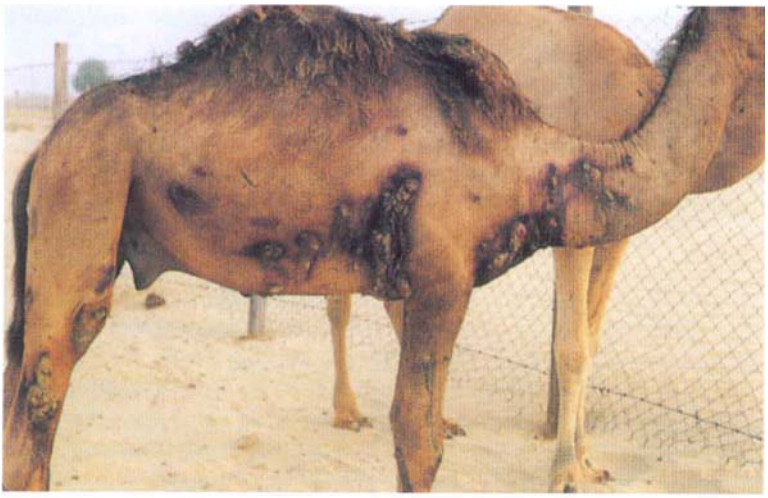

Figure 75 Dermatophilosis in a dromedary bull

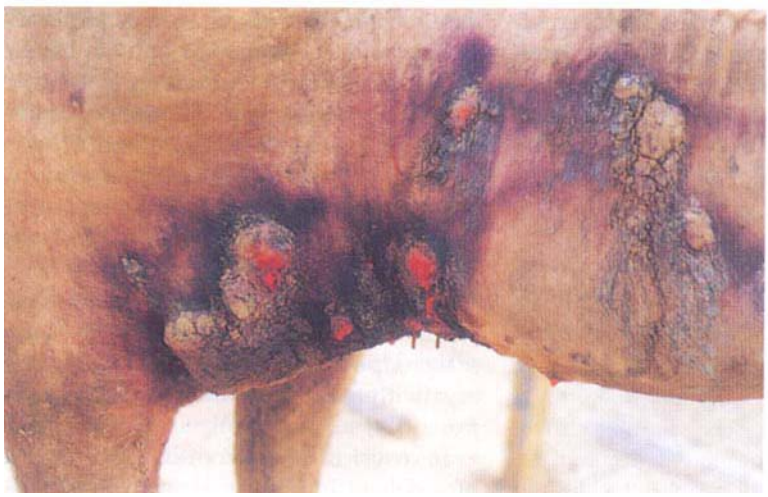

Figure 76 Dermatophilosis in a dromedary bull. Some of the crusts have been removed revealing a raw bleeding area; these lesions are seen in areas with short hair

branching, septated, bacterial filaments or coccoid zoospores are found in the epidermis down to the stratum basale.

Diagnosis The bacterium is comparatively easy to culture and grows well on sheep and ox blood agar. The plates should be incubated at 37°C for up to 5 days in a CO_2 atmosphere. Gram-stained smears of scab material show Gram-positive microorganisms arranged in rouleaux form (Fig. 77).

Gitao (1993b) developed an ELISA for the detection of antibodies against dermatophilosis in camels. The test detected antibodies to dermatophilosis 21 days after the experimental infection with *D. congolensis*. It is planned to use this test in the field.

Treatment and Control Successful treatment of dermatophilosis with terramycin or procaine penicillin and streptomycin has been reported. Infected dromedaries

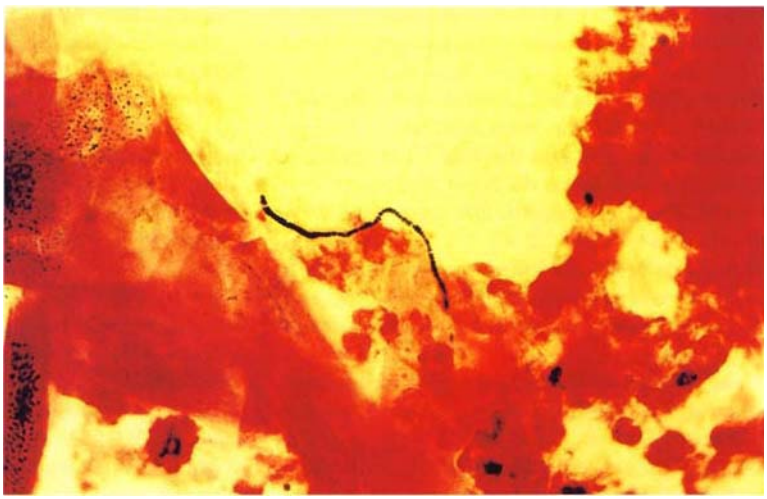

Figure 77 *Dermatophilus congolensis*: smear from underneath a scab of a dromedary. Gram-positive microorganisms arranged in rouleaux form (x1000)

are treated twice with Terramycin LA intravenously. The scabs are removed and the areas cleansed daily with an iodine solution for 7 days. The lesions should be fully healed within 4 weeks. Shearing of badly affected areas with long hair is often an important additional method of further reducing the development of lesions. Isolating clinically affected animals and controlling ectoparasites are methods used to break the infective cycle.

As dermatophilosis is on the rise in camelids, sometimes in connection with dermatophytosis (Gitao et al., 1998a) and because it is a zoonosis, the establishment of a vaccine should be considered.

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1.6 Udder

1.6.1 Infectious Mastitis

In the drought-stricken areas of the world where continuous severe drought decimates cattle, sheep and goat populations, only the camel survives and continues to produce milk. One of the most remarkable features of dehydrated camels is the ability to continue lactation and to secrete milk that is highly diluted with over 90% water content (Yagil and Etzion, 1980a,b). In true ruminants the reservoir for milk-water is lost for cooling and via fecal and urinary excretions. In cattle, sheep and goats, the lack of drinking leads to cessation of lactation or to a very concentrated high fat and low water content milk after a short period. Camel can secrete 20 L of milk daily for at least 10 days without drinking water. Lactating camels will therefore guarantee ample food with the desired content for their offspring and humans alike. However, the let-down of milk must be stimulated by massage or calf suckling. It is of short duration and milking must be as fast as possible. Nomads are aware of this fact and thus milking is carried out on both sides simultaneously. It is estimated that good milk camels can produce 30 to 40 liters of milk daily, which can only be achieved by regular milkings (3 to 4 times daily), rapid milking (milker on each side), and retention of the calves of the best milkers in the herd. The lactation period can last over 2 years.

In India, Pakistan and the Middle East, dromedaries are known to produce well over 20 liters daily with a lactation period lasting 8 to 18 months (Al-Sultan, 1996). Large concentrations of insulin and vitamin C have been found in camel milk. The milk is also unaffected by acid and will virtually pass untouched through the acid environment of the human stomach to the intestines, where it is available for absorption. These and many other features make the camel favorable over other ruminating domesticated animals. However, in many parts of the world the prejudice against the camel family still exists.

Inflammation of the udder occurs less frequently in the camelids than in other domesticated animals (Leese, 1927; Ramadan et al., 1987; Fowler, 1998). This may explain why there are so few publications regarding mastitis in the camel. There might be several reasons why mastitis is more uncommon in camelids than in other domesticated animal species used for milk production. The mammary glands of both OWC and NWC possess four quarters and one teat per quarter. Each teat has two streak canals that enter into separate teat and gland cisterns. Each teat is associated with a non-communicating double gland. The streak canals are very narrow and a 1 mm tomcat catheter is required for penetration. This twin duct anatomy with its narrow streak canals might in some way protect against infection. Milking camels are often fitted with udder covers to restrict suckling. These covers might reduce injuries to the teats and the udder and protect against gross contamination. However, the more likely explanation why udder infections in camelids are less frequent lies in the milk itself. Several scientists have found substances in camel milk that inhibit the growth of pathogenic bacteria (Kosparkov, 1975; Barbour et al., 1984; EL-Agamy et al., 1992; Farah, 1996; EL-Agamy, 1998; Kappeler, 1998). These inhibitors are proteins and have been described as lysozyme, immunoglobulins, lactoferrin and lactoperoxidase, which are already well characterized. These proteins have been shown to have higher concentrations or higher activity in camel milk

than in bovine milk. Kappeler (1998) found a novel minor whey protein, peptidoglycan recognition protein (PGRP), which has a beneficial influence on establishing favorable gut microflora in the newborn and seems to especially inhibit the growth of Gram-positive bacteria.

Etiology ▦ Reports of inflammation of the camel udder have appeared from various countries, such as Egypt (Mostafa et al., 1987), India (Kapur et al., 1982), Saudi Arabia (Barbour et al., 1985; Hafez et al., 1987), Somalia (Arush et al., 1984; Abdurahman et al., 1991), Sudan (Obied, 1983) and the UAE (Quandil and Ouadar, 1984).

Peracute (Kapur et al., 1982), subacute (Quandil and Ouadar, 1984) and gangrenous mastitis with lymph node enlargement (Bolbol, 1982) have been described in the camel. In acute cases, the mammary secretions are watery, yellowish or blood-tinged (Tibary and Anouassi, 1997). Ramadan et al. (1987) reported chronic unilateral mastitis in 3 dromedaries' lactiferous ducts blocked by accumulations of keratin. This obstruction caused a reduction in milk production, enlargement of the affected quarter and, in 2 cases, a secondary bacterial infection with *Pasteurella haemolytica* and *Staphylococcus aureus*. Milk samples from the third dromedary were sterile.

Barbour et al. (1985) examined 205 milk samples from dromedaries in Saudi Arabia using the California mastitis test (CMT). They showed that in the majority of the dromedaries examined, an increase in somatic cells in the milk samples occurred simultaneously with a bacterial mastitis. As in cattle, a correlation between mastitis and the number of somatic cells in the dromedary milk samples was confirmed. A similar observation was made by Abdurahman et al. (1992), Abdurahman et al. (1994) and Abdurahman et al. (1995), who examined 391 udder quarters from Sudanese dromedaries. The 391 milk samples from 101 dromedaries from eastern Sudan were studied to evaluate the value of the CMT, the somatic cell count (SCC) and the adenosine triphosphate (ATP) tests for the detection of subclinical mastitis. It was found that the mean values of all three tests were generally higher for quarters infected with major pathogenic bacteria, although a significant number of quarter milk samples had elevated values from which no pathogenic bacteria were isolated, indicating that subclinical mastitis seems to occur more often than is realized. Bakhiet et al. (1992), who examined milk samples from 49 healthy dromedaries in Sudan, found bacteria in 45% (22/49). *Staphylococcus* spp. and *Streptococcus* spp. were the most frequently isolated udder microorganisms. Guliye (1996), who investigated subclinical mastitis in dromedaries in the Negev desert, found that 81% of 86 milk samples from clinically healthy camels were positive for bacteria, with 40.7% revealing 2 or more bacterial species. *Staphylococcus aureus*, *Micrococcus* spp., *Bacillus* spp., *Streptococcus dysagalactiae* and *E. coli* were the most important organisms isolated. SCC ranged from 1.0×0^5 to 11.8×10^6 cells/mL. Quarter milk samples with bacterial isolates had significantly higher mean SCC values. Quarter samples from which *St. aureus* was isolated showed the highest mean values. Similar results of subclinical mastitis in Bactrian camels were reported from Abdurahman (1996). Of 160 milk samples originating from 7 clinically healthy Bactrians, 22.5% were found to be positive for bacteria. *St. aureus* and coagulase-negative *Staphylococci* were the main organisms found. Quarters from which cocci were isolated had significantly higher SCC and CMT values. Both the SCC and the CMT are of value in predicting the infection status of the camel udder. Mody et al. (1998) investigated the prevalence of mastitis in 146 adult Indian dromedaries using the CMT and cultivation. Thirty subclinical cases of mastitis were

found, of which 28 possessed bacterial pathogens including *Staphylococcus* spp., *Streptococcus* spp. and *Corynebacterium*. The authors also tested the pathogens for their antimicrobial susceptibility and found gentamycin and chloramphenicol highly effective.

Several scientists have studied the properties and products of camel milk (Whabi et al., 1987; Hashi, 1989; Farah and Ruegg, 1991; Farah, 1996).

There are divergent opinions as to which bacteria are potentially the primary causal organisms of infectious mastitis in the camel. Barbour et al. (1985) views *Micrococcus* as an important causative agent of mastitis whereas Obied (1983) did not consider this bacterium pathologically relevant. Obied et al. (1996) found *Streptococcus, Staphylococcus, Micrococcus, Aerobacter* and *E. coli* to be the main bacterial species causing mastitis. The authors did not find any correlation between the SCC and an udder infection. Al-Ani and Al-Shareefi (1997) found that 38% of lactating camels from three different herds in Iraq suffered from mastitis. *St. aureus* and *Corynebacterium pyogenes* were the main causes of chronic mastitis, whereas *St. epidermidis, Streptococcus* spp., *Pasteurella haemolytica, E. coli* and *Micrococcus* spp. were responsible for subclinical mastitis. Very little is known about fungal mastitis. Al-Ani and Al-Shareefi (1997) failed to isolate any fungi from 50 milk samples, but Quandil and Quadar (1984) cultured *Candida albicans* from milk originating from subclinical mastitis samples.

From the multitude of bacteria isolated from milk samples taken from camels with mastitis, *Staphylococcus aureus, Pasteurella haemolytica* and *Streptococci* were found most frequently. Numerous authors believe them to be primary causative organisms in the pathogenesis of mastitis in the camel (Barbour et al., 1985; Ramadan et al., 1987; Hafez et al., 1987). The following bacteria were considered secondary agents:

- *Micrococcus* spp.,
- *Actinomyces* spp.,
- *E. coli,*
- *Pseudomonas aeruginosa,*
- *Klebsiella pneumoniae,*
- *Bacteroides* spp.,
- *C. perfringens.*

These bacteria were isolated as both pure and mixed cultures from milk samples of camels with mastitis (Kapur et al., 1982; Hassanein et al., 1984; Quandil and Ouadar, 1984; Barbour et al., 1985; Mostafa et al., 1987). It is also of great significance that *Brucella* organisms have been isolated from fresh camel milk (Radwan et al., 1992) and cases of human brucellosis have been attributed to the consumption of raw camel milk (Mousa et al., 1988) (see also chapter Brucellosis). In NWC, no specific bacteria that cause mastitis have been reported. Several bacteria species were isolated from peracute NWC mastitis including *E. coli, Klebsiella pneumoniae, Aerobacter enterobacterium*. So far no mycoplasmas have been isolated from NWC (Fowler, 1998) and OWC udders.

Pathology Very little is known about the pathological alterations occurring during infection of the mammary gland. The affected udders are often swollen, hard, reddened and painful to the animal on palpation. In chronic mastitis, necrosis and abscessation might be observed with discharge of greenish pus. Al-Ani and Al-Shareefi (1997) described some of the lesions observed histologically in mastitis.

Treatment When mastitis occurs, prompt attention is necessary to avoid severe damage to the mammary gland or even loss of the animal. Mastitis treatment should be based on culture and sensitivity and the treating person must be fully aware of the anatomical particularities of the camelid's mammary gland. The streak canals can be easily traumatized when using bovine an-

tibiotic mastitis ointments. The nozzles of the bovine infusion tubes are too big. Streak canals should only be penetrated with a 1 mm tomcat catheter to avoid any injuries. Before the infusion is carried out, the teats should be cleaned and disinfected with an alcohol wipe. Before infusion, the udder or the infected quarters should be emptied. In severe cases, the exudate should be removed from the gland three to five times daily by gently massaging the udder. This is sometimes difficult to do when there is a lot of pain. Camelids should be restrained and then rolled on their sides with the hind legs roped back. Commercial mastitis infusions are Ampiclox®, Orbenin LA® and Mastalone®, which should be infused according to the manufacturers' recommendations. It is also important to follow the withholding time of milk after treatment. Peracute and sometimes acute mastitis require parenteral treatment with antibiotics along with local infusion.

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1.7 Nervous System

1.7.1 Tetanus

Almost all mammals are susceptible to tetanus, but there is a wide variation in the susceptibility to the tetanus toxin. Horses are the most sensitive of all species, with the exception of humans. Tetanus in camelids is rare and the degree of susceptibility of OWC and NWC is unknown. Since external wounds are very common and antibodies to tetanus have been detected in dromedaries with no disease, it may be concluded that camelids are quite resistant to tetanus.

Etiology and Epidemiology *Clostridium tetani*, an anaerobe with terminal spheric spores, is found in soil and feces. In most cases, it is introduced into tissues through wounds, particularly deep puncture wounds, which provide a suitable anaerobic environment. The toxemia often occurs in sheep following castration or cropping of the tail (e.g. especially when using rubber bands), leading to great losses. Cattle are resistant to tetanus infections.

Infection with tetanus in camelids occurs via a contaminated wound, and/or frequent puncture wounds due to the long hard thorns of the acacia bush. Small amounts of material contaminated with *C. tetani* spores may be introduced into the puncture channel. The spores multiply in the tissues only under certain conditions, especially when the oxygen partial pressure is reduced in the surrounding tissues. This may occur immediately following introduction of *C. tetani* into the wound if, for example, aerobic bacteria are also introduced simultaneously. *C. tetani* is also able to vegetate for months in the wound until suitable conditions for growth develop. This may be the case when a second trauma occurs to the initial site of infection (Blood and Radostits, 1990). The initial injury may even have long healed. After the oxygen partial pressure of the surrounding tissue falls, the strictly anaerobic *C. tetani* can multiply. *C. tetani* spores can then spread from the site of infection into the blood vessels and lymph system and from there into the liver and spleen (idiopathic tetanus). The highly active neurotoxin is released following multiplication and lysis of the bacteria in the organs and may reach the central nervous system by retrograde axonal transport, producing the typical ascending clinical signs of tetanus. Through massive toxin production following severe infection, the toxin may directly breach the blood-brain barrier, thereby reaching the CNS and then producing the descending clinical signs of tetanus (Seifert, 1992). These relationships are shown schematically in Fig. 78.

Tetanus in camels is considered to be insignificant (Rabagliati, 1920; Curasson, 1947; Mustafa, 1987). Rabagliati (1920) diagnosed only 4 cases of tetanus among 25,000 Egyptian dromedaries over 3.5 years, although the majority of the animals had received external injuries, some of them severe. Ramon and Lemetayer (1934) identified tetanus antibodies in dromedaries that did not exhibit any signs of the disease.

Schwartz and Dioli (1992) described a disease similar to tetanus in their book. Dromedary owners in East Africa call this the "stiff neck syndrome". An acute and chronic form of the disease occurs. The acute form is supposedly very similar to classical tetanus with muscle spasms, neck stiffness and the characteristic disturbances of mastication. Reflex activity is increased and the animals may suffer a tetanic seizure at the slightest provocation, be it noise or physical contact. Dromedaries of any age may develop clinical signs. How-

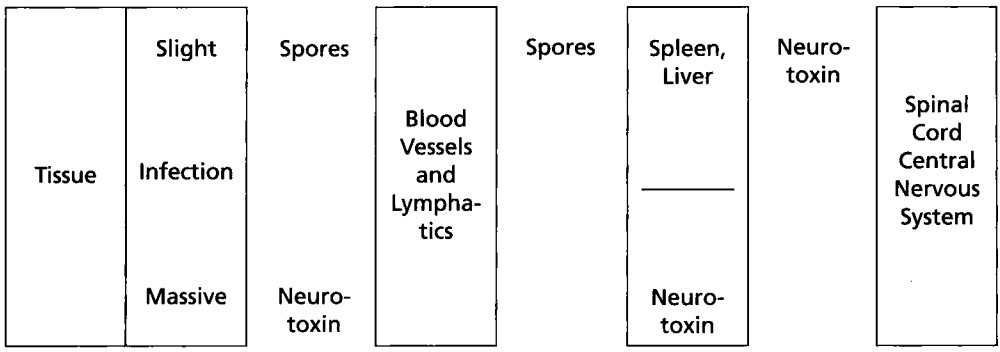

Figure 78 Development of ascending (slight infection) and descending (massive infection) forms of tetanus

ever, only individual animals are affected. Rabagliati (1920) and Morcos (1965) have described similar clinical signs. Both authors observed stiffness of the neck muscles, increased muscle tonus in the entire body, a stiff tail, a drooping nictitating membrane and all four extremities extended sideways (sawhorse).

Two cases of tetanus have been reported in alpacas in Peru (Moro Sommo, 1961 to 1962a), one case of tetanus in a llama in Argentina (Toucedo, 1965), and two cases in llamas in the USA (Keller, 1995; Lopez and Snyder, 1995).

Clinical Signs As clinical signs of tetanus in NWC and OWC are very similar, the following clinical signs describe a racing dromedary suffering from tetanus (tetanus occurs sporadically in racing dromedaries in the UAE). Abu Bakr (1992, personal communication) described a racing dromedary that initially had a deep laceration on the hind foot. The typical jaw spasms, stiff neck, rigid tail and stiff gait developed 14 days later. Other signs included dyspnea, erected ears and fixed stare. The camel was recumbent for 3 weeks while it was treated.

Control and Prevention The UAE dromedary recovered within 3 weeks following intravenous application of 2×100 mL tetanus antitoxin during the first 72 hours, wound debridement, antibiotic treatment and artificial feeding via a gastric tube.

Morcos (1965) treated two dromedaries in Egypt for tetanus with Combelen® and penicillin. Both animals recovered within 12 days. Only one dromedary was given $2 \times 30{,}000$ IU of tetanus antitoxin. The author is of the opinion that the quick recovery of the dromedary was primarily due to the Combelen® therapy.

Specific tetanus antitoxin is available and should be used in valuable animals. The dose is unknown for camelids, but 300,000 IU of tetanus antitoxin in conjunction with tranquilizers or barbiturate sedatives have been effective in the treatment of horses. One llama suffering from tetanus was given the treatment recommended for tetanus in cattle: tetanus antitoxin at a dose of 225 IU/kg body weight (half i.v. and the other half i.m.), antibiotics and chlorpromazine 2.2 mg/kg/6 hours as a tranquilizer (Fowler, 1998). *Clostridium tetani* is susceptible to penicillin and a full dose of this antibiotic should be administered for 7 days. During the acute phase of the disease muscle relaxants might also be used.

All sick animals should be placed in a quiet, darkened box-stall and good nursing is invaluable during the acute period of spasms. If animals are unable to drink or eat, artificial feeding via a gastric tube is recommended.

Tetanus toxoid vaccines are readily available and should be administered before any surgery. Llamas respond to toxoid vaccination with a rise in titer.

1.7.2 Listeriosis

Listeria bacteria are widely distributed in the environment and can be isolated from soil, plants, decaying vegetation and silage with pH of over 5.5. In silage, *Listeria* can multiply and it is commonly implicated in outbreaks of listeriosis in cattle and sheep. *Listeria monocytogenes* can also infect humans via food including soft cheeses, milk and poultry meat and coleslaw. Listeriosis has been reported from NWC but not from OWC.

Etiology *L. monocytogenes* is a medium seized Gram-positive rod, non-spore-forming, measuring about 0.4 to 0.5 µm in diameter. Five serotypes and a number of subtypes have been identified. The bacteria are readily cultured on ordinary media and some strains grow only at 4°C.

Epidemiology Listeriosis has a worldwide distribution. It is, however, more common in regions with cold temperatures and it is often associated with the feeding of poor-quality silage with a pH higher than 5.5. Silage is not fed to OWC and this might be the reason why OWC do not contract listeriosis. The disease occurs most frequently in sheep, goats and cattle. It causes sporadic outbreaks in NWC (Fowler, 1998). The mortality rate can reach 100%. Only individual animals are commonly affected. Haenichen and Wiesner (1995) described two cases of septic listeriosis in 10 day-old alpacas after feeding poor-quality corn silage.

Clinical Signs and Pathology Listeriosis causes a meningoencephalitis in NWC with circling, trembling of the head, running into objects and fever. Some cases develop unilateral facial nerve paralysis in association with drooping lips, ears, eyelids and paralysis of the jaw and pharynx, which interferes with mastication and swallowing. The course of the disease is usually 3 to 5 days (Moro Sommo, 1961 to 1962b; Tapia Cano, 1965; Mayer and Gehring, 1975; Butt et al., 1991). A listeriosis outbreak in a German zoo occurred in 1975 during which six llamas died. Three animals had developed encephalitis from which *L. monocytogenes* was isolated from the brain. The other three llamas died from septic listeriosis from which the organism was isolated from different organs. From this outbreak different serovars were cultured, three of them only through the cold incubation. It was mentioned that heavy rains had flooded the zoo and there was an acute outbreak also in other ungulates. An emergency vaccination with a live *Listeria* vaccine was administered which stopped the further spread of the epizootic (Mayer and Gehring, 1975). Two adult llamas contracted encephalitic listeriosis in New York with abortion, ataxia, depression, and facial paralysis followed by death (Butt et al., 1991). *L. monocytogenes* was isolated from one of the llamas, but both were positive in the immunofluorescent test. Hamir and Moser (1998) described lesions found in a 2-year-old female llama at post mortem. They were confined to the brain and the spinal cord. The surface of the leptomeninges was rough, dark red and thickened with a thin layer of yellowish exudate. Not only meningoencephalitis is caused by *L. monocytogenes*, but also septicemia with polyarthritis (alpaca) (Wisser, 1989), otitis media and interna with suppurative meningoencephalomyelitis (llama) (Van Metre et al., 1991), abortion (llama) (Mc Laughlin et al. 1993) and septicemia with thrombocytopenia and hepatopathy (llama) (Semrad, 1994).

Microscopic changes are confined to the white and/or gray matter of the brain stem, particularly the pons and the medulla ob-

longata. In the medulla oblongata, perivascular infiltrations of mononuclear cells and microabscesses can be detected. Hamir and Moser (1998), however, are of the opinion that the encephalitic form of listeriosis in NWC is not manifested as microabscesses in the brainstem, but as a suppurative meningitis. The authors also observed a multifocal acute necrotizing splenitis and on immunohistochemistry there was a positive immunoperoxidase reaction to *L. monocytogenes*.

Diagnosis Listeriosis can be confirmed by isolation and identification of *L. monocytogenes*. Specimens of choice are brains from animals with CNS involvement, aborted placenta and fetuses. In the septicemic form, the liver and spleen should be cultured. If primary isolation attempts fail, samples should be incubated at 4°C for several weeks and re-cultured weekly. Immunofluorescence and immunohistochemistry (Hamir and Moser, 1998) are two fast methods for the diagnosis of listeriosis. Rabies must always be considered in the differential diagnosis.

Treatment and Control Once nervous signs have developed the prognosis is poor. Penicillin at a dosage 44,000 IU/kg twice daily for 1 or 2 weeks may be tried. A live vaccine has been successfully used in a German zoo.

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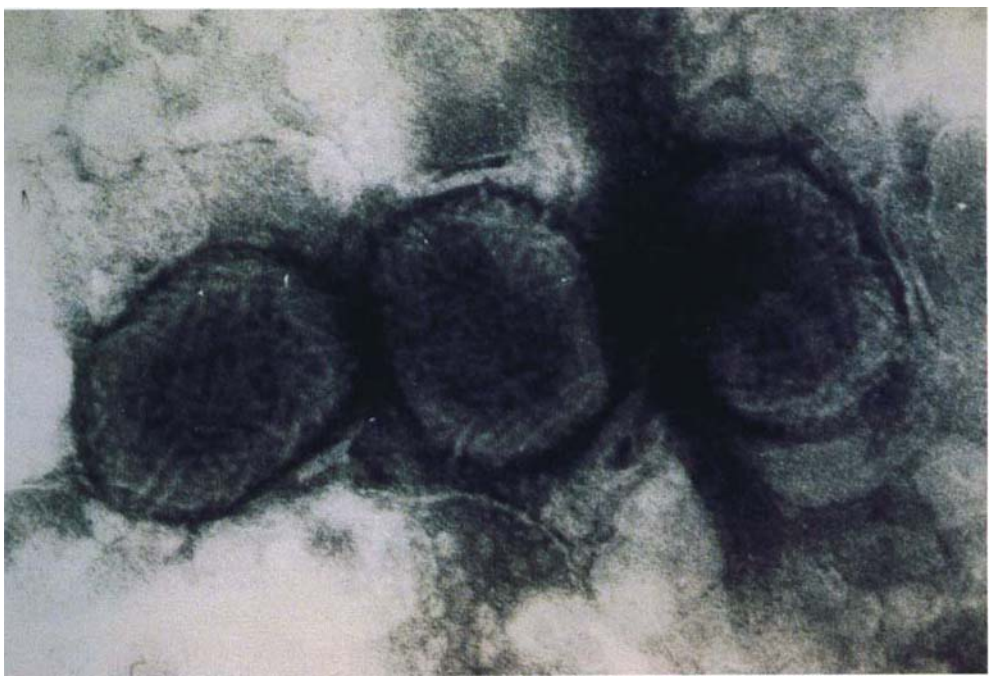
2 Viral Diseases



With the exception of the camelpox complex, a grave lack of information exists regarding viral diseases in camelids. Although all camelid species possess multiple physiological and anatomical similarities, and it is believed that they do not differ in their susceptibility to viruses, comparison of NWC with OWC is important to indicate any possible familial susceptibility. Only a few viruses appear to cause disease in camelids. They include:

- rabies,
- camelpox,
- ecthyma contagiosum,
- papillomatosis,
- influenza,
- rotavirus,
- equine herpesvirus,
- Borna disease.

Although several viral diseases mentioned under the chapter "Nonpathogenic Viral Infections" might cause mild clinical signs in camelids (e.g. foot-and-mouth disease, rinderpest, bluetongue), the authors prefer to keep them under this heading. It is worthwhile to mention that bovine herpesvirus-1 (BHV-1) does not seem to cause diseases in camelids, whereas equine herpesvirus (EHV-1) has been reported as being pathogenic to camelids. Bovine viral diarrhea virus could come under "Viral Infections Causing Disease", but the authors still prefer to keep it in the chapter "Nonpathogenic Viral Infections", as very little is known about its pathogenicity in dromedaries.

A great number of sero-epidemiological virus studies have been performed in the camel. A summary of the viral antibodies and isolates found is given in Table 35.

Table 35 Virus isolation and identification of viral antibodies in camels (except camelpox) – a summary of the literature arranged alphabetically by disease

Disease/Virus	Antigen	Antibody	Prevalence (%)	Country	Author	Year
Adenovirus	–	×	1.3	Nigeria	Olaleye et al.	1989
BAd VIII	–	×	93.0 llamas	USA	Picton	1993
Isolate 7649	×	–	– llamas	USA	Galbreath et al.	1994
	×	×	– llamas, alpacas	USA	Mattson	1994
	–	×	5.13 llamas	Argentina	Puntel et al.	1999
African horse Sickness	–	×	5.0	Egypt	Awad et al.	1981
	×	×	23.2	Sudan	Salama et al.	1986
	–	×	5.6	Egypt	Salama et al.	1986
	×	×	23.0	Sudan	Foreign Animal Report	1988
	–	0	0.0	East Africa	Binepal et al.	1992
	–	×	10.4	Nigeria	Baba et al.	1993
Bluetongue disease	–	×	14.3	Egypt	Hafez and Ozawa	1973
	–	×	5.9	Iran	Afshar and Kayvanfar	1974
	–	×	4.9	Sudan	Eisa	1980
	×	×	5.6–14.6	Sudan	Abu Elzein	1984
	–	×	16.6	Sudan	Abu Elzein	1985
	–	×	13.0	Yemen	Stanley	1990
	–	×	81.0	Botswana	Simpson	1979

Table 35 *(cont.)*

Disease/Virus	Antigen	Antibody	Prevalence (%)	Country	Author	Year
Bluetongue disease	–	×	23.0	Israel	Barzilai	1982
	–	×	67.0	Saudi Arabia	Hafez et al.	1984
	–	×	21.0 alpacas	Peru	Rivera et al.	1987
	–	×	13.0	Yemen	Stainley	1990
	–	×	1.5 llamas	USA	Picton et al.	1993
	–	×	5.0	UAE	CVRL Annual Report	1998
	–	0	0.0 llamas	Argentina	Puntel et al.	1999
	–	×	58.0	Saudi Arabia	Ostrowski	1999
Borna disease	×	–	NWC	Germany (Zoo)	Altmann	1975
	×	–	NWC	Germany (Zoo)	Altmann et al.	1976
	×	–	NWC	Germany (Zoo)	Schueppel et al.	1994
Bovine diarrhea virus	–	×	3.9	Tunisia	Burgemeister et al.	1975
	–	×	6.7	Oman	Hedger et al.	1980
	–	×	15.7	Sudan	Bornstein et al.	1987/88
		×	3.4	Somalia	Bornstein	1988
	–	×	0.0	Djibouti	Bohrmann et al.	1988
	–	×	9.2 breeding) 3.6 racing)	UAE	Wernery and Wernery	1990
	–	×	11.0	Egypt	Hegazy et al.	1993
	–	×	4.3	Egypt	Tantawi et al.	1994
	×	–	–	Egypt	Hegazy et al.	1995/98
	–	×	6.4 breeding) 0.5 racing)	UAE	CVRL Annual Report	1998
	–	×	2.05 llamas	Argentina	Puntel et al.	1999
Bovine herpes mammilitis virus	–	0	0.0	Oman	Hedger et al.	1980
	–	×	4.4 llamas	USA	Picton	1993
	–	×	11.0 alpacas	Peru	Rivera et al.	1997
	–	×	52.5	Egypt	Zaghhana	1998
Bovine herpesvirus (BHV–1)	–	×	5.8	Tunisia	Burgemeister et al.	1975
	–	0	0.0	Oman	Hedger et al.	1980
IBR/IPV	–	0	0.0	Sudan	Bornstein and Musa	1987
	–	0	0.0	Djibouti	Bohrmann et al.	1988
		0	0.0	Somalia	Bornstein	1988
		0	0.0	UAE	Wernery and Wernery	1990
	–	×	5.0 alpacas	Peru	Rivera et al.	1987
	×	–	– llamas	USA	Williams et al.	1991
	–	×	16.7 llamas) 16.2 alpacas)	Peru	Rosadio et al.	1993
	–	–	0.7 llamas	USA	Picton	1993
	×	–	– llamas	USA	Mattson	1994
	–	0	0.0	UAE	CVRL Annual Report	1998
	–	×	0.77	Argentina	Puntel et al.	1999
Malignant catarrhal fever	–	0	0.0	Argentina	Puntel et al.	1999

Table 35 *(cont.)*

Disease/Virus	Antigen	Antibody	Prevalence (%)	Country	Author	Year
Ecthyma	×	0	0.0 alpaca	Peru	Preston Smith	1940/47
contagiosum	×	–	0.0	Kazakhstan	Buchnev et al.	1969
	×	–	–	Russia	Tulepbaev	1969
	×	0	0.0 NWC	South America	Moro	1971
	×	–	–	Mongolia	Dashtseren et al.	1984
	×	–	–	Kenya	Munz et al.	1986
	×	–	–	Somalia	Moallin and Zessin	1988
	×	–	–	Sudan	Ali et al.	1991
	–	×	37.9 sick herds	Kenya	Gitao	1994
	–	×	0–68 healthy herds	Libya	Azwai et al.	1995
	×	–	–	UAE	Wernery et al.	1997
	×	–	–	Saudi Arabia	Abu Elzein et al.	1998
	×	–	– NWC	South America	Fowler	1998
	×	–	–	Libya	Azwai et al.	1998
Equine	×	–	alpacas	USA	Pursell et al.	1979
herpesvirus-1	×	–	llamas	USA	Jenkins	1985
(EHV-1)	×		llamas	USA	Rebhun et al.	1988
	×	–	llamas (exper.)	USA	House et al.	1991
	×	–	Bactrian	USA	Bildfell et al.	1996
	–	0	0.0	UAE	CVRL Annual Report	1998
FMD virus	×	–	–	Afghanistan	Pringle	1880
O,A,C,As1,SA	×	0	0.0	Oman	Hedger et al.	1980
T1	–	×	2.6	Niger	Richard	1986
O,C,SAT2	×	–	–	Egypt	Moussa et al.	1987
O	–	0	0.0 llamas	Argentina	Puntel et al.	1999
Influenza A	×	–	–	Mongolia	Lvov et al.	1982
A	–	×	4.7	Sudan	El-Amin and Kheir	1985
A	–	×	0.6	Nigeria	Olaleye et al.	1989
B	–	×	12.7	Nigeria	Olaleye et al.	1989
Influenza-like	×	–	–	Somalia	SOMAC-SAREC	1982
Influenza	×	–	–	Mongolia	Yamnikova et al.	1993
	×	–	–	Mongolia	Anchlan et al.	1996
	–	0	0.0	UAE	CVRL Annual Report	1998
Papillomatosis	×	–		India	Sadana et al.	1980
	×	–		Somalia	Munz et al.	1990
	×	–		UAE	Wernery and Kaaden	1995
	×	–		UAE	Kinne and Wernery	1998
	×	–		Sudan	Khalafalla et al.	1998
Parainfluenza	–	×	22.3	Nigeria	Nigeria	1989
1	–	×	2.5	Nigeria	Nigeria	1989
2	–	×	18.5	Nigeria	Nigeria	1989
3	–	×	3.8	Egypt	Singh	1967
3	–	×	99.0	Chad	Maurice et al.	1968

Table 35 *(cont.)*

Disease/Virus	Antigen	Antibody	Prevalence (%)	Country	Author	Year
3	–	×	80.8	Tunisia	Burgemeister et al.	1975
3	–	×	66.0	Somalia	Frigeri and Arush	1979
3	–	×	80.0	Oman	Hedger et al.	1980
3	–	×	66.7	Somalia	Arush	1982
3	–	×	37.0	Niger	Richard et al.	1985
3	–	×	81.1	Sudan	Bornstein and Musa	1987
3	–	×	17.3	Djibouti	Bohrmann et al.	1988
3	–	×	81.3	Sudan	Bornstein et al.	1988
3	–	×	42.8	Somalia	Bornstein	1988
Rabies	×	–	alpaca	South America	Moro Sommo	1958/59
	×	–		Mauritania	Bah et al.	1981
	×	–	similar	Somalia	Arush	1982
	×	–		Oman	Ata et al.	1993
	×	–		UAE	Wernery and Kumar	1993
	×	–		UAE	Afzal et al.	1993
	×	–	llama	South America	Miller	1994
	×	–		Niger	Bloch and Diallo	1995
	×	–		Israel	Perl et al.	1996
	×	–		India	Kumar and Jindal	1997
Respiratory syncytial virus	–	×	0.6	Nigeria	Olaleye et al.	1989
Retrovirus	–	0	0.0	India	Chauhan et al.	1986
Bovine leukosis	–	0	0.0 alpacas	Peru	Rivera et al.	1987
	–	0	0.0	UAE	Wernery and Wernery	1990
	–	0	0.0 llamas	USA	Picton	1993
Rift Valley fever	–	×	45.0	Kenya	Scott et al.	1963
	×	–		Egypt	Imam et al.	1978
	×	–		Sudan	Eisa	1981
	–	×		Tunisia	Slama	1984
	–	×	22.0	Kenya	Davies et al.	1985
	–	×	29.0	Mauritania	Saluzzo et al.	1987
	–	×	33.0	Nigeria	Olaleye et al.	1996
Rinderpest	×	–		India	Haji	1932–33
	×	–		Russia	Samartsev and Arbuzov	1940
	×	–		India	Dhillon	1959
	–	0	0.0	Kenya	Scott and MacDonald	1962
	–	–	experimental	Egypt	Singh and Ata	1967
	–	×	9.7	Sudan	Singh and Ata	1967
	–	–	7.7	Chad	Maurice et al.	1967
	–	–	experimental	Kenya	Taylor	1968
	–	×	0.5	Kenya	Wilson et al.	1982
	–	0	0.0	India	Chauhan et al.	1985
	–	x	5.2	Egypt	Abou Zaid	1991

Table 35 *(cont.)*

Disease/Virus	Antigen	Antibody	Prevalence (%)	Country	Author	Year
Rotavirus	–	×	50.0	Morocco	Mahin et al.	1983
	–	×	alpacas	S. America	Rivera et al.	1987
	–	×	87.7 llamas	Argentina	Puntel et al.	1999
	×	–	–	UAE	Mohammed et al.	1998

× = positive; – = not done; 0 = negative

Unusual Arboviruses	Origin	Country	Author	Year
Kadam virus, *Togaviridae, Flavivirus*	camel ticks	Saudi Arabia	Wood et al.	1982
Quaranfil virus	camel ticks	Kuwait, Iraq, Yemen	Converse and Moussa	1982
Akabane virus, *Bunyaviridae*	serology	Arabian Peninsula	Al-Busaidy et al.	1988
Dhori virus	camel ticks	India	Anderson and Casals	1973
Wanowrie virus, Thogoto virus, Dhori virus	camel ticks	Egypt	Williams et al.	1973
Congo hemorrhagic virus	camel ticks	Iran	Saidi et al.	1975
		Russia	Hoogstraal	1979
		UAE	Suleiman et al.	1980
		Iraq	Tantawi et al.	1980
		Egypt	Morrell et al.	1990
		Oman	Scringeour et al.	1996

Serological results have a limited predictive value since they only confirm whether or not the animal has come in contact with a viral agent and has produced antibodies. The results do not indicate whether the exposure has produced manifest disease or how severe the disease response may be. The sero-epidemiological studies have confirmed that the camel produces antibodies against a great number of pathogenic viruses without developing the disease.

At the end of the section on viral diseases we report on "Unusual Arboviruses", which are widespread in the tropics and subtropics. Although many have been isolated from camels and their ticks, their significance to camelids is not yet known. Many can severely affect humans.

2.1 Viral Infections Causing Disease

2.1.1 Rabies

Rabies is a fatal disease for humans and all other warm-blooded vertebrates which is generally transmitted by the bite of a diseased animal. It causes encephalitis. Camelids are susceptible to rabies and the disease has been extensively studied in NWC due to its zoonotic aspect.

Etiology The rabies virus belongs to the family *Rhabdoviridae,* which includes the genus *Lyssavirus* and the genus *Vesiculovirus.* The genus *Lyssavirus* includes the rabies and bovine ephemeral fever serogroups. Within the rabies serogroup, the rabies virus (*lyssavirus* serotype 1) and the rabies-related viruses (Lagos bat, Mokola, Duvenhage = *lyssavirus* serotypes 2, 3, 4) are biologically and antigenically different from seven other viruses of this group which are isolated from birds and hematophagus dipterids in Africa, South America and Australia. *Rhabdoviruses* are rod or bullet-shaped. The genome consists of a single segment of single-stranded RNA and there are five structural proteins. Replication occurs in the cytoplasm of the infected cell and viral proteins accumulate here, constituting the inclusions seen histologically as Negri bodies.

Epidemiology Rabies is an infectious disease transmitted via the saliva of infected animals and is characterized by disturbances of the central nervous system, paralysis and death. The transmission of the rabies virus from animal to animal and to man usually occurs through a bite. Herbivores and man are the final hosts and do not normally play a role as vectors. Carnivores or vampire bats only sustain the cycle of infection.

Warm-blooded mammals as well as birds are susceptible to the rabies virus; however, there are substantial variations in susceptibility to the virus. Foxes, cotton rats and prairie wolves are extremely susceptible; cattle, camels, rabbits and cats are very susceptible; dogs, sheep and goats are less susceptible. Opossums are most probably not susceptible to rabies (Blood and Radostits, 1990).

Although rabies in dromedaries has supposedly been observed in many African and Asian countries (Richard 1980 and 1986), little has been published on this subject. Recent reports of rabies in camels have appeared from Morocco (Chevrier, 1959), Mauritania (Bah et al., 1981), Oman (Ata et al., 1993), the UAE (Wernery and Kumar, 1993; Afzal et al., 1993), Niger (Bloch and Diallo, 1995), India (Kumar and Jindal, 1997) and Israel (Perl et al., 1996). Rabies-like diseases with hindquarter paresis have been reported by Arush (1982) and Somac/Sarec (1982) in Somalian dromedaries.

Little is known about the epidemiological interdependencies of rabies in the camel. Three types of rabies have been differentiated, depending on which animal species serves locally as the main reservoir and vector: the urban form, the sylvatic form and the bat form (paralyssa). The sylvatic form plays the greatest role on the Arabian Peninsula. Rabies is most probably transmitted by the red fox in the UAE and Oman (Wernery and Kumar, 1993; Ata et al., 1993) and by wild dogs in Yemen (Stanley, 1990). It can only be presumed that these animal species are the vectors of rabies on the Arabian Peninsula as there is very little available research.

In Niger, Bloch and Diallo (1995) reported that a rabid dog was responsible for a rabies outbreak in 7 camels in a herd of 40.

In America, several vectors are responsible for the spread of rabies. They include dogs, foxes, raccoons, skunks and bats. However, only dogs were responsible for outbreaks in alpacas in Peru (Moro Sommo, 1958–1959). Transmission of rabies from alpaca to alpaca by bites has also been reported (Franco, 1968).

The incubation period in NWC that had been bitten by dogs was between 15 days and 3 months; affected lamoids died 6 to 8 days after the development of clinical signs. Experimental rabies has also been produced in llamas (Tamayo, 1905) and there are several reports from the United States (Moro Sommo, 1958–1959; Anonymous 1990a, 1990b, 1991; Reid-Sanden et al., 1990; Krebs et al., 1992; Krebs et al., 1993; Miller, 1994; Krebs et al., 1995).

Clinical Signs and Pathology Peck (1966), Mustapha (1980) and Bah et al. (1981) have described two forms of rabies in the dromedary: the "raging fury" and the "silent fury". The latter form is seldom seen in the camel (Leese, 1927; Curasson 1947; Mustapha, 1980). After an incubation period of 3 weeks to 6 months (Higgins, 1986), the following clinical signs are seen in cases of the "raging fury": restlessness, aggression, biting and snapping, itching/scratching together with self-mutilation, hypersalivation and muscle tremor. This excitative state lasts 1 to 3 days in the dromedary and is followed by the paralytic phase. During the paralytic stage, the rabid dromedaries lie on their sides and flail with their limbs. During this stage, which can last one or two days prior to death, the dromedary attempts to yawn continuously (Fig. 79).

The attempted yawning is a typical symptom of rabies in the dromedary (Wernery and Kumar, 1993). Blood and Radostits (1990) consider these motions to be aphonic bellowing. Perl et al. (1996) reported an unusual form of rabies in an 8-year-old dromedary belonging to a herd of 150 camels in Israel. The animal showed the "silent fury" of rabies with weakness, trembling and sternal recumbency. Post mortem examination revealed a mild edema around the spinal cord at L7. Direct fluorescent antibody testing of hippocampus, cerebellum and medulla for rabies was negative, but the mouse inoculation test was positive. The intracerebrally infected mice showed paralysis 12 days after the infection and the brains were positive for rabies in the fluorescence test. An immuno-

Figure 79 Rabid dromedary: the attempted yawning is typical for rabies

Figure 80 Foreign bodies found in compartment 1 in a dromedary with rabies

histochemical investigation of the camel's brain was also negative, but when the lumbosacral to thoracic sections of the spinal cord were tested, rabies virus antigen-containing cells were detected. The authors stress that in rabies-suspicious camels, the spinal cord should be included in the diagnostic procedures.

In NWC, the aggressive form is also usually recorded and seldom the paralytic syndrome (dumb form). The major signs of furious rabies in lamoids are attacks on people, penmates and offspring and self-mutilation. The rabid animals may also bite inanimate objects. Anorexia, salivation, circling, facial paralysis and pharyngeal paralysis characterize paralytic rabies in NWC. It is worthwhile mentioning that lamoids suffering from rabies cannot spit due to the paralysis of the pharynx (Fowler, 1998).

There are no consistent macroscopic lesions in animals that die of rabies. The only visible abnormality is congestion of the leptomeningeal blood vessels. Animals may

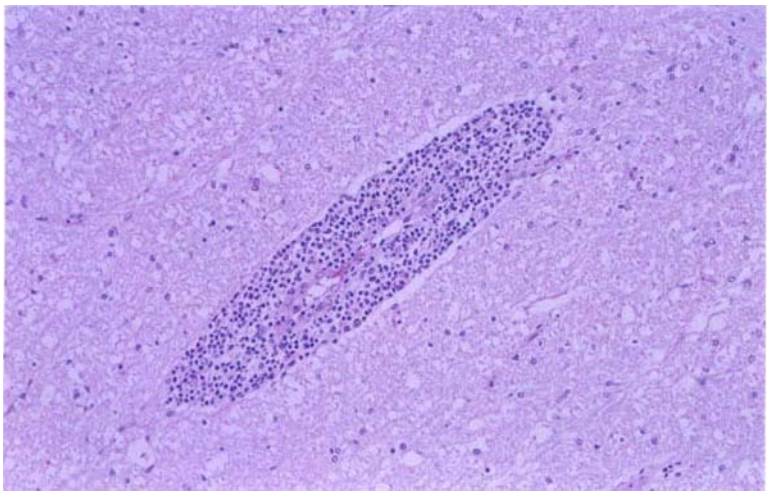


Figure 81 Non-suppurative, non-purulent encephalitis in a dromedary with rabies

be emaciated and there may be self-inflicted wounds or injuries sustained during fights. Foreign bodies such as nails, stones, small batteries, pieces of glass or porcelain have been found in compartment 1 in dromedaries (allotriophagia) (Fig. 80).

The most significant microscopic lesions of rabies are in the central nervous system and cranial and spinal ganglia. The rabies virus causes a nonsuppurative, nonpurulent encephalitis with perivascular cuffing by mononuclear cells (Fig. 81). There is focal and diffuse gliosis, neuronal degeneration and intracytoplasmic inclusions (Negri bodies) in the neurons.

Diagnosis Any abnormal behavior of camelids should be considered suggestive of rabies. Suspicion of rabies is heightened when the affected animal comes from an area where the disease is known to be endemic. Veterinary officials should be notified and they should decide whether to confine the animal and keep it under observation for a period of 14 days, and then, only if it develops overt signs of the disease, euthanize it for laboratory examination.

Animals should be euthanized in such a way as to avoid any damage to the cranium. The hippocampus is commonly used for the diagnosis of rabies, but the distribution of lesions or virus antigen varies and it is recommended that tissue samples be taken from a variety of sites in the brain and spinal cord (Perl et al., 1996). The diagnosis of rabies can only be made on dead camelids.

The standard method of making a diagnosis of rabies is to demonstrate rabies virus antigen in impression smears of fresh brain by immunofluorescence. In all of the rabid dromedaries examined by the authors, massive viroplasms of rabies virus antigen conglomerates of varying sizes were seen immunofluorescently in the brain, particularly Ammon's horn (Fig. 82).

Negri bodies can be demonstrated in impression smears prepared from fresh glycerol-saline preserved brain tissue or histologically in formalin-fixed tissue (Fig. 83).

The third diagnostic method for rabies is the isolation of the virus by intracerebral inoculation of brain suspension into weaned mice. This method is very sensitive and it may take up to 4 weeks or even longer to obtain a result. Isolation of the virus is confirmed by histopathological examination of the mouse brain and by immunofluorescence.

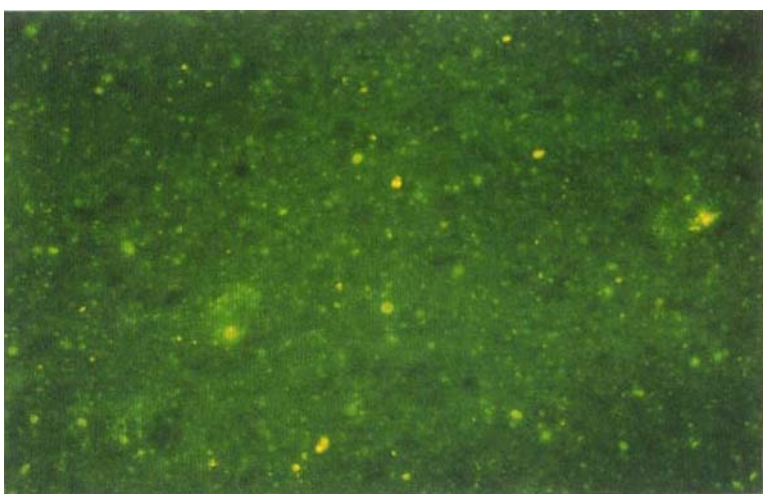


Figure 82 Rabies in the dromedary: masses of virus antigen in the hippocampus (immunofluorescent stain)

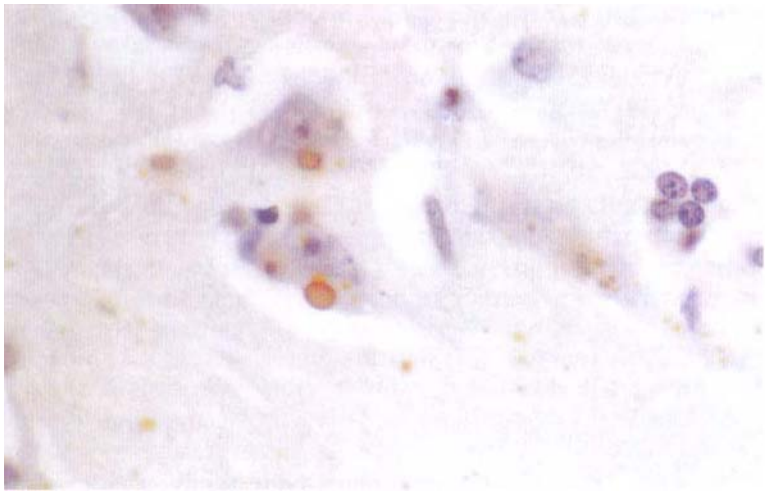


Figure 83 Rabies in the dromedary: Negri bodies in the hippocampus (HE stain, courtesy of Prof. Dahme, Germany)

As mentioned earlier, spinal cord samples should be included in all investigations (Perl et al., 1996). Fowler (1998) stresses that no single test should form the basis of a definitive diagnosis of rabies in NWC. He recommends histological investigations, fluorescent antibody staining and rodent inoculation for the diagnosis of rabies in NWC.

Several serological tests like ELISA may demonstrate antibodies to the rabies virus. These tests are mainly performed to assess a response to vaccine or to identify virus isolates.

Treatment and Control ▦ There is no treatment for rabies infections in animals. Rabies is a viral disease that can be effectively controlled by vaccination. Active immunoprophylaxis is possible with both live attenuated virus vaccines in foxes and vaccines from inactivated virus. All domesticated animals can be given only inactivated vaccines. A range of highly effective, safe and thermostable, inactivated rabies vaccines for animals prepared from virus grown in a variety of primary and permanent cell line cultures are available. A neutralizing antibody produced in response to vaccination is an important factor in protection against rabies infection. It is recommended that antibody titers equivalent to at least 0.5 IU/mL be obtained to protect animals from rabies (Barrat et al., 1992; Sihvonen et al., 1993). The duration of protective immunity to challenge with rabies virus generally varies from 1 to 3 years. Young herbivores should be vaccinated at the age of 4 months and/or 9 months if the dam has been immunized. Boosters should be administered annually. Killed rabies vaccines have been used with success in OWC (Wernery and Kaaden, 1995; Kalanidhi et al., 1998) and in NWC (Fowler, 1998). Some serological results following application of an inactivated aluminum hydroxide vaccine (1.0 mL subcutaneously) to a small herd of dromedaries in the UAE are presented below. Serological tests were performed four times on the dromedaries during a 13 month period. The results are summarized in Table 36.

The response of dromedaries to a single shot of an inactivated rabies vaccine at 14 days post vaccination can be regarded as satisfactory. Seven months post vaccination, however, rabies antibody titers had declined to low levels or disappeared altogether. Similar results are shown by Sihvo-

Table 36 Serological test results (Rapid Focus Fluorescent Inhibition Test, RFFIT)* before and after rabies vaccination with an inactivated aluminum hydroxide vaccine (1.0 mL sc). Titers are given in international units (IU/mL)**. Animals 21 to 25 are controls

Dromedary	24 hours before vaccination	14 days after vaccination	7 months after vaccination	13 months after vaccination
1	< 0.1	18.5	0.5	0.3
2	0.1	9.5	1.5	1.5
3	< 0.1	3.5	< 0.1	< 0.1
4	0.3	18.5	0.5	0.3
5	< 0.1	2.5	< 0.1	< 0.1
6	< 0.1	1.5	0.5	0.5
7	0.2	7.5	< 0.1	< 0.1
8	< 0.1	2.5	0.1	0.1
9	< 0.1	4.5	0.3	0.3
10	< 0.1	4.5	0.1	0.1
11	< 0.1	7.5	0.1	0.1
12	0.1	5.5	0.5	0.5
13	0.1	28.5	< 0.1	< 0.1
14	< 0.1	4.5	< 0.1	< 0.1
15	< 0.1	1.5	< 0.1	< 0.1
16	< 0.1	2.5	< 0.1	< 0.1
17	0.1	3.5	< 0.1	< 0.1
18	0.1	18.5	0.5	0.5
19	< 0.1	5.5	< 0.1	< 0.1
20	0.1	4.5	< 0.1	< 0.1
21	< 0.1	< 0.1	< 0.1	< 0.1
22	< 0.1	< 0.1	< 0.1	< 0.1
23	< 0.1	< 0.1	< 0.1	< 0.1
24	< 0.1	< 0.1	< 0.1	< 0.1
25	< 0.1	< 0.1	< 0.1	< 0.1

* Performed by Rhone Mérieux, Lyon, France and Federal Research Institute for Animal Virus Diseases, Tübingen, Germany

** Titers higher than 0.5 IU/mL are considered protective against rabies in cattle (Barrat et al., 1992).

nen et al. (1993) in reindeers. The data shows that one dose (1 mL) of inactivated rabies vaccine induces good, but short-term serological conversion in dromedary camels. Therefore, a booster dose of vaccine is necessary 6 to 8 months after primary vaccination to guarantee sufficient protection against rabies. The duration of the immunological response to vaccination was quite different in dromedaries from India. The authors showed that an inactivated tissue culture rabies vaccine induced a much longer lasting immunity. Kalanidhi et al. (1998) presume that the reason for this discrepancy may lie in the difference of camel breeds used in the study, or in the individual animal's response to the vaccine.

Fowler (1998) recommends administering only killed rabies vaccines (also to NWC) as a modified live virus vaccine (MLV) given to 290 alpacas following an outbreak of rabies caused postvaccinal paralysis in 10% of the vaccinees within 14 to 30 days. Killed rabies vaccines administered to llamas produced titers that are considered protective in other species. Llamas have contracted rabies in a num-

ber of different areas in South America and should therefore be vaccinated annually.

Among the rabies-related viruses, Duvenhage is antigenically closest to *lyssavirus* serotype 1 and rabies vaccines afford the greatest protection against this virus, and least protection against Mokola virus. Rabies viruses isolated from camels in the UAE were indistinguishable from the *lyssavirus* serotypes. It would be interesting to determine if camelid rabies viruses from different countries share the same antigenic structure, especially those inducing the "silent fury".

2.1.2 Borna Disease

Borna disease (BD) is a progressive viral polioencephalomyelitis predominantly affecting horses and donkeys (rarely other *Equidae*), sheep and a variety of other animal species. The disease is restricted to localities in Central Europe. BD was diagnosed in NWC in Germany (Altmann, 1975; Altmann et al., 1976; Schueppel et al., 1994).

Etiology The viral etiology of BD has been known since 1927. Recently, Borna disease virus (BDV) was shown to be an enveloped virus containing a single-stranded RNA of negative polarity. The virus replication occurs in the nucleus of infected cells. Although the virus shares some physicochemical and physical properties with members of the order *Mononegavirales*, it was classified by the International Committee on Taxonomy of Viruses as a member of the newly established family *Bornaviridae*, genus *Bornavirus*. All virus isolates seem to be antigenically identical but there are obvious differences in the degree of virulence. Under natural conditions, the host range of the virus includes horses, camels, sheep, cattle, dogs, cats, and also very likely humans.

Epidemiology Many animal species and different cell cultures can be infected experimentally with BDV. However, the mode of transmission is still unknown. Since the virus has been detected in nasal secretions, saliva and urine, it might be possible that the infection occurs by direct or indirect contact. BDV-specific antibodies have recently been shown in sera and cerebrospinal fluid from human patients suffering from psychiatric disorders.

Clinical Signs and Pathology In two German zoos, llamas and alpacas that were affected by BD exhibited anorexia and severe weight loss at the beginning of the outbreak. The animals later died as a direct result of the disease. The lamoids affected did not develop any neurological signs. Diagnosis of BD was confirmed by histopathological investigations. Four alpacas revealed a non-suppurative meningoencephalitis. In two of these four animals, Schueppel et al. (1994) also confirmed the disease by immunohistochemistry. Positive labeling for BDV was observed in the nuclei of ganglion cells of the hippocampus, Gyrus dentatus and Corpus striatum in the vicinity of inflammatory infiltrates (Fig. 84).

Furthermore, intranuclear inclusion bodies (Joest-Degen bodies) were detected in the hippocampus typical of a BDV infection.

Diagnosis BDV can be isolated from homogenates of infected brain or cerebrospinal fluid by infection of embryonic rabbit or rat brain cell cultures or by intracerebral inoculation of rabbits. Viral antigen might also be detected by immunohistochemical methods. Intranuclear Joest-Degen bodies, if present in neurons, are also useful for a diagnosis of BD.

Diagnosis of BD can also be confirmed by serological methods using indirect immunofluorescence in infected cell cultures.

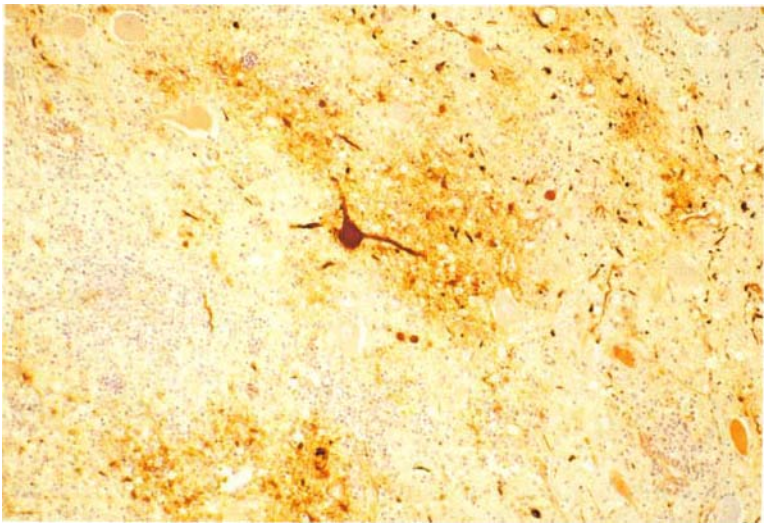


Figure 84 Positive labeling for BDV of the hippocampus in an alpaca

Treatment and Prevention BD is a reportable disease and controlled by a stamping-out policy.

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2.1.3 Camelpox

Camelpox occurs in the dromedary and the Bactrian camel and has also been experimentally induced in NWC (Kinne and Wernery, 1999; Wernery et al., 2000). The camelpox virus causes a proliferative skin disease that primarily affects younger animals (Rohrer, 1970; Richard, 1979, 1980; Mahnel and Munz, 1987; Schwartz and Dioli, 1992). Pox-like lesions in camelids may also be induced by a yet-unnamed parapoxvirus and papillomavirus.

Etiology Poxviruses are classified in the family *Poxviridae,* which are divided into two subfamilies: *Chordopoxvirinae,* which infects vertebrates and *Entomopoxvirinae,* which are found in insects. Camelpox virus (CaPV) is a large, enveloped, doublestranded DNA virus that represents 1 of 11 species currently assigned to the genus *Orthopoxvirus.* Poxviruses are the largest and most complex viruses and have a brick-shaped appearance. The infective agent of camelpox is the *Orthopoxvirus cameli.*

Epidemiology Camelids may become infected with the poxvirus through small abrasions of the skin, by aerosol infection of the respiratory tract or mechanical transmission by biting arthropods. Several scientists have reported an increase in camelpox outbreaks during wet seasons (e.g. Munz, 1992; Wernery et al., 1997a and b) when the disease becomes more severe. During the dry season, it usually follows a milder course (Pfahler and Munz, 1989). Since the camelpox virus has been isolated from the camel tick *Hyalomma dromedarii,* it is now believed that a larger arthropod population builds up during rainy seasons, forcing a greater virus pressure and virus doses onto the camel populations. Differences in the virulence of camelpox strains have also been suggested (Munz, 1992; Otterbein, 1994; Otterbein et al., 1995; Pfeffer et al., 1996; Munz et al., 1997; Wernery and Zachariah, 1999), which may explain the phenomenon that some strains produce generalized pox infections and others only a localized form (Wernery, 1994). DNA restriction enzyme analyses have shown that camelpox virus strains from different African countries possess different genomes, which may explain why virus strains differ in their virulence (Munz, 1992); an important factor in the production of vaccines and in performing test exposure experiments (Baxby et al., 1975).

Animals that have recovered from infection appear to develop a lifelong immunity. Epidemics occur in regular cycles dependent on the rainy season and relationship of the density of the insect population to the number of immune camels in the population.

Camelpox is most probably not a zoonosis, although clinical observations in various articles have reported the possibility of transmission of *Orthopoxvirus cameli* to humans. Even very recent reports of skin eruptions in camel herdsmen could not identify camelpox as the causative agent using current laboratory methods (Kriz, 1982; Jezek et al., 1983; Wernery and Kaaden, 1995).

Pox is the most frequent infectious viral disease of the camel and therefore the most widely reported. The disease occurs wherever camel husbandry is practiced (Table 37). An exception is the Australian dromedary population where, so far, camelpox has not been observed (Doerges and Heucke, 1992, personal communication; Hafez et al., 1992).

In both localized and systemic poxvirus diseases, initial multiplication of the virus occurs at the site of entry. In those infections characterized by systemic disease, further viral multiplication in the draining lymph nodes is followed by a primary viremia and multiplication of virus in organs and tissues. This results in a secondary viremia and subsequent infection of the skin.

Serological studies in different countries have revealed a high prevalence of CaPV. Davies et al. (1985) showed, using the SNT, that there is a high prevalence of antibodies to camelpox in herds kept by nomadic pastoralists and by ranchers. Antibodies were found in five out of six camel herds in Kenya using SNT, although there was no clinical disease seen in the herds investigated. Munz et al. (1986) reported 95% positive cases in Sudan, which was confirmed in 72.5% by Khalafalla et al. (1998). Pfeffer et al. (1998) found a prevalence between 88% and 100% in 1,000 dromedaries

Table 37 Outbreaks of camelpox, arranged by country and author

Country	Author	Year
Afghanistan	Odend'Hal	1983
Bahrain	Higgins et al.	1992
Egypt	Tantawi et al.	1974
	Tantawi	1974
	Tantawi et al.	1978
Ethiopia	Shommein and Osman	1987
India	Leese	1909
	Cross	1917
	Chauhan et al.	1985
	Chauhan et al.	1986
	Chauhan and Kaushik	1987
	Khanna et al.	1996
Iran	Baxby	1972
	Ramyar and Hessami	1972
Iraq	Al Falluji et al.	1979
Kenya	Davies et al.	1975
	Schwartz et al.	1982
	Wilson et al.	1982
	Kropp	1985
	Gitao	1997
Libya	Carter and Azwai	1996
Mauritania	Wardeh	1989
Morocco	Fassi-Fehri	1987
	El-Harrak et al.	1991
Niger	Richard	1986
	Ba-Vy et al.	1989
Oman	Shommein and Osman	1987
Pakistan	Odend'Hal	1983
	Ghulam et al.	1998
	Al-Hendi et al.	1994
Russia	Vedernikov	1893
	Vedernikov	1902
	Amanzhulov et al.	1930
	Bauman	1930
	Ivanov	1934
	Sarmatsev and Praksein	1950
	Vyshelesskii	1954
	Likhachev	1963
	Borisovich and Orekhov	1966
	Buchnev and Sadykov	1967
	Semushkin	1968
	Vedernikov	1969
	Borisovich	1973
	Marennikova et al.	1974
	Buchnev et al.	1987
Saudi Arabia	Hafez et al.	1986
	Hussein et al.	1987
	Hafez et al.	1992
Somalia	Kriz	1982
	Arush	1982
Sudan	Shommein and Osman	1987
	Khalafalla and Mohammed	1996
	Khalafalla et al.	1998
UAE	Kaaden et al.	1992
	Wernery et al.	1997a/b
Yemen	Odend'Hal	1983

tested with the ELISA in the UAE. In Libya, Azwai et al. (1996) investigated 520 dromedaries from 6 different herds and found only 10% positive animals. Serological investigations are of little value for the evaluation of the immune status of camel populations since it is known that in orthopox infections, the cell-mediated immunity seems to protect animals from disease rather than circulating antibodies (Fenner et al., 1988).

Diagnosis Various authors have concerned themselves with the characterization and systematization of the camelpox virus (e.g. Roslyakov, 1972; Mahnel and Bartenbach, 1973; Bartenbach, 1973; Mahnel, 1974; Guenther, 1990; Munz 1992; Binns et al., 1992; Renner-Mueller et al., 1995; Chandra et al., 1998). The results have shown that the camelpox virus is a typical representative of the genus *Orthopoxvirus*, family *Poxviridae*, based on morphological, chemical, physical and biological characteristics. The virus is closely related immunologically to other representatives of this group such as, for example, the *vaccinia/variola* virus subgroup of *poxviruses*. The systematization and laboratory differentiation was of great importance in demarcating the *orthopoxvirus* from the *para-*

poxvirus, as both viruses can be found in the same camel (Wernery and Kaaden, 1995) (Fig. 85).

Utilizing a relatively simple set pattern, laboratory methods also permit the differentiation of other closely related *orthopoxvirus* species (Baxby, 1974; Mahnel, 1974). Some criteria used to differentiate between viral species include the inoculation of embryonated eggs, cytopathic effects in cell cultures (Bedson, 1972), the intracutaneous test in rabbits and the feather follicle test in chickens. Newer methods include the ELISA technique with monoclonal antibodies, DNA restriction enzyme analysis (Munz et al., 1986; Munz et al., 1992) and a dot blot assay using digoxigenin-labeled DNA probes (Meyer et al., 1993). Czerny et al. (1989), Johann and Czerny (1993) and Pfeffer et al. (1998) have described various laboratory methods for the diagnosis of camelpox. They include electron microscopy, ELISA, immunohistochemistry and polymerase chain reaction. CaPV-antigen detection by immunohistochemistry is a new method for camelpox diagnosis which can easily be performed in laboratories not possessing an electron microscope (Fig. 86). In addition to the diagnosis, immunohistochemistry is of particular interest for histopathologists because it visualizes the morphological changes induced by the poxvirus. Another advantage of this method is that embedded tissue blocks can be inves-

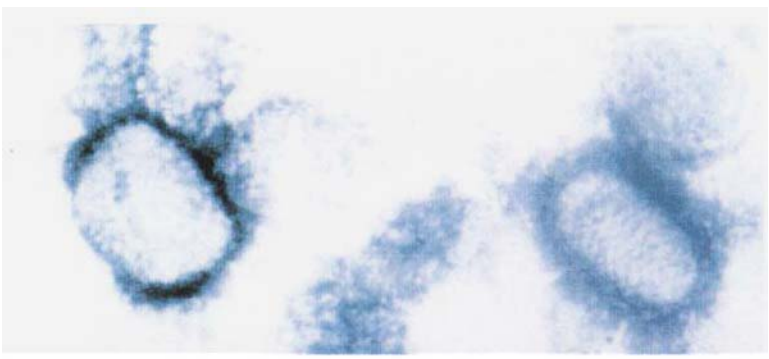


Figure 85 Electron microscopy of camelpox (left) and parapox (right) in a dromedary (courtesy of Prof. Mahnel, Germany)

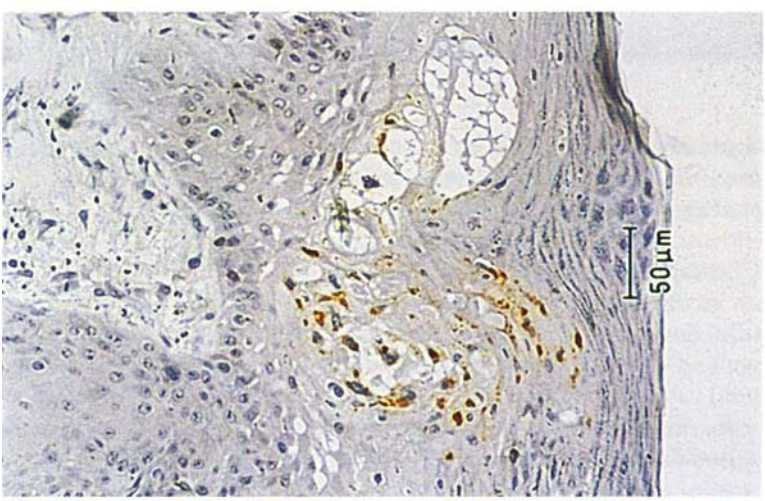


Figure 86 Acute lesions of camelpox within the dermis. Positive staining of CaPV-antigen (golden-brown granula) is found in macrophages, fibrocytes and endothelial cells

Figure 87 Camelpox on nasal mucosa

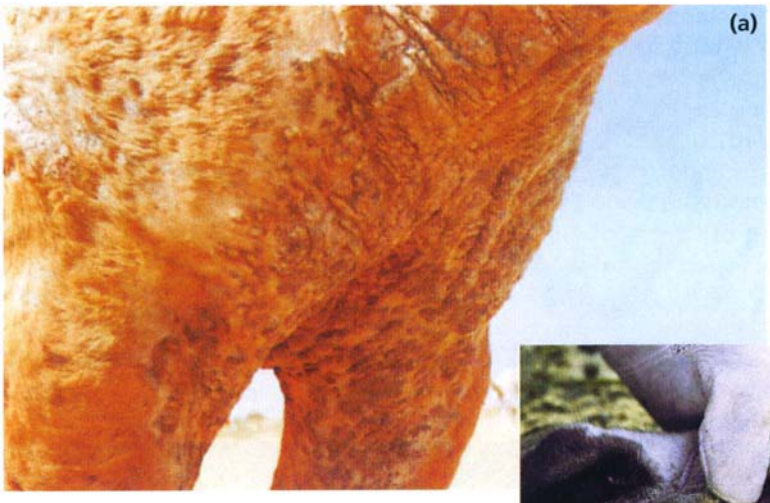


Figure 88a, b Generalized camelpox in a dromedary and a guanaco after experimental infection with *Orthopoxvirus cameli*

tigated years after they have been made, thus making it suitable for retrospective studies (Kinne et al., 1998).

Clinical Signs and Pathology Following an incubation period of 9 to 13 days, pustules develop on the nostrils and eyelids as well as on the oral and nasal mucosa in mild cases (Fig. 87).

In more severe cases presenting with generalized clinical signs such as fever, lassitude, diarrhea and anorexia, the eruptions are distributed over the entire body (Fig. 88a and b).

Buchnev and Sadykov (1967) have described abortions in camels caused by the *Orthopoxvirus cameli* and they have isolat-

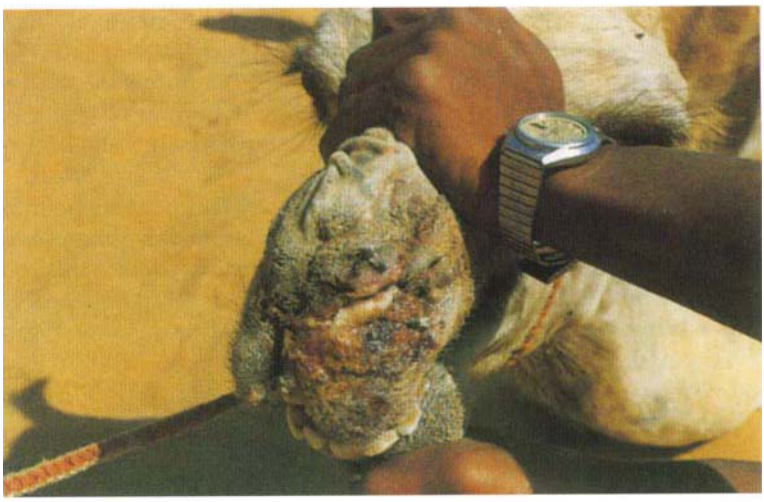


Figure 89 Camelpox with secondary *Staphylococcus aureus* infection

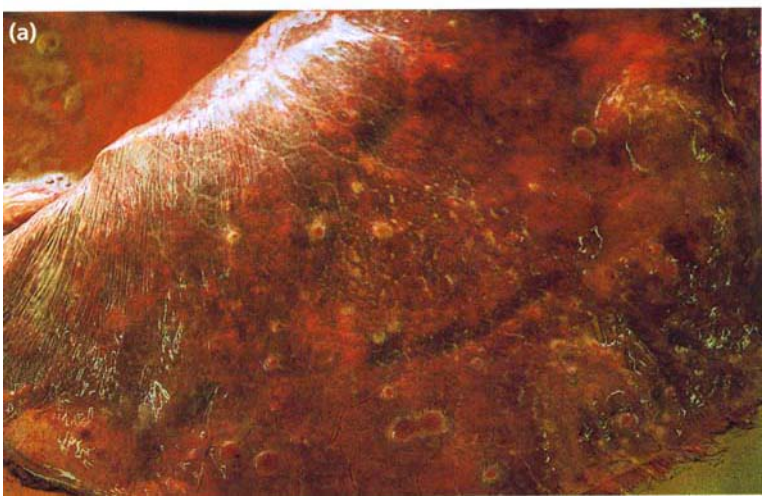
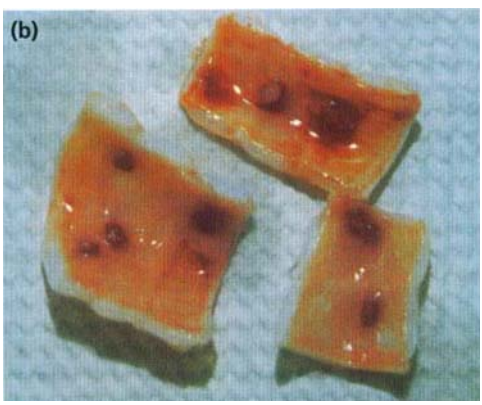


Figure 90a, b Camelpox lesions in the trachea and lung of a 9-month-old dromedary



ed the virus from the aborted fetuses. Mortality can reach 28% in generalized forms of the disease (Jezek et al., 1983). Secondary bacterial and mycotic infections can complicate the course of camelpox (Fig. 89).

Pox-lesions were also observed in the trachea and lungs of young dromedaries (Fig. 90a and b) (Wernery and Kaaden, 1995; Kinne et al., 1998).

Classical lesions in the skin start as erythematous macules, which develop into papules and vesicles. Vesicles develop into

pustules with depressed centers and raised erythematous borders, the so-called pock. After the pustules have ruptured, they become covered by crusts. Healing of pustules might take 4 to 6 weeks with or without scars. *Poxviruses* are generally epitheliotropic and the skin lesions are characterized by swelling, vacuolation and ballooning of keratinocytes, particularly in the stratum spinosum. Rupture of these cells leads to the formation of vesicles. Marked hyperplasia of epithelial cells surrounding pustules contributes to the raised borders of pustules. Perivascular mononuclear cell infiltrations, neutrophils and eosinophils are often observed in the dermis as well as an edema. Kinne et al. (1998) described camelpox lesions of the respiratory system in dromedaries. The disease caused scattered focal lesions in the trachea, esophagus and lungs. The pulmonary lesions, consisting of sparse foci of pulmonary consolidation, varied in diameter from 1 to 10 mm. HE-stained lung sections revealed confluent foci of proliferated alveolitis and bronchiolitis in which the normal architecture had been partly or completely obliterated with necrosis and fibrosis (Fig. 91).

Immunohistochemical examination of these foci showed numerous *poxvirus* antigen-positive cells in the bronchial epithelia (Fig. 92).

Immunohistochemistry technique was also applied for pox lesions of the skin (Nothelfer et al., 1995; Pfeffer et al., 1998).

Treatment and Control There is no treatment for camelpox infections; in order to minimize secondary infections it is advisable to treat severe cases by local application or parenteral administration of broad spectrum antibiotics and vitamins.

Although camelpox has a great economic significance, only a few scientists have concerned themselves with the production of a specific vaccine. Camel owners recognizing the importance of camelpox have created numerous names for this disease. Even today, these owners protect their calves by dissolving scabs from affected animals in milk and rubbing the mixture on the calves' scarified lips (Leese, 1909; Higgins, 1986).

Reports of the existence of a camelpox vaccine first originated in the Soviet Union (Samartsev and Praksein, 1950; Buchnev and Sadykov, 1967; Sedov, 1973; Borisovich, 1973). However, the details regarding the virus strain and the safety and effectiveness of the vaccine are insufficient.

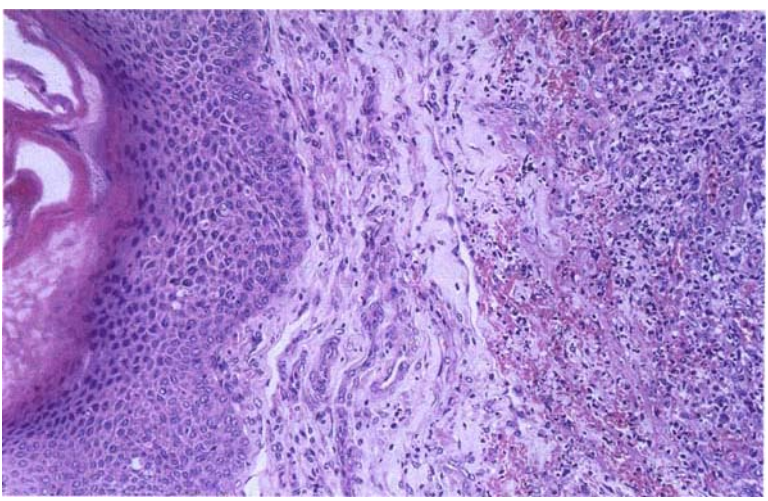


Figure 91 A consolidated focus consisting of a mixture of tissues including some residual alveoli, fibrous tissue and mature collagen. Note the infiltrating mononuclear cells and the cytoplasmic and nuclear debris (HE x220)

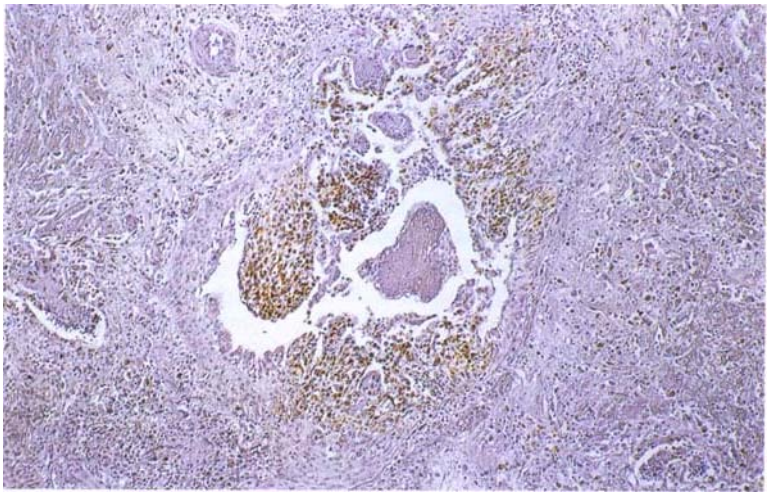


Figure 92 Proliferated and desquamated bronchial epithelium with numerous cells showing labeling for *poxvirus* antigen. Note the intraluminal necrotic mass (ABC method, x120)

Buchnev and Sadykov (1967) immunized camels with an aluminum hydroxide vaccine, but this vaccine did not protect camels from a camelpox infection. Mayr (1999) states that inactivated pox vaccines do not possess a protective efficacy against any *poxvirus* infection. Because of the inability of the vaccine virus to multiply in the host, not enough specific pox antibodies can be produced. A pox vaccine can only be protective when the vaccine titer is greater than $10^{7.0}$ $TCID_{50}$ and if the animals are revaccinated after 3 to 5 weeks. Newer reports of attempts at producing a vaccine come from Morocco (EL-Harrak et al., 1991; EL-Harrak, 1998), Saudi Arabia (Hafez et al., 1992) and the UAE (Kaaden et al., 1992; Wernery and Kaaden, 1995; Wernery et al., 1997a; Wernery and Zachariah 1999; Wernery et al., 2000). All three groups were successful in producing a camelpox vaccine. An inactivated vaccine was developed in Morocco and has been used in prophylactic campaigns since 1991. This vaccine has to be administered annually. In further developments, several clones were selected and clone A28 is now used because of its safety and good immunity (El-Harrak et al., 1991; El-Harrak, 1998). Attenuated virus strains were employed in Saudi Arabia and in the UAE. The UAE group established a permanent fetal dromedary skin cell line (Dubca) for the isolation of the camelpox virus (Kaaden et al., 1992; Klopries, 1993; Kaaden et al., 1995). The UAE attenuated camelpox vaccine (called Ducapox® = Dubai camelpox vaccine) has been used since 1994 with success (Wernery, 1994). Ducapox® is commercially produced in South Africa. It also protects NWC against camelpox (Wernery et al., 2000). In a recent experiment, Wernery and Zachariah (1999) showed that a single dose of Ducapox® given at the age of 12 months can protect dromedaries from camelpox infection for 6 years and even longer. However, the authors have stressed that only a small number of camels were used in this long term experiment. It is important to mention that the vaccine producer recommends a booster dose in 6 to 9-month-old camels to avoid any vaccine breakdown because of maternal antibodies. Because of the antigenic relationship between the camelpox virus and the vaccinia virus, it is possible to immunize camels with known *vaccinia* strains. Higgins et al. (1992) brought an outbreak of camelpox in Bahrain under control with the Lister strain, and Baxby et al. (1975) were able to

show that dromedaries in Iran, vaccinated with the *vaccinia* strain EA8, were able to withstand test exposure to camelpox. It should be mentioned, however, that the vaccination program against human *variola* using *vaccinia* virus was terminated worldwide by a recommendation implemented by the WHO in Geneva, Switzerland.

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2.1.4 Contagious Ecthyma

Parapoxviruses are not related to *orthopoxviruses* and there is no crossimmunity as such between the two viral species. Contagious ecthyma (ORF) causes a localized, vesiculo-pustular exanthema with a worldwide distribution. It is a disease in sheep, goats and wild ruminants (Buettner et al., 1995). In sheep, it is regarded as one of the most serious viral diseases.

Etiology Contagious ecthyma virus is a member of the genus *Parapoxviridae*. The

current members of the genus *Parapoxvirus* are:
- *Parapoxvirus* ovis (ORFV),
- bovine papular stomatitis virus (BPSV),
- pseudocowpoxvirus (PCPV),
- parapoxvirus of red deer in New Zealand (PVNZ).

Separation of the *parapoxviruses* into four distinct groups has been based on natural host range, pathology and more recently on restriction endonuclease and DNA/DNA hybridization analysis. The latter studies have shown that the *parapoxviruses* share extensive homology between central regions of their genomes, but much lower levels of relatedness within the genome termini (Mercer et al., 1997).

Parapoxvirus ovis (ORFV), the causative agent of contagious ecthyma in sheep and goats, has also been described in dogs, OWC, NWC and seals (Hartung, 1980). All three *parapoxviruses*, as anthropozoonoses, can also be transmitted to humans (Liess, 1962; Hartung, 1980; Hartmann et al., 1985; Mahnel and Munz, 1987; Mercer et al., 1997). Only the recently reported PVNZ has yet to be recorded as infecting humans.

Epidemiology The ORFV can cause a disease in OWC and NWC (Ali et al., 1991; Gitao, 1994; Wernery and Kaaden, 1995; Fowler, 1998).

Contagious ecthyma in the camel is very difficult to differentiate clinically from true camelpox. Contagious pustular dermatitis or "scabby mouth", as contagious ecthyma is also called, has been described in OWC from many different countries. In Kazakhstan this disease is called "Auzdyk" in the Bactrian camel, and has been intensively studied by Tulepbaev (1969 and 1971). The opinion that the disease is not contagious and due to the consumption of thorny plants has been a long held belief among camel owners (Borisovich and Orekhov, 1966). Later it was realized that the thorny plants damaged the lips, allowing transmission of the *parapoxvirus* (Buchnev et al., 1987). Roslyakov (1972) showed, using electron microscopic studies, that the ultrastructure of this *parapoxvirus* is similar to the virus found in contagious ecthyma and named the latter virus *"Dermovirus cameli"* and the disease "pustular dermatitis of the camel". Kokhoo (1982) also studied the biological characteristics of this virus.

Other reports of outbreaks of contagious ecthyma in OWC have originated from Russia (Buchnev et al., 1969), Mongolia (Dashtseren et al., 1984), Kenya (Munz et al., 1986; Dioli and Stimmelmayr, 1992; Gitao, 1994); Somalia (Kriz, 1982; Moallin and Zessin, 1988); the Sudan (Ali et al., 1991; Khalafalla, 1998); Libya (Azwai et al., 1995; Azwai et al., 1998); UAE (Wernery et al., 1997) and Saudi Arabia (Abu Elzein et al., 1998).

Camel contagious ecthyma occurs mainly in young animals up to 3 years of age. Several scientists from different countries have recorded the morbidity and mortality rates (Table 38), which differ extremely.

Table 38 The morbidity and mortality rates of dromedaries suffering from contagious ecthyma

Author(s)	Year	Country	Camels examined	Age in months	% Morbidity	% Mortality
Dashtseren et al.	1984	Mongolia	478	Adult	10–80	0
Munz et al.	1986	Somalia	–	Adult	100 / 10–20	0
Ali et al.	1991	Sudan	700	14	6	0
Gitao	1994	Kenya	600	8	100	0
Wernery et al.	1997	UAE	30	16	20	0
Abu Elzein et al.	1998	Saudi Arabia	700	Young and adults	24	0
Khalafalla	1998	Sudan	–	12	60.2	8.8

Very little is known about the transmission of the *parapoxvirus*. It is believed that natural transmission occurs by direct contact or indirectly from the environment or fomites. New findings indicate that the carrier animal is probably very important in the spread of the disease in sheep. Considerable evidence has shown that uninfected flocks grazing on pastures abundant in thistles, on which no sheep have grazed for many years, still succumb to the disease (Lewis, 1996). Azwai et al. (1998) found that the seropositivity rate (ELISA) in Libyan camel herds with clinically affected dromedaries was 38% (and was related to clinical signs) and in apparently healthy herds was between 0% and 7%. Gitao (1994) believes that the common practice of keeping all camel calves in the same shelter at night could be responsible for the spread of the virus by contact, and he also proved that the outbreaks in camel calves occurred when *parapoxvirus* infections were also observed in goat kids raised nearby. Similar observations were made by Munz et al. (1986) and by Robertson (1976) who examined ORF infections in alpacas. Abu Elzein et al. (1998) reproduced camel contagious ecthyma experimentally in susceptible dromedaries, but experimentally-infected sheep were refractory to the camel virus. On the other hand, experimental infection with the ovine ORFV in dromedaries did not produce any disease in this animal species (Wernery, pers. com.). Wernery and Kaaden (1995) reported three 8-month-old dromedaries in the UAE that developed and died from a mixed infection of true camelpox and parapox during the course of an experimental camelpox vaccination program. The camels used as control animals were artificially infected with the camelpox virus. They might have developed a super infection with the contagious ecthyma virus or were latent carriers of the virus. Both viral species were seen situated next to one another upon electron microscopy (see Fig. 85).

NWC are also susceptible to contagious ecthyma virus (Preston Smith, 1940 and 1947; Moro, 1971; Ramirez, 1980; Thedford and Johnson, 1989; Fowler, 1998). Affected NWC develop typical proliferative lesions of the epidermis at the commissures of the mouth, which might spread to regions of the face and perineum. It is also possible that crias become infected when they suckle their dams that have developed lesions on their teats. ORFV from lamoids has produced severe ulcerating lesions on fingers, limbs and face in man (Fowler, 1998).

Clinical Signs and Pathology Two to 6 days post infection, primary lesions develop at the point of entry of the virus to the body. The lesions consist mainly of localized skin lesions of different magnitude, severity and location. Single or multiple primary pox lesions develop on the skin of the lips and muzzle. They frequently extend to the skin of the eyelids and other parts of the head as well as to the buccal cavity, such as the palate and the gums below the incisor teeth. The lesions develop as reddish papules that change to yellowish pustules within a few days before becoming nodular, ulcerated and hemorrhagic. Secondary bacterial and fungal infections as well as myiasis may aggravate the lesions on the lips and mouth. Enlargement of some superficial lymph nodes is also often observed.

Microscopic examination of the affected skin reveals parakeratosis, acanthosis, ballooning degeneration of keratinocytes and inflammation and edema of the dermis. The lesions are often accompanied by focal ulcerations, neutrophilic and eosinophilic infiltrations and superficial bacterial and fungal colonies. Microscopic lesions are diagnostic in early and acute stages, when cytoplasmic inclusion bodies are found in swollen epidermal cells but disappear in older lesions (6 days or more).

Contagious ecthyma in camels is usually characterized by local pox-like lesions

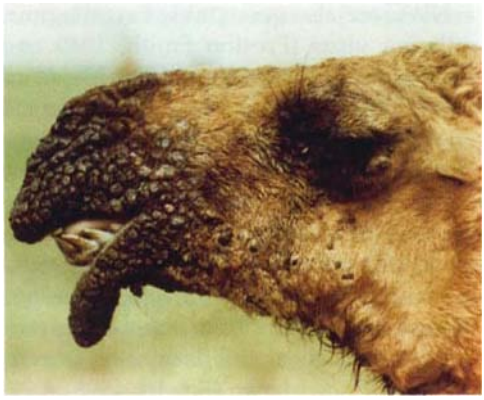

Figure 93 Camel contagious ecthyma in a young dromedary (courtesy of Dr. Khalafalla, Sudan)

on the face (Fig. 93). Recently, further reports have indicated that severe generalized forms of parapoxvirus infections seen in East African dromedaries cannot be differentiated from true camelpox (Mahnel and Munz, 1987).

Munz et al. (1986) described an outbreak of parapox in a 450-head dromedary herd in Kenya. Primarily, proliferative lesions on the lips were seen occasionally spreading to the nasal and oral mucosa. There was a tendency to generalization in calves and young dromedaries. Initially, papules emerged; then progressed into pustules before encrusting. The scabs finally became dark brown in color and dropped off after 6 to 10 weeks. In severely affected dromedaries, round, black hairless areas with slightly thickened epidermis remained up to 6 months. Some animals also developed edema of the eyelids, lips and alae of the nose or even the entire head. Similar clinical signs have also been reported by Moallin and Zessin (1988) from Somalia, and Gitao et al. (1994) observed swollen and edematous cervical and mandibular lymph nodes in many Kenyan dromedary calves. The majority of the skin lesions became infected with thick yellowish pus beneath the scabs. The authors did not detect any lesions on the udders of the dams or on the skin of any adult camel.

The morbidity among the young Kenyan dromedaries reached 100%. The disease was also described by Dashtseren et al. (1984) in Mongolian Bactrian camels, but without any deaths. The authors described small elevations around the mouth, which within 4 to 12 days developed to larger papules about 4 mm in diameter. These skin lesions became confluent and within 2 to 5 months scabs developed 5 to 15 mm thick, occasionally subdivided by many furrows. The percentage of adult camels that developed the disease lay between 10–80% in Mongolia and 10–20% in Kenya.

Diagnosis As it is extremely difficult to differentiate camel contagious ecthyma from true camelpox, mange or dermatophilosis, it is important that biopsies of fresh proliferative lesions are submitted to a veterinary diagnostic laboratory for diagnosis. Electron microscopic examination of biopsies or crusts is essential since virus isolation on the chorionallantoic membrane of chick embryos and in tissue cultures requires many passages. A recently developed PCR for the detection of *parapoxvirus* infection can also be recommended, especially when electron microscopy shows negative results (Buettner et al., 1995).

Indirect immunofluorescence, ELISA and western blotting technique for the detection of antibodies to camel contagious ecthyma can be used (Azwai et al., 1995), but are unreliable indicators of the immune status of the animal as it is known that immunity is mainly dependent on cellular mechanisms.

Treatment and Control Treatment is unrewarding as contagious ecthyma is caused by a virus. Management of infected animals plays a very important role. Because of public health considerations and the sometimes chronic form of ORF, animals

suspected of harboring the virus should be kept isolated from the herd until they recover fully. Stocking density should be reduced as much as possible and attention directed at reducing any secondary infection. Systemic treatment with high doses of synthetic penicillin against staphylococci is probably the best approach. Veterinarians examining or treating camels suffering from ORF should always wear gloves.

Neither vaccination nor natural infection produces a long-lasting immunity. Recovered sheep, for example, are only immune to re-infection for about 8 months after a primary infection. Attenuated vaccines are routinely used in sheep and goats and might also be used in camelids. Studies by Dashtseren et al. (1984) have shown that neither *vaccinia* virus nor the *parapoxvirus* ovis vaccines protect camels against *parapox* disease. However, the authors have achieved protection against this disease using a camel *parapoxvirus* strain adapted in eggs. Vaccinated camels were protected for at least 6 months. It seems to be possible that a bivalent vaccine against two of the most important viral diseases of camelids can be developed in the future.

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2.1.5 Papillomatosis

Papillomas (warts) are benign neoplastic growths of the skin and mucous membranes and are observed worldwide in humans and a variety of animals. They are caused by species-specific *papillomaviruses* that have also been associated with the development of squamous cell carcinomas.

Cattle are more affected by warts than any other domestic animal species: 6 types of *bovine papillomaviruses* having been identified. More than 70 *papillomavirus* serotypes are recognized in humans and cattle, while only 1 virus type has so far been identified in each of the other animal species.

The *papillomavirus* can also affect camels and cause typical skin lesions (Munz et al., 1990; Munz, 1992; Wernery and Kaaden, 1995; Khalafalla et al., 1998; Kinne and Wernery, 1998; Khalafalla, 1998).

Etiology *Papillomaviruses* are classified within the genus *Papillomavirus* within the *Papovaviridae* family. The virions are about 50 nm in diameter, spherical with icosahedral symmetry and possess 72 capsomeres composed of at least 3 proteins.

Epidemiology Papillomatosis has only been reported in OWC, where it is rare and of little economic significance. The disease usually occurs in camels less than 2 years old and the wart lesions, which are quite distinct from pox lesions, are commonly found on the lips and submandibular area without impairing the affected animal's health (Khalafalla et al., 1998). However, Munz et al. (1990) reported an outbreak of papillomatosis in Central Somalia primarily affecting animals from 6 months to 2 years old. The lesions were difficult to differentiate from true pox and parapox infections as generalized forms of papillomatosis had also been observed. Only laboratory procedures, such as electron microscopy, could clarify the disease agent. Sadana et al. (1980) reported a rare case of papillomatosis in a dromedary in India. The wart, located on the fetlock of a 15-year-old dromedary and weighing 2 kg, was removed surgically without complications. It is believed that this growth was not papillomatous, but rather a tumor (fibropapilloma).

Figure 94 Papillomatosis in a young dromedary (courtesy of Prof. Munz, Germany)

Cases of papillomatosis in young dromedaries have also been reported in the UAE (Wernery and Kaaden, 1995). Generalized forms have not been observed, only individual lesions on the lips and nostrils that, as pedunculate warts, were easily differentiated from other diseases involving pox viruses (Fig. 94).

Kinne and Wernery (1998) described papillomatosis in a small camel population of 10 dromedaries in the UAE of which 3 camels displayed proliferative, pedunculated warts on and in the mouth. These lesions that were examined by electron microscopy contained *papillomavirus*-like particles (Fig. 95).

Transmission of papillomavirus between animals usually occurs via abrasions or microlesions of the skin. Grooming equipment, ropes and contaminated instruments may transmit the virus. Mechanical transmission by arthropods might also be possible. Khalafalla et al. (1998) believe that there is a close relationship between papillomatosis and camel contagious ecthyma. The authors found most cases of camel papillomatosis during the rainy season, coinciding with outbreaks of contagious ecthyma. Dioli and Stimmelmayr (1992) found a relationship between camelpox and papillomatosis in Kenya.

Pathology The pathological picture of camel papillomatosis has been described by several researchers (Munz et al., 1990; Dioli and Stimmelmayr, 1992; Wernery and Kaaden, 1995; Khalafalla et al., 1998; Kinne and Wernery, 1998). The wart lesions appear as round cauliflower-like papillomas 0.3 to 4 cm in diameter and are usually pedunculated without affecting the health of the camels. This clinical picture is quite distinct from that produced by camelpox and parapox, in which the skin lesions usually undergo vesicle and scab formation. In the early stages of papillomatosis, the lesions appear as rosy, hyperemic elevations of the skin. Munz et al.

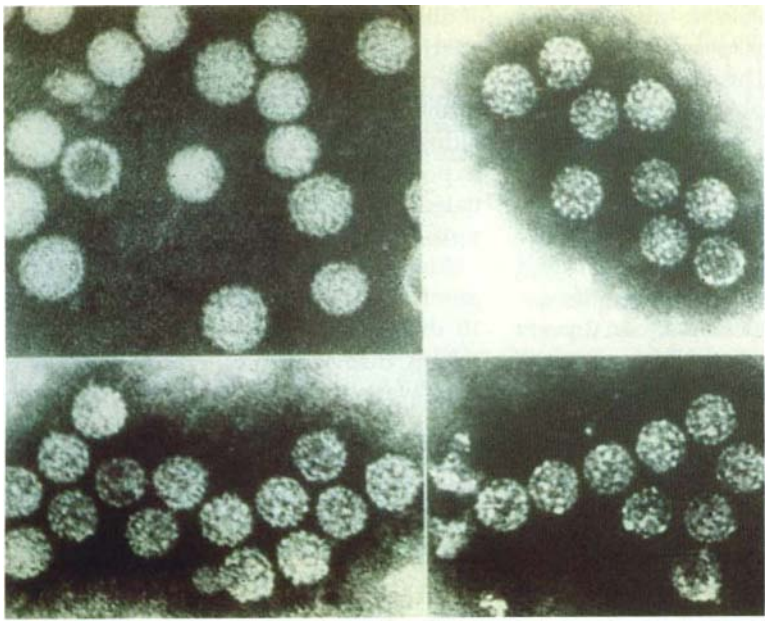

Figure 95 *Papillomavirus*-like particles from a wart in electron microscopy (x125,000)

(1990) described an outbreak of papillomatosis in Somalia where many dromedaries revealed pustules and scabs on lips and nostrils and generalized proliferative small and large nodules and tumor-like lesions. Some camels had lesions on the ears, eyelids, inguinal and genital regions and on their legs. The morbidity was high, but mortality was zero. Microscopically, the affected epithelium is hyperplastic with excessive folding that leads to the formation of proliferative outgrowths. The epithelial hyperplasia is characterized by marked acanthosis, para- and hyperkeratosis with

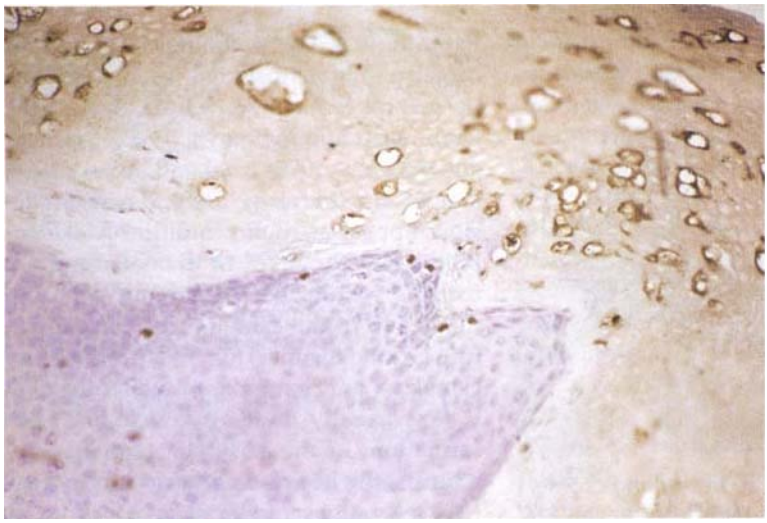

Figure 96 *Papillomavirus*-antigen-positive labeled cells of the epithelium of a wart. Virus antigen is visible in a few nuclei of the upper stratum spinosum and in numerous cells of the stratum granulosum and corneum (PAP-method, x220)

elongation of the rete ridges. These ridges extend deep into the underlying dermal connective tissue, which might turn hyperplastic. Within the stratum granulosum individual and/or clusters of cells might appear with swollen, clear cytoplasm and large pleomorphic keratohyalin-like granules (hollow cells).

Diagnosis Papillomas can usually be differentiated by their typical microscopic features. However, Kinne and Wernery (1998) were the first to develop an immunohistochemistry method using polyclonal rabbit-antibovine-*papillomavirus* serum (Fig. 96).

Treatment and Control Papillomatosis is generally a mild, self-limiting disease and therefore neither prevention nor treatment is usually necessary. Also in camels, wart lesions are often self-limiting and fall off within 3 to 6 months. However, in two outbreaks of papillomatosis in the UAE, the affected animals were treated with a formalinized autovaccine produced from surgically removed warts. The dromedaries were given between 3–7 mL (depending on body weight) of the wart vaccine subcutaneously. The warts receded within 8 to 10 days. Due to the antigenic variants of the *papillomavirus*, development of a specific vaccine for each individual herd is recommended.

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2.1.6 Influenza

The family *Orthomyxoviridae (Influenzaviruses)* consists of four genera, A, B, C and D, of which D comprises tick-borne viruses, e.g. Dhori and Thogota (see under Unusual Arboviruses). Group D differs biologically from A, B and C which are transmitted directly, usually by aerosols.

Although ruminants in general and camelids as intermediates are not considered susceptible to the influenza virus, severe outbreaks were reported among Bactrian camels in Mongolia (Lvov et al., 1982; Yamnikova et al., 1993; Anchlan et al., 1996).

Etiology The influenza viruses belong to the family *Orthomyxoviridae.* Humans are infected by all four groups of *Influenzaviruses:* A, B, C and D, but only Group A viruses produce epidemics. In animals too, Group A is the most important one. Epidemics occur particularly in poultry, pigs and equines, but infections have also been observed in minks, seals and whales.

Virions are 20 to 120 nm in diameter and are surrounded by a host cell-derived envelope with "spikes" formed by the glycoproteins of hemagglutinin and neuramini-

dase in a ratio of 4:1 or 5:1. Viral-protein synthesis occurs in the cytoplasm of host cells and during replication. Genetic reassortment may occur in mixed infections with viruses of the same species resulting in antigenic shift, but true recombinations have also been described.

Epidemiology Influenza A outbreaks have not been reported in NWC, but the disease has been observed in two-humped camels. Nineteen outbreaks of severe respiratory diseases were recorded in camels between 1978 and 1988 in 61 farms in different parts of Mongolia (Lvov et al., 1982; Yamnikova et al., 1993). The outbreaks started in 1979 and were caused by H1N1 influenza A virus. Thirteen virus isolates were obtained from a total of 92 nasopharyngeal swabs cultured in the allantoic fluid of infected embryonated chicken eggs. The isolates were identified by the hemagglutination test as H1N1 influenza A viruses. Four influenza A viruses of the same subtype H1N1, isolated from Mongolian patients during the same time as the influenza epidemics in camels, were found to be highly related in all genes sequenced to the camel strains. It is believed that the camel influenza isolates were derived from an UV-light inactivated reassortant vaccine (PR8 × USSR/77) prepared in Leningrad in 1978 and used in the Mongolian population at that time (Anchlan et al., 1996). The questions still remain as to how the viruses were introduced into the camel population and how they spread and attained pathogenicity in a formerly non-susceptible species. The outbreak occurred on 61 camel farms between 1978 and 1988 in different parts of Mongolia. One of the outbreaks involved about 4,000 camels affected with severe respiratory symptoms, occasionally with fatality. The clinical signs observed were as follows: lethality 9.1%, abortion 2.6% and cachexia 6.7%. Further clinical signs during the acute stages involved a dry cough, bronchitis, pneumonia and fever. There was a mucous ocular and nasal discharge. The clinical course lasted about one week.

A total of 34 healthy, 3 to 4-year-old Bactrian camels were infected experimentally with the H1N1 influenza isolates from affected camels. These test camels were confirmed to be free of pre-existing specific influenza antibodies. Groups of three camels were each infected by either the intranasal, intratracheal or intramuscular route. In three independent experiments performed between 1985 and 1986, no severe clinical signs were observed after the experimental infections, although the challenge virus strain was re-isolated and the experimental animals seroconverted, exhibiting hemagglutination inhibition titers between 1:16 and 1:128. The experimentally infected Bactrians developed clinical signs similar to those found during natural

Figure 97 Bactrian camel with nasal discharge caused by influenza

infection (but milder): fever, coughing, bronchitis and discharge from nose and eyes (Fig. 97). All infected animals recovered. No further outbreaks among Bactrian camels have been reported since.

The influenza outbreak in Mongolian camels is convincing evidence that a reassortant between two human strains has caused severe epizootics among camels, which are not regarded as natural hosts for influenza A viruses. Safety requirements for cold-adapted reassortants must therefore be adopted, because new strains may have a high pathogenicity for other species (Scholtissek, 1995).

Influenza-like epidemics in Somali camels have been reported by Auguadra (1958) and Somac-Sarec (1982) without any attempts to isolate the virus. The authors reported respiratory symptoms in conjunction with rhinitis and conjunctivitis. Serological studies to identify antibodies to the influenza virus have been performed in various African countries. Olaleye et al. (1989) found 0.6% of the samples positive for the influenza A virus and 12.7% for the influenza B virus taken from slaughtered dromedaries in northeastern Nigeria. El-Amin and Kheir (1985) reported 7.8% of Sudanese camels positive for the influenza A virus.

Among influenza viruses, 12 different hemagglutinins (H) and 9 neuraminidases (N) have been identified. In the Mongolian influenza outbreak in Bactrian camels, only one combination H1N1 occurred. Only two combinations have so far occurred in horses: H7N7 (influenza A/equine-1/Prague/56) and H3N8 (influenza/equine-2/Miami/63). Major antigenic drift has been observed among H3N8 viruses when mutations in the gene sequence result in amino acid substitutions, particularly in the hemagglutinin. One must be extremely cautious in countries where horses and camels are kept in close vicinity, as not only antigenic drift occurs but also recombinations of influenza viruses (antigenic shift). Antigenic shift gives rise to new influenza viruses which might result in pandemics in susceptible populations. One of the countries where valuable horses and camels are kept in close vicinity is the UAE. In this country, the authors have experienced annual outbreaks of respiratory disease in racing dromedaries caused by coccal infections. Furthermore, a serological survey on 500 UAE camels using the HIT with the equine strains Miami and Prague revealed no positive cases (CVRL Annual Report, 1998). However, the influenza outbreaks in Mongolia proved that other influenza serotypes than Miami and Praha may infect camelids.

Diagnosis In horses, for example, influenza must be differentiated from other respiratory diseases like EHV-1 and EHV-4, equine rhinoviruses, equine arteritis virus, *Streptococcus equi equi* (Strangles) and *Rhodococcus equi.* However, the rapid spread, the harsh cough and high temperature are sufficient to make a preliminary diagnosis. In vaccinated animals or in animals that have overcome the disease, it is extremely difficult to diagnose. It is therefore essential to carry out virus isolation and identification or serological tests.

Specimens for virus isolation should be collected after the onset of pyrexia and coughing, as virus excretion might be very short. Nasopharyngeal swabs have to be collected in virus transport medium and sent cooled to the laboratory as soon as possible. Influenza viruses should be cultured in embryonated eggs or in Madin-Darby canine kidney cells (MDCK). Several passages may be required in order to isolate the virus. The virus is identified by hemagglutination and subtyped using HIT with specific antisera. The HIT is also used for the detection of antibodies to the influenza virus. A rapid diagnosis in equines is done with the Directogen FLU-A test kit (Becton Dickinson, USA) and should also be tried in camelids with influenza-like

clinical signs. However, the serological diagnosis of infection in a vaccinated population is complicated by the presence of vaccine-induced antibodies.

Treatment and Control The most effective means of control in the face of an influenza outbreak are vaccination and restriction of movement of animals. No influenza vaccines have been administered to camelids, but in case of an outbreak, vaccination programs should be considered.

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2.1.7 Neonatal Diarrhea

Neonatal diarrhea in calves is one of the greatest sources of loss in animal breeding. Field and laboratory investigations have indicated that there is not a single etiology. The cause is complex and usually involves an interplay between enteropathogenic bacteria, viruses and parasites. On a clinical basis it is not usually possible to differentiate between the common known causes of diarrhea in newborns, which include enterotoxigenic E.coli (ENTEC), rotavirus, coronavirus, *Cryptosporidia* spp. and *Salmonella* spp. Rota- and coronaviruses have been identified as having characteristic localizations on the mucosal epithelium of the jejunum, ileum (rotavirus) and colon (coronavirus). The presence of viruses in the feces is not always indicative of manifest disease. Viral replication leads to the loss of function of the villous epithelium, causing the clinical signs (Freitag et al., 1984).

Etiology Rotaviruses are classified in the family *Reoviridae*, genus *Rotavirus*. Each rotavirus is named after the species in which it occurs.

Coronaviruses belong to the order *Nidovirales*, family *Coronaviridae*.

Epidemiology Very little is known about the cause of neonatal diarrhea in camelids, but there is agreement that the most common cause of death in camel calves up to 6 months of age is diarrhea (Khanna et al., 1992). In Sudan for example, Agab and Abbas (1998) reported a mortality rate higher than 30% in dromedary calves caused by diarrhea. There are only few reports that camelids might be susceptible to both ro-

tavirus and coronavirus. Mattson (1994) believes that neonatal diarrhea in NWC occurs with a lower incidence than in cattle, pigs and sheep. Rotavirus has not been isolated from NWC, but Rivera et al. (1987) detected antibodies to rotavirus in alpacas. Coronaviruses have been seen by electron microscopic examination of feces in two llamas with diarrhea, but attempts to isolate the virus in cell culture failed (Mattson, 1994).

Rota- and coronaviruses were detected in fecal samples in UAE dromedary calves suffering from diarrhea using electron microscopy (Mohamed et al., 1998; Ijaz et al., 2000 in prep.).

Rotaviruses were detected in a number of fecal samples from eastern Sudanese camel calves suffering from diarrhea using electron microscopy, ELISA and Latex agglutination (Khalafalla, unpublished). Eight out of 200 samples examined by Latex agglutination test, 11 out of 117 by ELISA and 4 out of 87 by electron microscopy were positive for group A rotaviruses. A seasonality of rotavirus infection in camel calves was observed. Most of the positive samples were recorded in early winter (October). Attempts to isolate rotavirus from eight positive fecal samples using MA 104 cell line (fetal monkey kidney) were unsuccessful up to the eighth blind passage. The genome of the camel rotavirus was analyzed by polyacrylamid gel electrophoresis (PAGE), in comparison with group A human and equine rotavirus isolates. The results indicated that the profile of the camel rotavirus RNA was different.

Mahin et al. (1983) found in serological studies of Moroccan dromedaries that 50% of the animals (27/55) had antibodies to rotavirus. This proves that dromedaries are susceptible to rotavirus infection. Rotavirus antibodies were also detected by Puntel et al. (1999) in 390 llamas from 9 farms located in 3 different Argentinean provinces. The antibody prevalence was 87.7% (342/390), which indicates that this species is highly susceptible to rotavirus. Chang-Say et al. (1985) showed that alpacas are also susceptible to rotavirus.

In the UAE, corona-like agents were detected by electron microscopic investigations in fecal samples of dromedary calves with diarrhea.

Diagnosis Rota- and coronavirus particles can be demonstrated in preparations of fecal samples from diarrheic calves by transmission electron microscopy. However, ELISA tests are more reliable and sensitive than electron microscopy and are nowadays widely used for the diagnosis of viral neonatal diarrhea. These tests also have the advantage of handling larger numbers of specimens. Virus isolation in cell cultures is difficult and often fails.

Several methods are used for the detection of antibodies to rota- and coronaviruses such as SNT and HIT.

Neonatal diarrhea is often caused by a secondary immunoglobulin deficiency and it is therefore important to comment in this chapter on the passive transfer of immunity.

Immunoglobulins (Ig) are divided into classes or isotypes (IgG, IgM, IgA, IgE, IgD) and further subdivided into subclasses. Most studies on animal immunology deal, however, with IgG, IgM or IgA (Table 39).

It is worthwhile mentioning that IgG antibodies in camelids differ from all other known antibodies and contradict all common theories on antibody diversity. At present, three subclasses of camelid IgG have been identified (IgG 1,2,3), of which IgG2 and IgG3 lack the light chains (Fig. 98) (Hamers-Casterman et al., 1993; Azwai and Carter, 1995):

- IgG1 binding strongly to protein A and G, composed of conventional antibodies, totaling 25% of serum IgG;
- IgG2 and IgG3 consisting of dimers of short heavy chains, which are characterized by a normal Fc region without CH1 domain, totaling 75% of serum IgG.

Table 39 IgG, IgM and IgA subclasses in different domesticated animals (after Huelsebusch, 1999)

Species	IgG subclasses	IgM	IgA	Source
Horse	Ga, Gb, Gc, G(B), G(T)	M	A	
Cattle	G1, G2 (G2a, G2b)	M	A	Tizard (1992)
Sheep	G1 (G1a), G2, G3	M	A1, A2	
Pig	G1, G2, G3, G4	M	A1, A2	
Alpaca	G	M	– *	Garmendia and McGuire (1987)
Llama	G 1a, G1b (conventional) G2a,G2b,G3 (heavy chain)			Ghahroudi et al. (1997)
	G1a, G1b G2a, G2b, G2c, G3			Woolven et al. (1999)
Camel	G1, G2	M	A	Grover et al. (1983)
	3 subclasses	M	– *	Azwai et al. (1993); Carter and Azwai (1996)
	G + associated protein	– *	– *	Ungar-Waron et al. (1987)
	G1, G2**, G3**	– *	– *	Hamers-Casterman et al. (1993)
	G1a, G1b (conventional) G2a, G2c, G3 (heavy chain)			Nguyen and Muyldermans (pers. commun.)

* = not identified
** = heavy chain antibodies

Ghahroudi et al. (1997) described that llamas possess IgG1a and IgG1b conventional antibodies and at least three heavy chain antibodies: IgG2a, IgG2b and IgG3. An existence of a fourth heavy chain antibody, IgG2c, has in the meantime been reported by Woolven et al. (1999).

In dromedaries at least five IgG isotypes have been detected: IgG1a, IgG1b (conventional antibodies) and IgG2a, IgG2c and IgG 3 (heavy chain antibodies, correspond to llama isotopes) (Nguyen and Muyldermans, 2000, in press). A recent paper by Linden et al. (2000) describes how different antigens (cell-lysate or haptens conjugated to carrier proteins) induce a variable response of different camelid isotypes.

It has been demonstrated that up to 75% of all serum proteins in camelids were IgG molecules lacking light chains (Hamers-Casterman et al., 1993). IgG 2 and IgG3, which only consist of heavy chains, show a

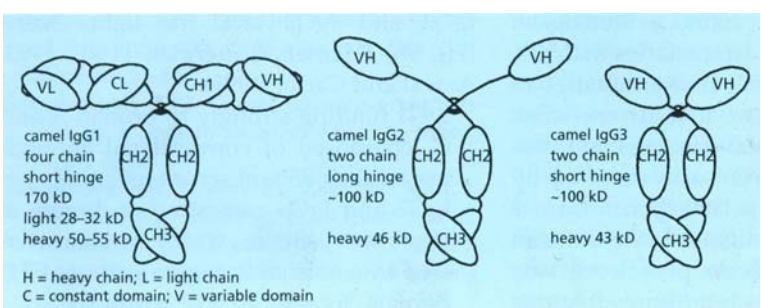

Figure 98 Structure of camelid IgG molecules (Huelsebusch, 1999)

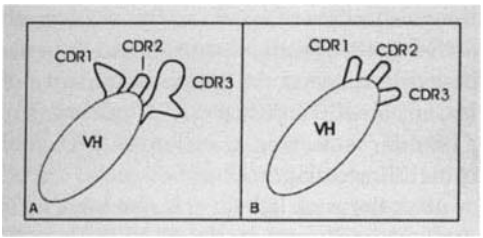

Figure 99 Molecular structure of variable heavy chain domains of camelid heavy chain IgG antibody (A) and common IgG antibody (B), VH: variable heavy domain, CDR: complementary determining region

molecular weight of 100 kD. These antibodies and their antigen-binding domain (referred to as VHH) have advantages over common antibodies, because their smaller size improves biodistribution and allows better tissue penetration. Moreover, the third complementary determining region (CDR) loop can be inserted deep into the active site of an enzyme, enabling it to neutralize enzymes fully (Muyldermans et al., 1994; Hoelzer et al., 1998; Lauwereys et al., 1998; Riechmann and Muyldermans, 1999; Fig. 99).

In general, it seems that camelids possess a unique class of antibodies which show a great advantage over common antibodies in applications where enzyme neutralization, size or stability is an issue (Nguyen et al., 2000). In the latter respect, it was also shown by Linden et al. (1999) that antigen-specific llama VHHs are stable at extreme temperatures. Two of the six llama VHHs were able to bind antigen at temperatures as high as 90°C.

Passive acquisition of antibodies is an important survival mechanism for the newborn. Immunoglobulins, principally IgG, are transferred from the dam by colostrum intake after birth. Protection is afforded rapidly, since the rate of decay of antibodies in serum is fast. For IgG, the half-life is 9 to 21 days; for IgM it is 3 to 5 days. Failure of passive transfer (FPT) of maternal immunoglobulins is the most important immunologic deficit in veterinary medicine because it is significantly correlated to numerous infections in postnatal life. The transfer of maternal antibodies from serum to colostrum to the intestinal tract and finally to the neonatal vascular system is a complex process with many sites for disruption.

Camelids have a thick-layered epitheliochorial placenta which prevents transplacental transfer of IgG. The camels therefore must obtain passive immunity by intestinal absorption of IgG from the colostrum. Fig. 100 demonstrates that newborn dromedary calves have very little demonstrable serum IgG prior to ingesting colostrum.

Although the neonate camelid is immunocompetent at birth, it is immunologically naïve and therefore dependent on

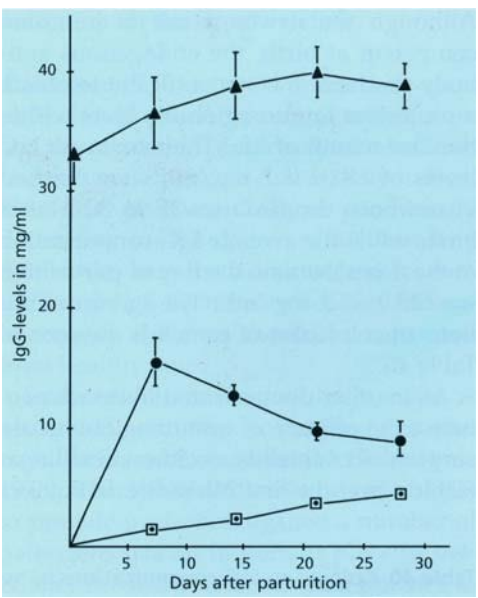

Figure 100 Serum immunoglobulin values in dromedary mothers (solid triangle), dromedary calves that have ingested colostrum (solid circle) and dromedary calves that have not ingested colostrum (square with central dot) (Graph modified after Ungar-Waron et al., 1987)

passively acquired humeral immunity. Newborns that fail to acquire adequate passive immunity are at greater risk of developing diseases such as diarrhea, enteritis, septicemia, arthritis, omphalitis and pneumonia. Successful passive transfer is achieved when neonates have IgG serum levels of greater than 9 mg/mL at 48 h of age (Barrington et al., 1999). Studies have determined that the concentration of IgG in NWC colostrum is approximately 220 mg/mL (Bravo et al., 1997). It was calculated that a 10 kg cria would require 20 g of IgGs to obtain an IgG level of greater than 9 mg/mL. To obtain 20 g of IgG from colostrum with an average IgG concentration of 220 mg/mL, a cria would need to consume approximately 100 mL of colostrum.

As in other domesticated animals, very low IgG concentrations were observed in 68 camel calves before intake of colostrum (0.26 ± 0.23 mg/mL) (Huelsebusch, 1999). Although the newborn calf is immunocompetent at birth, the endogenous antibody production is not sufficient to obtain a protective immunoglobulin level within the first month of life. The maximum IgG levels of 21.1 ± 11.7 mg/mL were reached in newborn dromedaries 18 to 30 h after birth, while the average IgG concentration of the dams' sera on the day of parturition was 23.9 ± 7.5 mg/mL. The Ig concentrations of colostrum of camelids are seen in Table 40.

As in other domesticated livestock neonates, the efficacy of immunoglobulin absorption in camelids declines in a linear fashion over the first 24h of life. In bovines the mechanism of IgG transfer involves an active IgG1 specific receptor, and it is believed that based on the predominance of IgG in camelid colostrum (7:1 IgG vs IgM), a similar selective transfer of IgG into camelid colostrum occurs.

After the peak IgG level is obtained, IgGs decline rapidly and reach low levels 2 weeks after birth. The calves' own antibody production does not start before 2 weeks, and a marked increase in serum IgG above 10 mg/mL is found between 1 and 2 months, meaning that the critical period of calves for infections lies between 2 and 5 weeks. Serum IgG concentrations stabilize at a plateau around 4 months after birth, indicating that the immune system has matured.

Ungar-Waron et al. (1987) and Hannant et al. (1992) were the first to examine this problem in dromedaries. Fowler (1998) described serum protein levels in NWC. Total protein levels of less than 5 g/dL are suggestive of FPT. Levels between 5 and 6 g/dL are equivocal and levels over 6 g/dL indicate a successful passage of IgG. In NWC as in OWC the lowest level of globulins is reached 3 to 5 weeks post partum.

Assessment of passive immune status of compromised camelid neonates is essential to enable prompt administration of IgG. Several methods are available to measure passive transfer such as zinc sulphate turbidity (ZST), sodium sulphate precipitation (SSP, commercially available for NWC: Llama-STM, VMRD Inc. Pullman, WA, USA). None of these tests measure serum IgG concentrations specifically. The single radial immunodiffusion (SRID) is

Table 40 Colostrum IgG concentrations in mg/mL of camelids (after Huelsebusch, 1999)

Species	IgG	Authors
Alpaca	10–280	Garmendia et al. (1987)
Camel	70–220	Ungar-Waron et al. (1987)
	58.6 ± 15.4	Kamber (1996)
	25.56–84.25 (Ig1)	El-Agamy (1998)
	1.81–6.02 (Ig2)	El-Agamy (1998)

the only method that specifically measures serum IgG concentrations. For NWC the test is commercially available in two kits: Llama IgG Test Kit, Triple J. Farms, Redmond, WA, USA and Llama Vet-RID, Bethyl Laboratories, Montgomery, TX, USA. Hutchison et al. (1995a) compared these tests on 528 llama plasma samples and found that each test kit provided significantly different IgG levels when compared to the other.

Bourke (1996) has studied the application of three tests for the determination of the passive immune status in llama neonates:
- zinc sulfate turbidity (ZST),
- total protein (TP),
- globulin (G).

Table 41 IgG status in camelids based on ZST, TP and G (after Bourke, 1996)

IgG transfer status	ZST I	TP g/dL	G g/dL
Nil or low	< 30	< 5	< 0.25
Moderate	30–40	5–5.5	0.3–1.2
Adequate	> 40	> 5.5	> 1.2

The study indicates that all three tests can be used to assess the IgG status in neonatal camelids, as seen from Table 41.

An ELISA has recently been developed for the quantification of camelid IgG in blood serum; an important tool in tackling FPT (Huelsebusch, 1999; Erhard et al., 1999). This assay is designed as an indirect ELISA carried out in 96-well microtiter plates. The anti-camel-IgG antibodies were raised by immunization of layer hens with camel IgG and were subsequently extracted from the egg-yolk.

The ingestion of colostrum is essential for the survival of the newborn. FPT is the major determinant of septicemic disease, and it also modulates the occurrence of mortality and severity of enteric and respiratory disease in early life and performance at later ages. Many important factors exist which influence the level of serum IgG achieved by the newborn. However, the amount of circulating IgG acquired from colostrum is primarily dependent upon two factors: the amount of IgG in the colostrum and the efficacy of its absorption by the calf.

Literature on camelid IgG deficiency is limited. Few reports indicate that FPT is the major factor in neonatal mortality in alpacas (Garmedia et al., 1987; Garmendia and McGuire, 1987; Murphy, 1998; Kennel and Wilkens, 1992; Hutchison et al., 1995b; Barrington et al., 1997), and hardly any reports exist on FPT in OWC. Wernery et al. (2001, in press) described a secondary IgG deficiency in young dromedaries in the UAE which died from septicemias. This syndrome was caused by copper deficiency. The calves did not suckle, but consumed variable amounts of sand as compensation for the copper deficit.

Treatment Treatment of diarrheic camelids should primarily aim at rehydration and the correction of electrolyte imbalance, as death mainly occurs due to dehydration. Fluid may be given orally or parenterally depending on the degree of dehydration. Antibiotics may be administered to control secondary bacterial infections, and animals with diarrhea should be separated from healthy ones.

Successful immunoglobulin transfer is associated with low infection rates and high likelihood of survival (McGuire et al., 1976). Therapeutic administration of IgGs to provide protection against a number of pathogens has an important place in veterinary medicine. It is common practice to establish a colostrum bank and feed 10% colostrum in milk during outbreaks of neonatal diarrhea. This procedure will provide passive protection for a 2 to 3-week period of risk. If no camelid colostrum is available, goat colostrum (up to 20% of body weight) may be administered to lla-

mas as a substitute (Pugh, 1992; Pugh and Belknap, 1997). For the treatment of FPT, it is also possible to give 20 to 40 mL of camelid plasma intravenously. It should be given over a 30 to 60 min time frame and warmed to 37°C. Llama hyperimmunplasma is commercially available at Triple J. Farms, Redmond, WA, USA.

Viral Neonatal Diarrhea in calves is difficult to control because the etiology is often complex and the disease has a rapid course. Maternal vaccination may be an alternative approach. The use of commercial vaccines 1 to 3 months before calving can significantly reduce the prevalence of rota- and corona diarrhea in affected animals. In cases of viral Neonatal Diarrhea in camelid calves, vaccination programs should therefore be considered.

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2.1.8 Equine Herpesvirus

Interestingly, no herpesvirus unique to camelids has been identified so far. Equine herpesvirus type 1 (EHV-1) produces rhinopneumonitis and abortion in horses and the disease has also been reported in OWC and NWC (Jenkins, 1985; Torres et al., 1985; Rebhun et al., 1988; Thedford and Johnson, 1989; House et al., 1991; Bildfell et al., 1996).

Etiology At least seven different herpesviruses were isolated from horses. The most important pathogens are EHV-1, the causative agent of equine abortion and neurological disorders, and EHV-4, the etiological agent of equine rhinopneumonitis. Both viruses are members of the genus *Varicellovirus* within the family *Herpesviridae.*

Epidemiology and Pathology EHV-1 has worldwide distribution and although it is considered a disease of equids, has been isolated from other animal species including bovines, zebras and antelopes. In equines, it causes a respiratory disease most frequently seen in foals and yearlings. In pregnant mares, it is a major cause of abortions in late gestation. Additionally, EHV-1 can also cause neurological disturbances associated with encephalomyelitis. When herpesviruses infect a non-adapted host, serious disease or death is likely to result (Fowler, 1996). This has occurred in both NWC and OWC. EHV-1 infections have been described in a group of 100 alpacas and llamas on an exotic animal farm (Pursell et al., 1979; Jenkins, 1985; Rebhun et al., 1988). One hundred lamoids had been brought to the USA from Chile where they had been in close contact with other llamas, camels, gnus and various species of antelopes. A herpesvirus indistinguishable from EHV-1 was isolated from dead llamas and alpacas that had suffered from blindness and central nervous system disturbances such as nystagmus, torticollis and paralysis. The blindness was believed to have been caused by chorioretinitis or optic neuritis.

EHV-1 appears to have followed an unusual pattern in alpacas and llamas exposed to the virus. In a subsequent study (House et al., 1991) three llamas were experimentally infected intranasally with EHV-1 isolated from the brain of an alpaca with severe neurological signs. Two of the three llamas developed severe neurological disorders: one died and one was euthanized. The third llama showed only mild neurological signs. EHV-1 was only re-isolated from a sample of the thalamus of the llama that had died acutely. These investigations demonstrate a difference between EHV-1 infection in equids and NWC. In equines, a viremia occurs after initial virus replication, whereas in NWC the virus is believed to replicate in the cells of the mucous membranes of the nasal cavity, where it infects the olfactory nerve and optic nerve and progresses to the central nervous system. There are no reports that EHV-1 induces abortions in camelids. EHV-1 infects not only NWC but also OWC. Bildfell et al. (1996) cultured EHV-1 from the brain of a Bactrian camel suffering from severe neurological disease prior to death.

Microscopic lesions in the brain of the Bactrian camel included a non-suppurative meningoencephalitis with vasculitis, necrosis and edema. These features are similar to those in EHV-1-induced neurological disease in llamas and horses. However, there was no ocular damage detected in this case. These investigations show that NWC and OWC can suffer from EHV-1 infections associated with nervous system signs, blindness and death. EHV-1 can cause seroconversion in NWC, but antibodies to EHV-1 have not been reported in OWC. Antibodies to EHV-1 were found in 21 serum samples from llamas and alpacas suffering an EHV-1 infection (Rebhun et al., 1988). Only 1 llama of 270 from Oregon possessed antibodies. Puntel et al. (1999) reported an antibody prevalence of 0.77% in 390 (3/390) llamas from 9 farms in 3 different Argentinean provinces. No antibodies to EHV-1 were found in 500 UAE dromedaries when tested with a sandwich ELISA (CVRL Annual Report, 1998).

Camelids are susceptible to EHV-1. With the increase in breeding of NWC in different countries and an increased opportunity for both NWC and OWC to come into contact with equids, not only EHV-1 but also other equine viral infections should be considered in a differential diagnosis. Efforts should be undertaken to clarify the role of EHV-1 in abortions and neonatal diseases in camelids.

Diagnosis EHV-1 infections cannot be diagnosed solely on the basis of clinical signs. Confirmation of the disease can be achieved by virus isolation on a wide

range of cell cultures, including rabbit kidney (RK 13), Vero and EDMIN (equine dermal cells), by IFAT and by immunohistochemical staining for viral antigen in endothelial cells of the central nervous system. Serological testing of acute and convalescent sera is also important for the diagnosis of EHV-1 in camelids.

Treatment and Control There is no specific treatment for EHV-1 infection. Although steroids and antibiotics were administered to sick lamoids suffering from EHV-1, there was no response.

Vaccination with a killed vaccine to EHV-1 induces antibodies in llamas (Mattson, 1994). However, so far no challenge infection has been conducted after vaccination to determine the efficacy of such a vaccine. EHV-1 vaccines have not been used in OWC. Live attenuated EHV-1 vaccines are not recommended in camelids.

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2.2 Nonpathogenic Viral Infections

The growing interest in camelids is documented in the rapidly increasing number of publications since the 1970s. More than 50% of all references appeared after 1970. More than 5,500 papers have been published on OWC and 2,400 NWC veterinary references appeared in the world literature (Wernery et al., 1999). For both NWC and OWC, less than 1,000 publications concerning microbiological subjects have been published, most of which are cited in this book.

Viruses such as African horse sickness, rinderpest, foot-and-mouth disease, Rift Valley fever, bovine viral diarrhea and bluetongue have been isolated from camelids; some of them have caused mild diseases, especially through experimental infection. The authors therefore prefer to keep these viral infections under the chapter "Nonpathogenic Viral Infections".

As seen in Table 35, sero-epidemiological viral studies on camelids were primarily performed in the last 2 decades and investigations have reported a number of positive findings on viruses in camelids, indicating exposure and antigenic response. However, for several viral diseases only antibodies to the virus have been identified. Many of these reports were of OWC used for slaughter with little or no background regarding either their origin or condition.

Little is known about the unusual *Arboviruses* in camelids, which have been identified through serological investigations or isolated from the camel tick. They will be examined in a separate chapter.

2.2.1 Respiratory Viruses

- Adenovirus
- parainfluenza virus 1,2,3
- bovine respiratory syncytial virus (BRS)
- infectious bovine rhinotracheitis virus (BHV-1)

Antibodies to the respiratory viruses mentioned here have been found in camelids all over the world. In northeastern Nigeria, Olaleye et al. (1989) found 1.3% positive reactants to adenovirus among dromedaries kept for slaughter. The same authors have identified antibodies to parainfluenza viruses 1, 2 and 3 (22.3%, 2.5%, 18.5%) and the respiratory syncytial virus (0.6%). The epidemiological significance of these results is still unclear and requires further study.

In a serological survey involving 270 llamas from 21 ranches in Oregon, USA, the prevalence of one of the adenovirus species (isolate 7649) was 93% (Picton, 1993). The incidence of exposure in llamas appears high, but the infection is mostly subclinical in nature. However, Galbreath et al. (1994) isolated an adenovirus from the lungs of a 5-month-old llama with pneumonia and hepatitis. Intranuclear inclusion bodies characteristic of adenovirus were detected in the lung and liver. Adenoviruses have been isolated from llamas and alpacas with diarrhea in the USA (Mattson, 1994). A llama that died revealed severe necrotizing enteritis and colitis. Because it registered a very low IgG level, it was diagnosed as having an immunodeficiency syndrome as well as a secondary adenovirus infection. Puntel et al. (1999) found a prevalence of antibodies to bovine adenovirus (Bad VIII) of 5.13% (20/390) in llamas on a single farm in Argentina.

It is interesting to note the high prevalence rate of antibodies to parainfluenza virus 3 in dry desert conditions (El-Amin and Kheir, 1985): 81% in Tunisia (Burgemeister et al., 1975), 99% in Chad (Maurice et al., 1968), 81% in Sudan (Bornstein and Musa, 1987) and 42.8% in Somalia (Bornstein, 1988). Only 5.6% of racing camels in the UAE were positive for parainfluenza 3.

The difference in the prevalence of parainfluenza in different countries is probably due to different environmental conditions and management practices (Afzal and Sakkir, 1994). In spite of the high incidence rate, the parainfluenza virus has not yet been isolated.

NWC can also become infected with parainfluenza 3 and respiratory syncytial viruses, but there have been no reports that these viruses can cause clinical signs (Rivera et al., 1987; Picton, 1993).

The role of bovine herpesvirus type 1 (BHV-1) in diseases of NWC and OWC is not well established and is therefore referred to in the chapter "Nonpathogenic Viral Infections".

The dromedary does not seem to be susceptible to the BHV-1 virus. Hedger et al. (1980), Bornstein and Musa (1987), Bornstein et al. (1988), Bohrmann et al. (1988) and Wernery and Wernery (1990) were not able to detect any antibodies to the causative bovine herpesvirus (BHV-1). Only Burgemeister et al. (1975) found low antibody titers (1:5) in 5.8% of Tunisian dromedaries. In a second serological survey conducted in the UAE (using a sandwich ELISA), no antibodies were found to BHV-1 in 804 dromedaries (717 racing camels, 77 breeding camels, 10 yearlings) (CVRL Annual Report, 1998). In an experimental trial, the authors infected two dromedaries intranasally with a BHV-1 strain that had a titer of 10^5 $TCID_{50}$/mL. Both camels and a control camel failed to develop any clinical signs and all three camels failed to seroconvert.

It seems that NWC are more susceptible to BHV-1 than OWC. NWC can become infected. BHV-1 was isolated from a 3-year-old llama revealing bronchopneumonia in association with *Pasteurella haemolytica* (Williams et al., 1991). It was not clear if the virus had caused the death of the animal. Histological changes revealed an acute, multifocal neutrophilic bronchopneumonia consistent with an early inflammatory response to a bacterial infection. BHV-1 was also isolated from three separate cases of bronchopneumonia in llamas and also confirmed by immunofluorescent antibody test (IFAT) (Mattson, 1994). The clinical signs of disease in these cases included progressive cough. BHV-1 was also isolated by the same author from the brain tissue of a 1.5-year-old llama with acute neurological disease associated with diffuse nonsuppurative encephalitis.

BHV-1 antibodies were found by Rosadio et al. (1993) in Peruvian llamas and alpacas. The authors found the highest prevalence (16.7% in llamas and 16.2% in alpacas) when the herds grazed on the same pasture together with cattle, sheep and goats. When the alpacas were separated from other ruminants, the prevalence was only 5.1%. In other serological surveys of alpacas, in Peru Rivera et al. (1987) detected 5% reactors, while only 0.7% reactors to BHV-1 of 270 llamas were diagnosed in Oregon (Picton, 1993).

Since it is known that malignant catarrhal fever virus (MCF), a *gammaherpesvirus*, can infect more than 150 species in the suborder *Ruminantia*, HongLi et al. (1996) tested 41 llama sera with a competitive-inhibition ELISA from the USA. All tested llamas were negative.

From all this data, it can be concluded that NWC are susceptible to BHV-1 and develop a disease, although the incidence of infection does not appear to be high. OWC, in contrast, seem resistant to BHV-1. However, the authors believe that additional studies are needed to clarify this issue.

Prevention ❋ Adenovirus, parainfluenzavirus, bovine respiratory syncytialvirus and bovine herpesvirus 1 are of minor importance to *Camelidae*. However, intranasal vaccination with live virus vaccines has been shown to be very effective in controlling respiratory tract disease in cattle caused by bovine herpesvirus 1 and para-

influenza 3. In case of outbreaks in camelids, these two vaccines may be used in these animal species.

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2.2.2 African Horse Sickness

African horse sickness (AHS) is a highly fatal, insect-borne viral disease affecting horses, mules and donkeys. It has been known in Africa for hundreds of years. AHS is a disease of the vascular endothelium resulting in a variety of different forms of the disease. Different clinical presentations are seen in the horse, depending on the virulence of the virus.

Etiology The African horse sickness virus (AHSV) belongs to the genus *Orbivirus* of the family *Reoviridae*. It shares many properties with other orbiviruses such as bluetongue and equine encephalosis. Virions of AHS contain 10 double-stranded RNA genome segments encapsulated in a double-layered capsid. Nine different serotypes of AHS are known to give cross-reactions between the serotypes. The size of plaques produced in cell culture indicates the virulence: small plaque viruses are more virulent than large plaque variants. The virus grows in BHK 21, Vero and MS cell cultures with a cytopathic effect (CPE).

Epidemiology AHS is endemic in eastern and central Africa from where it regularly spreads southwards. The disease has also been seen in North Africa, the Middle East and Spain. The spread of the disease is greatly influenced by favorable climatic conditions for the breeding of *Culicoides* midges, the main vector of AHS. It has now been confirmed that there is a strong link between the timing of epizootics of AHS in South Africa and the climatic changes brought about by El Niño (Baylis et al. 1999). With the increasing impact of El Niño as a result of global warming, there is real concern about important insect-borne diseases spreading worldwide. Serotype 9 of AHS is already widespread and occurs in North and West Africa as well as in the Middle East. Serotypes 1 to 8 are highly virulent for horses and cause up to 95% fatalities, whereas with serotype 9, the mortality rate reaches 70%. AHSVs affects all equines as well as canines. Horses followed by mules are most susceptible to the disease. Donkeys and zebras are resistant and the disease is often subclinical in these equids. The virus is transmitted by *Culicoides* spp., of which *C. imicola* is the most significant vector. These midges can travel hundreds of kilometers on air currents. It is believed that the outbreak in Spain in 1992 was caused by midges from Morocco (Coetzer et al., 1994). Ticks do not play an important role in the transmission

of the virus. However, the AHSV can replicate in *Hyalomma dromedarii,* which is usually parasitic to camels (Awad et al., 1981a). The reservoir host of AHSV is unknown.

Clinical Signs and Pathology Serological studies identifying antibodies to the AHSV in African dromedaries yielded a prevalence of 5% in Egypt (Awad et al., 1981b) and 23% in Sudan (Foreign Animal Disease Report, 1988). Salama et al. (1986), who examined 134 Sudanese and 266 Egyptian camels serologically, found 23.2% positive and 5.6%, respectively. In Nigeria, Baba et al. (1993) detected 10.4% positive dromedaries out of 96 with the HIT. The authors believe that camels and dogs are an important reservoir of AHSV. In 24 East African dromedaries, there were no antibodies detected by Binepal et al. (1992) and serological studies performed by Wernery (unpublished, 1992) on 500 dromedaries in the UAE also yielded a negative result for antibodies using the AGID. There are no reports of serological investigations of AHS in NWC.

Salama et al. (1986) isolated two AHSV strains, serotype 9, from blood of two healthy camels in suckling mice. Mouse brain suspensions of the fifth passage of these viruses were injected into two susceptible horses that subsequently developed typical clinical signs of AHS.

The AHSV was also isolated in Egypt from dromedary ticks *(Hyalomma dromedarii).* Those animals infested with infected ticks showed no signs of illness. Of 2089 ticks, 17% carried the AHSV, type 9, confirmed by the mouse inoculation test (Salama et al., 1979 and 1980). The infected larvae and nymphs of *Hyalomma dromedarii* transmitted the causative agent to susceptible animals that then developed the AHS. Infected nymphs are also able to transmit the disease later in the adult stage (Foreign Animal Disease Report, 1988). All these studies appear to prove that the dromedary can serve as a reservoir for the AHSV.

There are four clinical and pathological disease forms seen in equines. The *pulmonary form* occurs after an incubation period of 3 to 5 days and is associated with fever and severe respiratory distress. Pathologically, there is a marked pulmonary edema with widened, edematous interlobular septa. The *cardiac form* occurs after a slightly longer incubation period and is characterized by intermittent fever and heart failure. At necropsy, there is enormous subcutaneous edema throughout the anterior portion of the body with petechial and ecchymotic hemorrhages on organ surfaces accompanied by hydropericardium. A rare third form of AHS, the *mixed form,* is a mixture of the pulmonary and cardiac forms. Lastly, a mild form of AHS, the *horse sickness fever,* can be observed in partially immune animals with an influenza-like syndrome followed by total recovery. This form occurs in species such as donkeys and zebras that are resistant to the development of clinical disease. No clinical signs of AHS have been described in camelids, although the virus has been isolated from the blood and ticks of dromedaries.

Diagnosis Clinical signs and macroscopic lesions of AHS are often sufficiently specific to allow a preliminary diagnosis in equines. However, to confirm the diagnosis virological investigations must be performed. AHSV should be isolated from heparinized blood during the febrile stage and from the lung, spleen and lymph nodes of necropsied horses. Virus isolation should be done on BHK 21, Vero, MS and by intracerebral inoculation of suckling mice. Virus isolates are identified by group-specific tests such as CFT, AGID, IFA or ELISA. Serotyping of AHSVs is carried out by virus neutralization using type-specific antisera. ELISA is the best serological test for the detection of antibodies. Antibodies in horses vaccinated 9 years prior were still positive when tested in the ELISA (CVRL

Annual Report, 1998). AGID, CFT and HIT have been used for the detection of antibodies to AHS in camels. A sandwich ELISA used at CVRL, Dubai, showed no antibodies to AHSV in 293 UAE dromedaries (CVRL Annual Report, 1998). New techniques, like polymerase chain reactions (PCR) or genomic probes that are more rapid, sensitive and specific, will soon become available for the diagnosis of AHS.

Treatment and Control There is no specific therapy for AHS. Horses suffering from AHS should be euthanized and disposed of properly.

Since there are many serotypes, the use of a polyvalent vaccine is recommended to protect horses from AHS in endemic regions. Infection of susceptible horses can be prevented to a large degree by stabling them some hours before sunset and letting them out a few hours after sunrise. *Culicoides* midges are nocturnal and will not enter buildings. The application of insecticides will also have a positive effect on the control of AHS. Racehorses are generally not vaccinated against AHS, because they might be excluded from international trade or racing. Since camels seem to be resistant to AHSV there is no necessity for vaccination.

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2.2.3 Bluetongue

Bluetongue (BT) is an acute arthropod-borne viral infection of sheep, cattle and wild ruminants. The virus is transmitted by *Culicoides* species. Twenty-four serotypes of the virus are known. The disease is characterized by cyanosis of the mucous membranes of the oronasal cavity, laminitis, coronitis, edema of head and neck, inflammation and ulceration of the mouth. The disease, originally confined to Africa and only affecting sheep, has spread during the last decades to America and Australia.

Etiology BT virus (BTV) belongs to the genus *Orbivirus* in the family *Reoviridae.* BTV was the first domestic animal virus to possess a double stranded RNA genome. All 24 serotypes possess cross-immunity. A

large number of related orbiviruses, (mainly from insects in Australia), as well as epizootic hemorrhagic disease (EHD) virus in deer, have also been detected.

Epidemiology The distribution of BT is mainly confined to the tropics and subtropics and to areas with high rainfall in association with a sufficient number of game and cattle. With these conditions, large numbers of *Culicoides* transmit the virus to sheep, which are the most susceptible species. BTV has been isolated from various parts of the world from a variety of *Culicoides*, of which *C. imicola* is the most important. After a female gnat has ingested blood, the virus replicates in its salivary glands. Infected midges remain infective for the rest of their lives. Ten days after becoming infected with BTV, the midges can transmit the virus to animals (by biting). Midges live 30 days and feed every 3 to 5 days.

Although reports of BTV seropositive NWC and OWC exist, there is only one statement (Fowler, 1998) of a suspected BT case in a llama associated with respiratory distress followed by abortion. Paired serum samples taken after the abortion demonstrated a fourfold increase in BTV antibody titer. However, it remains unknown what role camelids play in the epizootiology of BT.

Reports of BTV seropositive dromedaries have appeared from many different countries. In Sudan, where BT is endemic, Eisa et al. (1979) and Eisa (1980) identified 4.9%, Abu Elzein (1984) 14.6% and Abu Elzein (1985a) 16.6% positive dromedaries. According to Abu Elzein (1985b), that is a very small prevalence in a country where 93% of the cattle, 86% of the goats and 73% of the sheep exhibit positive titers. This small percentage may be explained by the slight susceptibility of the dromedary to the BTV. In Egypt, where the virus is also endemic, Hafez and Ozawa (1973) were only able to identify 14.3% reactors. However, Hafez et al. (1984) found 67% reactors in Saudi Arabian dromedaries, although it should be noted that the authors only examined three animals. In Botswana, Simpson (1979) found a prevalence of 81% of the dromedary population showing antibodies to the BTV. Seropositive dromedaries were also diagnosed on the Arabian Peninsula. Stainley (1990) found that 13% of the dromedaries in Yemen had antibodies to this virus. In a serological survey conducted in the UAE, less than 1% of 1023 dromedaries were found positive to BTV with the agar gel immunodiffusion test, and 5% of 211 camels positive with the competitive ELISA, although 35% of sheep from the same area reacted positively to the virus (CVRL Annual Report, 1998). No BTV was isolated from sheep and camels from this region. It is worthwhile mentioning that Ostrowski (1999) found 58% serological reactors in Saudi Arabian camels (an arid country almost identical to the UAE). The reason for the great difference in the prevalence of BT between these countries is unknown. Afshar and Kayvanfar (1974) diagnosed 5.9% reactors in Iran. Antibodies to the BTV have also been found in Israeli dromedaries (Barzilai, 1982). Twenty-three percent of the dromedaries examined had positive titers to type 4.

Abu Elzein (1984) was the first to identify BT antigens in Sudanese dromedaries using the immunodiffusion test. In 5.6% of 89 animals, BT antigen was detected. Based on these results, the author is of the opinion that the dromedary might play a role in the spread of BT.

Several serological studies have also been conducted in NWC. In a seroprevalence study carried out in Peru by Rivera et al. (1987), 21% of 114 alpacas possessed antibodies to BTV, and in Oregon, USA, 1.5% of 270 llamas reacted positively (Picton, 1993). However, most of the llamas originating from Oregon were from areas where BT is not enzootic in livestock. Puntel et al. (1999) did not detect any antibodies to BTV

in 390 llama sera from 9 farms in 3 different Argentinean provinces.

Clinical Signs and Pathology No clinical signs or pathological lesions caused by BTV have been described in camelids, except in one llama with a respiratory syndrome and abortion (Fowler, 1998). Clinical manifestation of BT possesses an extreme variability not only between different ruminant species, but also between different breeds of sheep. BT in sheep in the USA is much milder than in Africa and the authors have not seen any clinical cases in sheep in the UAE, although more than 30% of the sheep population have antibodies to BTV. In sheep the disease is characterized by fever, dyspnea, and hyperemia of the muzzles, lips and ears. Other signs include a swollen, cyanotic "blue" tongue, lameness and muscle necrosis. Ulceration, erosions and necrosis of the mouth mucosa and of the dental pad may appear. The coronary bands may become inflamed and swollen. Lameness is an early sign of infected flocks and might be confused with FMD. Cattle are commonly latently infected, but some may develop clinical signs similar to those seen in infected sheep.

Diagnosis BT is often misdiagnosed as photosensitization, FMD, BVD/MD, IBR, MCF, EHD and Orf, and it is therefore often necessary to confirm the disease by either virus isolation or serology. Direct isolation of the virus can be done in embryonated chicken eggs, certain cell cultures or susceptible sheep. The virus can also be propagated in suckling mice by intracerebral inoculation. Viruses are then identified by serum neutralization or by FA. Serological tests include ELISA, CFT, AGID and SNT.

Prevention and Control Measures to reduce the *Culicoides* populations in endangered areas can make a significant contribution towards the control of BT. This can be done by the use of insecticides and sterilization of *Culicoides* males by irradiation. However, the most effective and practical approach to endemic BT is prophylactic immunization. Attenuated vaccines are highly effective, but problems might arise in areas where several serotypes exist. No BT vaccines have been used in camelids.

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2.2.4 Retrovirus Infection

Several retroviruses have been isolated which can cause leukemias, lymphomas and sarcomas in mice, rats, chickens, cats and a variety of other animals. There are several diseases in livestock caused by viruses belonging to the *Retroviridae* family:

- enzootic bovine leukosis (BVL),
- ovine pulmonary adenomatosis (OPA),
- caprine arthritis encephalitis (CAE),
- equine infectious anemia (EIA),
- visna/maedi strain.

Several serological studies have been conducted in NWC and OWC for the detection of antibodies against BVL and OPA. Chauhan et al. (1986) found no antibodies to BLV in 283 sera from Indian dromedaries. Wernery and Wernery (1990) examined sera from 986 UAE dromedaries with the agar gel immunodiffusion test. All these sera were negative. However, the authors had diagnosed lymphatic leukemia in UAE dromedaries. Ten dromedaries affected by lymphatic leukemia within a six year period did not have antibodies to the bovine leukemia virus in their serum or in organ homogenates (Wernery and Kaaden, 1995). The affected dromedaries were all above 8 years of age and all exhibited a very high leukocytosis (Table 42), composed primarily of lymphoblasts (Fig. 101).

All of the dromedaries diagnosed with leukemia died within 6 months. Upon

Table 42 Cases of lymphoblastic leukemia in the dromedary in the UAE

Cases	WBC x 10^3/mm^3	RBC x 10^6/mm^3	Hb g/dL	Differential Cell Count			
				% Lympho-cytes	% Neutro-phils	% Eosino-phils	% Mono-cytes
1	818.5	8.5	12.0	99	1	0	0
2	126.4	9.2	12.6	99	1	0	0
3	157.4	9.6	13.7	100	0	0	0
4	142.0	9.6	14.0	99	1	0	0
5	44.7	6.8	11.0	98	2	0	0
6	949.3	7.5	12.0	94	6	0	0
7	45.1	7.3	11.6	98	2	0	0
8	204.8	5.3	8.9	92	8	0	0
9	226.0	7.9	10.3	98	2	0	0
10	217.0	3.3	7.0	98	2	0	0

WBC = white blood cells; RBC = red blood cells; Hb = hemoglobin

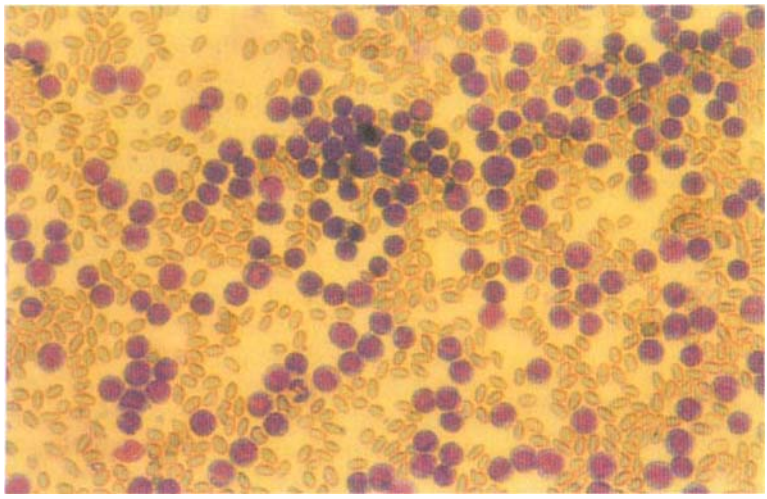

Figure 101 Lymphoblastic leukemia in a dromedary (Sudan black stain, case No. 1 from Table 42)

autopsy, enlarged lymph nodes, secondary pyelonephritis, bronchopneumonia and endometritis were seen (Afzal and Hussein, 1995; Wernery and Kumar, 1996). Histopathological examinations showed extensive infiltration with neoblastic lymphoid cells in the lungs (Fig. 102), spleen and lymph nodes. Two pregnant dromedaries diagnosed with leukemia gave birth to healthy offspring with no abnormalities in their blood cell counts.

Ten mL of heparinized blood was drawn from each of two dromedaries with leukemia and given to two test camels intravenously. The blood of both experimental camels was examined regularly over a 1 year period. During this time, no hematological changes were detected, indicating that the disease is probably not infectious.

Antibodies to OPA were not detected in a serological survey conducted in Peru where alpacas grazed with sheep (Rivera

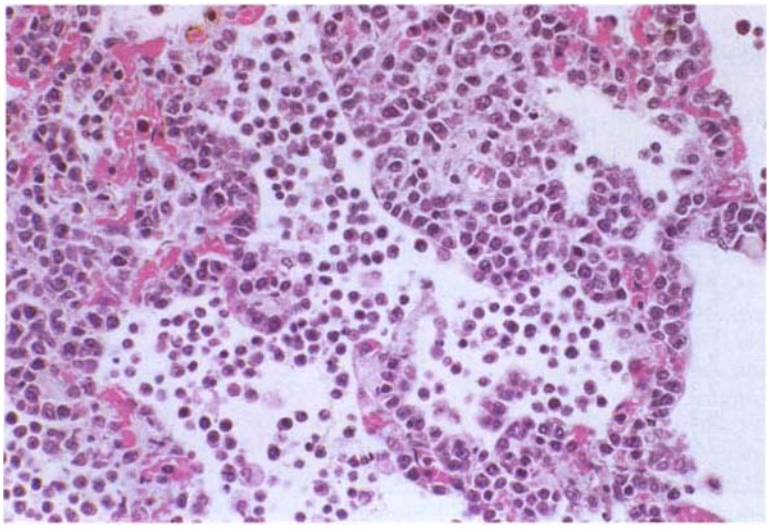

Figure 102 Neoblastic lymphoid cells in the lung of a racing camel suffering from lymphoblastic leukemia (HE stain)

et al., 1987), and none of the 270 llamas tested in Oregon, USA, seroconverted, although sheep in that region were infected with OPA (Picton, 1993). A serological study conducted in Peru also showed no evidence of antibodies to BVLV (Rivera et al., 1987). No antibodies to BVLV were found in 390 llamas from 9 farms located in 3 Argentinean provinces by Puntel et al. (1999). However, as in dromedaries, there have been several reports that llamas develop lymphosarcoma similar to that induced by BVLV (Mattson, 1994). Until a leukemia virus is identified from such cases, the susceptibility of camelids to leukemiavirus remains uncertain. A retrovirus has been isolated from a llama in association with an immunodeficiency syndrome (Underwood et al., 1992), but it is not proven that this virus has caused this syndrome (Vogel, 1992). The rare incidence of lymphatic leukosis in camels detected in the UAE, the similarities of the pathohistological findings and the apparent lack of a retrovirus led us to speculate that the described cases may be related to sporadic forms of bovine leukosis.

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2.2.5 Foot-and-mouth Disease

Foot-and-mouth disease (FMD) is a highly contagious disease occurring almost exclusively among cloven-hoofed animals. This most feared disease inflicts great economic (productivity and operating) losses worldwide because of international embargos. Opinions vary widely whether camelids are susceptible to the disease or not; therefore it is important to ascertain if they can develop the disease or serve as viral reservoirs.

Etiology FMD is caused by a RNA *aphthovirus* of the family *Picornaviridae*. At least

seven immunologically distinct serotypes of FMDV have been identified, of which A, O and C are the three most common strains in Europe. O appears to be the most common and C the least common. There is no crossimmunity between strains. Within the 7 serotypes, over 60 subtypes have been identified.

Epidemiology FMD was enzootic in parts of Europe, and still is in the Middle East, India, the Far East and South America. North America, Australia, New Zealand, and many countries in western Europe are free of the disease. These countries have stringent regulations preventing the introduction of the FMDV. FMD is of great interest with regard to NWC and OWC, because the disease is enzootic in many countries where camelids are reared. Saudi Arabia, for example (with a camel population of 800,000), imports approximately 6.5 million live animals, mainly sheep and goats, from Africa, Asia and Australasia. Animals from Africa and Asia bring their own FMDV strains, which spread within the nomadic herds of Saudi Arabia and neighboring countries. It would be of great importance to know if camels play a role in transmitting FMDV. The natural hosts of the virus are artiodactylids including cattle, sheep, swine and goats, but also many different wild animals. Most transmission is via aerosols, usually when animals are in close contact with each other, although under certain circumstances it may spread over long distances. Some ungulates can harbor the virus for a long period without developing vesicular lesions. The study of the epidemiology of FMD has been transformed by the use of molecular techniques to characterize individual strains of virus. By these methods, it has been possible to trace the movement of individual strains of FMDV from one country to another (Kitching, 1998).

Clinical Signs and Pathology Earlier studies have reported FMD epizootics in camels. This observation was based on clinical manifestations of the disease. After observing thousands of Afghani dromedaries, Pringle (1880) reported that the disease was widespread and that foot lesions were the most prevalent symptom. Steele (cited from Curasson, 1947) also described outbreaks similar to FMD in dromedaries.

According to Kowalevsky (1912), who observed outbreaks of the disease among Bactrian camels in Kazakhstan, the disease affects the lips, the buccal mucosa and the feet. Rohrer (1970) also believes that all artiodactyl ungulates can develop FMD. However, Leese (1918) and Curasson (1947) are skeptical of the earlier reports. They believe that these epizootics were all outbreaks of camelpox. Leese (1927) was not able to confirm FMD in dromedaries following inoculation attempts with material from bovine aphthae.

Recent studies by Moussa et al. (1987) in Egypt have shown that dromedaries are susceptible to FMD. The authors described ruptured vesicles and ulcera on the upper lips of four dromedaries. Additionally, ulcerations were seen between the teeth and on the teats. Two viral strains were isolated from these lesions and both were identified as FMDV, serotype O. A calf, a ram, a goat and a dromedary were artificially infected with this FMDV, type O. The calf and the goat did not develop any lesions, the ram developed hyperemia of the eyes and buccal cavity and the dromedary developed ulcera on the scarification site and two to three vesicles on the inside of the upper lip. The dromedary did not develop any antibodies to the artificial infection. However, the other animals seroconverted 3 weeks later. After an additional 5 weeks, all four animals were infected with a known bovine FMDV, type O. The calf developed a vesicle at the inoculation site and the goat developed ulcera on the dorsum of the tongue and the upper lip. Again the dromedary did not develop any antibodies following the second viral exposure.

Additional experimental studies on the dromedary by Nasser et al. (1980) and Moussa (1988), following intranasal infection with serotype O FMDV strains, yielded only slight or clinically inapparent manifestations. However, the virus was re-isolated from the pharynx and the feces over the course of 6 days following infection, whereby the highest titers were observed between the 3rd and 4th day p.i. Four weeks later, a second excretion of the virus was noted lasting for a week. Thereafter, virus detection was negative. Again there was no evidence of humoral antibody production. Similar reports in Saudi Arabia from Hafez et al. (1993) again showed that, following intranasal infection with the Egyptian strain Sharkia 0/2/72, neither clinical signs nor seroconversion were seen in the infected dromedaries.

Only Richard (1986) was able to identify antibodies to the FMDV serotypes O, C and SAT_2 in 2.6% of the sera from Nigerian dromedaries. However, Moussa (1988) is of the opinion that the substances identified were nonspecific inhibitory substances in the sera, frequently seen in camel serum, as opposed to specific antibodies.

Dromedaries kept for weeks in close contact with severe cases of FMD in cattle, sheep and goats in various FMD epizootics in Ethiopia (Richard, 1979), Oman (Hedger et al., 1980), Niger (Richard, 1986), Saudi Arabia and Egypt (Hafez et al., 1993) did not develop any signs of clinical signs. Further studies carried out by Abou Zaid (1991) revealed that dromedaries could contract FMD after experimental infection. Three camels were infected with FMDV strain 01/3/87 Egypt IDL intradermolingually and the fourth intradermally in the footpad. One camel and three bovine calves were kept as contact animals. At the same time, three bovine calves were inoculated with the same FMDV strain and one bovine calf and two camels were kept as contact animals. The three infected camels showed signs of FMD and the virus was isolated from blood, esophageal-pharyngeal (OP) fluid, feces and ruptured vesicles. The fourth camel that was inoculated with FMDV into the footpad did not develop any FMD symptoms. The contact camel did not show any lesions and no virus was isolated, but the three bovine calves contracted the disease and the virus was isolated. In the second group, the three infected bovine calves as well as the contact bovine calf developed FMD, but the contact camel did not show any clinical signs. These investigations showed that camels in contact with cattle or camels with FMD did not contract the disease. However, camels could contract FMD when intradermolingually infected. These experiments also showed that infected, diseased camels seroconverted to FMDV during the first week, but the antibodies disappeared after 6 weeks. The camel not showing any lesions did not develop antibodies to FMD.

Several studies of NWC susceptibility to FMDV have been carried out (Mancini, 1952; Konigshofer, 1971; Moro, 1971; Moussa et al., 1979; Lubroth and Yedloutschnig 1987; Lubroth et al., 1990; Callis and Craig, 1992). As with OWC, the opinions on whether NWC are susceptible to FMD or not vary widely. A few surveys suggest that NWC are resistant to natural FMD infection (Mancini, 1952; Paling et al., 1979; Tantawi et al., 1984) while others describe the susceptibility of NWC to experimental infection with FMDV (Moussa et al., 1979; Lubroth et al., 1990) in a limited number of animals and with variable results. Unlike cattle, which are known to carry FMDV for long periods of time, little is known about the carrier ability of camelids. In a well-executed study by Fondevila et al. (1995), further evidence was provided that llamas are resistant to FMD infection. The authors conducted an experimental trial with FMDV serotypes A79, O3 and O1 to evaluate the ability of FMD to infect susceptible llamas exposed either directly to affected livestock or indirectly to llamas that had been

directly exposed to affected livestock. Six pigs were inoculated with three different types of FMDV by different routes. Thirty llamas were placed together with the infected pigs and later interspersed with an additional 30 llamas after the exposure to the pigs. Forty susceptible livestock (pigs, bovine calves, goats and sheep) were then added to the entire group of 60 llamas to detect possible transmission of FMDV from llamas. Only 2 of 30 llamas directly exposed to the FMD pigs developed minor lesions, seroconverted and yielded virus in blood or OP fluid. A third llama from this group also seroconverted, but showed no lesions and did not shed the virus. All control animals introduced to the 30 contact-exposed llamas failed to develop lesions or antibodies and failed to yield any FMDV.

The divergent results from scientists of various countries underline the importance of examining and qualifying the pathogenesis of FMDVs in camelids. There is now agreement that NWC and dromedaries can contract the disease after experimental infection and by very close contact with FMD-diseased livestock. NWC and dromedaries are not very susceptible to FMD and do not present a risk in transmitting FMDVs to other susceptible animal species. However, all FMD antibody-positive animals should be considered to be potentially infected, as it is known that immune animals in contact with live FMDV can become carriers. The pandemic serotype O virus (now named the PanAsia strain) has currently reached a global extension and has out-competed all other strains of FMD. It has also caused disease in Bactrians in Mongolia. It is now believed that NWC and dromedaries are more or less resistant to FMD infection, and that they play no role in transmitting the virus to domestic livestock. *Camelus bactrianus*, however, is susceptible to FMD and may transmit the virus to other artiodactylids (Indian elephants are susceptible to FMD, Africans not).

Stehman et al. (1998) reported a *picornavirus* infection in llamas that caused abortion in 15 llamas. This occurred over a three and a half month period at an average 220 days gestation. Along with the *picornavirus* infection, diabetes mellitus was observed in the adult llamas. The virus was isolated from two fetuses; serum neutralizing antibodies to the *picornavirus* were found in the fetal fluids as well as in two llama herds with a similar clinical syndrome of diabetes mellitus.

An encephalomyocarditis virus (EMCV), which is a *picornavirus*, has been isolated from a 2-year-old dromedary in an American zoological collection (Wells et al., 1989). Gross pathology consisted of excessive pericardial fluid, epicardial hemorrhages and pale foci within the myocardium. The virus was isolated from the heart. It is believed that rodents may have transmitted the EMCV.

Diagnosis The clinical signs of FMD are indistinguishable from vesicular stomatitis, vesicular exanthema of swine *(calicivirus)* and vesicular disease in pigs (enterovirus of the *Picornaviridae* family). Laboratory methods are therefore necessary for diagnosis. These methods include complement fixation test, ELISA, virus neutralization and agar gel precipitation. The virus can be isolated on different cell lines, including fetal camel kidney (Farid et al., 1974).

Treatment and Prevention There is no cure for FMD. The most effective preventive measure is to prohibit introduction of animals or animal products into FMD-free countries from countries that have the disease. Many European countries have banned routine vaccinations against FMD because most of the outbreaks have been traced to improperly inactivated vaccines or escape of the virus from the production site. Furthermore, ruminants (in particular cattle), continue to carry live FMDV in their

pharynx after contact. Animals immune to FMD after vaccination can still become carriers after contact with field strains during outbreaks. Cattle can harbor FMDV for up to 3 years. FMD vaccine is an inactivated preparation; attempts to take advantage of new molecular biological technology to produce better FMD vaccines have been unsuccessful. The duration of immunity after FMD vaccination is rarely longer than 6 months (Kitching, 1998). In countries where vaccines are used, the virus from the outbreak must be isolated and typed to determine whether the field strain is homologous to the vaccine strain being used. FMD vaccines have not been used in camelids.

2.2.6 Vesicular Stomatitis

Vesicular stomatitis (VS) is another vesicular disease which is indistinguishable from FMD. VS is caused by a *rhabdovirus* and there are two major types: New Jersey and Indiana. Few studies involving the susceptibility of NWC to VSV have been conducted. It is believed that natural infection rarely occurs, as llamas that had been in close contact with diseased cattle did not contract VS (Thedford and Johnson, 1989). The llamas even shared the same watering and feeding facilities with the diseased cattle and they did not seroconvert to VSV. Two hundred and seventy llamas which were serologically tested to strains Indiana and New Jersey in Oregon were also negative (Picton, 1993). However, one natural case of VS in lamoids has been reported (Fowler, 1998). Alpacas and llamas have been shown to be susceptible to an experimental infection with VSV. Vesicles appeared at the inoculation site at the dorsum of the tongue and the animals developed fever and anorexia (Gomes, 1964). Fluids taken from these vesicles of NWC caused disease in cattle. No reports exist on VS in OWC.

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2.2.7 Bovine Viral Diarrhea

Bovine viral diarrhea (BVD) and mucosal disease (MD) are epidemiologically different diseases of cattle that have different

pathogeneses, although both are caused by the same virus. BVD can occur at any age in postnatal life as a result of an acute mild infection. However, severe clinical disease with agalactia and diarrhea may also occur. In the late 1980s, an acute and fatal syndrome of calves was reported in America. The disease was characterized by a profound thrombocytopenia and hemorrhages. In contrast, MD is a severe disease with fatal consequences in cattle 6 months to 2 years of age. It occurs only in those cattle that have suffered a non-cytopathic viral infection in the early stages (40–120 days) of fetal life and in which the virus has persisted as a result of immunological tolerance of the fetus (Thiel et al., 1999). Superinfection, either through mutation of the non-cytopathic to a cytopathic BVDV or through an exogenic cytopathic infection, is believed to trigger MD. Brownlie et al. (2000) suggest the following definition for MD: "MD is a fatal condition, mainly of young cattle aged 6 to 18 months, with characteristic erosive pathology in the oral/intestinal mucosa from which the cytopathogenic biotype of BVDV can be isolated. The clinical disease is typically rapid in onset, although chronic debilitating forms can occur." After the persistently infected (p. i.) calf is born, it excretes the virus during its entire life. It is remarkable that both the non-cytopathic and the cytopathic forms can be isolated from MD cases. The virus is widespread in cattle populations worldwide and has also been isolated from NWC and OWC (Evermann et al., 1993; Mattson, 1994; Hegazy et al., 1998).

Etiology Bovine viral diarrhea virus (BVDV) is a small RNA virus of the *Flaviviridae*. Together with the viruses of border disease and classical swine fever virus it forms the genus *Pestivirus*. The three viruses are antigenically related. Strains isolated from newborn calves and persistently infected cattle are generally non-cytopathic (BVDVnc), while those from tissues of cattle suffering from MD are usually cytopathic (BVDVc). Today two genotypes of BVDV are recognized: BVDV-1 and BVDV-2. BVDV-1 has a worldwide distribution, whereas BVDV-2 is largely restricted to the USA and Canada.

Epidemiology Postnatal infection with the virus is acquired by ingestion or inhalation of contaminated material and results in the development of serum neutralizing antibodies. This is usually a clinically unrecognizable infection. On the other hand, with infection of a non-immune pregnant animal, the virus is capable of crossing the placental barrier and invading the fetus. While the dam seroconverts without showing signs of disease, the fetus is immunotolerant in the early stages of pregnancy. This congenital infection can result in a wide spectrum of abnormalities: fetal death, congenital defects, or a persistent lifelong infection without clinical signs. The outcome is mainly dependent on the stage of fetal development during which infection takes place.

Clinical Signs and Pathology Serological studies indicate that NWC and OWC are susceptible to infection with the BVDV. The results of serological studies identifying BVDV antibodies in the dromedary have appeared from Tunisia with 3.9% positive (Burgemeister et al., 1975), from Oman with 6.7% (Hedger et al., 1980), from Sudan with 15.5% and 15.7% (Bornstein and Musa, 1987; Bornstein et al., 1989) and Somalia with 3.4% (Bornstein, 1988). Bohrmann et al. (1988) did not identify any antibodies to BVDV in Djibouti. Using the serum neutralization test, Wernery and Wernery (1990) explained the higher incidence of BVD in UAE breeding camels (9.2%) when compared to racing dromedaries (3.6%) with their larger breeding herds and closer contact with cattle herds. In a later survey (CVRL Annual Report, 1998), these findings were confirmed using

an antibody ELISA. The incidence of BVDV antibodies in 552 camels tested was 0.5% in racing camels and 6.4% in breeding camels. The presence of neutralizing antibodies to BVDV was 11% in Egypt with a peak of 23% in one area (Hegazy et al., 1993). In another Egyptian survey, Tantawi et al. (1994) detected 4.3% BVDV positive dromedaries and Zaghhana (1998) found that camels from Egypt exhibited an even higher prevalence (52.5%) of neutralizing antibodies to BVDV. In a serological survey conducted in Peru involving 117 alpacas that grazed with cattle and sheep, the prevalence of antibodies to BVDV was 11% (Rivera et al., 1987) and Picton (1993) reported a prevalence of 4.4% in 270 llamas from Oregon in the USA. A recent study by Puntel et al. (1999) found 2.05% (8/390) reactors to the BVDV in llamas from nine farms located in three different provinces in Argentina.

Cattle suffering from BVD and MD show lesions in the alimentary tract. The pathological changes in MD are much more severe than in BVD. The MD lesions are often found only in the upper alimentary tract. In both BVD and MD, pathological changes consist mainly of erosions and ulcers of varying severity. In camels these lesions have not been described.

BVD infections have been described in dromedary calves from Egypt (Hegazy et al., 1998) causing intrauterine death, stillbirths, weak calf syndrome with congenital deformities, neonatal respiratory distress syndrome and acute hemorrhagic gastroenteritis. BVDV was isolated from lymphoid tissues, spleen, brain and kidney on bovine kidney cells causing a cytopathic effect (CPE). The virus was also demonstrated by immunofluorescence in different organs. In another publication, Hegazy et al. (1995) state that the main cause of abortions in dromedaries is the BVDV, which can reach 50% in some herds.

In the UAE, adult dromedaries and calves that have died of other causes are routinely virologically screened, including the fluorescence test for the presence of the BVDV. So far the results have always been negative (Wernery et al., 1992). VDV has been isolated from dead llamas that suffered excessive nasal discharge and diarrhea (Mattson, 1994), indicating an MD-like disease.

Over the last years our knowledge about BVD in camelids has increased and it seems that both NWC and OWC can contract the disease. However, extensive studies are necessary to elucidate the entire disease pattern in this animal species, as with bovines, through extensive field observations and laboratory studies. Investigations in bovines have led to a new understanding of the complex epidemiology and pathogenesis of BVD and MD and one can hope that this will also be the case in the camelid family. Since only one publication on BVD has been published each on NWC and OWC, the authors prefer to keep this chapter under "Nonpathogenic Viral Infections".

Diagnosis Diagnosis of BVD and MD requires laboratory support in the form of virus isolation, virus antigen detection and serum antibody determination. Skin biopsies are the tissues of choice for the diagnosis of BVDV using immunohistological techniques and are always positive in persistently infected animals (Braun et al., 1999). This method should also be applied in the diagnosis of this disease in camelids.

Treatment and Prevention Economic losses caused by BVD/MD mainly arise from prenatal infections. It is therefore essential to remove all persistently infected animals and to vaccinate heifers prior to first breeding. Since it is known that BVDV also causes abortions in camels, it may be necessary to adopt control and vaccination strategies similar to those carried out in cattle. Live and inactivated vaccines have been widely used in several countries. Live vaccines are not recommended in camelids

because a variety of adverse effects have been observed using live BVDV vaccines in cattle. Inactivated vaccines are safer and can provide good protection. It has been shown that NWC seroconverted after a regimen of three vaccinations using a inactivated-virus preparation (Mattson, 1994).

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2.2.8 Rift Valley Fever

Rift Valley fever (RVF) is an arthropod-borne viral disease of animals including humans, but mostly found in ruminants. Infection in humans is primarily due to contact with material from infected carcasses (Hoogstraal et al., 1979). In addition to the human health hazards, RVF epidemics regularly cause serious economic damage to animal owners through the loss in production and fatalities, exacerbated by the 100% abortion rate at all stages of pregnancy. Strikingly, all of the RVF epizootics described to date have followed unusually severe rainy seasons, probably indicating a very large insect population as a vector prerequisite (Huebschle, 1983). RVF does not occur in very arid areas.

Etiology The Rift Valley Fever Virus (RVFV) is a member of the *Phlebovirus* genus of the family *Bunyaviridae.* The bunyaviruses are spherical, 80 to 120 µm in diameter, and have a host cell-derived, bilipid layer envelope through which virus-coded glycoprotein spikes project. No significant antigenic differences have been detected between RVF isolates, but differences in virulence have been demonstrated.

Epidemiology For more than 70 years, RVF epidemics have occurred at prolonged intervals in eastern and southern Africa. It has been accepted that the virus is endemic in indigenous forests, where it circulates in mosquitoes and vertebrates spreading to livestock-rearing areas when heavy rains favor the breeding of mosquito vectors. Many different mosquito species can serve as vectors. The virus was first isolated in 1931 in livestock on a farm located in the Rift Valley of Kenya. The virus is now endemic in much of sub-Saharan Africa with epidemics in West Africa. It has also spread into Egypt and clearly has the potential to spread elsewhere.

Epidemiologic studies of RVF have always been performed during epizootics or immediately afterwards. This was the case following epidemics in Sudan, Kenya and Egypt. Several studies also included the respective local dromedary populations. Scott et al. (1963) reported outbreaks of RVF in cattle following severe rainfall in Kenya, parallel to a drastic increase in abortions in dromedaries. Antibodies to RVF were found in 45% of the dromedaries examined during this outbreak. The authors incrimi-

nate the RVFV for the increased rate of abortions; however, no virological studies were performed to substantiate this supposition. Meegan et al. (1979) also observed an increased abortion rate in dromedaries during a RVF epizootic in Egypt. In this case, the epidemic was supposedly carried by Sudanese dromedaries to Egypt (Hoogstraal et al., 1979), as severe epidemics were raging in northern Sudan at the time (Eisa et al., 1977). During this period, Hoogstraal et al. (1979) registered 31 RVF reactors in dromedaries. Other than the increased abortion rate during outbreaks of RVF, no other clinical signs have been so far observed in camels (Davies et al., 1985). Aly (1979) found antibodies with the HI-test in 15.6% dromedaries in Egypt and Walker (1975) described abortions and deaths in young one-humped camels during RVF outbreaks. Peters and Meegan (1981), however, observed only a subclinical form of RVF. Olaleye et al. (1996) examined 180 dromedaries with the hemagglutination inhibition test and serum neutralization test in Nigeria and detected 3.3% positive cases. The authors stressed the involvement of camels in the transmission cycle of RVFV.

Imam et al. (1978) and Eisa (1981) were able to isolate the virus from a healthy, naturally infected dromedary. Experimental infections with the RVFV have induced no clinical signs in non-pregnant dromedaries (Davies et al., 1985). In spite of high RVF antibody titers, the same authors were not able to determine an increased rate of abortion in infected dromedaries.

Severe RVF epidemics have recently occurred in East Africa (Anonymous, 1998). Many domestic animals and humans had been affected in vast areas of Kenya, southern Sudan and northern Tanzania in December 1997 and January 1998.

Clinical Signs During the last RVF outbreaks in East Africa, the WHO received many reports of high mortality in camels throughout the affected area. Some descriptions of morbidity and mortality were highly suggestive of camelpox or parapox *(Ecthyma contagiosum)*, with ballooning of the head and upper neck, swollen eyes and huge mucoid membrane sloughs in the mouth covering some ulcers.

However, the general disease pattern was that of fever and abortion, which were the predominant features, but early neonatal death and jaundice have also been observed. Since no RVFV was isolated from camels during these outbreaks it is not clear if the disease was caused by RVF. The authors therefore prefer to keep this part of RVF under the overall chapter "Nonpathogenic Viral Infections" until proven otherwise.

Diagnosis Definitive diagnosis of RVF depends on virological and serological investigations, since other arthropod-borne virus diseases tend to occur under the same climatic conditions. This is especially true for Wesselsbron disease, which can also cause mortality in lambs, kids and calves and abortion in ewes. However, RVF is associated with higher mortality and abortion rates. Lesions in the livers of young animals also differ in both RVF and Wesselsbron disease. Hepatic changes are usually less extensive in RVF compared to Wesselsbron disease. Specimens for laboratory confirmation should include heparinized blood, liver, spleen, kidney, lymph nodes and brain from aborted fetuses for virus isolation on Vero and BHK 21 cells or suckling and weaned mice. Antibodies to RVF can be demonstrated by CFT, AGID, HIT and ELISA. Viral antigen can also be detected by impression smears of infected tissues by immunofluorescence.

Treatment and Prevention Measures such as chemical control of vectors, movement of livestock to higher altitudes, or the confinement of animals to mosquito-proof stables are usually impractical or too late. Immunization remains the only effective way to protect livestock.

Although it has still not been determined decisively whether dromedaries actually develop RVF, Guillaud and Lancelot (1989) have concerned themselves with the production of a vaccine. The authors determined that the attenuated vaccine strain (MVP-22) has yielded satisfactory results in the dromedary. Following a single subcutaneous vaccination, 18 of 22 dromedaries developed neutralizing antibodies. A challenge infection with the RVFV was not performed. As in other viral diseases already described, the camel appears to be susceptible to RVFV. Further intensive research, however, is necessary to clarify the pathogenicity of this virus in the camel.

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2.2.9 Rinderpest

The clinical presentation and dangers of rinderpest, the scourge of cattle husbandry, have been known for centuries. This disease led to the foundation of the first European veterinary faculties and the implementation of laws governing contagious diseases. Rinderpest is an acute or subacute, highly contagious, febrile viral disease of cloven-hoofed mammals, the artiodactylids. The disease is characterized by high mortality and is typified by hemorrhagic/septicemic symptoms and mucosal erosions of the entire alimentary tract. Among the larger domesticated animals, cattle, water buffaloes and yaks are susceptible to the virus. Different breeds of cattle have varying degrees of resistance to

the virus. Of the smaller domesticated animals, sheep, goats and swine are vulnerable to the disease under natural conditions. Wild animals exhibit varying susceptibility and play an important role in the epizootiology of rinderpest as virus reservoirs. There is evidence that the primary means of infection is via a virus-containing aerosol (Munz, 1983).

Etiology The rinderpest virus belongs to the order *Mononegavirales,* subfamily *Paramyxoviridae,* genus *Morbillivirus.* It is closely related to the viruses that cause canine distemper, phocine distemper, measles and peste-des-petits-ruminants. Individual rinderpest virus strains vary in their pathogenicity in various species.

Epidemiology The natural hosts for the rinderpest virus are all members of the order *Artiodactyla.* Natural rinderpest has never been reported in NWC, but experimental studies have shown that llamas develop a mild febrile response with a short clinical course of 3–5 days (Fowler, 1998). As in foot-and-mouth disease, there are differing opinions as to whether OWC are susceptible to the rinderpest virus or not.

Clinical Signs and Pathology Reports from the turn of the century mention severe outbreaks of rinderpest among camels. Curasson (1947), who reported the big rinderpest epizootic in Niger in 1892, learned that dromedaries developed severe diarrhea and hematuria. Vedernikov (1902) and Tschegis (1902) (cited from Curasson, 1947) saw cases of rinderpest among Bactrian camels in the region around Baku in 1898. Tartakowsky (1899) produced rinderpest experimentally in four Bactrian camels, one of which died. However, he could only elicit slight clinical reactions in two dromedaries. Lingard (1905) inoculated five Indian dromedaries with cattle blood infected with the rinderpest virus. The dromedaries developed the following clinical signs: fever, vesicles and ulcera in the mouth, eruptive skin lesions and, in one case, diarrhea. One bull (bovine) developed rinderpest after being given blood from one of these dromedaries. Cross (1919) injected the rinderpest virus into three Indian dromedaries, one of which later died. Conti (1913) too diagnosed rinderpest symptoms in dromedaries in Eritrea. The author believed that the epidemic in dromedaries was more of a prophylactic problem and that disease control measures should be instituted for the Eritrean animal population.

According to Haji (1932–1933), dromedaries were also affected during outbreaks of rinderpest among cattle and buffalo in India. The affected animals had fever, ruminal atony, ocular discharge, depression, severe diarrhea occasionally mixed with blood, as well as vesicles on the lips and hard palate that developed into ulcera. The mortality was 20 to 40%. Dhillon (1959) reported similar discoveries in India. Between 1948 and 1958, the author observed more than 15 outbreaks of rinderpest among dromedaries with mortality rates of up to 100%. The dromedaries' clinical signs were similar to those in cattle. Srinivasan (1940) was successful in controlling an outbreak of rinderpest among dromedaries after "goat blood virus" inoculation from infected goats.

Contrary to all these statements, various groups have reported that the camel is not susceptible to rinderpest. Littlewood (1905) in Egypt, Pecaud (1924) in Chad and Samartsev and Arbuzov (1940) in the Asiatic region of Russia have reported that camels are not susceptible to natural infections. Leese (1927), traveling around India, neither observed nor heard of outbreaks of rinderpest in dromedaries. The author does not exclude the fact that slight clinical signs may be possible and that these may be overlooked.

Until the middle of the twentieth century, epidemics with clinical signs similar to

rinderpest were diagnosed on the basis of clinical signs as well as the tendency to spread among other ungulates living in close proximity with the dromedaries. Laboratory methods in the diagnosis of rinderpest were introduced later. Scott and MacDonald (1962) confirmed a severe outbreak of rinderpest among wild animals in northern Kenya in 1960. Dromedaries in this region did not develop the disease and antibody studies on 60 dromedary sera with lapinized rinderpest antigen were negative. Chauhan et al. (1986) examined 283 dromedary sera from India serologically and did not detect any antibodies. However, Maurice et al. (1967) found rinderpest antibodies in 7.7% of the dromedary sera examined from Chad. Singh and Ata (1967) also detected antibodies to the rinderpest virus in 10% of the Sudanese and Egyptian dromedaries examined, and Abou-Zaid (1991) found rinderpest-neutralizing antibodies in 5.2% of 536 dromedaries in Egypt.

Experimental infections in dromedaries with the rinderpest virus have yielded further information regarding the susceptibility of this species to rinderpest. Only one out of ten dromedaries infected experimentally with an aerosol of the rinderpest virus developed signs of an asymptomatic, non-contagious infection. Leukopenia and antibodies to the rinderpest virus were observed in the serum of this animal. Zebus, serving as contact animals, did not develop the disease and also developed no antibodies to rinderpest (Provost et al., 1968). Singh and Ata (1967) utilized two virulent and two attenuated (vaccine) rinderpest strains in their experimental trials. Dromedaries that were infected with these strains subcutaneously did not develop rinderpest. A slight increase in body temperature was observed following inoculation with the virulent strains. Dromedaries given the attenuated vaccine developed only a low antibody titer, whereby high neutralizing antibody titers were observed 28 days after experimental infection with the virulent strains. This experiment also showed that infected dromedaries did not transmit the virus to susceptible cattle. Taylor (1968) confirmed these results through further trials and performed additional experiments on dromedaries using the rinderpest virus. The results of these experiments were as follows:

1. Following experimental intravenous infection with a virulent rinderpest strain (Kabete O), the virus was re-isolated between the 3rd and 8th day from the blood of the infected dromedary. This animal also developed neutralizing antibodies.
2. One of two dromedaries infected subcutaneously with the virulent rinderpest strain RGK/1 developed a slight viremia lasting 6 days, though both animals developed neutralizing antibodies.
3. Slight viremia occurred in two out of three dromedaries that were in close contact to a bull infected with rinderpest. One dromedary developed slight pyrexia. This was the only clinical manifestation that was observed during the experiments.
4. Although the rinderpest infection originated in cattle, it was not possible to transmit the rinderpest virus from infected dromedaries to cattle or other dromedaries.

In order to determine the susceptibility of camels to experimental rinderpest infection, further experiments were carried out by Chauhan et al. (1985). The authors inoculated 10 mL of a 10% spleen suspension collected from a buffalo calf suffering from rinderpest, into two healthy 8 to 12-month-old camels. One camel was given subcutaneous and the other intravenous inoculation. No distinct clinical signs of rinderpest lesions were detected except a slight hyperemia of visible mucous membranes and mild diarrhea. A post mortem examination of one of the camels infected with rinderpest virus did not reveal any lesions.

Chauhan et al. (1985) further showed that blood which was collected from the experimentally inoculated camels at the height of febrile reaction and injected into two susceptible buffalo calves caused the development of typical clinical signs and lesions of rinderpest in these calves within 6 days of inoculation.

From all these experiments it can be assumed that OWC are susceptible to rinderpest, and might develop mild clinical signs, especially through contact with infected cattle. It is less likely that they serve as vectors for the rinderpest virus; therefore they do not appear to play a major role in the epizootiology of rinderpest.

A new epizootic disease has affected thousands of camels in Ethiopia in 1995 and 1996 characterized by a febrile, highly contagious respiratory syndrome. The morbidity rate reached over 90% with a mortality ranging between 5 and 70%. The major clinical signs were sero-mucopurulent nasal discharge, lacrimation, coughing, dyspnea and abdominal breathing. Swelling of the submandibular area and diarrhea was reported in some cases. Two *morbillivirus* strains, closely related to the pestedes-petits-ruminants (PPR) virus, were isolated from diseased camels and a similar disease was reproduced in goats and sheep after inoculation of the camel viruses. *Streptococcus equi* spp. *equi* was also isolated from diseased camels. Further investigations are currently being undertaken to reproduce the disease in camels (Roger et al., 2000).

Diagnosis Several handbooks and scientific papers detail the diagnosis of rinderpest. A presumptive diagnosis of rinderpest can be made on the basis of the clinical signs and gross pathology in cattle, but might be very difficult in camelids. In areas where the disease is not prevalent, it is essential to obtain laboratory confirmation of the diagnosis as soon as possible. Mirchamsy et al. (1971) reported that the rinderpest virus can be readily grown on camel kidney cells.

Treatment and Prevention Confirmed rinderpest outbreaks are controlled by the slaughter and disposal of all infected and contact animals as well as by the imposition of rigid quarantine and animal movement controls.

Prevention of rinderpest in endemic areas requires annual vaccination of all calves up to 2 years of age with the attenuated Kabete "O" strain. This is an inexpensive, freeze dried vaccine that is highly effective (Coetzer et al., 1994). It has not been used in camelids.

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2.2.10 Unusual Arboviruses

Arboviruses (**arthropod-born** viruses) are primarily vector viruses that multiply in blood-sucking insects and/or are transmitted to vertebrates via the insect's bite or sting. They are widespread in the tropics and subtropics, but their significance in camelids is not known.

Wood et al. (1982) isolated the Kadam virus from *Hyalomma dromedarii* ticks collected from the immediate vicinity of a dead dromedary in Saudi Arabia. It was not possible to determine whether this animal's death was due to the Kadam virus. The pathogenicity of the Kadam virus for the dromedary, cattle and humans has not yet been determined.

Five strains of the Quaranfil virus were isolated by Converse and Moussa (1982) from *Hyalomma dromedarii* ticks collected in Kuwait, Iraq and Yemen. The significance of this finding has not yet been determined.

The Akabane virus can cause epizootics and spontaneous abortions, premature births and congenital deformities in cattle, sheep and goats. The virus is widespread in Africa and Asia and appears to be en-

demic to the Arabian Peninsula. Al Busaidy et al. (1988) found that 50% of the dromedaries examined in Oman had neutralizing antibodies to the Akabane virus. It has not been ascertained whether the virus causes abortions in the dromedary.

Anderson and Casals (1973) isolated four strains of Dhori virus from ticks *(Hyalomma dromedarii)* found on Indian dromedaries. Additionally, the author discovered neutralizing antibodies to the virus in 48 out of 50 dromedary sera. No clinical signs of disease were observed in the seropositive dromedaries. The virus has also been isolated from dromedary ticks in southwest Asia and Africa. Williams et al. (1973) found three non-classified *Arboviruses* in *Hyalomma* ticks from Egyptian dromedaries: Wanowrie, Thogoto and Dhori viruses. These viruses have been widely spread by the migratory patterns of the indigenous animals, including the camel caravans. The veterinary importance of these viruses is unknown.

A survey for antibodies against *flaviviruses* in 269 slaughter camels was carried out in Nigeria (Baba et al., 1990). The antibody prevalence against *flaviviruses* was noted as follows: Wesselsbron: 60.2%, Yellow Fever: 54.0%, Potiskum: 66.2%, Dengue type 1: 4.5%, Banzi: 5.4% and Uganda S: 0%. The importance of the high prevalence of some of the *flavivirus* antibody in camels was not evaluated, but the authors believe that there is a potential for infected camels to play an important epidemiological role in the spread of these viruses to humans and livestock. Similar findings were reported by Kemp et al. (1973) who isolated the following viruses from camel blood injected into infant mice: Thogoto, West Nile and Wesselbron.

Crimean-Congo hemorrhagic fever virus (C-CHFV) is widely distributed throughout the arid regions of Africa, the Middle East, southern and eastern Europe and Asia (Hoogstral, 1979). The infection is enzootic, but mainly asymptomatic in many animal species such as cattle, sheep, goats, camels and hares (Schwarz et al., 1996). Thirty species of ticks, particularly the genus *Hyalomma,* act both as reservoir and vector. Humans become infected by tick bites or through close contact with infected animals or humans. Several reports deal with the detection of C-CHF-antibodies from different animal species as well as with the isolation of the virus from animals. Causey et al. (1970) isolated 35 virus strains in Nigeria from cattle blood, from liver and spleen of a hedgehog and from four species of ticks and *Culicoides* spp. C-CHF-antibodies were found in Iranian men (13%), sheep (38%), goats (36%), cattle (18%) and small mammals (3%) with the agar gel immunodiffusion test, but no positive cases were detected in camels (Saidi et al., 1975). However, C-CHF viral antibody was demonstrated in 14% (600/4301) of camels imported into Egypt by the agar gel diffusion and the indirect fluorescent antibody techniques (Morrill et al., 1990). Hassanein et al. (1997), who performed a serological survey on humans and livestock in Saudi Arabia using the reversed passive hemagglutination inhibition test, found antibodies in humans (0.8%), sheep (3.2%), goats and cattle (0.6%), but no positive cases in horses and camels. The *Hyalomma* tick was most probably responsible for epidemics in Iraq (Tantawi et al., 1980), the UAE (Suleiman et al., 1980) and Oman (Scrimgeour et al., 1996). However, C-CHFV was not isolated from camels during an epidemiological survey on ticks conducted in Saudi Arabia (El-Azazy and Scrimgeour, 1997), although camels had the highest rate of tick infestation. The importance of C-CHFV for the camel is unknown. Serological examination for the virus in a small number of dromedaries suffering from hemorrhagic diathesis in the UAE (see 1.1.4) was negative (Wernery et al., 1992).

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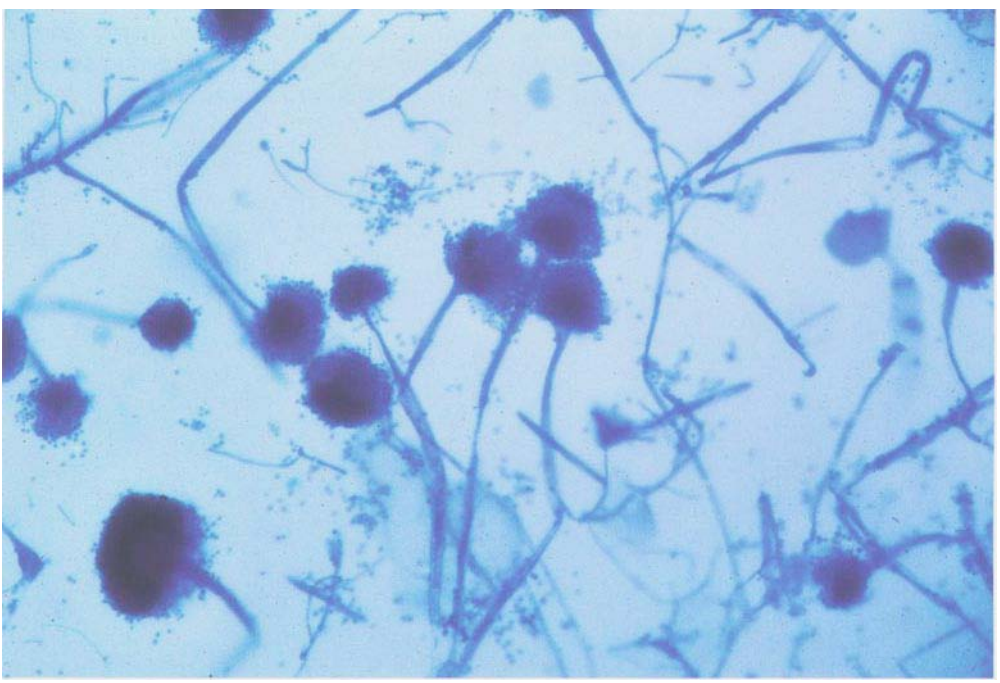
3 Fungal Diseases



Most agents of mycoses exist as saprophytes in soil, decaying vegetables and dung. The soil reservoir is the primary source of most fungal infections, which can be contracted by inhalation, ingestion or by contact with infected individuals or equipment. Pathogenic fungi establish infection in apparently healthy hosts and such diseases as histoplasmosis, coccidioidomycosis and blastomycosis are regarded as primary systemic mycoses. Opportunistic fungi usually require a host that is debilitated by stress, metabolic acidosis, malnutrition or neoplasia to establish infection. Prolonged exposure to antimicrobials or immunosuppressive substances can increase the likelihood of infection by opportunistic fungi like *Aspergillus, Mucor, Cryptococcus* and *Candida.*

Dermatophytosis (ringworm) is an infection of keratinized tissue (skin, hair, nails) by several genera of fungi called dermatophytes. All domestic animals are susceptible and the fungi are found worldwide. A few dermatophytes *(Microsporum [M.] gypseum)* normally inhabit soil (geophilic) and can cause disease in animals and humans. Some dermatophytes *(M. audouinii)* are adapted to humans and seldom infect animals (anthropophilic) and others are primarily animal pathogens *(M. canis, Trichophyton [T.] equinum),* but can also cause disease in man (zoophilic).

Very little is known about fungal diseases in camelids. This chapter tries to summarize current knowledge of these microorganisms.

3.1 Mycotic Dermatitis

Etiology Various fungal species can produce infection of the epidermis, of which the species causing dermatophytosis (ringworm) are the most common in camelids (Table 43).

Dermatophytes are a group of closely related fungi that utilize keratin for their growth. Over 38 species of dermatophytes are known and those that affect animals are placed in one of two genera – *Microsporum* and *Trichophyton.*

Table 43 Fungi isolated from mycotic skin lesions of camels

Dermatophytes	Authors
Trichophyton verrucosum	Curasson (1947) Nasser (1969) Torky and Hammad (1981) Khamiev (1981, 1982, 1983) El-Kader (1985) El-Tamavy et al. (1988) Mahmoud (1993) Fadlelmula et al. (1994) Abou Zaid (1995)
Trichophyton mentagrophytes	Refai and Miligy (1968) Kuttin et al. (1986) Mahmoud (1993)
Trichophyton schoenleinii	Kamel et al. (1977) Chatterjee et al. (1978) Al Ani et al. (1995)
Trichophyton sarkisovii	Ivanova and Polyakov (1983)
Trichophyton dankaliense	Dalling et al. (1966)
Microsporum gypseum	Boever and Rush (1975) Kamel et al. (1977) Fischman et al. (1987) Mancianti et al. (1988) Gitao et al. (1998)
Microsporum canis	El-Kader (1985) El-Tamavy et al. (1988) Abou Zaid (1995)
Others	
Sporothrix schenckii	Curasson (1947)
Candida albicans	unpublished
Penicillium vinaceum *Pseudorotium spp.* *Pseudoarachniotus spp.* *Allescheria spp.* *Mycelia sterile*	Singh and Singh (1969)
Cryptococcus neoformans	Ramadan et al. (1989)
Chrysosporium	Mahmoud (1993)

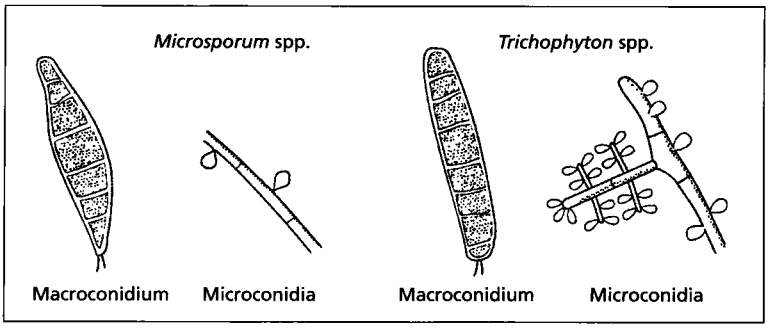


Figure 103 Microscopic differentiation of the dermatophyte genera affecting camels (after Quinn et al., 1994)

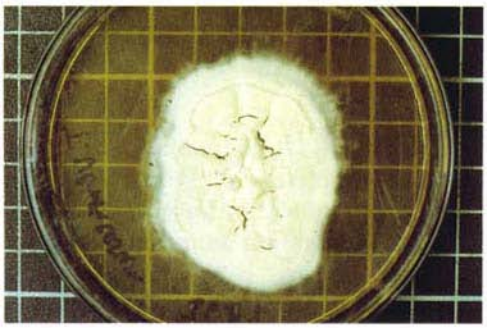
Macroconidia	Large thick-walled and divided into septa. Tend to be spindle or boat-shaped.	Few or absent in some species. If present they are cigar or pencil-shaped. Walls are thin. Divided by septa into 3 to 8 cells.
Microconidia	Relatively few or absent. If present they are tear-shaped and single on hyphae.	Usually numerous, single or in clusters.

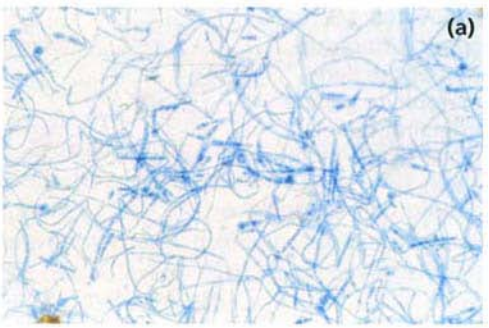
Dermatophytosis is a common skin disease in OWC under 3 years of age with a peak incidence age of between 3 to 12 months. In NWC it is, however, a very rare disease (Fowler, 1998) and only *T. verrucosum* and *T. mentagrophytes* have been isolated from NWC so far.

Macroconidia and microconidia are produced in laboratory cultures and their differences for *Microsporum* spp. and *Trichophyton* spp. are shown in Fig. 103.

The macroscopic (culture) and microscopic characteristics of camelid dermatophytes are shown in Fig. 104a–e. All fungal cultures presented here are 14-day-old cultures grown on Sabouraud agar at 27°C.

Epidemiology In most circumstances, dermatophytes grow only in dead, keratinized tissue; advancing infection halts upon reaching live cells or inflamed tissue. Infection begins in a growing hair or in the stratum corneum where thread-like hyphae develop from conidia. The hyphae penetrate and invade the hair shaft, thus weakening it. It grows downward as the hair grows upward. The dermatophytes produce clusters of arthrospores, primarily along the outer surface of the hair (ectothrix type) rather than within the hair (endothrix type). The epidemiology of ringworm in camelids is yet unexplored, but it is believed that direct and indirect contact with infected animals and fomites are the modes of transmission of dermatophytes. High humidity, overcrowding and nutritional imbalance (most probably Vitamin A deficiency) are conducive to the disease. As many as 80% of calves show clinical signs in affected herds (Wilson, 1998). Khamiev (1982) examined 200 camels with skin lesions, of which 90 were positive for *T. verrucosum*, which he named *T. camelius*. Of these 90 animals, 90% were younger than 2 years. The chlamydiospores of *T. verrucosum* and *T. mentagrophytes* may remain viable for up to 4.5 years in hair and cellular debris scraped off animals and left attached to fomites (Fowler, 1998).

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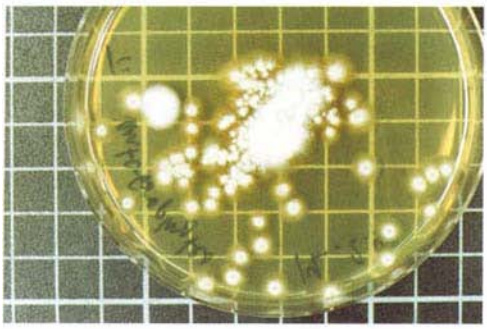

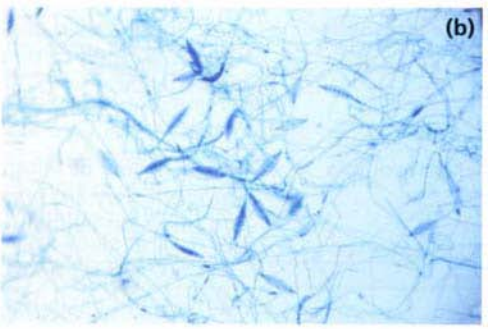
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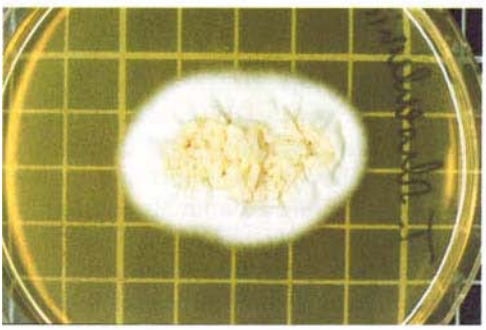

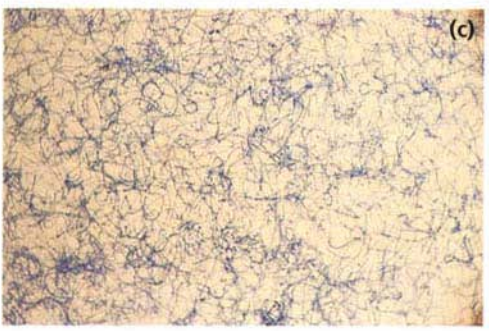
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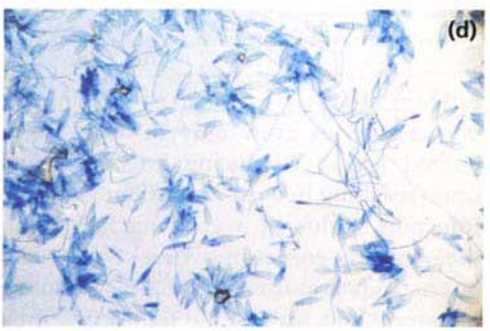
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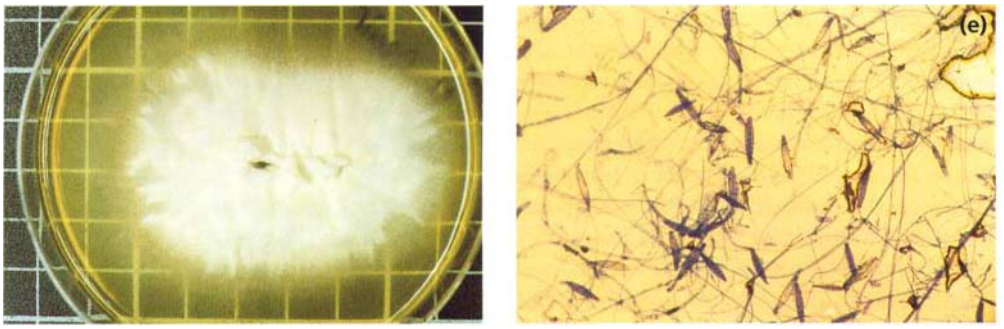

Figure 104a–e Macroscopic (culture, left) and microscopic characteristics (right) of camelid dermatophytes (after Kozlowska and Nuber, 1995): **(a)** *Trichophyton verrucosum*, **(b)** *Trichophyton mentagrophytes*, **(c)** *Trichophyton schoenleinii*, **(d)** *Microsporum gypseum*, **(e)** *Microsporum canis*

Clinical Signs Although camel owners are familiar with ringworm and are able to differentiate this dermatitis from other skin infections, dermatophytoses are extremely variable in their clinical appearances. There are two clinical types of ringworm in camels. The first shows typical lesions that are gray-white in color (Fig. 105).

Figure 105 Typical lesions of ringworm in a young dromedary

These lesions are characterized by small, round alopecic areas, which may coalesce and mainly occur on the legs, neck and head of young animals. The second is a more generalized infection on head, neck, limbs and flanks whereby these lesions may initially be confused with mange (Fig. 106) (Manefield and Tinson, 1996).

The disease is zoonotic and handlers often become infected, exhibiting typical ringworm lesions on their arms.

Figure 106 Ringworm, generalized infection of the hind limb of a dromedary

Pathology The epidermis is thickened with rete pegs extending downwards. The crusts consist of tissue fragments, inflammatory cells, dried serum and fungal elements. Fungal elements are often detected inside hair follicles associated with microabscesses, folliculitis and trichogranulomas (Fadlelmula et al., 1994). Histology reveals hyperkeratosis, parakeratosis and acanthosis in the stratum corneum. The characteristic hyphal filaments are difficult to see on HE-staining; they are best seen with PAS (Marks et al., 1986) and Grocott's methamine silver stain.

Diagnosis Direct microscopic examination of hairs or skin scrapings might reveal characteristic hyphae and/or arthrospores. However, fungal culture is the most effective and specific means of diagnosis, although growth usually requires 10 to 14 days of incubation.

Hairs or scrapings from the periphery of suspicious areas are examined for fungal elements in a wet preparation (20% potassium hydroxide, KOH in water) that has been warmed and squashed out under a coverslip (Fig. 107). A 10 to 20 min. incubation of the slide at room temperature should facilitate the microscopic examination. According to Hollaender et al. (1984) a fluorescent staining with Acridin Orange can make the identification of fungal spores and septate hyphae easier.

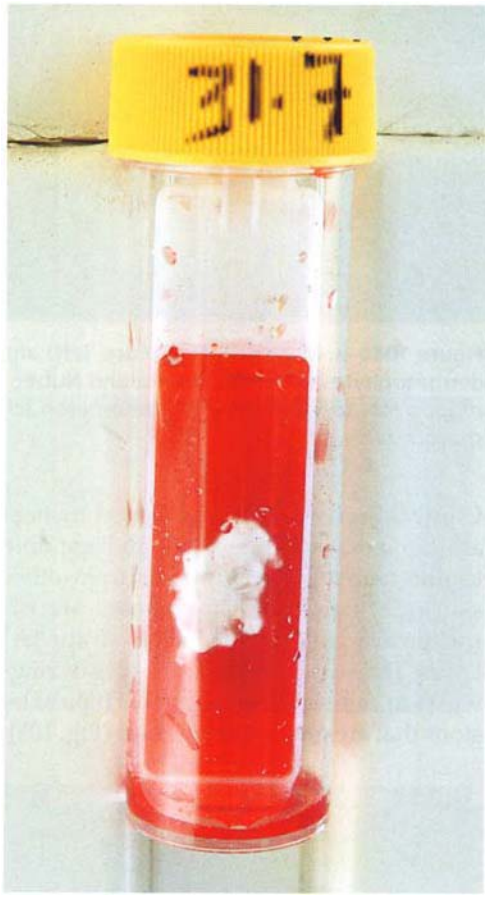

Figure 108 Mycoline agar slide culture of *T. verrucosum* (12 days incubation)

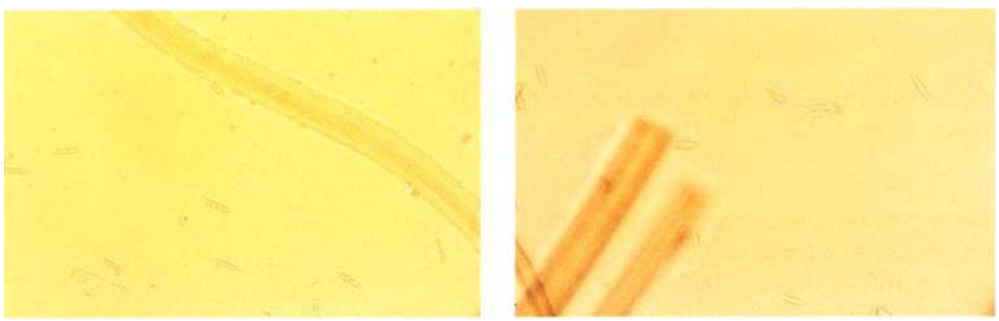

Figure 107 *Trichophyton* spp. (left) and *Microsporum* spp. (right) from camels suffering from ringworm dermatitis (wet preparation with KOH)

Microsporum spp. and *Trichophyton* spp. as well as other fungi should be cultured on Sabouraud dextrose agar and on Mycoline agar slide *(bioMérieux)* and incubated for 10 to 14 days at 27°C (Fig. 108). Definite diagnosis and species identification requires removal of hyphae and macroconidiae from the surface of the colony with acetate tape and microscopic examination with Lactophenol Cotton Blue (LPCB) stain. Culture on Mycoline agar slide is especially helpful when saprophytic contamination is expected.

A number of keratin-proliferative dermatoses have been seen in camelids, and not all are caused by dermatophytes (Table 43). It is therefore essential that any skin lesions should be carefully investigated and multiple deep skin scrapings (containing blood) should be dispatched for laboratory diagnosis.

Treatment and Prevention The spread of ringworm can be limited by early diagnosis and separation of infected from uninfected camels. To avoid recurrence of infection, it is also essential that stables and equipment be properly disinfected.

Lesions should firstly be scrubbed clean with warm soapy water and all scabs removed. A variety of common fungicidal and fungistatic agents such as iodine, 5% sulfur in sesame oil (w/v), 5% salicylic acid, coal tar phenols (3.25%) with copper acetate (0.58%) and hydroxyquinolines may be applied topically as ringworm ointments onto the affected areas.

Captan® is a fungicide for ornamental plants. The use of Captan® has been advocated (Ainsworth and Austwick, 1973) when sprayed on infected animals as a solution of 1:200. The mixture is stable for one week after mixing and the solution should be applied to the lesions and surrounding areas for 2 weeks.

Treatment of dermatophytoses with griseofulvin is very effective in cattle (Coetzer et al., 1994), but it causes side effects in camels such as nausea and diarrhea and is therefore not recommended (Schwartz and Dioli, 1992).

Successful vaccination programs against *Trichophyton* spp. and *Microsporum* spp. in camels have been reported from Kazakhstan (Toleutajewa, 1994).

Camelvac Tricho® (IDT Dessau-Tornau, Germany) has been used in the Republic of Kazakhstan, where 34,302 Bactrians from 12 farms were investigated. In these herds the following incidents of ringworm were found:

5-day to 4-month-old Bactrians:	21.5%
5 to 12-month-old Bactrians:	60.1%
13-month to 3-year-old Bactrians:	17.1%
4 years and older Bactrians:	1.3%

In these herds, 3,300 camel calves were vaccinated with Camelvac Tricho® and no ringworm cases reoccurred for several years. This vaccine is used with very good success not only for prophylactic but also for therapeutic purposes. Camels suffering from dermatophytoses were healed after one or two injections with Camelvac Tricho® (Toleutajewa, 1994). Camelvac Tricho® has also recently been successfully used by the authors in several camel herds in the UAE. Young dromedaries with ringworm lesions (see Fig. 105) were vaccinated once. The lesions receded within 14 days and disappeared after 4 weeks.

3.2 Aspergillosis

Aspergillosis spp., particularly *A. fumigatus*, are associated with infections of the respiratory system and of the placenta in livestock, but may also cause mastitis and rumenitis. Moldy litter and feed are often suspected as sources of infection in outbreaks of aspergillosis. Aspergillosis is an opportunistic fungal infection and has been reported in alpacas and dromedaries (Bhatia et al., 1983; Pickett et al., 1985; Severo et al., 1989; El-Khouly et al., 1992, Gareis and Wernery, 1994).

Etiology Several hundred species of *Aspergillus* have been described, but it is estimated that *A. fumigatus* is responsible for 90–95% of *Aspergillus* infections in animals. Other Aspergillus species that occasionally cause infections include *A. niger, A. flavus, A. terreus* and *A. nidulans. A. flavus* is involved in aflatoxicosis. *Aspergillus* infections are found worldwide in almost all domestic animals and birds as well as many wild species. *Aspergillus* spp. are rapidly growing molds with septate hyphae. Many of the *Aspergillus* species produce colored colonies (black, green or yellow) due to pigmented spores (conidia) (Fig. 109). *Aspergillus* species can be invasive, cause mycotoxicosis and are involved in allergic reactions in humans (Quinn et al., 1994).

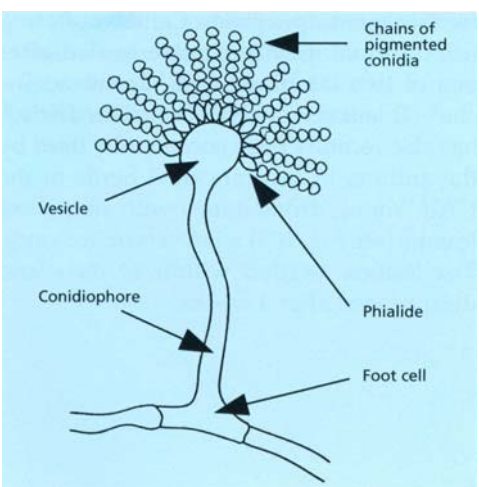

Figure 109 Head of an *Aspergillus* species (after Quinn et al., 1994)

Epidemiology *A. fumigatus* is an ubiquitous fungus and infection does not often occur in mammals. Aspergillosis is especially found in patients debilitated by stress, metabolic acidosis, malnutrition or neoplasia. Prolonged exposure to antimicrobials or immunosuppressive substances can also play an important role in the development of this fungal infection. Transmission is by inhalation and ingestion of fungal spores.

Diagnosis Tissue scrapings or any other material can be examined directly with KOH microscopically, and histopathological sections should be stained by the PAS stain. For the isolation of *Aspergillus* spp., Sabouraud, dextrose agar is used. Pieces of tissue are gently pushed into the agar and the culture is incubated at 37°C for up to 5 days. The colonies usually appear within 2 to 5 days of incubation. The identification is done by colonial morphology and microscopic appearance of the fruiting heads. Immunofluorescent procedures can be used to identify hyphae in tissue sections.

The agar gel immunodiffusion test (AGID) for serum fungal antibodies is a reliable technique for diagnosis and an improved sensitivity may be possible with techniques such as ELISA.

Clinical Findings and Lesions El-Khouly et al. (1992) reported a disease in racing camels in the UAE with a specific

respiratory and enteric syndrome. The diseased camels had a diminished appetite and were lethargic. Some animals developed a mild, dry cough. In many cases there was a swelling of the throat with enlargement of the submandibular lymph nodes. In terminal cases, some camels also developed bloody diarrhea. Affected camels showed a slight increase in body temperature. Death occurred 5 to 7 days after the onset of the first clinical signs. Consistent necropsy findings in 40 camels showed extensive bleeding into the intestines and into the internal organs. *A. fumigatus* was cultured from many organs of the dissected camels and fungal hyphae and conidia were demonstrated in direct smears from the lesions. In some of these cases, aflatoxin was also detected from tissues and sera. However, the authors claimed that it was not possible to determine whether these findings were due to a secondary infection with the fungus or were the primary cause of this syndrome.

A very similar disease has been described by Wernery et al. (1992) as hemorrhagic diathesis (see chapter Endotoxicosis). Gareis and Wernery (1994) described cases of mycotoxicoses characterized by severe watery diarrhea, hemorrhaging, low white blood cell count and deaths in one-humped camels. Heavy rainfall and improper storage resulted in the hay which was fed to breeding camels becoming moldy. Some hay contained high numbers ($> 10^8$ CFU/g) of *Aspergillus, Penicillium, Alternaria, Fusarium* and *Scopulariopsis* species. Extracts of the hay samples, body fluids and intestinal contents of necropsied camels proved to be highly cytotoxic using a cell-culture bioassay (MTT-test). Subsequent analyses of the extracts showed the presence of the epidithiodioxopiperazine mycotoxin gliotoxin, which was the first proven case of natural occurrence of this mycotoxin in feed.

Saad et al. (1989) and Osman and Abdel-Gadir (1991) found aflatoxin M1 and total aflatoxins in a number of milk samples from dromedaries in the UAE. The authors were concerned about the health hazard of camel milk for humans. They stress the need for continuous testing of camel milk to ensure that exposure of the human population to aflatoxins is kept at a minimum. Elmaraghy (1996) reported aflatoxin contamination of camel feed in Libya. Bhatia et al. (1983) reported pulmonary aspergillosis in a 9-year-old camel from India. Several nodules were found in the lung

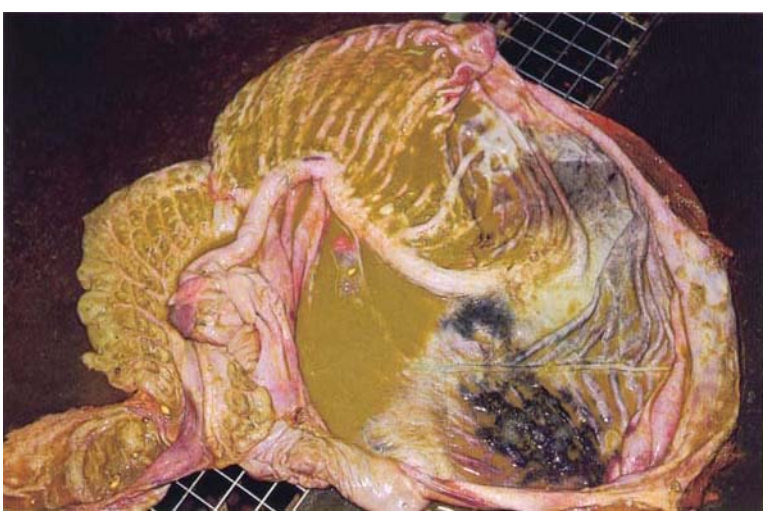

Figure 110 *Aspergillus* spp. granuloma in the lung of a breeding dromedary

surrounded by dark colored consolidated pulmonary tissue containing semisolid caseous necrotic material. Numerous abscesses were also scattered over the lung parenchyma. A necrotizing suppurative pneumonia was diagnosed and branching, septate fungal elements that resembled *Aspergillus* species were seen. *C. pyogenes* was also isolated from the lung.

Aspergillosis granulomas some 5 cm in diameter (Fig. 110) were detected in a breeding dromedary in the UAE which had suffered from generalized camelpox for several weeks and which was treated with tetracyclines for some time. Invasive aspergillosis in two alpacas was reported by Pickett et al. (1985) and Severo et al. (1989) with dissemination causing small abscesses and multifocal areas of necrosis in lung, heart, spleen and kidneys. In one of the cases, large numbers of branching, septate fungal hyphae were detected in the necrotic retina, ciliary body and posterior lens capsule of one eye. This caused blindness associated with head tilt and intermittent circling. In both cases, the morphology of the hyphae seen in histology sections was compatible with an *Aspergillus* species, but no cultivation of the fungus was attempted.

An *Aspergillus fumigatus* rumenitis was diagnosed in the UAE in a guanaco suffering from impaction of the stomachs due to an inflamed diverticle obstructing the duodenum (Fig. 111).

Aspergillus niger pyogranulomatous pneumonia with bronchiectasis was reported in an alpaca by Muntz (1999). The alpaca was euthanized due to poor prognosis. Gross post mortem examination revealed purulent material in the pulmonary airways from which *A. niger* was isolated along with high numbers of associated oxalate crystals. It was presumed that the crystals had been produced by the fungus.

Treatment and Prevention Treatment of aspergillosis has been unsatisfactory. Drugs used have included thiabendazole, flucytosine, and amphotericin B, but very little is known about their effect on camelids. The application of thiabendazole as an antifungal agent had no effect on the outcome of the disease in racing camels as experienced by El-Khouly et al. (1992) and Manefield and Tinson (1996). As a stress-related disease, prevention of aspergillosis can best be accomplished by minimizing factors that lead to stress.

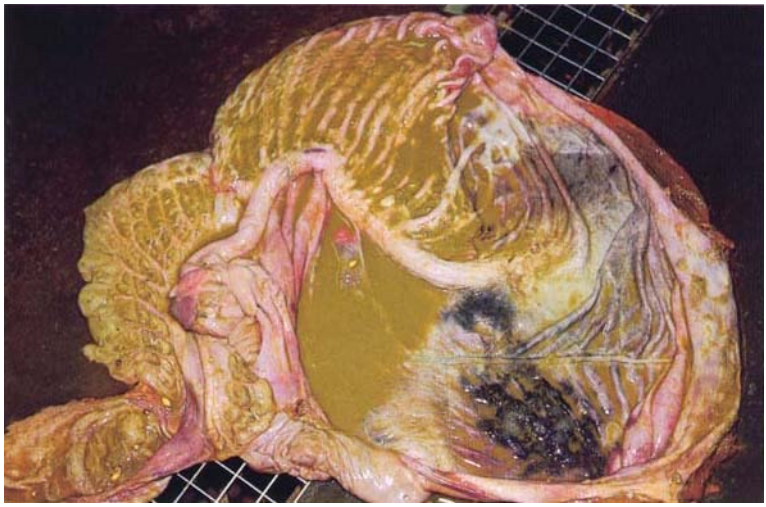

Figure 111 *Aspergillus fumigatus* rumenitis in a guanaco indicated by the black area in C1

3.3 Candidiasis

Candidiasis (moniliasis) is a common sporadic disease of the digestive tract caused by the yeast *Candida* spp. (most commonly *C. albicans*). The disease has been described worldwide in poultry, dogs, cats, horses, swine and wild animals (Merck Veterinary Manual, 1991). *Candida* infection can also cause bovine mastitis and abortion, mycosis of the oral mucosa (thrush), glossitis in infants, skin infections and vaginitis. Dissemination from the intestinal tract to other organs may occur. Infections are more common in young animals and often follow some predisposing factors. One case of gastric candidiasis in a neonatal llama in Europe (Hajsig et al., 1985) and cases in young dromedary calves in the UAE after prolonged treatment with antibiotics (Wernery et al., 2000, in press) have been reported. A *Candida* infection of a dromedary calf's skin was also observed (not published).

Etiology *Candida albicans* is the usual agent of infection, but other yeast-like species have been identified. *C. albicans* is a commensal of the mucous membranes of the intestinal and genital tracts of humans and many animal species. Therefore it is sometimes rather difficult to relate this fungal infection to a disease.

Epidemiology The isolation or demonstration of *C. albicans* from mucous membranes or tissue sections should not lead to a false diagnosis of candidiasis. In many cases *C. albicans* belongs to the normal flora of the digestive tract. It is known that *C. albicans* is not very pathogenic. Cell-wall glycoproteins seem to possess an endotoxin-like activity. The development of candidiasis often follows some predisposing factors such as malnutrition, or extended immunosuppressive or antibacterial therapy. Transmission of this fungus may be via ingestion of contaminated food or water.

Clinical Findings and Lesions Hajsig et al. (1985) reported a neonate llama that had developed a yellowish diarrhea; despite antibiotic treatment and electrolyte therapy it died 5 days later. On necropsy, the walls of C1 and C2 were thickened and

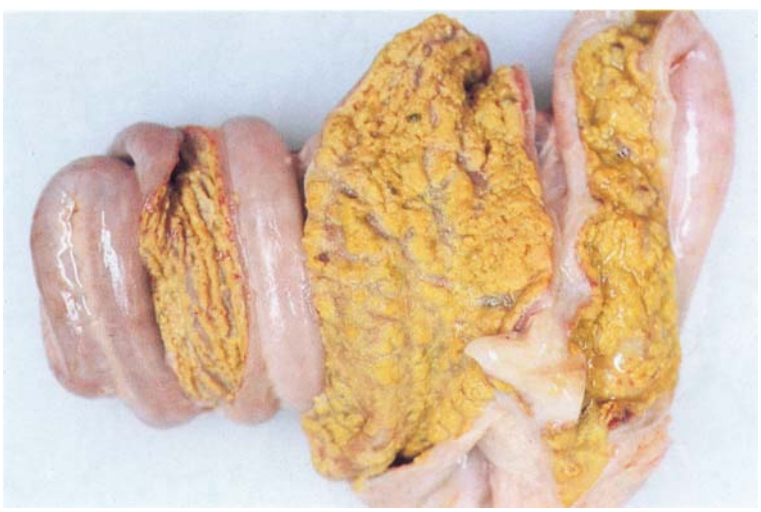

Figure 112 Yellow pseudomembrane of the small intestine of a camel calf with candidiasis

edematous. A white-grayish pseudomembrane several millimeters thick was diagnosed. Microscopically, the epithelium of the mucous membranes was necrotic and invaded by masses of pseudohyphae and budding yeast cells. Similar clinical findings and lesions were found by Wernery et al. (2000, in press) who reported candidiasis in 8 to 48 hour-old dromedary calves in the UAE. These calves developed yellowish diarrhea. On necropsy, yellow pseudomembranes were found in the small intestines (Fig. 112). There was no milk in their digestive system, but variable amounts of sand and water were seen in the abomasum.

Smears taken during necropsy from the intestinal mucosa showed *C. albicans* and *C. perfringens* organisms (Fig. 113). Microscopic investigation showed necrosis of the mucous membranes invaded by yeast

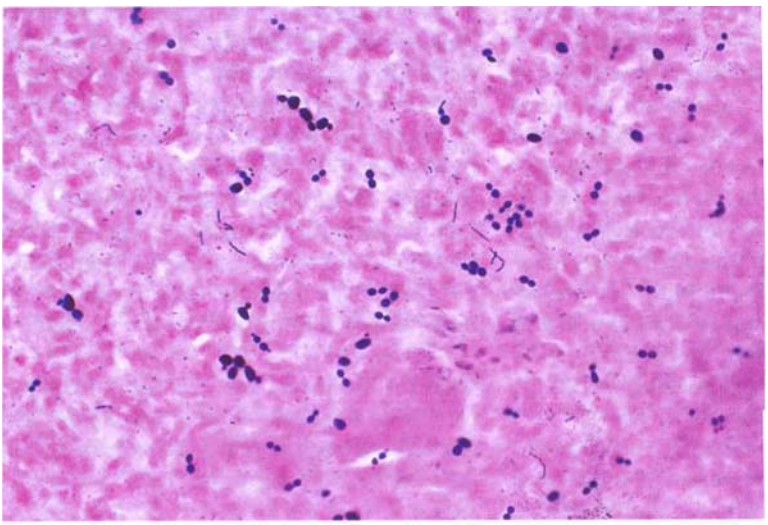

Figure 113 Direct smear from the intestine of a camel calf with candidiasis showing *C. albicans* budding yeast cells and *C. perfringens* rods

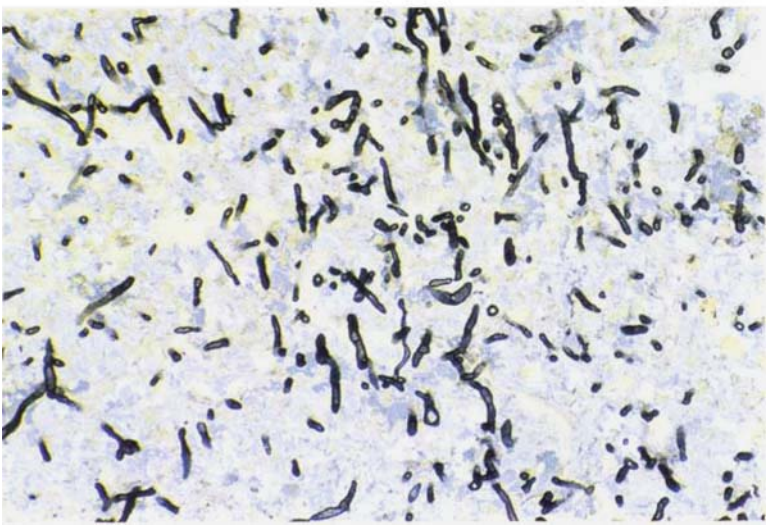

Figure 114 Histology of the infected mucous membrane invaded by *C. albicans*

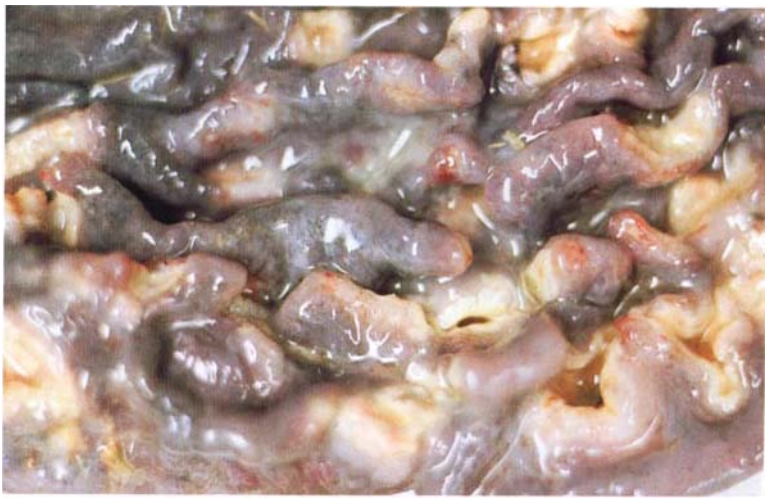

Figure 115 Multiple ulcers in the abomasum of a dromedary

cells that were limited to the epithelial tissue (Fig. 114).

The dromedary calves had also developed a colisepticemia and some of them a *C. perfringens* enterotoxaemia. The authors could prove that the calves possessed very low levels of copper and therefore had ingested sand with which they took up clostridial spores. In adult camels that had been treated with antibiotics over a long period, multiple ulcers have been observed in the abomasum (Fig. 115), invaded by masses of *C. albicans* organisms (Fig. 116).

The same authors have also diagnosed a skin lesion caused by *C. albicans* (Fig. 117). The lesions resemble infections caused by *D. congolensis* (see chapter Integument). The 6-week-old camel calf had developed thick crusts near the hump in which hyphae were demonstrated with PAS stain (Fig. 118).

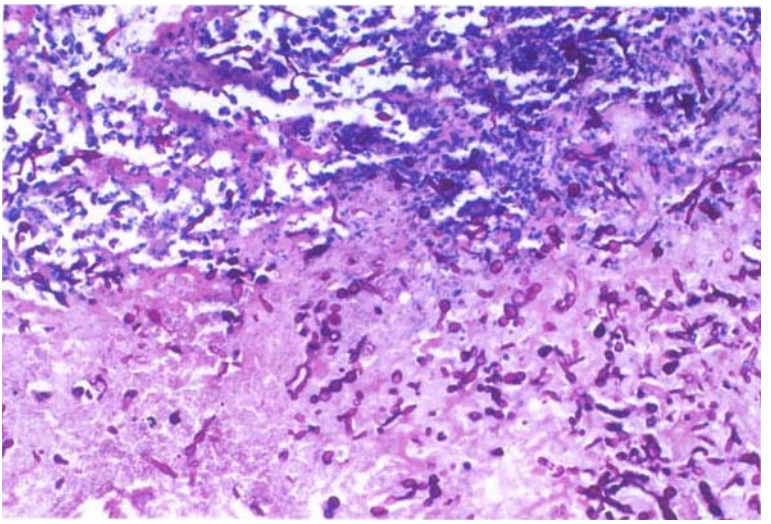

Figure 116 Histology of Figure 115 showing *C. albicans* invasion of the ulcers

Figure 117 Thickened crusts near the hump of a dromedary calf caused by *C. albicans*

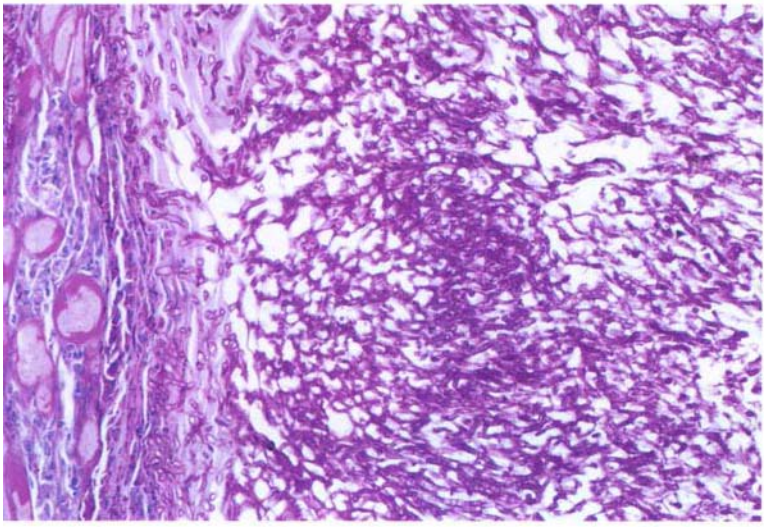

Figure 118 *C. albicans* hyphae from the skin of a camel calf

Diagnosis ▩ Fungal organisms were numerous in proliferating tissue, and diagnosis can be made either by culture or examination of mucosal scrapings or tissue sections. *C. albicans* are ovoid, budding yeast cells (blastospores, 3 to 6 mm in diameter) or occur in chains that produce pseudohyphae. Filamentous, regular, true hyphae may also be visible. The fungal organisms are well stained with LCBP, Giemsa or Gram stain. *C. albicans* can be cultured on Sabouraud's agar or ordinary agars, like blood and nutrient agars, at either room temperature or 37°C. The colonies are white, shiny and convex and grow within 24 to 72 hours.

Treatment and Prevention ▩ Nyastatin, miconazole and ketoconazole have been recommended for intestinal *C. albicans* infections in pigs and bovines, but no reports exist concerning these drugs in infected

camelids. In our cases, the camel mothers received copper and selenium treatment and the calves were given 10 mL of an *E. coli* autovaccine orally, 20 mL of a *C. perfringens* antiserum i.v. (Rhone Merieux), and 10 mg Stegantox® (Schering-Plough Animal Health) i.v. twice within 24 hours. The camels did not receive any antibiotics.

Prevention of candidiasis can best be achieved by minimizing predisposing factors. It is therefore essential to detect and to remove them. Optimal management of breeding herds, including vaccination (see chapter Vaccination Program), and proper mineral supplementation are crucial for the survival of young camelids.

3.4 Coccidioidomycosis

Coccidioidomycosis is a fungal infection of the respiratory tract of humans and animals and it may also appear in a disseminated form or as a dermatitis (Fowler, 1998). NWC seem to be highly susceptible to this fungus. There are no reports of coccidioidomycosis in OWC.

Epidemiology *Coccidioides (C.) immitis* is the cause of coccidioidomycosis, a dimorphic fungus that is not transmitted from animal to animal. The disease is acquired by the inhalation of arthrospores from the environment. In the USA, 100,000 cases of infection and 70 deaths are estimated to occur annually in humans (Salfelder, 1990). The disease has been diagnosed in many animal species, with the dog being the most frequently infected animal (Wolf and Pappagianis, 1981). Arthrospores are found in the infective stage and they convert into spherules in animal tissues. The life cycle of *C. immitis* has been described by Fowler (1998). Infection has never been confirmed in Europe and Asia and seems to be endemic in some areas of North and South America. Infection is restricted to specific geographic zones where climatic conditions of hot, arid weather favors the survival of the fungus in the soil. Disruption of soil exposes the organism to winds, creating an aerosol that can be carried for long distances. These aerosols are suitable for inhalation of arthrospores.

Coccidioidomycosis was described in llamas by Muir (1982) and Fowler et al. (1992).

Clinical Findings and Lesions The respiratory form with dyspnea and coughing, as well as the dermal form, with nodular lesions over most of the body surface, have been described. Muir (1982) reported on a llama with posterior paresis which was euthanized due to poor prognosis. At necropsy, disseminated visceral granulomas and an extradural pyogranulomatous mass compressing the spinal cord of T-10 were found. *C. immitis* was isolated from these lesions.

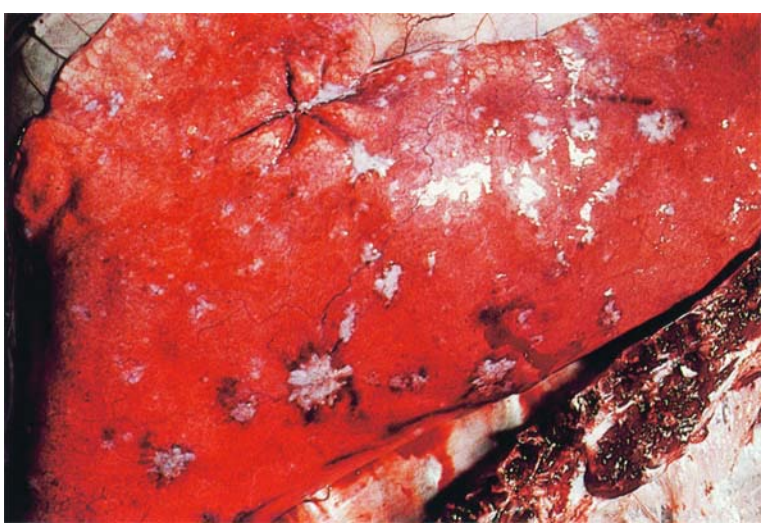

Figure 119 Lung granulomas caused by *C. immitis* (courtesy of Prof. M.E. Fowler, USA)

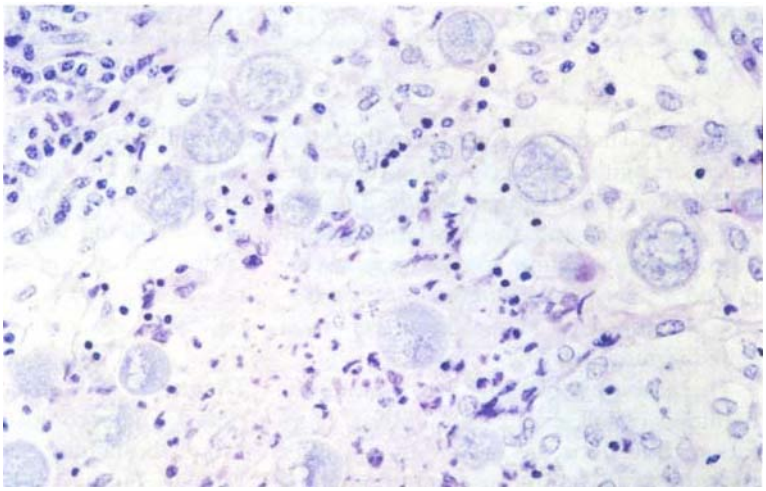

Figure 120 Thick-walled spherule filled with endospores (courtesy of Prof. M.E. Fowler, USA)

In the disseminated form, every organ of the body might be infected (Fig. 119).

The granulomas can range from 1 to 5 cm in diameter or coalesce into large masses. The nodules are gray and firm and contain numerous spherules when microscopically examined (Fig. 120).

Diagnosis Diagnosis of coccidioimycosis can be made by serological tests like complement fixation test, agar gel diffusion, fluorescent antibody or latex agglutination, or by microscopic observations of biopsies or during necropsies. The most sensitive and specific serological test used to date is the agar gel diffusion (Fowler et al., 1992). The fungus may be cultured on selective media such as cycloheximide-chloramphenicol agar, but this should be restricted to those laboratories equipped to handle dangerous infective cultures.

Treatment and Prevention Amphotericin B is the drug of choice but with poor response in lamoids. Treatment of a llama with this drug over a period of 6 weeks did not successfully eliminate the disease or prevent transplacental passage of the organism to the fetus.

NWC characteristically roll in dry soil, creating dust. Avoiding dust is the only way to avoid infection with *C. immitis*, but this is extremely difficult to achieve.

Vaccines for lamoids have not been established as they have for humans and non-human primates.

3.5 Mucormycosis

Etiology There are 11 genera of the order *Mucorales* with 22 species. The most important genera are *Mucor, Absidia, Rhizopus* and *Mortierella* and the most pathogenic thermotolerant *Mucor* spp. are now classified in a new genus: *Rhizomucor.* The disease produced by any of these genera is called "mucormycosis".

These ubiquitous fungi are common inhabitants of soil, manure and rotting vegetation. Infections are secondary to other disorders and might cause granulomatous lesions in several organs of various animal species. Mucormycosis is particularly important as a cause of placentitis and abortion in cattle.

Only one genus, *Rhizopus* spp., has been isolated from a llama (Fowler, 1998).

Clinical Signs The llama suffered from a disseminated, multisystemic infection in association with a facial paralysis of cranial nerve VII. During the course of the disease, swallowing became impossible and the llama began to lose weight. An endoscopic examination of the nasal cavity revealed a black membrane with white patches.

Diagnosis Mucormycosis can be diagnosed microscopically by demonstrating broad, branching, aseptate and irregular hyphae. Fungi can be identified in tissue sections by FA techniques with fluorescein antiglobulins specific for each genus of the *Mucorales.* In the reported case of the llama, filamentous growth was present on the surface of a necrotic rhinitis, the meninges on the ventral aspect of the brain were inflamed, and granulomas were present in the area of the cranial nerves.

3.6 Miscellaneous Fungal Infections

Table 44 Miscellaneous fungal infections in camelids

Disease	Organism	Species	Clinical Signs	Authors
Zygomycosis (Entomophtho-ramycosis)	*Conidiobolus coronatus*	llama	chronic, eosinophilic dermatitis of the nose	French and Ashworth (1994)
		llama	nodular dermatosis of external nares	Moll et al. (1992)
Phycomycosis	*not mentioned*	dromedary	ulcers of abomasum	Satir et al. (1993)
Histoplasmosis	*Histoplasma capsulatum*	dromedary	miliar necroses of the lung	Chandel and Kher (1994)
Cryptococcosis	*Cryptococcus*	vicuña	meningitis and pneumonia	Griner (1983)

Several other fungal infections have been described in OWC and NWC but they are rare. They are listed in Table 44.

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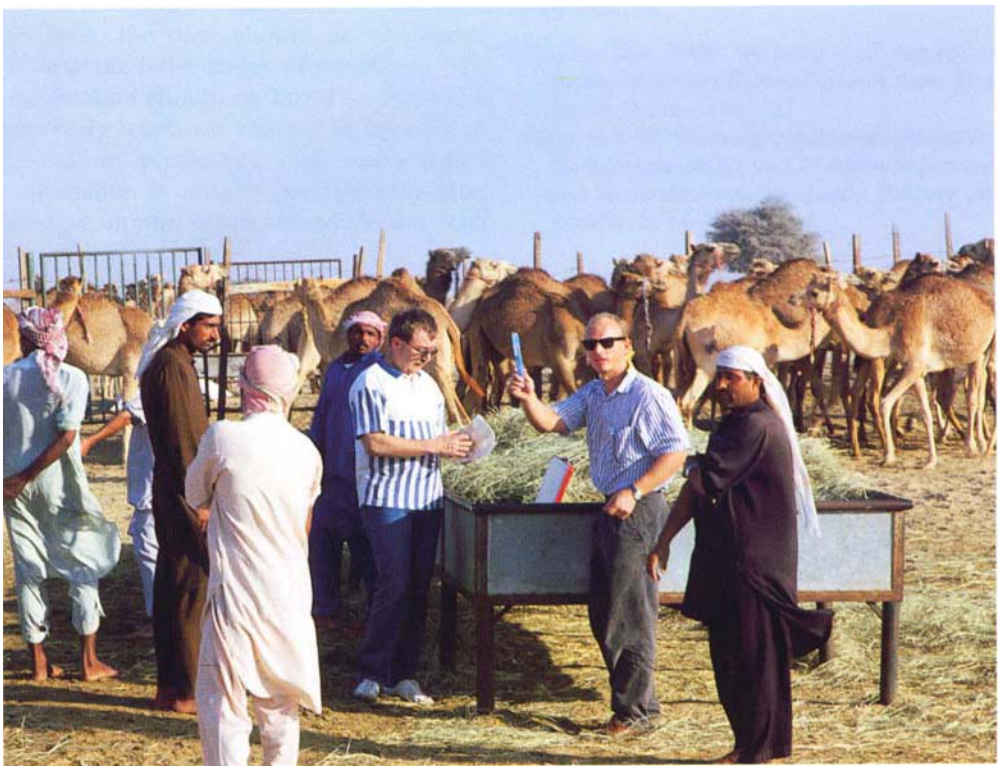
Vaccination Programs 4



The methods of reducing infection of economically important animals include a wide range of management practices, such as testing and slaughter, hygiene and sanitation and immunization. Preventing and controlling a large number of animal diseases by immunization is probably the outstanding achievement of veterinary medicine in the last century.

Although it is impossible to give exact schedules for each vaccine, certain principles are common to all methods of active immunization. As maternal antibodies may passively protect newborn animals, vaccination is usually not successful early in life. If immunity is necessary for newborn animals, the dam should be vaccinated during the latter stages of pregnancy. The vaccination should be timed so that peak antibody levels are reached at the time of colostrum production. Successful active vaccination is usually possible only after passive immunity has waned. As the exact time of maternal immunity loss cannot be predicted, young animals must be vaccinated at least twice to ensure successful immunization.

Very little is known about the efficacy of vaccines in camelids. In the United States of America for example no vaccines have been approved for use in camelids (Fowler, 1998). However, Fowler (1998) and Mayr (1998) recommend some vaccines in NWC. The following vaccine programs for viral, bacterial and fungal diseases are based on their recommendations and our own experience (Tables 45 and 46).

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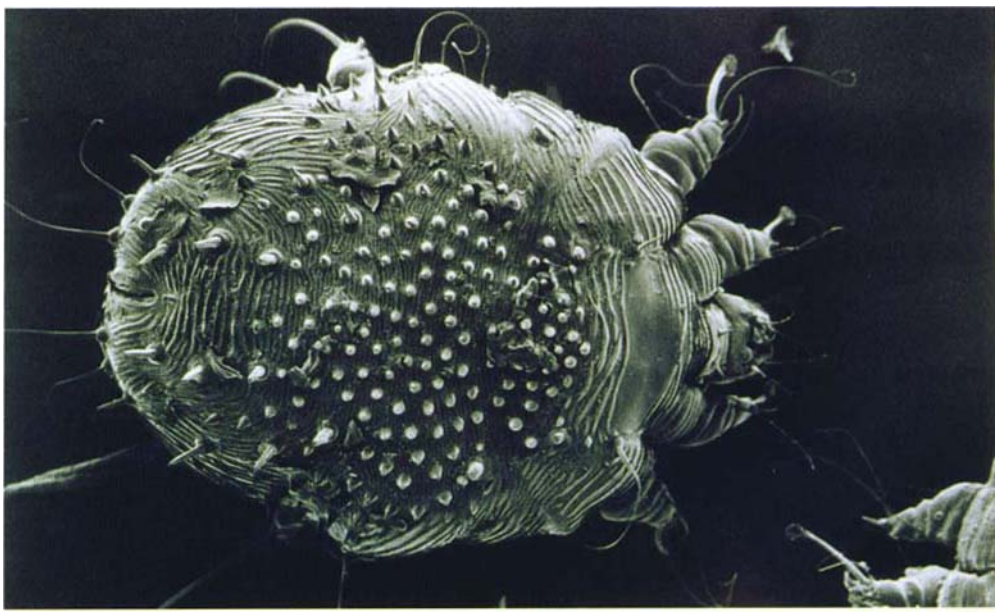
Table 45 Vaccination regime for *Camelidae* against bacterial diseases

Disease	Vaccine	1st vaccination at the age of	Booster	Repeated vaccination	Particularities
Tetanus *(Cl. tetani)*	toxoid vaccine	2–3 months	after 4 weeks	1–3 years	hyperimmunserum in suspected cases
Enterotoxemia (*Cl. perfringens* A,B,C,D)	toxoid and bacterial vaccine	1–2 months	after 4 weeks	annual	autovaccine with local strains hyperimmunserum 100 mL i.v.
Gas edema complex *(Cl. chauvoei, septicum, novyi)*	toxoid and bacterial vaccine	1–2 months	after 4 weeks	annual	in endangered areas
Anthrax *(Cl. anthracis)*	live attenuated	2–3 months	–	annual	in endemic areas
E. coli diarrhea	inactivated (doses > 10^{10} CFU)	oral application 20–50 mL	for 10 days	–	autovaccine for young calves
E. coli diarrhea	inactivated	6 weeks before parturition	2 weeks before parturition	annual	autovaccine for pregnant camels
Salmonellosis	inactivated (doses > 10^{10} CFU)	oral application 20–50 mL	for 10 days	–	autovaccine for young calves
Salmonellosis	inactivated	6 weeks before parturition	2 weeks before parturition	annual	autovaccine for pregnant camels
Leptospirosis	inactivated appropriate serovar	2 months	after 4 weeks	4 months	in endemic areas with appropriate strains

Table 46 Vaccination regime for *Camelidae* against viral and fungal diseases

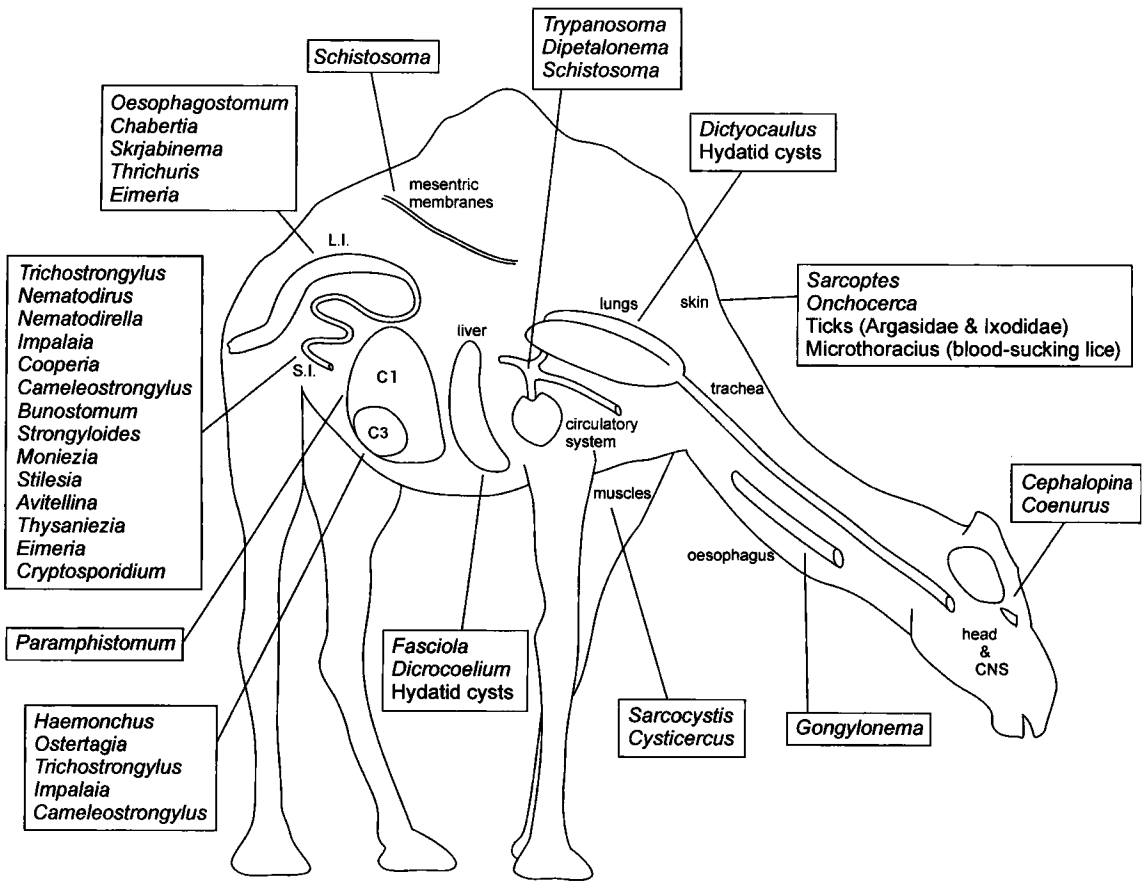
Disease	Vaccine	1st vaccination at the age of	Booster	Repeated vaccination	Particularities
Rabies *(Rhabdovirus)*	inactivated cell culture virus	3 months	after 3 weeks	annual	in endangered areas, Rabisin®
Camelpox *(Orthopoxvirus)*	attenuated cell culture virus	6–9 months	after 4 weeks	life-long immunity?	commercial – South Africa, Ducapox
Ecthyma contagiosum (Parapoxvirus)	attenuated cell culture virus	1–2 weeks	after 6 weeks	6–8 months	Turkey
Papillomatosis *(Papovavirus)*	inactivated Papilloma tissue	for treatment	3 times every 5 days with increased doses	–	autovaccine
BVD/MD *(Flavi/Pestivirus)*	inactivated vaccine	2–4 weeks	after 2 months	annual	in areas with MD abortions
Neonatal viral diarrhea *(Rota, Corona)*	inactivated vaccine	–	4 and 2 weeks before delivery	annual	if required
Equine Herpes (EHV-1)	inactivated vaccine	8–12 weeks	after 3–4 weeks	annual	if required
Dermatophytoses (Ringworm)	*Trichophyton verrucosum*, attenuated	4 months also for treatment	after 14 days	?	commercial – Camelvac Tricho, IDT, Dessau, Germany

Parasitic Diseases 5



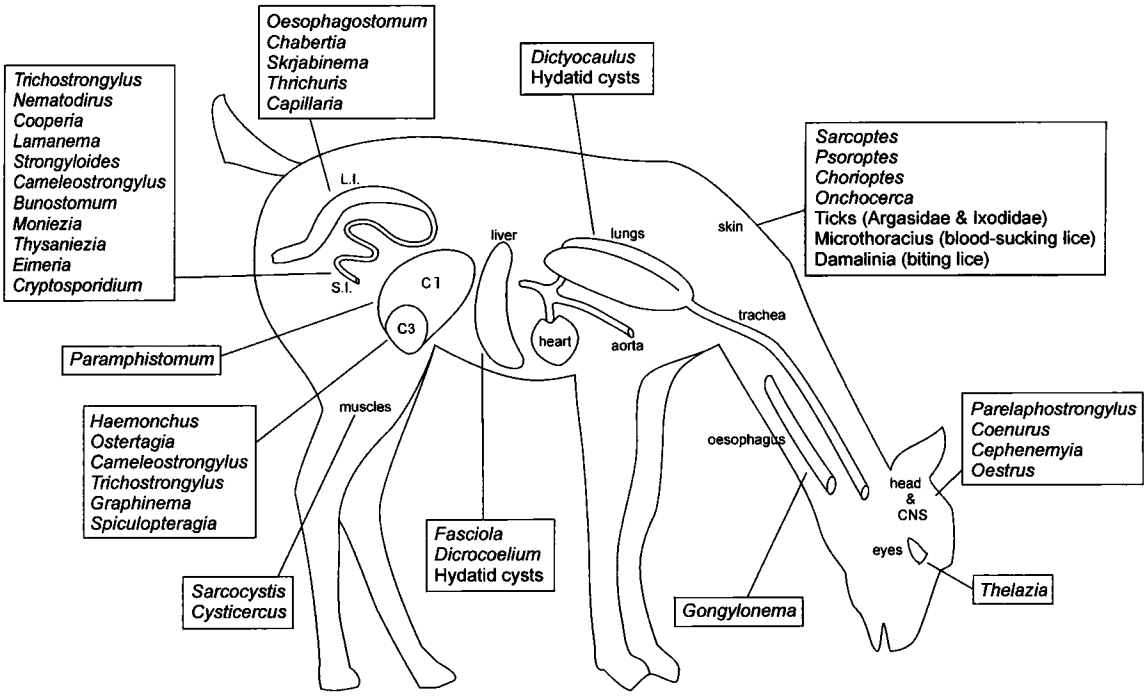
Parasites of Old World Camels

The organ localization of parasites of the OWC is illustrated below.



Parasites of New World Camels

The organ localization of parasites of the NWC is illustrated below.



Parasitic infections may significantly limit the productivity of camelids and other livestock by causing a substantial reduction in the provision of milk, meat, wool and fibers, as well as transport. Many conditions are of a subclinical nature. The economic losses due to parasitic infections can be substantial. For example in the 1970s, the estimated annual loss in meat from 3.02 million head of alpacas in Peru was more than $US 1.5 million (Table 47).

Table 47 Estimation of annual losses of meat due to parasitic infections in alpacas in Peru

Disease	Losses ($US)	% of total
Parasitic pneumo-gastroenteritis	695,400	46.3
Ectoparasites	337,555	22.5
Sarcocystiosis	296,822	19.7
Fasciolosis	170,911	11.4
Hydatidosis	1,489	0.1
Total	**1,502,177**	**100.0**

Source: Ministerio de Agricultura, Estudio de la Evaluación de Problemas de Carnes en el Perú, Tomo V. Lima, 1973. Cited by Leguia (1991)

5.1 Protozoal Infections

Introduction

The protozoal infections of camelids are listed in Table 48.

Protozoa are eukaryotic organisms. Their genetic information is stored in chromosomes contained in a nucleus that is surrounded by two membranes containing several pores. Besides the nucleus, they possess an endoplasmic reticulum, mitochondria, Golgi apparatus and lysosomes. In contrast with the prokaryotic cells of rickettsiae and certain algae, the nuclear apparatus of protozoa is not separate from the cytoplasm.

Additionally, the protozoa possess certain other structures with distinct features and functions, e.g. for locomotion; although the genus *Trypanosoma* has a single flagellum, other protozoa may have several. Some protozoa move by means of cilia, as does *Balantidium*. As a means of locomotion, *Entamoeba* uses pseudopods that are prolongations of the cytoplasm. Movement occurs as some of the cytoplasm flows into the prolongation. These pseudopodia also have phagocytic properties.

Nutrition of the protozoa occurs mainly by pinocytosis or phagocytosis. The metabolic by-products are excreted by diffusion through the cell membrane.

Among the protozoa are many species that are not parasitic, e.g. those found in the rumen. These are commensal or symbiotic organisms that assist in the digestion of cellulose and, after having passed the abomasum, act as a source of protein for the host.

5.1.1 Classification of Protozoa

Regnum Protozoa
Phylum Sarcomastigophora
Subphylum Mastigophora (Flagellates)
Order Kinetoplastida
Trypanosoma spp.
T. evansi (OWC)
T. simiae (OWC)
T. brucei (OWC)
T. congolense (OWC)
T. vivax (OWC)
T. cruzi (NWC?)

Order Trichomonadida
Tritrichomonas foetus (OWC)

Order Diplomonadida
Giardia sp. (NWC, OWC)

Table 48 Protozoa of camelids

Disease	Protozoa	Occurrence		Location
		OWC	NWC	
Trypanosomosis	*Trypanosoma evansi*	+		Blood
Trichomonosis	*Tritrichomonas foetus*	+		Genital tract
Giardiosis	*Giardia* spp.	+	+	Intestinal tract
Balantidiosis	*Balantidium coli*	+		Intestinal tract
Coccidiosis	*Eimeria* spp.	+	+	Intestinal tract
Cryptosporidiosis	*Cryptosporidium* spp.	+		Intestinal tract
Sarcocystiosis	*Sarcocystis* spp.	+	+	Muscle, brain
Besnoitiosis	*Besnoitia* spp.	+		Intestinal tract
Toxoplasmosis	*Toxoplasma gondii*	+	+	Multiple organs
Neosporosis	*Neospora caninum*	+		?

Phylum Apicomplexa (Sporozoa)
Class Sporozoea
Subclass Coccidia
Order Eucoccidiida
Family Eimeriidae
Eimeria alpacae (NWC)
E. bactriani (OWC)
E. cameli (OWC)
E. dromedarii (OWC)
E. auburnensis (NWC)
E. lamae (NWC)
E. macusaniensis (NWC)
E. pellerdyi (OWC)
E. peruviana (NWC)
E. punoensis (NWC)
E. rajasthani (OWC)

Family Cryptosporidiidae
Cryptosporidium sp. (OWC)

Family Sarcocystidae
Sarcocystis spp. (OWC, NWC)
S. aucheniae (NWC)
S. cameli (OWC)
S. tilopoidi (NWC)

Family Toxoplasmatidae
Besnoitia sp. (OWC)
Isospora cameli (OWC)
I. orlovi (OWC)
Toxoplasma gondii (OWC, NWC)
Neospora caninum (OWC?)
Hammondia heydorni (OWC)

Sublass Pirosplasmia
Order Piroplasmida
Babesia sp. (OWC)
Theileria sp. (OWC)

Phylum Ciliophora
Order Trichostomatida
Balantidium coli (OWC)

5.1.2 Trypanosomosis

Trypanosomosis is a disease of humans and animals caused by parasitic trypanosomes. The trypanosomes of mammals are subdivided into two sections: the Stercoraria and the Salivaria, based on the mode of development in their insect vectors and vertebrae hosts. They are further divided into subgenera and species on the basis of morphological differences.

The most important protozoal disease of camels is trypanosomosis (named surra), caused by *Trypanosoma evansi* (Cross, 1917; Leese, 1927; Richard, 1975, 1979). This parasite described by Evans was the first recognized pathogenic mammalian trypanosome. The parasite is widespread throughout tropical and subtropical areas. However, in Africa, where camels may contract tsetse-transmitted trypanosomes, infections may also occur with *T. brucei, T. congolense, T. vivax* (Bennett, 1933) and *T. simiae* (Mihok et al., 1994).

T. simiae was identified as the cause of an outbreak in dromedaries in a Kenyan national park, confirming the susceptibility of camels to this pathogen (Mihok et al., 1994). *T. simiae* was also documented as a camel pathogen in Somalia (Pellegrini, 1948), and isolated from camels in Kenya (Roettcher et al., 1987; Dirie et al., 1989).

Haerter et al. (1985) experimentally confirmed that dromedaries were sensitive to *T. brucei* and particularly to *T. congolense.* Their attempt to infect three camels intravenously with two different strains of *T. vivax* failed. However, an experimental infection with *T. congolense* resulted in an acute disease that led to death between days 22 and 37 with fever, progressive edema and general weakness. At necropsy, serous fluid was found in the body cavities and hemorrhages on the serous membranes. The response to infection with *T. brucei* was milder; parasitemia persisted throughout the three months of observation and the only changes seen were an ini-

tial rise in fever and declining packed cell volume values.

T. evansi may affect many different species of mammals. The disease was originally reported in India in 1880 and is most severe in horses, donkeys, mules, deer, camels, llamas, dogs and cats. Occasionally it occurs in sheep, goats, pigs and Indian elephants as a mild or subclinical infection. In addition there have been reports of *T. evansi* infections in tigers, foxes, tapirs, and orangutans (Molyneux and Ashford, 1983).

Etiology *T. evansi* is one of the salivarian trypanosomes. Morphologically it is indistinguishable from the long and slender form of *T. brucei*, having a prominent undulating membrane and a long, free flagellum and a small sub-terminal kinetoplast. It is hypothesized that *T. evansi* originated from *T. brucei* by adaptation to a non-cyclical mode of transmission and loss of ability to undergo growth and differentiation in the fly vector (Hoare, 1957). Brun et al. (1998) in a review confirmed the many similarities between *T. evansi* and *T. equiperdum* based on biological, biochemical and molecular studies. Electron microscopic investigation revealed no ultrastructural differences between the two species. However, the most prominent differences are the presence of maxi-circles in *T. equiperdum*, which are missing in *T. evansi*, and the route of transmission. In the host's blood it most often occurs as monomorphic trypanomastigote, 15–36 µm long (mean 24 µm) and 1.5–2.2 µm wide (Fig. 121).

Occurrence Surra is found within a wide range of climate and vegetation zones in Asia, the Middle East, the Far East, Central and South America and usually outside the tsetse belt in Africa. Just north of this belt the prevalence of surra in camels is roughly estimated at between 15 and 20%. Only few reliable data exist on the distribution and seasonal prevalence of the disease in endemic areas. In Kenya, the prevalence of *T. evansi* in 2000 camels was 48% (Olaho and Wilson, 1983) and 79% in a smaller herd comprising 174 camels (Rutagwenda, 1984). In Sudan, the prevalence of the infection was 25–50% in 948 dromedaries (Bitter, 1986). In Somalia, 58% of camels were found positive (Caille, 1987), and 7.2 to 56% depending on the diagnostic methods used (Baumann and Zessin, 1992). An epidemiological survey in Mo-

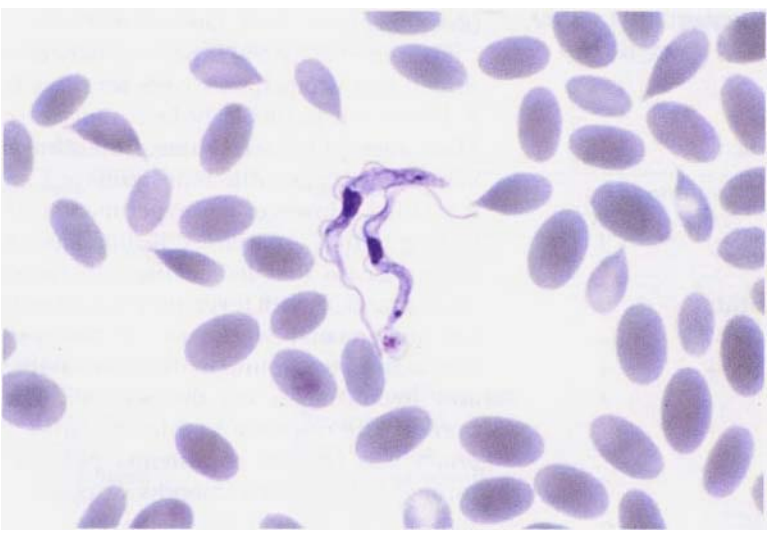


Figure 121 Two *T. evansi* parasites in dromedary blood

rocco during 1996–1999 revealed a prevalence of 6.6% (Atarorhouch et al., 2000). Two endemic foci were identified in the south of the country affecting sedentary camels husbanded in small groups. In Mauritania the prevalence varied between 1.3% employing blood smear examination, and 16.2–25.2% using different serological tests (Dia et al., 1997). On the Canary Islands 7 out of 745 dromedaries yielded *T. evansi*, while the seroprevalence was 4.8% (Guitierrez et al., 2000). As prevalence data are based on different tests which differ widely in sensitivity, these figures should only be regarded as rough estimates (Butt et al., 1998).

In the 1880s, surra spread from Asia into European Russia, where it killed an estimated 70% of the camel population (Molyneux and Ashford, 1983). In 1907, surra was diagnosed in Port Hedland, Western Australia. The infected camels were destroyed and since then no further evidence of the disease has been seen in Australia.

Trypanosomosis has not been reported in camelids in South America despite the presence of *T. evansi* in cattle and horses (mal de caderas) and in some wildlife species such as the capybara *(Hydrochoerus)*, the vampire bat *(Desmodus rotundus)*, the ocelot *(Felis pardalis)* and deer (*Odocoileus* spp.). However, trypanosomes have been demonstrated in llamas imported into the USA (Fowler, 1998).

Transmission *T. evansi* is transmitted mechanically by blood-sucking flies. Several biting or blood-sucking insects may serve as vectors. Mechanical transmission by contaminated hypodermic needles is also a potential means of transmission. The trypanosomes remain in the mouthparts of the fly. No cyclical development occurs in these flies in contrast to other salivarian trypanosome infecting tsetse flies. The main vectors involved are tabanids and *Stomoxys* (Molyneux and Ashford, 1983). Other insects may also transmit the parasite, although they are considered to be of less importance, e.g. *Lyperosia, Haematobia* and *Hypobosca* (Rutter, 1967). Experiments have shown that the vomit from lapping flies that have fed on parasitemic blood and exudates caused by biting flies can be infective to laboratory rodents. Hilali and Fahmy (1993) reported large numbers of *Cephalopina titillator* larvae in the nasal cavities of dromedaries in Egypt infected with two different sizes of epimastigote trypanosomes thought to be *T. evansi*. Smears obtained from the larvae contained the epimastigote stage, which was always observed in a dividing state. Mice and guinea pigs inoculated with the epimastigote form showed no parasites in their blood.

The efficacy of transmission depends on the interrupted feeding behavior of tabanids, i.e. on the interval between a fly feeding on an infected host and moving to a clean host. The aggressive feeding behavior of tabanids involves many attempts at feeding. Individual flies can therefore infect more than one host. The shorter the interval between two feeds the greater the chance of successful transmission, as the trypanosome has a restricted survival time in the vector. The infectivity of a fly is highest within minutes of feeding and decreases quickly with no transmission at all if the interval exceeds 8 hours (Losos, 1980). The trypanosomes remain alive in the mouthparts of some insects for not more than 15 seconds (Curasson, 1947), but as long as 44 h in the gut of tabanids, and 5 to 6 h in the gut of *Muscidae* flies (Rutter, 1967).

Eating parasitemic animal meat can infect carnivores. In South and Central America, the vampire bat *(Desmodus rotundus)* can be infected from blood meals and then act as a vector, transmitting the trypanosomes through its saliva. In addition it may act as a reservoir.

Other domesticated species like sheep and goats, which have only mild, subclinical infections and which often coexist with camels, might act as reservoirs. The para-

site isolated from naturally infected camels, horses, mules and dogs was found to be pathogenic to sheep and goats (Mahmoud and Gray, 1980).

Surra has a marked seasonal pattern in some areas in association with wet conditions, e.g. the development of the biting fly populations after rain. However, this was shown not to be the case in Sudan where the infection was more prevalent during the dry rather than wet season (Elamin et al., 1998). Tabanid flies are more abundant early in the dry season in Sudan (Elamin et al., 1998), and camel herds congregate in larger numbers at the few available water holes facilitating efficient transmission of the trypanosomes by flies.

Some other factors that may predispose to patent parasitemias are stressful climatic conditions and poor nutrition.

Clinical Signs Surra may be acute, subacute or chronic, with a mortality of up to 90%.

Acute cases often show signs of recurrent fever accompanied by progressive anemia and poor general condition. Edema and paralysis may also develop. Subacute infections occur with fever, edema, emaciation and high mortality. The edema varies from plaques on the neck and flanks to edema of the muzzle, chest wall, sheath and scrotum and on the legs up to the knees and hocks. Death may take a few days or months. An experimentally infected guanaco developed the subacute form of surra showing edema and wasting (Kinne and Wernery, 2000).

The chronic form of the disease leading to wasting and anemia is more common in camels. Many infected camels have a mild and protracted infection that can persist for several years, eventually ending in emaciation and death. It can cause abortion, premature birth and reduced milk production. Calves may be weak at full term. In an infected herd, the disease can vary between individuals: some die within a few months following the infection while others develop chronic or subclinical conditions lasting two or more years. Some camels may recover spontaneously.

Immunodeficiency may be a sequel to surra, thus making animals more susceptible to other infections which may complicate the clinical picture.

Pathology Gross lesions in the camel are not very specific. In acute and subacute cases, petechiae are seen on serous surfaces and within liver and kidney parenchyma.

In subacute cases in camelids, it is common to see severe hemorrhages on the cauda equina. In chronic cases, the carcass is anemic and often emaciated. Ascites and hydrothorax may be present and the lymph nodes are enlarged. A few scattered petechiae are found in the edematous meninges of the cerebellum and brain stem.

More pathognomonic in camelids are the histological lesions in the central nervous system. In most of the subacute and chronic cases, mild to moderate nonsuppurative meningitis and focal meningoencephalitis are found. Typical are broad, perivascular cuffs in the gray matter (Fig. 122). Eosinophilic, PAS-positive "corpuscular structures" in the meninges are often observed (Fig. 123). These structures represent "Russel or Mott bodies" and are characteristic of human African trypanosomosis, but are also observed in other causes of encephalitis (Salfelder et al., 1992). In horses, Seiler et al. (1981) described these structures as "morular cells". Similar structures are also found in the large infiltrates on the cauda equina. It is assumed that parasites hide in the meninges where they might survive for a long time, evading treatment.

Clinical Pathology The anemia is macrocytic and hemolytic. There is a decrease in erythrocytes and an increase in lymphocytes, eosinophils and monocytes. The infection is also accompanied by progressive

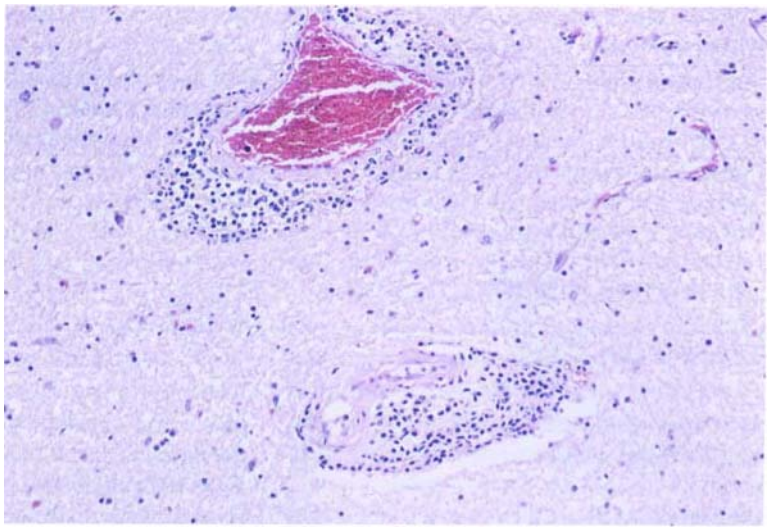


Figure 122 Non-suppurative meningoencephalitis caused by trypanosomosis; note the cuffing

changes in the serum protein concentrations, a decrease in albumin, an increase in γ-globulins and a five-fold increase of IgM levels during the course of the infection (Boid et al., 1980). In addition, there are changes in some serum enzymes resulting in an increase in sorbitol-dehydrogenase and glutamate-pyruvate-transaminase as well as glutamate-oxalacetate-transaminase (Boid et al., 1985).

Wernery (1995) compared blood parameters and iron of racing camels with chronic trypanosomosis with reference values (Table 49). Hemoglobin, packed cell volume, red blood cells and iron were significantly decreased, whereas the total white blood cell count was elevated. Similar results were obtained from dromedaries with subacute trypanosomosis. In acute trypanosomosis, a monocytosis of up to

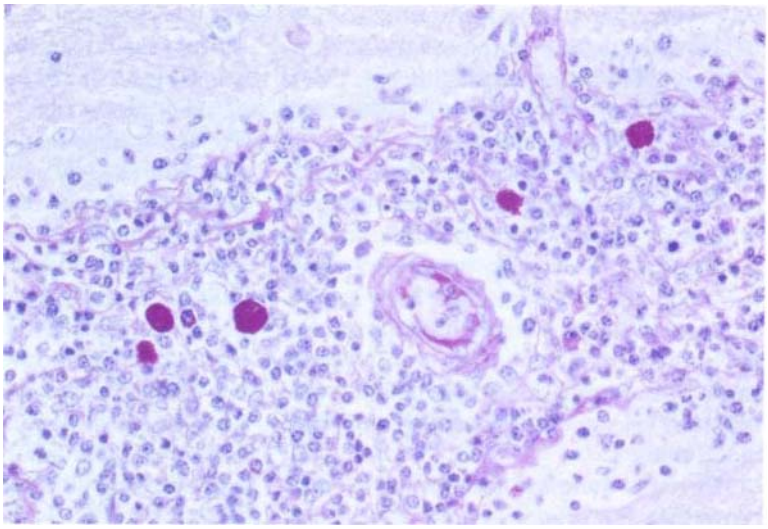


Figure 123 Eosinophilic PAS-positive structures ("Russel bodies") caused by trypanosomosis

Table 49 Blood parameters and iron of racing dromedaries with subacute and chronic trypanosomosis

Blood parameters	Unit	Reference values*	Camels with chronic trypanosomosis	Camels with subacute trypanosomosis
Hemoglobin (Hb)	g/dL	12–15	9.32	7.54
Packed Cell Volume (Hematocrit, PCV)	%	26–38	18.4	17.87
Red Blood Cells (RBC)	$\times 10^6$ µL	7.5–12.0	6.08	5.75
White Blood Cells (WBC)	$\times 10^3$ µL	6.0–13.5	17.2	16.2
Iron (Fe)	µg/dL	87–135	20.2	54.2

* Wernery et al. (1999)

15% was observed during the first four weeks of the disease.

Diagnosis Trypanosomosis can be confused with any other chronic wasting disease, notably helminthosis and malnutrition. A reliable diagnosis can be made on the basis of the demonstration and identification of trypanosomes in the blood, although that may be difficult due to the often low and fluctuating parasitemia. The parasites in the blood of the vertebrate host are often scarce, particularly in the chronic and subclinical stages. The severity of an infection is not necessarily related to the number of parasites seen in the blood. It may be difficult or impossible to find trypanosomes in the blood of an infected animal, even when it is in the moribund state.

There are no real pathognomonic clinical signs of infections with *T. evansi*. Clinical signs such as emaciation and anemia (PCV < 25%) (Table 49) are often used as a provisional diagnosis, but are unsatisfactory when considering successful measures of control. Parasitological techniques applied for demonstrating trypanosomes in the blood are only successful in 50 to 60% of infected camels. Confirmation of a tentative diagnosis in the field is still largely carried out by relatively insensitive methods such as examining wet, thin and thick blood films.

However, there are techniques for concentrating the blood samples. These improve the chances of demonstrating trypanosomes in the blood of infected animals with fairly low parasitemia. The most applicable and commonly used in the field is the microhematocrit centrifugation technique (MHCT). Microhematocrit tubes are filled with fresh blood and spun at 2,500 g in a microhematocrit centrifuge (MHC). The centrifugation separates the blood into three different layers: the packed red blood cells, the buffy coat and the plasma. The interface between the buffy coat and the plasma should be examined for motile trypanosomes under a microscope (Woo, 1969; Woo, 1971). The buffy coat may also be examined as a wet preparation on a microscope slide. Trypanosome species can be identified in a fresh preparation or after Giemsa staining.

The MHCT can detect trypanosomes in camel blood 6 to 10 days earlier than in wet or thick blood films (Kelley and Schillinger, 1983). This technique is easy to carry out in the field by a battery-powered MHC, which can also be run by a car battery.

Other methods for detecting very low parasitemia include the miniature anion exchange centrifugation technique (Lumsden et al., 1979, 1981) and the silicone centrifugation technique (Ogbunde and Magaji, 1982). The latter technique was shown to be as sensitive as the above-mentioned concentration methods and has the advantage of being simple and rapid (Nessiem, 1994).

Inoculation of laboratory rodents with blood from suspected infectious camels is a very sensitive method for detecting low parasitemia caused by *T. evansi* (Boid et al., 1985) and *T. brucei* (Godfrey and Killick-Kendrick, 1962). Mouse or rat inoculation increases the number of camels found positive by approximately 50% compared with blood film techniques (Molyneux and Ashford, 1983). However, this method is time-consuming, expensive and inappropriate for use in large-scale surveys. Development of patent parasitemia is 5 to 9 days in mice and 3 to 9 days in rats.

Despite improvements in parasitological techniques for the detection of trypanosomes, a high proportion of infections are never detected. One major reason for this is the constant antigen variations that occur in *T. evansi* (as in other salivarian trypanosomes) (Jones and McKinnell, 1984). This phenomenon makes it difficult to detect circulating antigens and antibodies – a tremendous advantage for the parasite, keeping it ahead of an attack by specific antibodies directed against the previous surface antigens. The identification of circulating variable antigen types (VSG) would be of great value in developing more sensitive diagnostic tests. The antigenic variation is also a major constraint for immunoprophylactic control methods.

The development of enzyme immunoassays (ELISA) detecting circulating antigens in animal sera provides an opportunity for an early diagnosis of trypanosomosis. This was an important breakthrough in the diagnosis of this disease (Rae and Luckins, 1984; Nantulya et al., 1987). However, the present ELISA has proven to be unsatisfactory in sensitivity as well as specificity (Antigen ELISAs for trypanosomosis – Evaluation of the performance: Proc. Workshop ILRI, Nairobi, Kenya 1996). A simpler test established for use under field conditions was the latex agglutination technique (Suratex®) (Nantulya, 1989). *T. evansi* antigen may also be detected in blood by the polymerase chain reaction (PCR) (Masiga and Gibson, 1992; Wuyts et al., 1994). In the near future, well-equipped laboratories may more efficiently use DNA-amplification technologies in the diagnosis of *T. evansi* in animals, while pastoralists still traditionally diagnose trypanosome infections by the smell of the infected animal's urine.

In the diagnosis of surra, antibody techniques like flocculation assays (including the formol gel and mercuric chloride tests) (Pegram and Scott, 1976) measure an increase in the level of serum globulins. However, these tests are non-specific and have yielded many inconsistencies (Luckins et al., 1979; Boid et al., 1980). As early as 1924, Schoening described a complement fixation test demonstrating antibodies to *T. evansi* (Schoening, 1924). This test has never been routinely used as a diagnostic test because it is too difficult to perform, and procedure standardization is not possible.

Another promising assay for the diagnosis of *T. evansi* antibodies (an indirect hemagglutination test) was developed by Jaktar and Singh (1971). However, this assay also had difficulties with the standardization of antigens and the presence of interfering, non-specific antibodies. Wilson et al. (1983) successfully used this assay to demonstrate antibodies to *T. evansi* in a serological survey of Kenyan camels. Another agglutination test also available is the modified card agglutination test (CATT/*T. evansi*), which was initially developed for *T. brucei gambiense* (Dialli et al., 1994). This test is not specific to *T. evansi* antibodies but can also detect antibodies to other salivarian trypanosomes, thereby complicating the interpretation of positive results when other salivarian trypanosomes are present.

Even the improved indirect fluorescent antibody test (Luckins et al., 1978) had inherent drawbacks. The development of an enzyme-linked immunosorbent antibody assay (ELISA) was a major breakthrough.

The ELISA has been used with good results in the serodiagnosis of *T. evansi* (Luckins et al., 1979; Boid et al., 1980; Rae et al., 1989). In the United Arab Emirates (UAE), where surra in dromedaries is endemic, a decrease in the seroprevalence was achieved (from 12.5% in 1990 to 2.5% in 1999) due to the treatment of positive cases (by the use of antibody ELISA) and the control of vectors (CVRL Annual Report, 1999).

Antibodies to *T. evansi* infections in camels as demonstrated by ELISA do not differentiate between acute and chronic infections (Rae et al., 1989). Antibody responses to *T. evansi* infections may vary and the levels may stay high for a considerable time after effective treatment (Luckins et al., 1978).

Treatment and Control Only a few drugs, e.g. Cymelarsan® (melarsomine, Merial), Triquin® (quinapyramine sulfate, quinapyramine chloride, distributor Wockhardt Ltd.) and Trypamidium-Samorin® (isometamidium chloride, Merial) have been approved by appropriate authorities for use in OWC or NWC. It is known that the pharmacokinetic behavior of drugs differs significantly among different species. Therefore it is important that drugs should be studied carefully in every species. This is especially true for camelids due to their unique physiological characteristics. As there are only very limited pharmacokinetic data available on camelids, drugs should be used with great caution. This also applies to the use of vaccines. They should undergo testing by regulatory agencies for safety and efficacy before they are used on camelids.

Monitoring for drug resistance is important and there are techniques available (Kaminsky and Zweygarth, 1989; Zhang et al., 1993; Brun and Lun, 1994) that should be employed when suspicion of resistance arises.

Surra is endemic in most countries where camels are reared. Chemotherapy alone will not have a permanent effect on the cycle of the disease, regionally or globally. The use of chemotherapy is often inadequate: e.g., underdosing is common. As a result of the limited number of drugs available for therapeutic or prophylactic use, the dependence on trypanocidal drugs for the control of surra is alarming. Also, not all compounds effective against *T. evansi* are suitable for use in camels. Many of the drugs used for cattle are either not curative or too toxic for camels: e.g. diminazene aceturate (Berenil®, Hoechst AG – production, however, was stopped recently), which is toxic to camels at doses of >3.5 mg/kg and should not be used in dromedaries. Berenil® has been successfully used in Bactrian camels with doses as high as 5 mg/kg bodyweight (Luckins, 1992). Alternative drugs are Trypan®, Atarost®, Veriben®, and Sanofi® (Rommel, pers. commun.).

In the early 1970s, Imperial Chemical Industries Ltd (ICI) stopped their production of the quinapyramines, Antrycide® and Antrycide® Pro-Salt, at that time the commonly used and successful trypanocides against *T. evansi* (Schillinger and Roettcher, 1984). Production of Antrycide® has been resumed by a few drug companies and it is available today. This meant that only one curative drug remained available for camels – Naganol® (Bayer AG, Leverkusen, Germany – production, however, was stopped recently), in use since 1925. After nearly 75 years of use, the effectiveness of Naganol® is decreasing in some areas due to drug resistance. However, Suramin (Naganol®) administered at a dose rate of 10–12 mg/kg by slow intravenous injection is still used in the treatment of camels. Due to its slow elimination from the body, it also has a prophylactic effect for between 6 to 12 weeks (Kaufmann, 1996). The preventive effect depends on the dosage used and the degree of the trypanosome challenge (Luckins, 1992). Leakage of the drug into the tissues may cause phlebitis.

Quinapyramine methylsulfate may also be used curatively at 3–5 mg/kg together with quinapyramine chloride at a ratio of 3:2 (5–8.3 mg/kg). Quinapyramine prosalt, administered subcutaneously, may be used prophylactically and has a prophylactic effect of 4 to 6 months. In cases of resistance to suramin and the quinapyramines (Zhang et al., 1993), isometamidium chloride (Samorin® or Trypamidium®) may be used but with great caution (0.5–0.7 mg/kg intravenously administered as a 2% solution). This drug is only curative when the trypanosomes are present intravascularly. Overdosing quinapyramines can cause side effects in camels such as tremors, salivation and collapse leading to death.

There is *in vitro* and *in vivo* evidence that most isolates tested for *T. evansi* are resistant (innate) or non-responsive to isometamidium.

The latest drug on the market, melarsomine (Cymelarsan®, Merial, Lyons, France) was developed about 10 years ago (Raynaud et al., 1989). It is effective against *T. evansi* when administered to camels by deep intramuscular injection at 0.25 mg/kg. The residual effect in the dromedary is not yet fully known. Camels have subsequently apparently remained free of *T. evansi* for 90 days. However, the degree of parasite challenge may be a factor to keep in mind.

Control and eradication of trypanosomosis is difficult because of the development of drug resistance; even more difficult is the control of vectors. Regular monitoring of infections is necessary to prevent large losses in endemic areas. Employing frequent (monthly) PCV estimations on well-managed herds has proven useful in keeping losses low. Some farm managers treat any camel with a PCV of < 25%.

Table 50 Drugs for treatment of *Trypanosoma evansi* infections in camels
C = curative; P = prophylactic; IV = intravenous; SC = subcutaneous; IM = intramuscular; Administ. = administration

Drug	Trade Name	Action	Administ.	Dose mg/kg
Melarsomine	Cymelarsan®	C	deep IM	0.25
Quinapyramine sulfate	Antrycide®	C, P	SC	5
	Trypacide®	C	SC	5
Quinapyramine sulfate/chloride[1]	Trypacide®	P	SC	5–8
Quinapyramine sulfate/chloride[1]	Triquin®	C, P	SC	5–6
Suramin[2]	Naganol®	C, (P)	IV	5–10[3]
Isometamidium chloride[4]	Samorin® Antrypol®	C, P	IV	0.5
Isometamidium chloride	Trypamidium®		IV	0.5–1
	Veridium®	C	IV	0.5
Diminazene accturate[5]	Berenil®	C	IM	3.5–5

[1] Is called Pro-Salt
[2] Drug resistant to *T. evansi* (reported in Sudan, India and the former USSR)
[3] 10 g/camel for treatment and 5 g/camel for prevention
[4] Toxic effects when used in camels resistant to Suramin and quinapyramine – not advisable for use in camels
[5] Not recommended for use in dromedaries; however, it is widely used in Bactrians in Asia

Table 50 lists the drugs that are used against *T. evansi* in camelids.

5.1.3 Tritrichomonosis

Only one report of *Tritrichomonas foetus* infection of camels has been published recently. The parasite was isolated from 24 out of 48 camel breeding herds with endometritis, exhibiting whitish-yellow, mucopurulent discharge (Wernery, 1991). The pathogen was also isolated from one of four bulls in these herds.

Tritrichomonas foetus belongs to a group of organisms, Trichomonadida, commonly found in the digestive and reproductive tracts but also in other organs of a variety of animals. It is a common pathogen in bovines, particularly in developing countries. Bovine trichomonosis is a venereal disease characterized by early fetal death in cows usually first seen as an infertility problem. Subclinically infected bulls transmit the infection. The parasite can be cultured in several different media and as it is motile the characteristic fast, jerky, rolling movements are readily seen in fresh preparations.

Tritrichomonas foetus is pear-shaped, and possesses three free flagella arising from a basal body at the anterior end. A fourth flagella extends backwards to form the undulating membrane along the side of the organism continuing as a free flagellum. A hyaline rod, the axostyle, is found extending throughout the cell, often with a slight posterior projection (Fig. 124).

Diagnosis Apart from animals exhibiting problems of infertility, diagnosis depends on finding the parasite in cervical and vaginal mucus and/or the preputial washings. The organism may be found in discharges from the uterus and in the aborted fetus (stomach). Microscopic examination of fresh smears from the above specimens is easily performed, but the organism may be only present intermittently and/or in minute numbers, requiring several repeat examinations. To enhance the chances of finding the organisms, clean samples may be cultured in special media for a few days allowing the organisms to multiply, thus making the parasites visible through a light microscope. The diagnosis can be confirmed by PCR (Polymerase Chain Reaction) (Kaufmann, 1996). Sero-

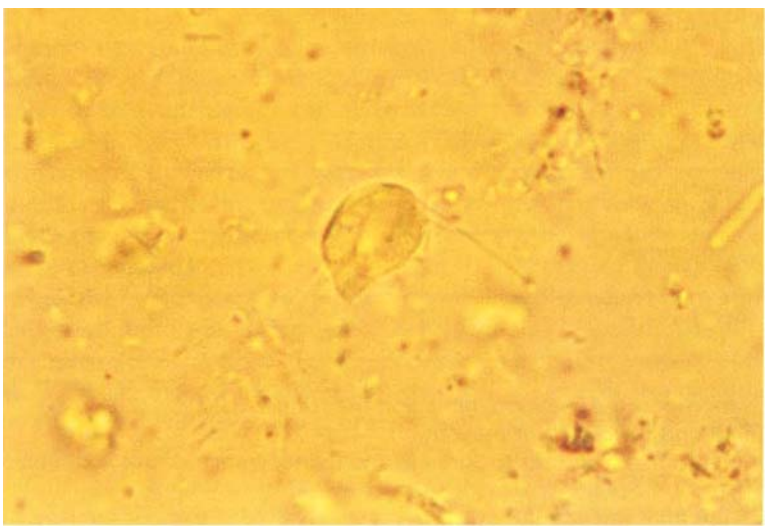


Figure 124 *Tritrichomonas foetus* from an endometrial smear of a dromedary with endometritis

logical tests are used for epidemiological surveys.

Treatment ▒ Chemotherapy is not regularly used because its effect is unreliable. Compounds used against trichomonads are dimetridazole, diminazene aceturate, ipronidazole and metronidazole. Rinsing the affected organs with acridin and iodine preparations may have a positive effect (Tibary and Anouassi, 1997).

5.1.4 Giardiosis

Giardia spp. have been found in a debilitated young llama with diarrhea (Kiorpes et al., 1987). Parasites have been observed in OWC.

Giardia spp. have been isolated in a variety of mammals. They appear morphologically similar with small differences. There has been considerable discussion concerning the significance of *Giardia* infections in mammalian hosts. Some species or strains are considered pathogenic in humans. Many infections are latent, but some are associated with acute or subacute to chronic diarrhea due to enteritis of the small and (sometimes) large intestine. Waterborne outbreaks of giardiosis may result in significant epidemics in humans and it is therefore one of the world's most common infectious intestinal parasites (Stevens, 1985). Contaminated food and untreated surface water polluted with cyst-containing animal feces in conjunction with inadequate filtration are the primary sources. Additionally, public health authorities consider giardiosis a sexually transmitted disease (Stevens, 1985).

Giardia spp. have been divided into three different groups based mainly on the morphology of microtubular structures (median bodies) in the trophozoites. The first group, *G. agilis*, is a parasite of amphibians with long, narrow trophozoites. The second group, *G. muris*, occurs in rodents as well as in birds and reptiles. The third group, *G. duodenalis (G. intestinalis)*, is a parasite in birds, reptiles and mammals (including humans).

The life cycle (LC) of *Giardia* is direct and includes two morphological forms: trophozoites (feeding stage) and cysts (infective stage). The oval, pear-shaped multinucleated (2 or 4 nuclei) cysts may be ingested via contaminated water or by direct transmission from feces. Excystation occurs in the small intestine where the cysts release motile trophozoites that multiply asexually. The cysts are sensitive to desiccation but can survive for months in a moist and cool environment.

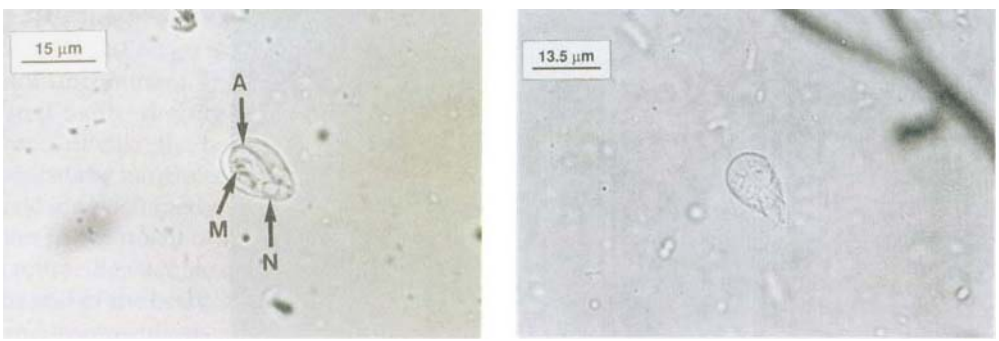


Figure 125 Giardia cyst (left) with nuclei (N), axostyle (A) and median bodies (M); *Giardia* trophozoite (right) in an unstained fecal smear (courtesy of Professors Sloss, Kemp and Zajac, Veterinary Clinical Parasitology, 6th ed., 1994, Iowa State University Press, USA)

Diagnosis Diagnosis of *Giardia* is based on the detection of pear shaped multi-nucleated cysts (Fig. 125).

Occasionally the trophozoites may be seen. The recommended method of cyst detection is by using the 33% zinc sulfate flotation technique with fresh feces. Repeated sampling and testing should be done because of the cyclical shedding of cysts. Microscopic examination of fresh diarrheic feces mixed with some saline may reveal motile trophozoites, recognized by their rapid "falling leaf" motion and concave ventral surface. Trichomonads are also mobile organisms and of similar size and may be differentiated from *Giardia* spp. by the undulating membrane, rolling form of movements, lack of concave surface and the presence of only one nucleus.

There are several fecal ELISAs that have been marketed for use in humans. These diagnostic tests demonstrate *Giardia*-specific antigens derived from trophozoites. Merifluor® from Meridian Diagnostics Inc., USA is an *in vitro*-direct immunofluorescent test for the simultaneous detection of *Giardia* cysts and *Cryptosporidium* oocysts in fecal material (Fig. 126).

Treatment Metronidazole and fenbendazole are recommended for treating infected dogs and cats. Infections in farm animals have been successfully treated with dimetridazole at a dose rate of 50 mg/kg daily for 5 days. In many countries this drug is forbidden for use in food animals. Some benzimidazoles, albendazole (20 mg/kg) and fenbendazole (10 mg/kg) daily for three days have proved effective in calves (Xiao et al., 1996). However, the efficacy of these drugs in camelids is unknown.

5.1.5 Balantidiosis

The ciliate (Ciliophora) *Balantidium coli* is the only species associated with disease in mammals. It is a parasite of the colon in man, pigs, monkeys and perhaps in other animals. Large numbers of nonparasitic ciliates take part in the digestive process and occur in the rumen of ruminants and camelids as well as in the colon of equines.

The pig is thought to be the primary host of *B. coli*, which is generally regarded as a commensal organism. Occasionally it may invade the mucosa and cause ulceration associated with mild to severe enteritis.

The cysts, which may remain viable for days and weeks in moist feces, usually infect the host. The trophozoites may also initiate infection but they are much less re-

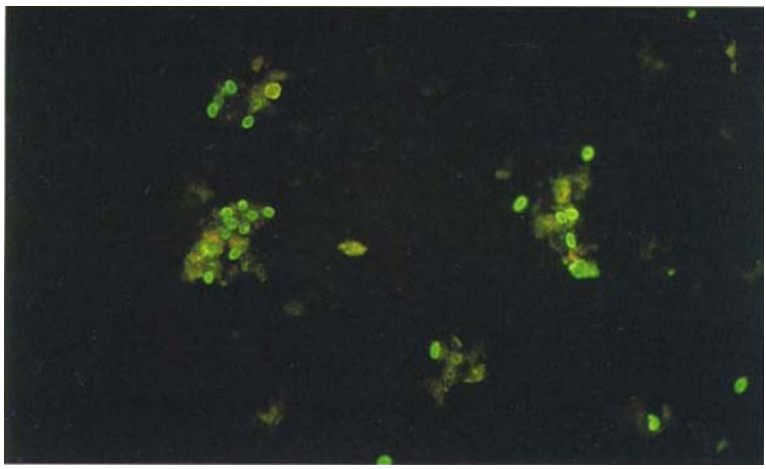


Figure 126 Giardia cysts in dromedary feces (immunofluorescent test)

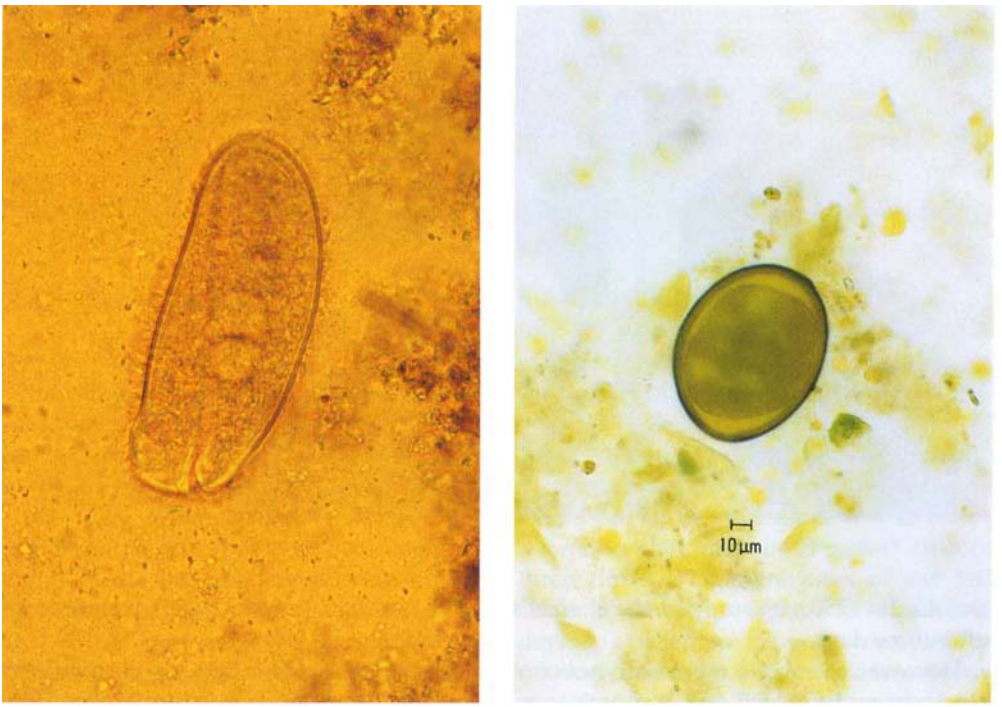


Figure 127 *Balantidium coli* trophozoite from a dromedary intestine (left) and *B. coli* cyst (right)

sistant to the microclimate than the cysts. Trophozoites die within 15 to 30 minutes in temperatures above 40°C. *B. coli* cysts are generally excreted, but large numbers of trophozoites have been observed in fecal samples in diarrheic camels (Kayum et al., 1992).

The trophozoite averages 50 to 60 µm in length, but larger forms up to 150 µm are not uncommon. The body surface is covered with slightly oblique longitudinal rows of cilia, the peristome is subterminal and at the narrower end, the macronucleus is kidney-shaped, and the micronucleus lies in the notch of the macronucleus. One contractile vacuole occurs near the posterior end of the body, another near the center, and the cytoplasm contains numerous food vacuoles. The organism is actively motile and moves quickly over the microscopic field (Fig. 127).

Ovoid to spherical cysts are produced, measuring 40 to 60 µm. They are faintly yellowish-green in color, and the organism can be recognized within the cyst by the macronucleus.

A limited number of cases of clinical balantidiosis in camels have been reported (Vosdingh and Vanniasingham, 1969; Ali and Abdelaziz, 1982; Shommein and Osman, 1987; Kayum et al., 1992). The authors reported large numbers of the organism in the feces of dromedaries, some with diarrhea for 3 months. Ali and Abdelaziz (1982) described a case of diarrhea in a dromedary in good condition (apart from having loose stools). Fecal examination revealed only 300 cysts per gram feces. It is important to know that trophozoites are destroyed by flotation solutions but can be observed in direct fecal smears. Symptomatic treatment with carbarsone (250 mg)

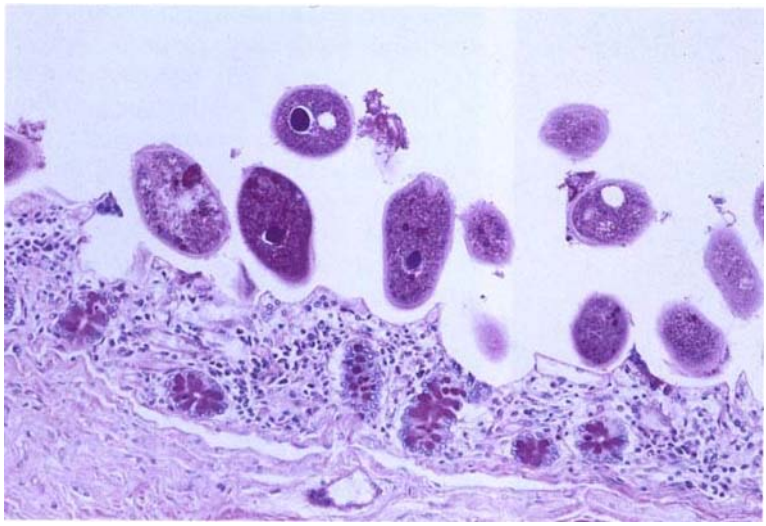


Figure 128 Balantidiosis in a young dromedary

and kaolin (250 mg) stopped the diarrhea after three days.

However, the above reports do not conclusively prove that *B. coli* is a pathogenic organism in camels. *Balantidium* often plays a secondary role in the pathogenesis of intestinal disorders. In central parts of Saudi Arabia, Magzoub et al. (1997) found *Balantidium* cysts in apparently healthy camels.

Severe cases of balantidiosis have been observed in young dromedaries in the UAE. The camels suffered from enteritis with loss of villi in the small intestine (Fig. 128).

5.1.6 Tick-borne Diseases: Babesiosis, Theileriosis

Pathogenic protozoa belonging to the order *Piroplasmida*, which include *Babesia* spp. and *Theileria* spp., are common pathogens transmitted by ticks and are of significant importance in many domestic animals. Although ticks are often found on camels in large numbers, very few reports have been published concerning tick-borne pathogens in camels. These few case reports are not considered reliable as they usually fail to give adequate taxonomic descriptions.

Reported *Theileria* spp. are *T. camelensis* and *T. dromedarii*; the former in Turkmenistan, Egypt, and Somalia (Barnett, 1977; Boid et al., 1985); however, no schizont stages were described. The latter *T. dromedarii* was reported in India (Rao et al., 1988) and thought to be non-pathogenic. In Egypt, Nassar (1992) examined 200 apparently healthy camels and found 30% infected with *Theileria* spp. Ten mL of bovine blood containing high numbers of *T. annulata* parasites were injected intravenously into five 2-year-old healthy dromedaries by the authors. The camels did not show any signs and *T. annulata* was not observed in blood samples taken over a period of one month.

Neither *Theileria* nor *Babesia* spp. have been found in NWC. Only one unconvincing report of *Babesia* infection in camels has been found (Egbe-Nwiyi, 1994). The author did not describe any parasite in the blood cells of the animals. However, the animals showed some signs seen in babesiosis of other animals, e.g. hemolytic anemia, hemoglobinuria, hemoglobinemia, anisocytosis and polychromasia.

Table 51 Taxonomic classification of the coccidia of veterinary importance

Family:	Eimeriidae	Cryptosporiidae	Sarcocystidae	Toxoplasmatidae
Genus:	*Eimeria*	*Cryptosporidium*	*Sarcocystis*	*Besnoitia* *Hammondia* *Toxoplasma* *Neospora* *Isospora*

5.1.7 Coccidiosis

Another group of protozoa are the coccidia; these organisms are intracellular and occur particularly in vertebrates. They are important within the Eimeriidae and Sarcocystidae families (Table 51).

The Eimeriidae are mainly intracellular gut-dwelling parasites (gut-dwelling coccidia) of the intestinal epithelium where they undergo both asexual (schizogony) and sexual (gametogony) multiplication. They complete their life cycle (LC) in a single host, in contrast to the Sarcocystidae (tissue cyst-forming coccidia), which have a two-host LC and which form tissue cysts in the intermediate hosts. The LC stages in both families ultimately result in the formation of oocysts, which are environmentally resistant forms that following sporulation may eventually infect susceptible new hosts.

The term coccidiosis is usually reserved for infections with *Eimeria* and *Isospora* spp. Coccidiosis occurs in all parts of the world that have substantial populations of *Camelidae*. Disease outbreaks characterized by enteritis are mostly associated with young animals living in crowded and wet conditions, after or during the rains, or close to where animals are watered (Kawasmeh and Elbihari, 1983).

Other species of importance to domestic animals are *Cryptosporidium* spp., pathogens which in mammals are parasites of the stomach and intestinal epithelium.

Life Cycle of Eimeria When the infective stage, the oocyst, is ingested by a host following excystation the sporozoites are released usually penetrating the epithelial cells of the mucosa in the small intestine. The sporozoite develops to a trophozoite within the cell and grows quite large, becoming a schizont. Merozoites form within the schizont and eventually rupture the invaded cell and invade other cells in turn – a process that may be repeated two or three times. The merozoites of the last schizont generation invade new cells and develop either to microgametocytes or macrogametocytes. Each macrogametocyte finally forms one macrogamete and each microgametocyte several microgametes. Zygotes result from the union of micro-(male) and macro-(female) gametes (fertilization). The zygotes become oocysts which are excreted with the feces. During sporogony, which takes place outside the host, four sporocysts are formed within the oocyst, each containing two sporozoites. The oocyst is the resistant stage, able to survive outside the host for many months under suitable conditions (Fig. 129).

Oocysts of *Eimeria* spp. are distinguished from those of *Isospora* spp. by the content of the sporulated oocyst. *Eimeria* oocysts have four sporocysts with two sporozoites each and *Isospora* have two sporocysts with four sporozoites each.

Diagnosis Young animals are particularly prone to infection of coccidiae. Verification of suspected cases of coccidiosis depends on the demonstration of unsporulated oocysts either in smears prepared from fresh feces or by concentration methods involving flotation in saturated salt solu-

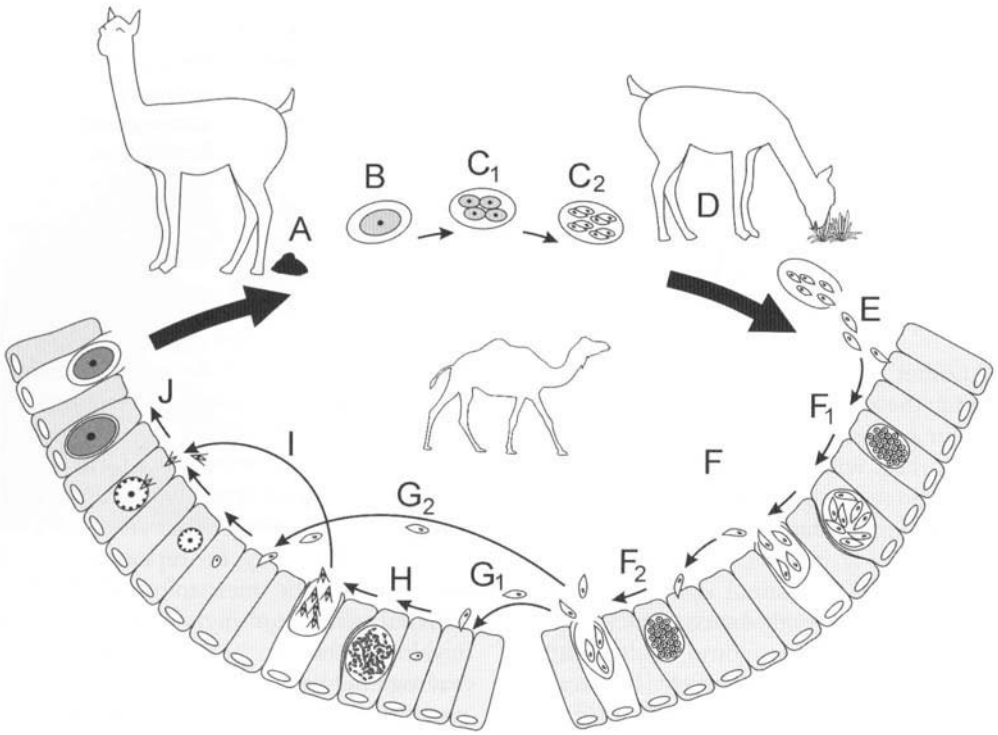


Figure 129 Life cycle of *Eimeria*: A = feces; B = oocyst; C_1, C_2 = oocyst sporulation; D = initial infection; E = invasion of intestinal mucosal cells by sporozoites; F = schizogony, several schizont generations; F_1 = first stage schizont; F_2 = second stage schizont; G_1, G_2 = end of schizogony; merozoites give rise to male and female gametocytes; H = microgametocyte; I = male (micro-) gamete fertilizes a female (macro-) gamete; J = cyst wall forms around the fertilized macrogamete (zygote) developing to oocyst

tions. Identification of the different species is usually conducted on the morphology of the oocysts. It is often necessary to sporulate the oocysts for species differentiation. At necropsy, lesions in the intestine may be recognized and asexual stages may be seen in scrapings of the intestinal mucosa and on histological sections. The main characteristics of the *Eimeria* species reported in camelids are listed in Table 52 a and b. Fig. 130 a–c shows the most important species found in dromedaries in the UAE.

Occurrence Intestinal coccidia found to infect *Camelidae* are *Eimeria* and less so *Isospora* (Ouhelli and Dakkak, 1987). *Eimeria cameli* and *E. dromedarii* are the most widespread species of camelid *Eimeria*, infecting both Bactrian and dromedary camels. There are five *Eimeria* spp. found in camels (see Table 52 a) and two *Isopora* spp.: *I. orlovi* and *I. cameli*. *I. orlovi* (Zigankoff, 1950) is thought by Péllerdy (1965) to be an avian form accidentally ingested. The same probably applies to *I. cameli*. Recently *Isospora* sp. was isolated from 1–3-week-old calves in a dromedary herd in Kenya. The calves exhibited profuse diarrhea. One isolation was from a calf which died from the infection. Raisinghani et al. (1987) isolated *Isospora* spp. from a dromedary calf showing abdominal pain and diarrhea. The sporulated oocysts were oval to ellipsoidal and measured 29.5 × 18.4 µm. Each

Table 52 a Oocyst morphology of *Eimeria* spp. reported in OWC (after Levine, 1985)

Species	Size (µm)	Shape	Wall	Micropyle
*Eimeria bactriani**	22–34 × 25–27	spherical	1 layer	present
*E. cameli***	81–100 × 63–94	piriform	thick	present
*E. dromedarii****	23–33 × 21–25	ovoid, brown	2 layers	present
*E. rajasthani*****	34–39 × 25–27	ellipsoidal	2 layers	not visible
*E. pellerdyi******	22–24 × 12–14	oval	2 layers	absent

* This species has been found in the small intestine of Bactrian and dromedary in Russia (Levine and Ivens, 1970), but Dubey and Pande (1964) do not recognize *E. bactriani* as a valid species while Pellerdy (1974) does

** This species is presumably common in the small intestine and to a lesser extent in the cecum of the dromedary and Bactrian camel (Henry and Masson, 1932; Reichenow, 1952; Soulsby, 1982; Levine, 1985)

*** This species is apparently quite common in feces of dromedaries and Bactrian camels in India, Iraq and Pakistan (Levine and Ivens, 1970)

**** This species is common in the feces of dromedaries in India (Dubey and Pande, 1964)

***** This species occurs in the feces of the Bactrian camel. Its prevalence and geographic distribution are unknown (Prasad, 1960)

Table 52 b Oocyst morphology of Eimeria spp. reported in NWC by Guerrero et al. (1967)

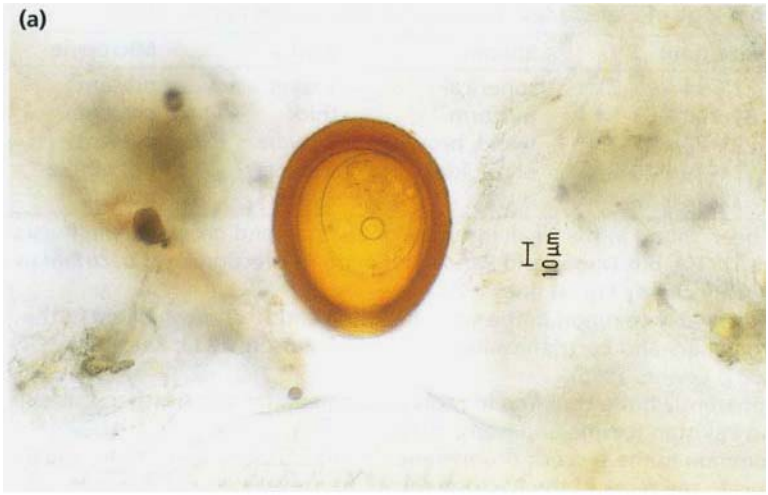
Species	Size (µm)	Shape	Wall	Micropyle
E. alpacae	22–26 × 18–21	ellipsoidal	thick	present
E. lamae	30–40 × 21–30	ovoid-ellipsoidal		present
*E. macusaniensis**	81–107 × 61–80	ovoid, brown		present
E. punoensis	17–22 × 14–18	ellipsoidal-ovoid	thick	present
E. peruviana	28–37 × 18–22	ovoid		absent
E. auburnensis	32–46 × 20–25	ovoid	smooth	present

* Pathogenic according to Rosadio and Ameghino (1994)

oocyst contained 2 sporocysts with 4 sporozoites. The parasite was believed to be *Isospora orlovi*. It is presumed that the parasite was accidentally ingested through avian droppings (Pellerdy, 1974). Recently in the UAE, a similar Isospora was found in dromedary calves' bloody diarrheic feces (Fig. 130 d).

The species associated with disease are primarily *E. cameli* and *E. dromedarii*. Hussein et al. (1987) also found *E. rajasthani* to be pathogenic in a survey conducted in Saudi Arabia. Several researchers have identified a sixth *Eimeria* species: *E. nolleri* (Partani et al., 1999) which is probably nonpathogenic. The pathogenic role of two *Isospora* spp., *I. orlovi* and *cameli* is according to Kaufmann (1996) unknown.

Six *Eimeria* species have been described from NWC (Table 52 b). A limited number of severe outbreaks of coccidiosis of OWC and NWC, some with mortality rates up to 10% in young dromedaries in Chad, have been reported (Gruvel and Graber, 1965). Haenichen et al. (1994) reported that 13 out of 16 adult llamas died from coccidiosis in Germany. The animals were emaciated and developed watery diarrhea shortly before death. Histology revealed an extreme invasion of the intestinal mucosa with different stages and oocysts of the genus *Eimeria*. The parasites were only observed in the jejunum, but not in the colon. Three different *Eimeria* species were identified: *E. macusaniensis, E. punoensis* and *E. spec.* (Minck, 1968). However, most reports are



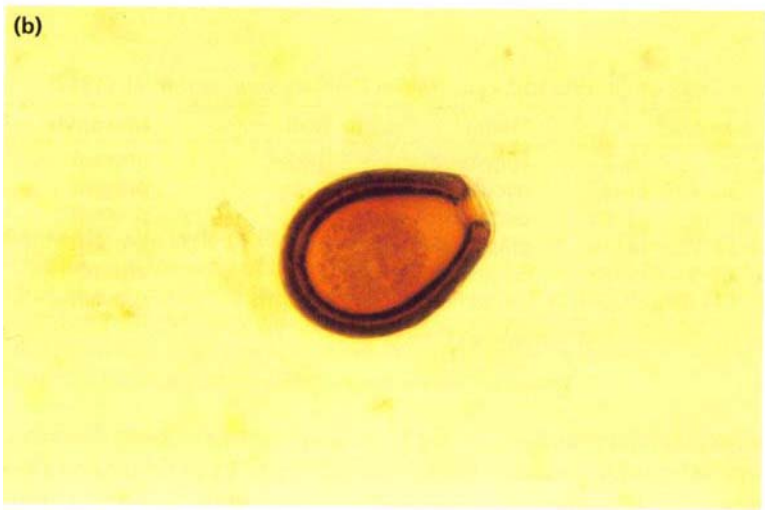
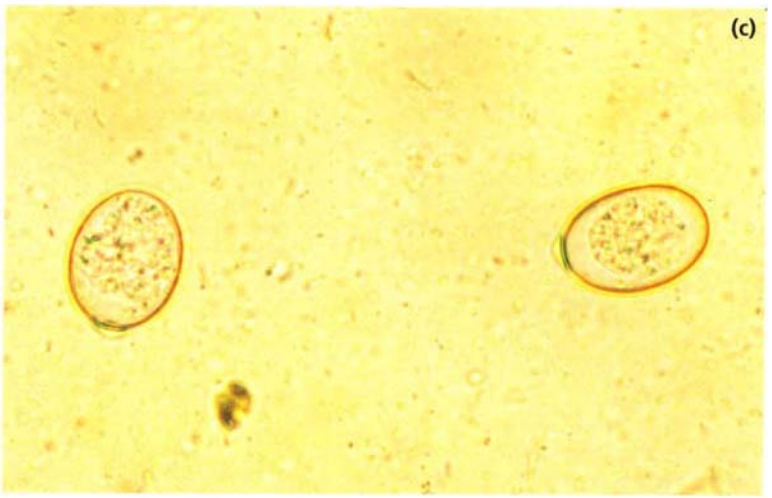


Figure 130 a–d Oocysts of Old World Camelids: **(a)** *Eimeria dromedarii* **(b)** *Eimeria cameli*

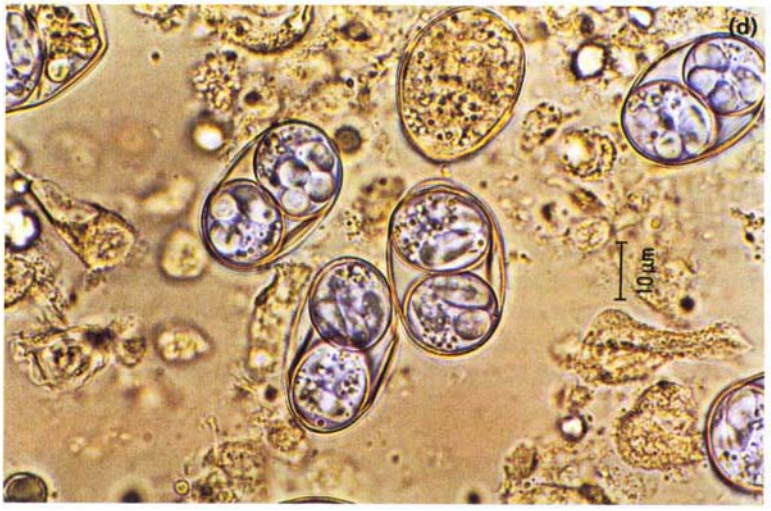
based on fecal examination of healthy camelids. A summary of the prevalence of *Eimeria* infections from different countries is shown in Table 53.

Clinical Signs Young animals suffer from hemorrhagic enteritis (Fig. 131) and diarrhea. The feces may be stained with blood and mucus (Hussein et al., 1987). Animals with severe infections show signs of inappetence, dehydration, and progressive weight loss. Their coat is rough and hair loss may occur. Anemia is often seen and respiration may be rapid. Secondary bacterial infections may severely aggravate the disease and cause mortalities in young camels (Kinne and Wernery, 1997).

Pathology Development stages of the parasites are found in the mucosa and lamina propria of the jejunum and ileum. Histological sections show destruction and disorganization of the mucosa together with hemorrhages and infiltration of inflammatory cells (mainly eosinophils and macrophages) (Figs. 132 and 133).



(c) *Eimeria* of probably goat origin often found in dromedary fecal samples in the UAE



(d) *Isospora orlovi* oocysts with 2 sporocysts containing 4 sporozoites from a dromedary calf with bloody diarrhea

Immunity In ruminant livestock immunity develops following infection, which is thought to be a combination of cellular and humoral factors. It is unknown whether the same principles can be referred to *Camelidae*. Both *Eimeria* spp. and *Isospora* spp. are host-specific and immunity to any one species is only effective for that species. Coccidial infections are generally self-limiting unless a re-infection takes place. Clinical coccidiosis must be treated, but finding oocysts in the feces is not a criterion for therapy. On the contrary, therapy of non-clinical infection may defeat the animal's ability to mount an immune response.

Diagnosis Diagnosis is based on clinical signs of diarrhea, dysentery and often the demonstration of very large numbers of oocysts in the feces (microscopic examination following flotation with e.g. salt or sugar solutions by Fuelleborn's method). A direct smear of diarrheic feces examined under a microscope may reveal oocysts. However, peracute and acute diseases may be exhibited before oocysts are excreted.

Table 53 Prevalence of *Eimeria* infections in camelids in different countries

Authors	Year	Country	Species	Prevalence
Yakimov	1934	Kazakhstan	Bactrian	22
Pellerdy	1956	Kazakhstan	Bactrian	40
Prasad	1960	India	Dromedary	
Dubey and Pande	1964	India	Dromedary	
Gruvel and Graber	1965	Chad	Dromedary	
Mirza and Al Rawas	1976	Iraq	Dromedary	86
Gill	1976	India	Dromedary	24
Chineme	1980	Nigeria	Dromedary	
Kawasmeh and El Bihari	1983	Saudi Arabia	Dromedary	14
Levine	1985	Africa	Dromedary	
Kasim et al	1985	Saudi Arabia	Dromedary	
Hussein et al.	1987	Saudi Arabia	Dromedary	
Yagoub	1989	Sudan	Dromedary	14.5
Daruish and Golemansky	1993	Syria	Dromedary	
Kinne and Wernery	1997	UAE	Dromedary	
Mahmoud et al.	1998	Saudi Arabia	Dromedary	13
Partani et al.	1999	India	Dromedary	25
Guerrero et al.	1967, 1971	South America	NWC	
Schrey et al.	1991	USA	NWC	28
Fowler	1998	USA	NWC	
Jarvinen	1999	USA	NWC	

Young animals may have had previous contact with the coccidia and there is a possibility, as is the case in some other animal species, that they have established an immune response.

Identification is done by the morphology of the freshly excreted oocysts as well as the sporulated oocysts. Sporulation of the oocysts, usually employing a 2.5% potassium dichromate solution, is achieved by in-

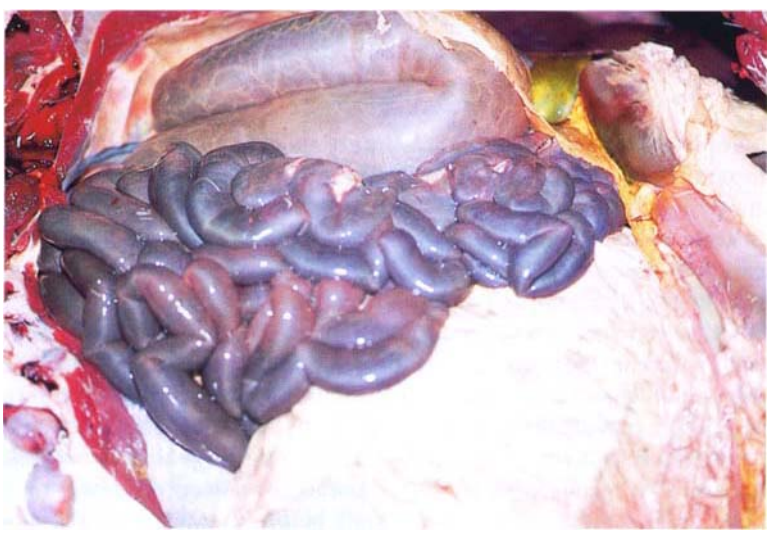


Figure 131 Hemorrhagic enteritis caused by *E. dromedarii* in a young dromedary

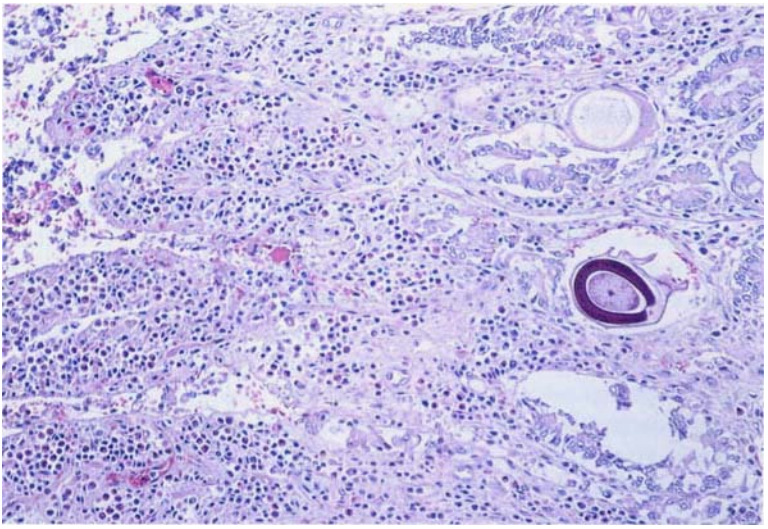


Figure 132 Severe eosinophilic enteritis in a 7-year-old dromedary caused by coccidiosis; note the different developmental stages of the parasite

cubating the oocysts at 25°C for about 10 days, depending on the species involved. The morphology of the sporocysts are helpful in the diagnosis of species. In many cases of necropsied dromedaries in the UAE, coccidiosis was only confirmed during necropsy by histological investigations. In these cases, masses of coccidial developmental stages (see Fig. 132) were seen histologically, but no oocysts were detected in feces. The simple flotation method might not be adequate to isolate the large and heavy oocyst of *E. cameli* (Kinne and Wernery, 1997).

At necropsy, mucosal scrapings may be directly examined as smears under the microscope and may often be diagnostic if oocysts and the different sexual stages are seen. Several scrapings should be taken from different sites of the small intestine.

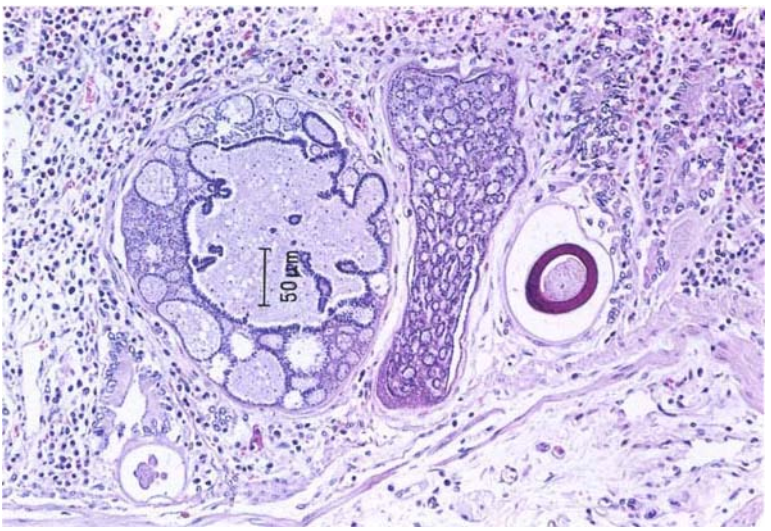


Figure 133 Eosinophilic enteritis; note the unsporulated oocyst of *E. cameli* (right)

Treatment Coccidiosis is a self-limiting disease. Following the multiplication stages in the intestine, recovery is often spontaneous and occurs without any specific treatment.

Anticoccidials are used to control coccidiosis outbreaks in livestock. However, very little is known about the doses and efficacy of anticoccidial drugs in camelids.

There are numerous anticoccidials used in ruminants. Their use has been recommended in NWC with caution because there is species sensitivity to some of the drugs (Fowler, 1998). With regard to OWC, Hussein et al. (1987) successfully treated infected animals with sulfadimidine. Haenichen et al. (1994) used the following drugs in llamas: sulfadimethoxin (Theracanzan®, 50 mg/kg i.m. for 3 to 5 days. In young animals, the authors recommend formosulfathiazol (Socatyl®), 100–200 mg/kg given orally for 3 to 5 days, and in severe cases in combination with Theracanzan®. Another drug is toltrazuril (Baycox®) which is given orally: 15–20 mg/kg for 3 to 5 days. Drugs recommended for treatment of coccidiosis in domestic ruminants are listed in Table 54.

Camels seem to be very susceptible to poisoning by ionophorous antibiotics, and overdosing with Salinomycin and Monensin has recently been reported in dromedaries (Wernery et al., 1998; Chaudhry et al., 1998) and in a Bactrian (Miller et al., 1990). Poisoning is characterized by skeletal and heart muscle degeneration and

Table 54 Anticoccidial drugs recommended for domestic ruminants

Drug	Usage
Amprolium	therapeutic and prophylactic
Sulfonamides	
Sulfamethazine	therapeutic
Sulfaquinoxaline	therapeutic
Sulfaguanidine	prophylactic (for sheep and swine)
Ionophorous antibiotics	
Monensin	prophylactic
Lasalocid	prophylactic
Miscellaneous compounds	
Nitrofurazone	therapeutic
Decoquinate	prophylactic (for cattle)
Toltrazuril	therapeutic (for sheep)
Diclazuril	therapeutic (for sheep and goats)

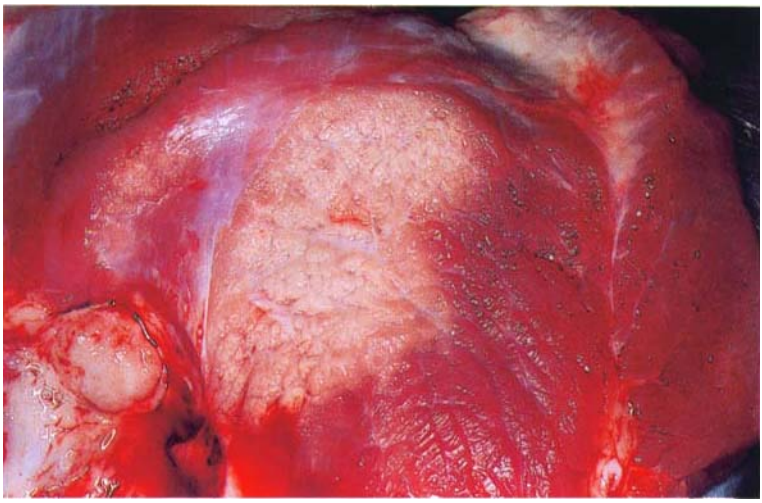


Figure 134 Degeneration of skeleton muscle in a dromedary caused by Salinomycin poisoning

splayed legs in conjunction with extremely elevated muscle enzymes (Fig. 134).

Control The control of clinical coccidiosis in young calves is essential and may be achieved by good management. Calving grounds should be well drained and kept as dry as possible. Stocking rates should be kept to an acceptable level so as to avoid overcrowding, reducing the risk of a build-up of infections. Feed and water troughs must be kept free of contamination from feces. Frequent rotation of pastures is a prerequisite for keeping most parasites at bay.

5.1.8 Cryptosporidiosis

Other Coccidia of importance to domestic animals are *Cryptosporidium* spp., pathogens of mammals that are usually confined to the microvilli of the intestinal mucosa of the host. The small oocysts (4.0–4.5 µm) sporulate within the host and are infective when released in the feces. The infective oocyst contains 4 sporozoites. According to Kaufmann (1996), *C. parvum* may infect young camels. An infection can lead to severe diarrhea, emaciation, dehydration and death. Oocysts of *Cryptosporidium* sp. were found in 15 dromedary camels in an epidemiological survey in Egypt (Abou-Eisha, 1994). Fayer et al. (1991) reported a zoo Bactrian chronically infected with a *Cryptosporidium* sp. resembling *C. muris*. Isolates of this organism were found to colonize gastric glands in experimentally infected mice. Histologically, epithelial hyperplasia with mucosal hypertrophy without any inflammation was seen (Anderson, 1991). These changes were considered consistent with chronic gastric cryptosporidiosis in cattle (Anderson, 1987). Transmission is via feces contaminating drinking water.

Diagnosis Oocysts are demonstrated in stained smears of fresh feces (Fig. 135). The most commonly used is a modified Ziehl-Neelsen stain (counter-stained with carbol-fuchsin). The oocysts stain deep red against a green-blue background. Several diagnostic enzyme immunoassays (EIAs) and direct and indirect immunofluorescent antibody tests have been developed (Graczyk et al., 1996).

Treatment and Control Although a large number of chemotherapeutic antimicrobial compounds have been tested for their

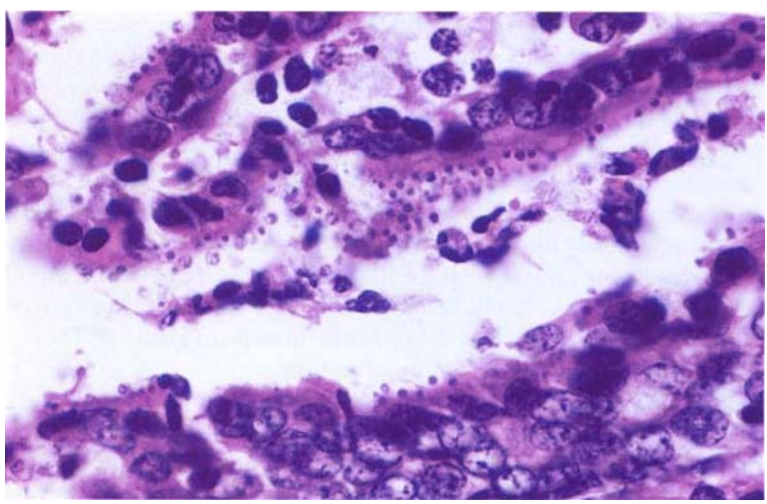


Figure 135 *Cryptosporidium* oocysts in the intestinal mucosa

efficacy against cryptosporidiosis, no really effective compound for therapy or prophylaxis has been found (Fayer et al., 1991). However, recently a few antiprotozoal drugs have been recognized as having some therapeutic and prophylactic properties, e.g. halofuginate lactate (Yvore and Naciri, 1989; Peeters et al., 1993), and paromomycin (Fayer and Ellis, 1993) in dairy calves. Halofuginate lactate is commercially available as Halocur® vet (Intervet). Another compound, lasalocid, showed promising anticryptosporidial effect both in *in vitro* and *in vivo* trials when employed at a low dosage (Castro Hermida et al., 2000). Lasalocid is an ionophoric antibiotic produced by *Streptomyces lasaliensis*. It is not known whether these drugs are tolerated by camels.

Rehydration may help in mild to moderate cases. Oocysts are resistant to most disinfectants except formalin (5%) and ammonia. They can survive for months in the environment if kept cool and moist. Optimal management with well-drained calving grounds and the avoidance of overcrowding will prevent infection.

5.1.9 Sarcocystiosis

Species of Sarcocystidae have a two-host LC: an asexual stage in an intermediate host and a sexual in the final host.

Etiology *Sarcocystis* spp. are parasites using two hosts to complete their LC. Carnivores commonly act as final hosts and herbivores as intermediate hosts. *Sarcocystis* spp. are mostly host-specific for their intermediate hosts, but less so for their final hosts (Dubey et al., 1989). The general developmental cycle in the final hosts in which sexual stages occur is similar to that of the *Eimeria* spp., except that there is no asexual multiplication, sporulation takes place in the intestinal wall and sporocysts are excreted for several weeks. In the intermediate host, the infective sporocysts, following ingestion of contaminated feces of the final host, release sporozoites into the intestine. The sporozoites invade many organs via the blood stream. Schizogony occurs in the endothelial cells of blood vessels of many organs before typical cysts in the striated muscles develop.

Clinical Signs Usually the definitive hosts carry the infection without showing any signs of disease. Most infections by *Sarcocystis* spp. in the intermediate hosts are subclinical; however, some infections may cause losses (recorded in domestic animals). Acute clinical disease may occur (referred to in cattle as Dalmeny disease) causing abortion, reduced milk production, wool breakage, lameness, suboptimal growth rate, and sometimes death in cases of heavy infection. Experimental studies have shown that even subclinical infections may have a negative effect on growth and blood parameters in young animals (Leek et al., 1977; Giles et al., 1980).

According to Fowler (1998), light infections give no clinical signs in NWC, but in heavily infected animals the schizogony cycles in endothelial cells may give acute febrile disease, resulting in abortion and death. Also, mild myositis with myalgia may be seen interfering with muscular function. Some llamas have shown clinical signs similar to those in horses with protozoal myeloencephalitis caused by *S. falcatula* (Fowler, 1998). Recently, La Perle et al. (1999) described Dalmeny disease in an alpaca caused by *S. aucheniae*. It revealed an eosinophilic myositis associated with macroscopic sarcocysts and aborted two hours before death. The animal had been imported five years earlier to the USA from Peru.

Occurrence Myocardial lesions have been attributed to *Sarcocystis* spp. in camels (El-Etreby, 1970). Mason (1910) who found the cysts primarily in the myocardi-

um and esophagus of camels, and first reported *Sarcocystis cameli*, described two different thin-walled and thick-walled cysts, and thought that they belonged to the same species. Since then *S. cameli* has been reported in Afghanistan, Egypt, Iran, Sudan and the former USSR (Dubey et al., 1989). The latter authors reviewing the previous studies on camel sarcocysts named the thick-walled cysts *S. cameli*. Fatani et al. (1996) found two morphologically distinct sarcocysts. The thin-walled cyst was found in all three indicator organs: diaphragm, heart and esophagus, but the thick-walled cyst was only present in the esophagus. Both types were microscopic. Dogs have been found to be the final host of at least one of the parasites (Hilali and Mohamed, 1980; Kuraev, 1981; Hilali et al., 1995; Fatani et al., 1996). The cysts, according to Hilali and Mohamed (1980) and Hilali et al. (1995), are up to 12 mm long and 2 mm in diameter. The crescent-shaped bradyzoites that fill the cysts are 15–20 by 4–6 µm (Fig. 136).

Several scientists have fed *Sarcocystis* spp.-infected camel meat to dogs and cats.

The sporulated *Sarcocystis* sporocysts were only recovered in dog feces (Hilali et al., 1982; Warrag and Hussein, 1983).

The prevalence of infection with *Sarcocystis* in camel carcasses at slaughter varies from 4.5% in Sudan (Ouhelli and Dakkak, 1987) to 88% in Saudi Arabia (Fatani et al., 1996). The infection is of economic importance because part of or the entire carcass may be condemned at meat inspection.

Three *Sarcocystis* species have been reported in NWC (Leguia, 1999). *Sarcocystis aucheniae* was demonstrated in Bolivia and Peru (Guerrero, 1967; Fernandez-Baca, 1975) in alpaca, llama and vicuña and *Sarcocycstis tilopoidi* (syn. *S. guanicoe-canis*) in guanacos (Gorman et al., 1984; Leguia, 1999). Both species produce macrocysts in the muscles. A third species, *S. lama-canis* (Leguia et al., 1989) is found as microcysts in alpacas in the myocardium and muscles. The final host of at least one of the species is the dog (Schnieder et al., 1984). The prevalence of *Sarcocystis* sp. in certain areas of Peru was estimated to be over 50% in animals above two to three years of age (Fernandez-Baca, 1975; Fowler, 1998). The infections are of economic significance. Losses in alpacas are estimated to reach $US 300,000 annually.

Diagnosis *Sarcocystis* spp. may be identified by the typical ultrastructure of their

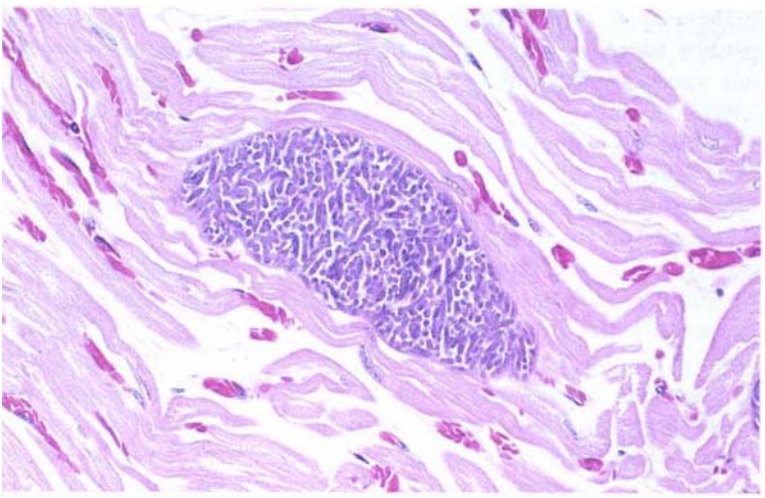


Figure 136 *Sarcocystis cameli* in the heart muscle of a dromedary

cyst wall (Dubey et al., 1989). The oocysts of the *Sarcocystis* spp. lack a micropyle and they have a fine colorless wall. Sporulated oocysts contain two sporocysts each with four sporozoites. The sporocysts are released in the intestine before being shed through the feces.

Macroscopic cysts are seen during meat inspection or at necropsy. Microscopic cysts are often found accidentally in a histologic section of muscle, including the myocardium. Cysts or free bradyzoites may be demonstrated in squash preparations of small pieces of fresh meat samples followed by stereomicroscopy (magnification 10–60) (Gut, 1982). Peptic digestion of minced muscular tissue followed by examination by a light microscope preferably equipped with phase contrast may be used to diagnose released bradyzoites (Dubey et al., 1989). This method can also be used to extract the bradyzoites for further antigenic or molecular biological investigation (Lunde and Fayer, 1977). It is not possible to identify the particular species based only on the morphology of the bradyzoites. Histological examination is often used to demonstrate the presence of microscopic sarcocysts in the tissues of a host.

There are several serological tests that have been developed for the diagnosis of *Sarcocystis* infections in different intermediate hosts. Reported techniques so far are usually based on crude antigens that are not species specific (Uggla and Buxton, 1990).

Molecular techniques mostly based on PCR have proved to be useful in detecting infection and species identification.

Oocysts and sporocysts may be found during fecal examinations of the final host by using traditional flotation techniques based on saturated sodium chloride, sucrose or zinc sulfate solutions. Additionally, the organisms may also be found in mucosal scrapings of the small intestine by a flotation-concentration technique (Dubey et al., 1989). However, species differentiation based on the morphology of the oocysts or sporocysts is not possible.

Treatment and Control *Sarcocystis* spp., with few exceptions, are considered to be non-pathogenic. Some infections may be subclinical and only detected after slaughter during meat inspection. Although experimental studies have shown that subclinical infections may also have negative effects on the growth of young animals (Leek et al., 1977; Giles et al., 1980), treatment and control are very seldom applied.

The only way to control *Sarcocystis* infections is to break the LC of the parasite. Domestic dogs and cats should not receive uncooked meat or offal. Therefore, at abattoirs it is important to keep offal away from predators. Bradyzoites are readily killed by freezing and by heating to approx. 65°C. The organisms may remain infective in uncooked or poorly cooked meat. Freezing to –18°C and cooking were effective for inactivating Sarcocystis in guanaco meat (Gorman et al., 1984).

5.1.10 Besnoitiosis

In India, Kharole et al. (1981) reported finding *Besnoitia* cysts at the base of the lamina propria in the intestine of a dromedary. Numerous different-sized cysts were found, ranging from 10 µm to several 100 µm. There was no systemic or cutaneous involvement as was reported by Fazil and Hofmann (1981) as is often the case in other animal species. No inflammation was seen although the intestinal tissue was damaged by pressure from the large cysts. Similar parasitic infections had been reported in buffalo calves, cattle and sheep in the same area. Fazil and Hofmann (1981) stated that besnoitiosis (which they called globidiosis) often occurred in camels with typical skin lesions on the distal part of the legs. The infection often became general-

ized with high fever and diarrhea indicating involvement of the intestines. Morbidity was low, but the mortality could reach 10% of clinically affected animals. Small and large cysts of *Besnoitia* was seen in the mucosa of the small intestine of six dromedaries in Iran (Tafti et al., 2000). Some of the cysts were surrounded by inflammatory reactions. It is most likely that the cysts in the intestinal mucosa described as *Besnoitia* are developmental stages of *Eimeria* species. The life cycle of the *Besnoitia* species occurring in the skin is still unknown.

Intracellular cysts, mainly within fibroblasts, characterize *Besnoitia*. A cyst wall is found around the infected cell with bradyzoites in a parasitophorous vacuole. The nucleus of the host cell undergoes hyperplasia and hypertrophy (Soulsby, 1982).

Cysts of several species of *Besnoitia* infect different domestic animals and wildlife. The best-known species of this genus is *B. besnoiti*, found particularly in Africa. The final host is the cat and the intermediate hosts are mainly cattle, in which the parasites are found in the dermis, subcutaneous tissues and fascia as well as in the laryngeal, nasal and other mucosa.

5.1.11 Toxoplasmosis

Toxoplasmosis is caused by the cyst-forming coccidial parasite *Toxoplasma gondii*, an important worldwide zoonotic pathogen. It is an intestinal coccidial parasite of *Felidae*, particularly cats, which become infected by ingesting *Toxoplasma*-infected animals, containing cysts of the organism. The parasite in the intermediate hosts (which can be almost any mammalian species including man) may cause a severe disease. Generally, however, *Toxoplasma* infections are subclinical, although in pregnant individuals the infection may cause abortion or congenital disease in the offspring. In sheep, abortions and perinatal mortality are commonly attributed to the infection. *T. gondii* is one of the most common cat zoonoses.

Life Cycle ▦ Two separate stages of multiplication of *T. gondii* may be recognized. The sexual cycle is only completed in the intestinal epithelium of felines (entero-epithelial phase) (Hutchison et al., 1970). This results in the development of oocysts, excreted in cat feces (felids) (Fig. 137).

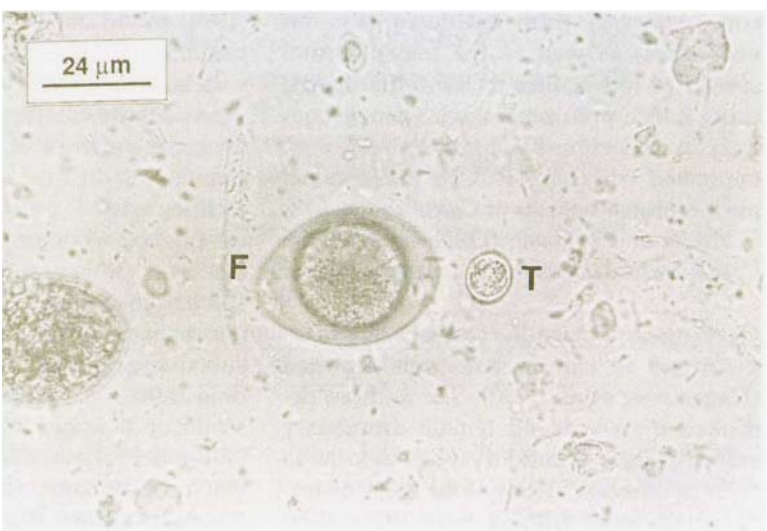


Figure 137 Oocyst of *Toxoplasma gondii* (T) next to an *Isospora felis* oocyst (F) from cat feces (courtesy of Professors Sloss, Kemp and Zajac, Veterinary Clinical Parasitology, 6th ed., 1994, Iowa State University Press, USA)

The oocysts are highly resistant when sporulated and can stay infective for a year or longer (Yilmaz and Hopkins, 1972). When the sporulated oocyst is ingested by a susceptible host (over 200 spp. have been recorded, including rodents, lagomorphs, insectivores, carnivores, marsupials, primates and many birds species (Levine, 1985)), the sporozoites emerge and enter tissues via the blood and lymph. Any type of cell may be invaded producing tachyzoites by endodyogeny, an asexual multiplication (extra-intestinal phase). This may also occur in the final host – the cat – parallel to the entero-epithelial phase.

In the acute infection, the tachyzoites rapidly multiply within any nucleated cell. New cells are invaded after rupture of the infected cell, which contains large numbers of tachyzoites. As the infection proceeds, cysts within cells are formed containing hundreds of organisms named bradyzoites. The tissue cysts measuring up to 300 µm may be found in any tissue, but are most commonly found in the brain, skeletal and heart muscle. These cyst formations are characteristic of the chronic infection.

Transmission Human infection may result from ingestion of cysts with bradyzoites, oocysts from cat feces (e.g. on vegetables) as well as the transplacental spread of tachyzoites to the fetus during acute infection in pregnancy. Camels contract the infection by ingesting feed contaminated with oocysts. Cats given camel meat excreted oocysts of *Cystoisospora felis*, *C. rivolta* and *T. gondii* (Hilali et al., 1995). *C. felis* and *C. rivolta* are coccidia of cats.

Occurrence Only one case of acute toxoplasmosis in camels has been reported (Hagemoser et al., 1990). The authors described a six-year-old female dromedary showing signs of mild dyspnea associated with pyothorax. Twenty-four liters of turbid fluid were drained from the pleural cavity and *Toxoplasma* tachyzoites were found in macrophages and neutrophils in smears. The camel had become anorectic a month earlier and aborted a near-term fetus. Several serological tests, including the Sabin-Feldman dye test, performed on the pleural fluid and the serum showed antibody titers to *T. gondii*.

Several seroepidemiological toxoplasmosis surveys in *Camelidae* have been reported (Table 55).

Both in India and Saudi Arabia there was a higher prevalence of antibodies in adults than in younger animals similar to findings in other hosts. This was attributed to a longer period of exposure to the parasite in older animals (Gill and Prakash, 1969). Hussein et al. (1988) found an association between husbandry methods and the seroprevalence of *T. gondii* infections. Housed camels had a much higher prevalence due to exposure to the final hosts (cats) than camels in the desert. This has been confirmed by a serological survey in the UAE (unpublished) using the recently established *T. gondii* ELISA (Chekit Toxotest® Dr. Bommeli, Switzerland). A seroprevalence of 38% in 521 racing dromedaries was detected. Other researchers using an indirect hemagglutination test also confirmed this result. Afzal and Zakkir (1994) found 36.4% reactors in dromedaries in the UAE.

It is difficult to determine the significance of the results from these surveys. However, the presence of antibodies shown in camels is indicative of past or present infections with *T. gondii*. It still needs to be established whether the *T. gondii* infection has any clinical significance in camels. Clinical toxoplasmosis-like signs were experimentally induced in three camels by subcutaneous injections of peritoneal exudate from mice infected by a pathogenic strain of *T. gondii* (Galuzo, 1965 cited by Gill and Prakash, 1969). However, earlier similar trials failed to produce the clinical disease in camels (Blanc et al., 1951).

Table 55 The prevalence of *T. gondii* in camelids in different countries

Authors	Year	Country	Test	Camels/ Llamas	% positive
Kozojed et al.	1976	Afghanistan	Micromodification of Indirect Hemagglutination Test	19	73.6
Gorman et al.	1999	Chile	Indirect Hemagglutination Test	447	16.3
El-Ridi et al.	1990	Egypt	Indirect Hemagglutination Test	19	26.3
Fahmy et al.	1979	Egypt	Sabin-Feldman Dye Test	119	24.4
Michael et al.	1977	Egypt	Sabin-Feldman Dye Test Complement Fixation Test	80	83.7 2.5
Rifaat et al.	1977	Egypt	Sabin-Feldman Dye Test	43	67.4
Rifaat et al.	1978	Egypt	Sabin-Feldman Dye Test	73	63
Maronpot and Botros	1972	Egypt Egypt	Indirect Fluorescent Antibody Test Indirect Hemagglutination Test	49	6
Hilali et al.	1998	Egypt	Direct Agglutination Test	166	17.4
Okoh et al.	1981	Nigeria	Indirect Hemagglutination Test	159	0
Hussein et al.	1988	Saudi Arabia	Indirect Hemagglutination Test	227	16
Bornstein and Musa	1987	Sudan	Sabin-Feldman Dye Test	102	22.5
Abbas et al.	1987	Sudan	Indirect Hemagglutination Test	95	12
Eldin et al.	1985	Sudan	Indirect Hemagglutination Test Micromethod	204	54
Elamin et al.	1992	Sudan	Latex Agglutination Test	482	67
Berdyev	1972	Turkmenistan	Complement Fixation Test	200	4.5
Chaudhry et al.	1996	UAE	Indirect Latex Agglutination Test	100	18
Afzal and Sakkir	1994	UAE	Direct Agglutination Test Indirect Hemagglutination Test	– –	30.9 36.4
Dubey et al.	1992	USA	Modified Agglutination Test	283	33.5
Unpublished	2000	UAE	ELISA	521	38.0
Leguia et al.	1991	Peru	Indirect Agglutination Test	–	25
Leguia et al.	1984	Peru	Indirect Agglutination Test	–	50

This ubiquitous parasite has also been reported in NWC. Abortions have been associated with *T. gondii* (Cheney and Allen, 1989; Johnson, 1993) and seroprevalent studies have been undertaken. Two llamas, experimentally infected orally with *T. gon-*

dii oocysts, remained clinically normal and one delivered a healthy offspring (Jarvinen et al., 1999). Antibodies to *T. gondii* were shown in the two adult animals, employing several tests, but no specific antibodies were detected in precolostral sera obtained from the offspring suggesting that there was no fetal *T. gondii* infection.

Diagnosis Oocysts in the final host may be found in the feces. Demonstration and isolation of the parasite may be achieved by testing material from the intermediate hosts by histology, immunohistology, serology, molecular biological techniques (PCR), animal inoculation and pepsin digestion. In addition the organism may be cultured *in vitro*. In abortion cases the parasite may be isolated from the placenta.

A large number of different serological methods have been used for the demonstration of antibodies to *T. gondii*. The most commonly used serological test has been the classical dye test of Sabin and Feldman (1948) traditionally regarded as the definitive test. However, it is a time-consuming and expensive test and has been replaced by a range of others such as: complement fixation test, indirect fluorescent antibody test, indirect hemagglutination test, direct agglutination test and ELISAs. Detectable levels of the antibodies will not be found until the end of the short period of oocyst shedding in the final host (Dubey and Frenkel, 1972). In some intermediate hosts such as sheep and pigs, the antibodies may be demonstrated when the viable *T. gondii* organisms are present in the muscles and other organs (Work, 1967; Boch and Neurohr, 1982).

Public Health Concern Although, there is great uncertainty whether camelids harbor *T. gondii* cysts in their muscles and/or organs, hitherto no pathological evidence of such infections has been reported. Consumption of undercooked camel meat may constitute a risk of infection to humans and should therefore be of public health concern. Bradyzoites in the cysts do not survive heating to 65 °C nor freezing (−20 °C) with subsequent thawing (Frenkel, 1982).

Treatment and Control Effective treatment of toxoplasmosis is difficult to achieve. The antimalarial drug pyrimethamine (2,4-diamino-5-p-chlorophenyl-6-ethyl-pyrimidine) in combination with sulphadiazine is effective against tachyzoites (acute form of the disease) (Soulsby, 1982; Urquhart et al., 1996). Oocyst shedding is reduced and partly inhibited in infected cats given this combination (Frenkel, 1975; Sheffield and Melton, 1976) and almost completely inhibited in cats given 5 mg/kg toltrazuril daily (Daugschies, 1996).

In livestock, treatment of ovine toxoplasmosis with a combination of sulfamezathine and pyrimethamine proved successful (Buxton et al., 1993). There is to the authors' knowledge no reported treatment of toxoplasmosis in camelids. However, if the infection is diagnosed in a herd of camels, control measures should be employed. Foodstuff should be stored so that cats, mice and insects cannot contaminate it. Cats should not be given raw meat. A live vaccine (Toxovac®, Intervet) is available for use in sheep.

5.1.12 Neosporosis

Neospora caninum is a protozoan parasite earlier confused with *T. gondii*. The sexual stage occurs in a final host from which oocysts are excreted. Experimental studies have recently been able to identify domestic dogs as the final host (Dubey and Lindsay, 1996).

N. caninum was first recognized in dogs in 1988 and has since been reported worldwide (McAllister et al., 1998). Neosporosis is severe in transplacental infected puppies. The most characteristic signs are progressive ascending paralysis, particularly

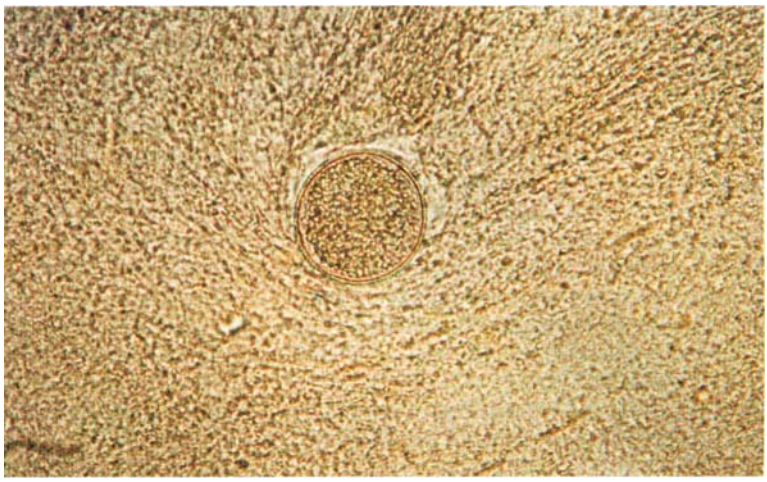


Figure 138 *Neospora caninum* cyst in the brain of a mouse (courtesy of Prof. P. Fioretti, Italy)

of the hind limbs. Polymyositis and hepatitis may also occur.

Neosporosis also affects cattle (intermediate host) and is a relatively common cause of abortion and neurologically associated limb disorders in calves. It is regarded as the most common cause of cattle abortions in the USA.

Whether this parasite infects camels has not yet been properly documented. Preliminary studies indicate that camels may become infected with the parasite (Naeslund, unpublished). Antibodies to *N. caninum* have been demonstrated employing an ELISA developed earlier for serology in dogs and cattle (Bjoerkman et al., 1997). Hilali et al. (1998), employing a direct agglutination test, reported finding antibodies to the parasite in a few of 161 camels in Egypt. Although the parasite is closely related to *T. gondii* there is no convincing evidence that *N. caninum* will infect or cause disease in humans (Fig. 138). There are at present no effective control measures to prevent disease or infection.

5.1.13 Hammondiosis

Previously only one species, *Hammondia hammondi* with a rodent-cat cycle had been known. However, *H. heydorni* which has ruminants as intermediate and dogs as final hosts has now been also isolated from dromedaries. Two dogs fed 500 g each of musculature from esophagus collected from 30 camels slaughtered at a Cairo abattoir started shedding *H. heydorni* oocysts from day 8 and 10 respectively for 5 and 7 days (Nassar et al., 1983). Warrag and Hussein (1983) and Hillali et al. (1992, 1995) also found that dogs experimentally fed dromedary camel meat shed *H. heydorni* oocysts.

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5.2 Infestations with Ectoparasites

Camelids like other livestock are exposed to and affected by a range of ectoparasites (Table 56), which may directly or indirectly cause a great diversity of health problems. Some ectoparasites play a significant role in many disorders. For example, some biting insects are vectors of disease agents such as *T. evansi*, and the mite *Sarcoptes scabiei* is the cause of sarcoptic mange. Both are regarded as the two most economically important diseases in camelids, the latter especially in Peru, which has the largest NWC population (Alvarado et al., 1966).

The ectoparasites of camelids can be classified into two zoological classes, the Arachnea and the Insectea, both within the phylum Arthropoda.

5.2.1 Classification of Arachnea

Phylum Arthropoda
Class Arachnea
Subclass Acaria
Order Astigmata (Mites)
Family Sarcoptidae
Sarcoptes (OWC, NWC)

Family Psoroptidae
Psoroptes (OWC, NWC)
Chorioptes (OWC, NWC)

Table 56 Arthropods of camelids

Disease	*Species*	Occurrence		*Location*
		OWC	NWC	
Sarcoptic mange	*Sarcoptes scabiei*	+	+	Skin
Psoroptic mange	*Psoroptes* sp.	+	+	Skin
Chorioptic mange	*Chorioptes* sp.	+	+	Skin
Demodectic mange	*Demodex* sp.	+	+	Skin
Tick infestation	*Hyalomma* spp.	+		Skin
	Ambylomma spp.	+		Skin
	Ixodes spp.	+		Skin
	Rhiphicephalus spp.	+		Skin
Spinose ear tick	*Otobius megnini*		+	Ear canal
Sucking lice	*Microthoracius* spp.	+	+	Skin
Biting lice	*Damalinia breviceps*		+	Skin
Fleas	*Vermipsylla* spp.	+	+	Skin
Flies	Sarcophagidae	+	+	Wound, orifices
	Calliphoridae	+	+	Skin, perineum
	Oestridae	+	+	Nose, pharynx
	Glossinidae	+		Skin
	Tabanidae	+		Skin
Biting midges	*Culicoides*			Skin
Tongue worm	*Linguatula serrata*	+		Lymph nodes

Order Prostigmata
Family Demodicidae
Demodex (OWC, NWC)

Order Metastigmata (Ticks)
Family Argasidae (Soft ticks)
Ornithodoros savigny (OWC)
O. lahorensis (OWC)
O. tholozani (OWC)
Otobius megnini (NWC)

Family Ixodidae (Hard ticks)
***Hyalomma* spp.**
H. asiaticum (OWC)
H. dromedarii (OWC)
H. scupense (OWC)
H. franchini (OWC)
H. rufipes (OWC)
H. anatolicum (OWC)
H. detritum (OWC)
H. impressum (OWC)

***Amblyomma* spp.**
A. lepidum (OWC)
A. gemma (OWC)
A. variegatum (OWC)

***Boophilus* spp.**
B. decoloratus (OWC)

***Rhipicephalus* spp.**
R. pulchellus (OWC)
R. appendiculatus (OWC)
R. sanguineus (OWC)

***Dermacentor* spp.** (OWC, NWC)

***Ixodes* spp.**
I. holocyclus (OWC)

5.2.2 Sarcoptic Mange

Sarcoptic mange occurs in more than 100 species of mammals including humans. The disease in humans is generally referred to as scabies. The causative mite is *Sarcoptes scabiei*. The mite is thought to have a number of subspecies or variants, each designated according to which host it has been isolated from *S. scabiei* var. *hominis*, *S. scabiei* var *cameli*, *S. scabiei* var. *aucheniae* etc. However, the host-specificity is not complete and transmission from one host species to another may occur. The different isolates or subspecies are morphologically indistinguishable.

Morphology *Sarcoptes scabiei* belongs to the burrowing mites (Fain, 1978). It has an oval, ventrally flattened and dorsally convex tortoise-like body (Fig. 139).

Life Cycle The developmental cycle of *S. scabiei* consists of egg, larval, protonymphal and tritonymphal stages. The sarcoptic mites differ from most other mange mites; they inhabit the epidermis of the skin excavating tunnels in the outer cell layers. The mites burrow in the stratum corneum through the dead cell layers until they reach living cells in the stratum granulosum and stratum spinosum. Due to the continual outgrowth of the epidermis the burrows containing the mites and eggs are mostly found in the corneum. The mites are rarely found beneath the stratum germinativum.

The fertilized female lays her eggs in tunnels. Her lifespan is about four weeks and the development time from egg to adult is about 12 to 16 days. The eggs are produced at a rate of three to four daily. The eggs hatch in 3–5 days and larvae with three pairs of legs emerge (Fig. 140).

Epidemiology and Transmission Infection is mainly through direct contact. All three developmental stages (including the adults) are capable of migrating on the skin surface. However, infection occurs when the mites become dislodged by their host scratching or rolling on the ground, whereby infection may take place indirectly. Fomites also play an important part in the transmission of the mites. Sarcoptic

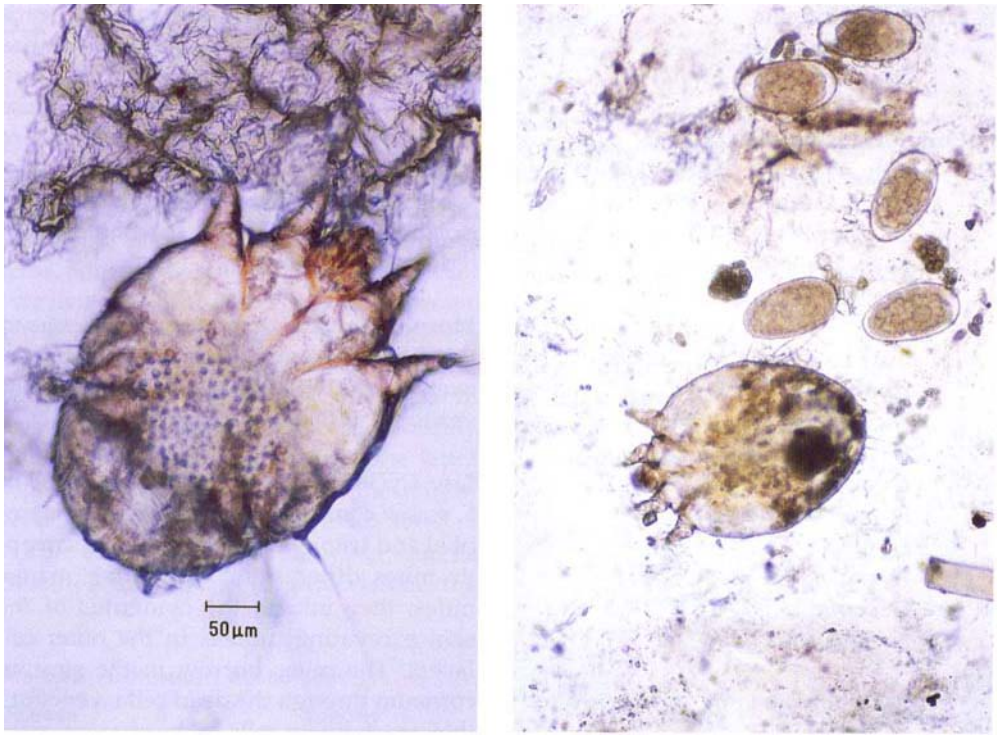


Figure 139 *Sarcoptes scabiei* with eggs from the skin of a dromedary (right), and a close-up of a female *S. scabiei* mite (left)

mites can survive outside their host for several days and remain infective (Arlian, 1989) if the microclimate is sufficiently moist and cool. During the dry season in the tropics, the mites most likely do not survive for long off the host. However, in crowded wet places such as waterholes, indirect transmission may occur, most probably during the cool and moist part of the night and early morning hours. Nayel and Abu-Samra (1986 a) found that *S. scabiei* of camels remained viable away from their host for 4 days. Observations indicate that when dislodged from their host, *S. scabiei* mites may remain infective between one half and two-thirds of their survival time (Arlian, 1989).

S. scabiei isolated from naturally infected sheep and goats have been successfully transferred to dromedaries (Nayel and Abu-Samra, 1986 a, b). Transmission of *S. scabiei* var. *auchenia* to sheep, horses and humans has been reported (Mellanby, 1946; Alvarado et al., 1966).

The infection is regarded as highly contagious and common among many animal species. It is particularly prevalent in swine, dogs and camelids, and less so in cattle, equines, sheep and goats. The disease also occurs worldwide in a wide range of wildlife species (Bornstein, 1995). The infection is endemic in some areas with epizootics occasionally resulting in high mortality in wildlife.

Sarcoptic Mange in Camelids

Sarcoptic mange is regarded as one of the most prevalent and serious camel diseases (Lodha, 1966; Higgins, 1983). It is often

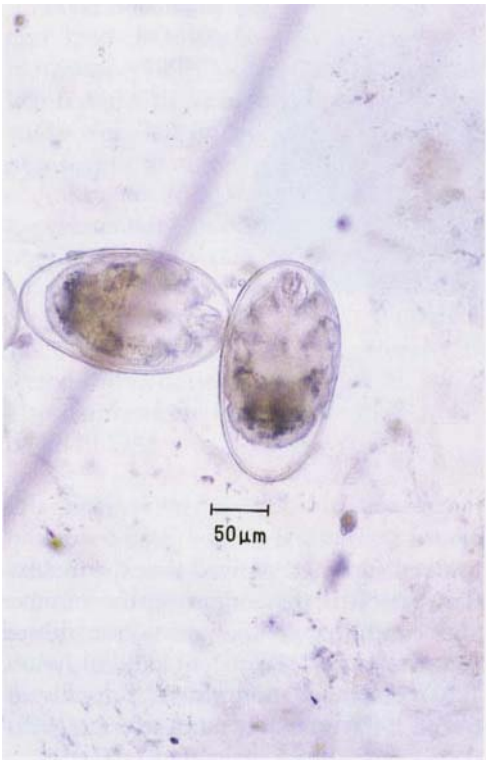


Figure 140 Larvated *Sarcoptes scabiei* eggs from the skin of a dromedary

ranked second in importance to all the disorders in dromedary camels (Pegram and Higgins, 1992), and second only to trypanosomosis. It can generally be regarded as a chronic debilitating condition with high morbidity and low mortality. The infection is also common among NWC (Fowler, 1998). The disease "sarna sarcoptica" was previously widespread in North American captive camelids where it appears to be decreasing, probably through routine deworming with ivermectin (Rosychuk, 1989).

A few decades ago Peruvian veterinarians and farmers considered sarna the most important disease affecting NWC for centuries (Alvarado et al., 1966). The infestation is still highly prevalent among the herds of the campesinos and is considered to be the main cause of financial losses (Guerrero and Alva, 1986, cited by Windsor et al., 1992). According to an old monograph by Cardozo (Alvarado et al., 1966) there were outbreaks of this disease in 1544, 1545, 1548, and 1826 killing two-thirds of the animal population.

Any camelid regardless of sex and age may be affected by *S. scabiei* (Nayel and Abu-Samra, 1986c). However, some reports state that the infection is more prevalent in younger animals (Rathore and Lodha, 1973). It is often cited that animals in poor condition are more prone to infection (Lodha, 1966; Higgins, 1983, 1984). However, this is controversial as others report that animals in very good condition can also become infected (Nayel and Abu Samra, 1986 c).

There are conflicting opinions regarding the seasonality of the disease. Some authors describe a quiescent phase usually coinciding with winter (Pegram and Higgins, 1992), others finding a higher incidence in the winter (Lodha, 1966; Rathore and Lodha, 1973; Nayel and Abu-Samra, 1986 c). Higgins (1984) on the other hand found a higher prevalence in Saudi Arabia during the hot summer months.

Clinical Signs The first signs of infection are small hyperemic papules often appearing on the medial aspect of the thighs or inguinal region, the head and neck, medial areas of the flanks, udder, and shoulder (Fig. 141). In severe cases any part of the body may be affected. Most authors report that the humps and dorsal aspects of the neck are usually free of any signs of mange (Lodha, 1966; Rathore and Lodha, 1973; Higgins, 1983, 1984). However, Nayel and Abu-Samra (1986 a, b, c) found mangy lesions on the dorsum (including the hump) both in naturally and experimentally infected camels. These lesions are often accompanied by intense pruritus with excoriation and secondary infections. The itching and rubbing causes alopecia.

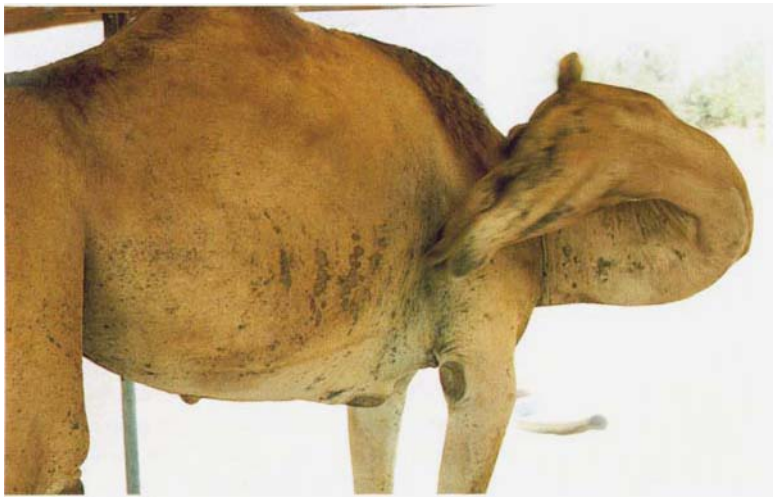


Figure 141 First signs of camel mange

Hairless areas with serous exudation forming scabs follow the first acute signs and itching may increase, seriously disturbing the animals. Grazing and even milk production may show a rapid decrease. The camels desperately rub, bite and scratch trying to alleviate the extreme pruritus. The lesions spread and aggravate excoriation, alopecia, and crusting, resulting in more scabs. The latter may be rubbed away revealing a "red raw surface", erosions and wounds. Localized or generalized acute exudative dermatitis develops.

If untreated, camels with severe acute sarcoptic mange decondition. Within a few weeks, the acute disease may develop to the chronic stage (Fig. 142), which is the stage most often encountered in the field. Hyperkeratosis and proliferation of the dermis leads to the skin becoming thicker, fissured, and corrugated-appearing like a dried cracked field of clay.

Camels with generalized mange may eventually die from extreme wasting caused by the reduction in normal feed intake due to intense irritation and pruritus (Abu-Samra and Imbabi, 1981).

The incubation period is believed to be around 2 to 3 weeks (Lodha, 1966; Higgins, 1983). Experimental transmission studies in dogs and pigs showed that the incubation period is dependent on the number and condition of the mites transmitted (Bornstein, 1991; Bornstein and Zakrisson, 1993). The incubation period is greatly reduced if the animal is reinfested after clinical recovery from a previous infection.

Immunity Absolute protective immunity following recovery after treatment is not known. However, in experimentally infected dogs and rabbits Arlian et al. (1994, 1996) demonstrated partial immunity or protection against challenge infections.

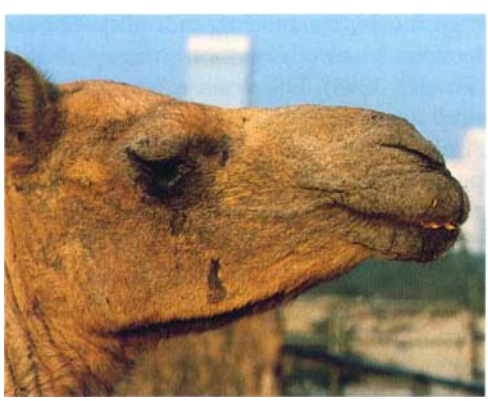


Figure 142 Severe chronic camel mange

It was shown by Alvarado et al. (1966) that some animals in alpaca herds were more susceptible. Lesions in three naturally infected alpacas in the above-mentioned study were left unchecked. All three died of sarcoptic mange.

Antibodies to *S. scabiei* in naturally and experimentally infected dogs, red foxes, pigs and guinea pigs have been demonstrated 2 to 5 weeks following infection (Bornstein and Zakrisson, 1993; Bornstein, 1995; Bornstein et al., 1995). In naturally infected dromedaries Bornstein et al. (1997) also demonstrated antibodies to *S. scabiei* by an ELISA.

Diagnosis Any pruritic skin disease may be caused by *S. scabiei*. The earliest lesions are often unnoticed. Apart from the characteristic clinical signs of pruritus, alopecia and hyperkeratosis, demonstration of the mite is possible by taking deep skin scrapings from several affected areas. Higgins (1984) stressed the importance of taking proper and adequate numbers of skin scrapings from the individual mangy animal. Care should be taken to scrape at least 1 cm^2 area of the mangy skin. In chronic lesions where the skin is thickened and corrugated, scrapings should be made in the "valley" areas (Higgins, 1984). The scrapings should be done by parallel strokes of a sharp scalpel blade at the margins of the mange lesions. This is to be followed by taking deeper scrapings until capillary oozing occurs on the whole scraped surface. All scrapings, keratinous and epidermal material are collected and placed into a broad mouthed centrifuge tube. At least three to four scrapings should be taken per animal.

Finding *S. scabiei* is often difficult. Studies of infected dogs have shown that even when applying multiple skin scrapings, the probability of verifying a diagnosis of sarcoptic mange is less than 50% (Hill and Steinberg, 1993). This similarly applies to camels (Higgins, 1984). Also, according to Higgins (1984) due to the seasonality of the disease, there is a quiescent period during which one may mistakenly think that the animals have been spontaneously cured.

The chances of making a correct diagnosis by skin biopsies are less likely because *S. scabiei* mites are rarely seen in biopsies. Histologically, lesions of acute sarcoptic mange often suggest a *S. scabiei* infection due to hypersensitivity reactions seen in the skin. However, these findings alone are not conclusive because other conditions may cause similar skin lesions (Lodha, 1966; Abu-Samra and Imbabi, 1981). In mange, varying degrees of superficial dermatitis, epidermal spongiosis, hyperplasia and para- and hyperkeratosis may be observed. Eosinophils and mast cells are sometimes intermingled with neutrophils and macrophages. The papillary layer and dermis often show proliferation of connective tissue and infiltration with lymphocytes, macrophages, some eosinophils and giant cells (Abu-Samra, 1999). Epidermal erosions and crusting are often seen due to self-trauma (Fig. 143).

The scrapings should first be examined with a stereomicroscope or a magnifying glass to search for living mites that are stimulated into movement when the environmental temperature is above 18°C. This is done by mildly heating the material to stimulate the mites into migrating from the skin-scabs and debris to the surface, making them easier to see. If no mites are observed, 10% potassium hydroxide (KOH) solution is added to each tube containing the skin scrapings, which are placed into a water bath of 37°C for a few hours until the material has disintegrated. Higgins (1984) recommends adding 20 mL of KOH solution to the skin material and placing the tube into boiling water for 30 minutes. The sample is then centrifuged at 1500 rpm for 5 minutes. The supernatant is discarded and one to two drops of glycerin are added to the sediment, which is then examined under a low power light microscope in search of the mites and their eggs.

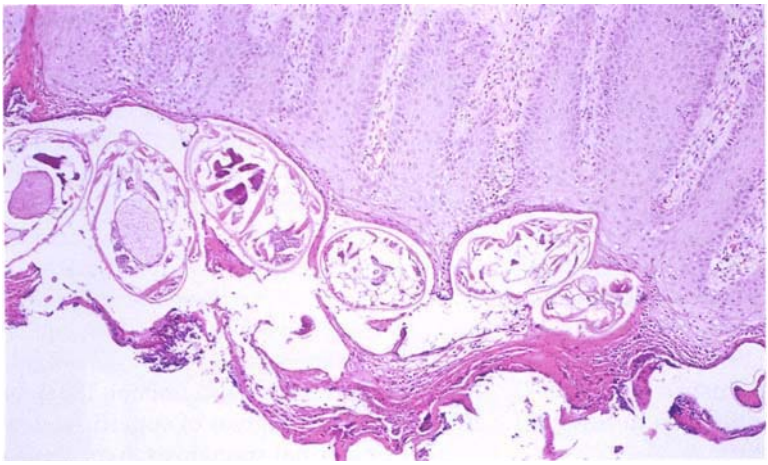


Figure 143 *S. scabiei* mites from skin biopsies (HE stain)

The lesions of mange are most probably caused by hypersensitivity reactions, as has been shown in sarcoptic mange of humans and pigs (Davies and Moon, 1990). Only a few sarcoptic mites burrowing into the skin of the animal can provoke a generalized hypersensitivity reaction leading to the typical acute signs of mange in the host.

An indirect diagnostic ELISA has been developed for dogs and pigs to detect antibodies to *S. scabiei* (Bornstein et al., 1995, 1996; Bornstein and Wallgren, 1997). Preliminary studies also show that a similar ELISA detects antibodies to *S. scabiei* in naturally infected camels (Bornstein et al., 1997).

Differential Diagnosis Several skin diseases may mimic sarcoptic mange. These are:

1. ringworm; note that mixed infection may occur;
2. *Dermatophilus congolensis* (contagious skin necrosis);
3. infestations with other ectoparasites (incl. *Chorioptes* sp.);
4. *Staphylococcus aureus* dermatitis;
5. endocrinal dermatopathy;
6. inhalant or food allergies (Rosychuk, 1989);
7. irritant dermatitis associated with contact with abrasive surfaces when lying down (Rosychuk, 1989);
8. camelpox, particularly the papule and scab formation stages;
9. idiopathic hyperkeratosis (associated with zinc responsive dermatoses recognized in NWC).

Zoonotic Potential Humans occasionally become infected with *S. scabiei* from camel, horse, pig, goat, sheep, chamois, ferret, fox and llama (Leese, 1927; Alvarado et al; 1966; Fain, 1978; Schillinger, 1987; Raisinghani and Kumar, 1991; Basu et al., 1996) and alpacas (Alvarado et al., 1966). Direct transmission between the herders and their animals is most likely during milking, riding, and handling of animals (Basu et al., 1996). Delafond and Bouguinon in 1895 were the first scientists to discover *S. scabiei* in llamas at the Muséum National d'Histoire Naturelle in Paris. Two students were accidentally infected by the affected llamas (Alvarado et al., 1966).

Cross-infections by *S. scabiei* from animals to humans are called pseudo-scabies, distinguished from true human scabies (infections by *S. scabiei* var *hominis*). Humans infected by the itch mite *S. scabiei* from camels exhibit signs similar to those of classical scabies: pronounced intensive itching during the night. Erythema and papule formation are seen mainly in the interdigital

spaces of the hands, the flexor surface of the wrists, the forearms, elbows and axillary folds (of milkers) and between the thighs (in riders). Secondary infections can occur leading to pyoderma. As long as there is continuous contact with mangy animals, the clinical signs will continue in the contact person. Pseudo-scabies is usually self-limiting. The clinical signs will gradually wane and disappear within about two weeks when contact with the infected animal/s is interrupted or the animals are treated, preventing a reinfection.

One of the authors accidentally became infected with *S. scabiei* var. *cameli* when a mangy camel (see Fig. 142) was walked for 6 h to a different location. The first red spots were detected on the right forearm 24 h later. It is believed that the mites had crawled over the lead rope onto the arm. Eight days later severe erythema and papules were observed on both legs (Fig. 144) and both arms with severe itching. There were no lesions on the head and very few papules on the body. Skin scrapings were taken from the leg and *S. scabiei* identified. After treatment with Jacutin® emulsion (lindane 0.3 g) or Prioderm® (malathion 0.5% w/v) for 3 consecutive days and Stromectol® 6 mg (ivermectin) orally, the lesions receded within 72 h.

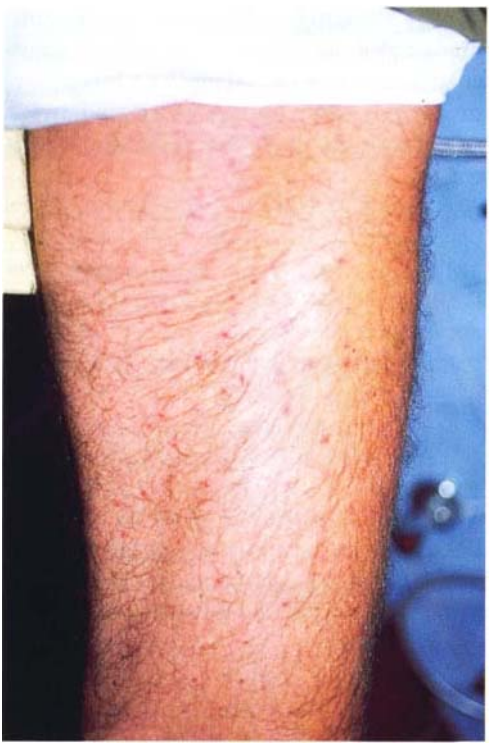


Figure 144 Erythema with papules on a human leg caused by *Sarcoptes scabiei* from a dromedary

Treatment and Control ▦ There are several effective acaricides available today. Some are conventional preparations for skin application: organochlorines, organophosphorous compounds and synthetic pyrethrins. More recent drugs are applied parenterally as well as topically. Also effective against nematode infections are endectocides or macrocyclic lactones (avermectins and milbemycins). In addition, old remedies have been recently reported to be affective against sarcoptic mange in dromedaries, e.g. the ayurvedio preparation "Charmil" gel (Pathak et al., 1995).

When using acaricides as dipwash or sprays, it is essential that the whole animal be covered with the solution. Local topical application of the acaricide only over visible lesions is an incorrect procedure. Additional hand-dressing of chronic, hyperkeratotic areas is often necessary. Before acaricides are applied, such areas should preferably be washed with lukewarm water and soap to soften the scabs and keratinized material. In addition, the application of a 15% solution of salicylic acid, a keratolytic agent, is recommended (Nayel and Abu-Samra, 1986 c). The salicylic acid solution is applied a few times at an interval of 2–3 days followed a day or two later by washing with soap and water. Scales and detritus may be removed with a firm brush. Extra local hand-dressing with the acaricide solution employing a hard brush may also be applied on the parts of the skin

particularly thickened, scabby and corrugated (Higgins, 1983).

The animals should be treated 3 times within an interval of 7 to 10 days, but sometimes 4 or more applications are needed until a cure is reached.

Nayel and Abu-Samra (1986 c) using the acaricide 0.1% hexachlorocyclohexane (Gammatox®) on chronically infected camels found that 3 days following the first wash with Gammatox®, most of the treated camels (75%) became calm with reduced signs of pruritus. Two days after the second wash most of the scales had been shed, the cracks and fissures started to heal, and the edema on the legs had subsided. Six days after the second wash, there was no pruritus and the restless animals behaved normal. Hair began to grow 5 days after the third wash.

The topical application of acaricides is very laborious and difficult to carry out under nomadic conditions, but may more easily be applied in sedentary herds.

The injectible modern endectocides or macrocyclic lactones (like ivermectin, doramectin) have made the treatment of sarcoptic mange much easier. Ivermectin® has been shown to be effective and safe in *Camelidae* and cattle when the same dose and regime is employed (Ibrahim et al., 1981; Boyce et al., 1984; Raisinghani et al., 1989; Kumar and Yadav, 1993; Kuntze and Kuntze, 1991; Oukessou et al., 1996). The recommended dose is 200 mg/kg given subcutaneously and repeated after 15 days. The subcutaneous injection is painful to camelids and some diffuse swelling at the injection site may appear after 24 h. Camelids need to be well-restrained in the couched position. After treatment, clinical improvement is gradual. Pruritus completely ceases after one week to 10 days following the second injection. Four weeks after the second injection all previously alopectic areas are covered with growing hair (Hashim and Wasfi, 1986; Raisinghani et al., 1989). Complete healing of skin lesions was reached on day 145 (Raisinghani et al., 1989). Unfortunately, this treatment protocol is not always successful. Depending on the severity of lesions, a combination of topical and injectible treatments is necessary.

New endectocides have recently reached the market. Some have a longer period of bioavailability in the animal than the ivermectins. There are indications that one injection of these new drugs (e.g. moxidectin, doramectin) may cure sarcoptic mange in pigs and cattle. If the same applies to camelids, these drugs would be of great advantage to nomadic camel owners. One intramuscular injection of doramectin (Dectomax®, Pfizer, NY, USA) was enough to successfully eradicate sarcoptic mange in a herd of mangy pigs (Jacobsson et al., 1998).

In a trial on 15 camels (9 juveniles, 6 adults) showing mild to severe sarcoptic mange, doramectin was applied intramuscularly at a dose of 200 µg/kg. Only two of the severe cases had to be treated twice. All 15 camels were cured (Mumin, pers. comm., 1999).

Abu-Samra (1999) reported even better results with 0.1% solution of phoxim (Sebacil® E.C., Bayer) applied topically three times, one week apart, following thorough application of 15% salicylic acid solution, resulting in complete recovery from chronic mange after 3 weeks.

Another promising form of endectocides is the pour-ons, which are poured onto the skin of the dorsal part of the body. The drug is absorbed through the skin. Both ivermectin and moxidectin are marketed for use as pour-ons for cattle with very good acaricidal as well as nematodicidal properties.

5.2.3 Psoroptic Mange

Psoroptic mange mites spend their entire life on the skin, feeding superficially. They

Figure 145 *Psoroptes* sp. from the skin of a dromedary

reportedly infest camelids, but are less commonly found on camelids than *S. scabiei*.

Morphology *Psoroptes* sp. is larger than *S. scabiei*, about 0.75 mm long and is oval shaped with all four legs projecting beyond the body. Some of the features that distinguish *Psoroptes* from the other common non-burrowing mite *Chorioptes* are the pointed mouthparts, the male's rounded abdominal tubercles, and the three jointed pedicels bearing funnel-shaped suckers on most of the legs (Fig. 145). The female's third pair of legs end in bristles instead of suckers.

It was recently shown that *Psoroptes* sp. isolates of different phenotypes, hosts and geographic origins are conspecific (Zahler et al., 1998) and therefore only one species is mentioned in the text.

Clinical Signs *Psoroptes* sp. (originally named *P. communis* var. *aucheniae*) has been isolated from the ears of alpacas in South America (Chavez and Guerrero, 1965; Fowler, 1998) and found in the ears and necks of llamas (Alverado et al., 1966; Foreyt et al., 1992; Guerrero and La Rosa, 1962). Common lesions consist of dry flakes in the ears. The ears may occasionally be filled with purulent discharge responsible for head shaking and poor coordination. Mites were also found in the perineum, nares, axillae, groin, neck and legs (Alverado et al., 1966).

The piercing and chewing mouthparts of the mite can severely damage the skin. This stimulates a local inflammatory reaction that exudes serous exudate. The exudate coagulates forming a crust or scab. The dermatitis causes intense pruritus and fiber loss. Lesions are generally found around the shoulder and along the back, flanks and base of the tail. Early lesions are small papules about 5 mm in diameter, yellowish, with a moist surface. Within 5 days, a characteristic scab will form. The dermatitis does not become hyperkeratotic to the extent seen in sarcoptic mange.

Gabaj et al. (1992) recorded the only documented case of psoroptic mange in dromedaries and Werner et al. (1989) in Bactrians in Mongolia. In many countries sarcoptic and psoroptic mange are reportable diseases.

Diagnosis Skin scrapings reveal the mites. A mite may be found in the center of the first papules seen. However, mites are usually found at the edges of the lesions. For laboratory procedure, see sarcoptic mange.

5.2.4 Chorioptic Mange

The mange mite *Chorioptes* commonly infests cattle, sheep, goats and equines and, unlike *S. scabiei*, lives on the skin. Unlike *Psoroptes* sp., its mouthparts allow the mite to feed on scales and other skin debris.

Chorioptes sp. closely resembles *Psoroptes* sp., but has rounder mouthparts and tarsal cup-shaped suckers on short unsegmented pedicels. The abdominal tubercles of the male are clearly truncate. Adult mites are about 3.5 to 4.0 mm in length. Only recently has one species been recognized (Essig et al., 1999).

Chorioptes sp. causes pruritic mange mostly seen on the neck, tail, udder and legs in cattle and on horses' legs below the knees and hocks. It is usually regarded as a mild condition. However, lesions may resemble those caused by *Psoroptes* sp. having hyperemic skin covered by scabs 0.5–1.5 mm thick.

Infestation with *Chorioptes* is most probably rare in camels. It has been reported on a Bactrian camel (Higgins, 1984) and in the Netherlands on one llama, three alpacas and two camels, one of which had "foot mange" (Cremers, 1984). An infestation of *Chorioptes* sp. was also responsible for mange in a herd of alpacas from Chile recently imported into France (Petrowski, 1998).

Treatment All the acaricides used topically are effective against the *Psoroptes* and *Chorioptes*. It has been shown that pour-ons may be used. Bayticol, Pour-on 1% (flumethrin), 1 mL/10 kg applied on Bactrian camels with psoroptic mange proved to be effective. Five days after the single topical treatment was applied, no more living mites were found and the healing process of the skin lesions began a few days later.

5.2.5 Demodectic Mange

The preferred site of the burrowing mite of the genus *Demodex* is at the hair follicles and sebaceous glands of the skin. It is a cigar-shaped, elongated 0.2 mm long mite. The thorax has four pairs of short stumpy legs. The LC is only partially known. It includes eggs (70–90 µm × 19–25 µm), one larval stage and two nymphal stages, and lasts 3 weeks. The mite is most probably transmitted from the dam to the offspring during nursing. *Demodex* sp. (Fig. 146) is found in all domestic mammals and hu-

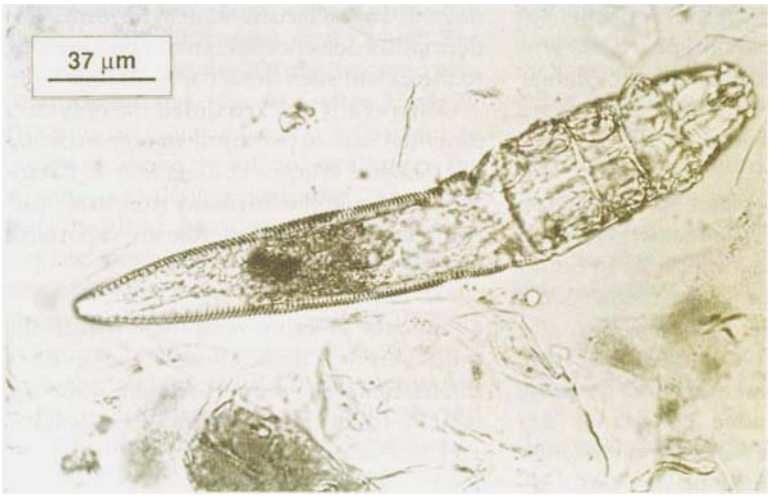


Figure 146
Demodex mite from the skin of a dromedary (courtesy of Professors Sloss, Kemp and Zajac, Veterinary Clinical Parasitology, 6th ed., 1994, Iowa State University Press, USA)

mans worldwide. Most of the species are named after their hosts, i.e. *D. canis, D. bovis* etc. These follicular mites mainly live as commensals in the skin. In some animals, these mites may cause mange, of particular severity in dogs. In bovines, the most significant sequela to infestation is the damage to the hide, causing economic loss.

Demodex sp. has been reported on dromedaries in Iran where the eyelids of 15% of the camels were infested (Rak and Rahgozar, 1975). There was no evidence of any secondary bacterial infection in the investigated camels, nor were there any significant histological changes other than distention of the hair follicles. *Demodex* sp. was isolated from camels exhibiting mange on a ranch in Kenya (Bornstein, pers. commun.). *Demodex* sp. commonly occurs in llamas and alpacas in Bolivia (Sqire, 1972). The mite most probably also infests other NWC in other countries.

5.2.6 Infestations with Metastigmata (Ticks)

Ticks are important vectors of protozoal, bacterial, viral and rickettsial diseases in many animal species. However, their vector role appears to be much less important in camelids than in other livestock. Being blood-feeders, ticks may cause debility and anemia in camels and other animals. There is a significant loss of blood: about 1 to 3 mL for every tick completing its blood meal. Thousands of ticks may be found on the same infested animal. A *Hyalomma* sp. was said to have caused the death of a camel (Steward, 1950) that had been infested by 100 nymphs and adults per 2.5 cm^2 skin surface. The high calf mortality rate of 20% encountered in some camel herds in Kenya has been attributed on "tick-anemia" (Rutagwenda, 1984).

Lesions, although small, are made by the tick's mouthparts and may attract flies; some causing myiasis and are also gateways to secondary bacterial infections. The attachment sites of ticks commonly show dried blood with scabs (inflammatory reaction) and frequently these sites, following infestations by *Amblyomma lepidum*, develop into large ulcerations (sores) (Hoogstraal, 1956). Infested camels are often irritated and exhibit pruritus. Allegedly, some ticks also cause paralysis in camels as well as in other livestock: 43 species of 10 different genera have been incriminated in causing toxic reactions according to Fowler (1998), and 60 species according to Hoogstraal (1985) and Gothe and Neitz (1991). In general, tick infestations may cause local and generalized disease causing damaged hides and mortalities.

Ticks belong to two families, the Ixodidae, the hard ticks and the Argasidae, the soft ticks. The former have a rigid chitinous scutum that covers the entire dorsal surface of the adult male. This chitinous scutum covers only a small area in the adult female, the larvae and nymph allowing the abdomen to swell after feeding. The soft ticks lack a scutum.

Ixodids spend a relatively short period on the host. The number of hosts to which they attach during their parasitic life cycle varies from one to three. According to the number of hosts they require to fulfill their lifecycle, ticks are classified into the following three groups:

The one-host ticks: All the three instars engorge (take their blood meals) on the same host. The two ecdyses also take place on the same animal: e.g. *Boophilus* spp.

The two-host ticks: The larva engorges and moults on the host. The nymph after feeding drops onto the ground where it moults and the imago then seeks a new host: e.g. some *Rhipicephalus* spp.

The three-host ticks: These need a different host for every instar, which drops off the host after engorging and then moults on the ground: e.g. some *Ixodes* (e.g. *I. ricinus*) and *Rhipicephalus* (e.g. *R. appendiculatus*) spp.

As the name implies, the soft tick lacks a scutum and its integument is leather-like. There are three genera of veterinary significance in the family Argasidae: the bird ticks, the ear ticks and the sand tampans. The latter genera *Ornithodoros* live in sandy soils, in cracks and crevices seeking shade. Masses of these ticks may be seen on the sand in places where large numbers of animals congregate, such as holding grounds and marketplaces.

5.2.6.1 Ticks Found on Camelids

A large number of tick species may infest OWC. However, there are only a few tick species (adults) that are camel host-specific. It is thought that these species only survive where camels are present, although they can infest other mammals (Higgins, 1984). For comprehensive information regarding tick distribution, prevalence, biology and epidemiological significance in camels in the Middle East and North African regions, the excellent study on ticks of Saudi Arabia by Hoogstraal et al. (1981) is recommended. Comprehensive checklists of ticks found on camels in Ethiopia, Yemen Arab Republic and Kenya have been published by Pegram et al. (1981, 1982), Dolan et al. (1983) and Pegram and Higgins (1992). Van Straten and Jongejan (1993) recently reported on camel ticks in Sinai, Egypt, and Singh and Chhabra (1999) on ticks in Haryana in India.

The most important tick genus infesting camels is *Hyalomma* with the species *H. asiaticum, H. dromedarii, H. franchini* and *H. scupense* (Pegram and Higgins, 1992). Singh and Chhabra (1999) found *H. dromedarii* to be the most common followed by *H. anatolicum*. Other genera of hard ticks found on camels are *Amblyomma, Rhipicephalus* and *Dermacentor*. The cattle tick *Boophilus microplus* has been reported attacking dromedaries in Australia (Kennedy and Green, 1993) and in India (Singh and Chahbra, 1999). Three camel soft tick species are recorded: the most important is *Ornithodoros savignyi*, followed by *O. lahorensis* and *O. tholozani* (Fig. 147 a–e).

Some species of ticks may adapt to different climates by adjusting their LCs accordingly. *H. dromedarii* is a desert-adapted two-host tick widely found in arid lands wherever camels are reared. This species sometimes uses three hosts for better survival (Hoogstraal et al., 1981). During January and July in the Yemen Arab Republic's hot arid lowlands the tick seems to produce two generations per year, but only one generation per year in the cooler highlands, with an adult peak in June and July (McCartan et al., 1987).

Another example of an extremely adaptive tick is *H. anatolicum anatolicum*, which is classified as a two-host tick infesting a wide range of domestic animals, particularly camels and cattle. On cattle, this tick uses three hosts for completing its LC (Hoogstraal et al., 1981). The tick is found to be active throughout the year, even in very hot areas, and the numbers may be very high.

The subspecies *H. excavatum* is also often found in large numbers wherever domestic stock is plentiful in the Middle East and northern Africa, and is found on camels in large numbers. It is reported that it may employ either a two-host or a three-host LC (Higgins, 1984). The immature stages are found in rodent burrows.

There are few reports that list particular species of hard ticks found on NWC. *Dermacentor* sp. and *Ixodes holocyclus* caused tick toxicosis in a llama (Vogel, 1995; Jonsson and Rozmanec, 1997). Hard ticks are reported to be a problem, particularly on llamas in the western USA, during treks (Fowler, 1998). *Amblyomma parvitarsum* Neumann was found parasitizing vicuñas in Peru (Dale and Venero, 1977).

Ornithodoros savignyi, the sand tampan, is a common soft tick on camels in hot and arid deserts. It can also attack humans and other livestock, particularly goats. Large

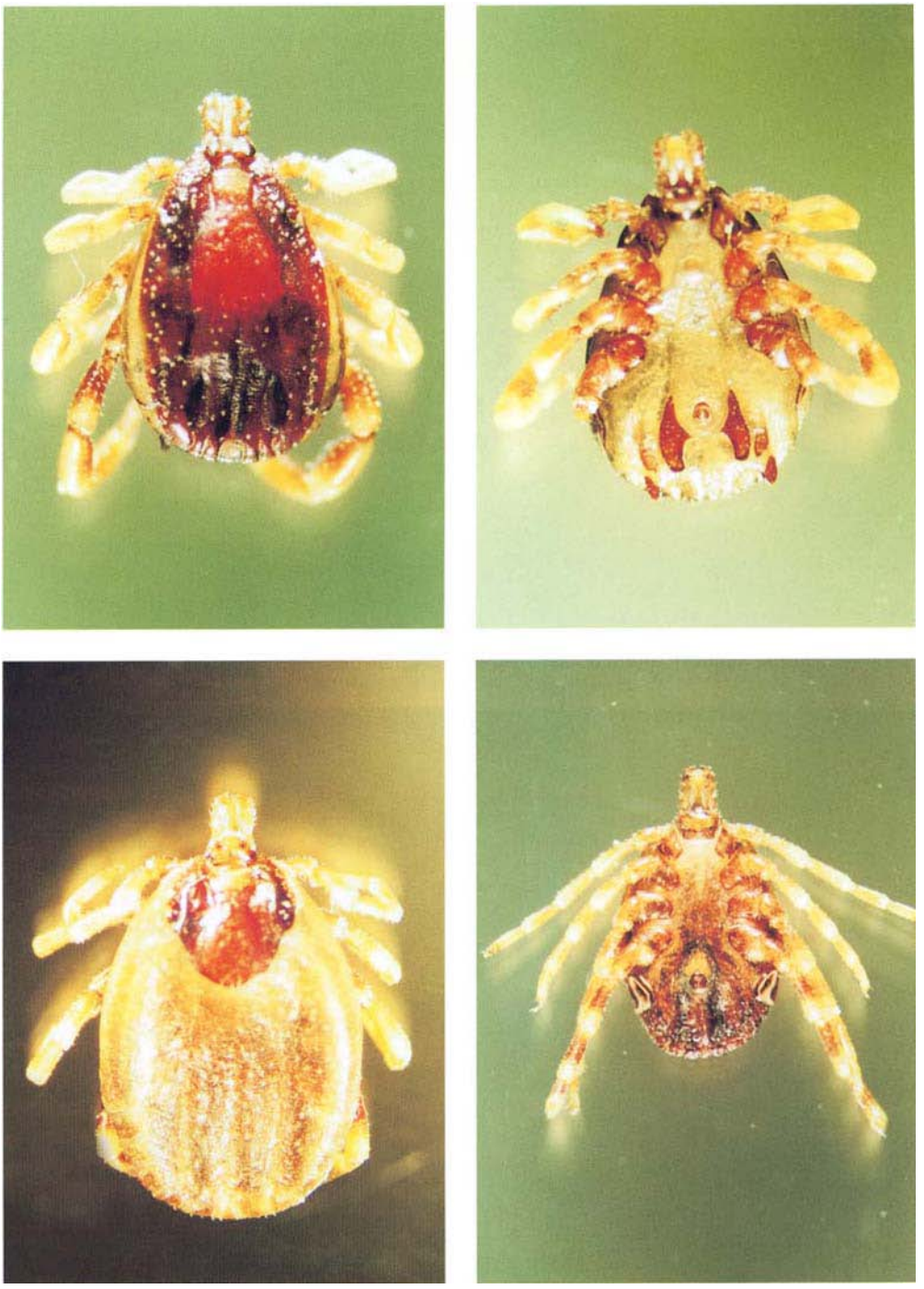
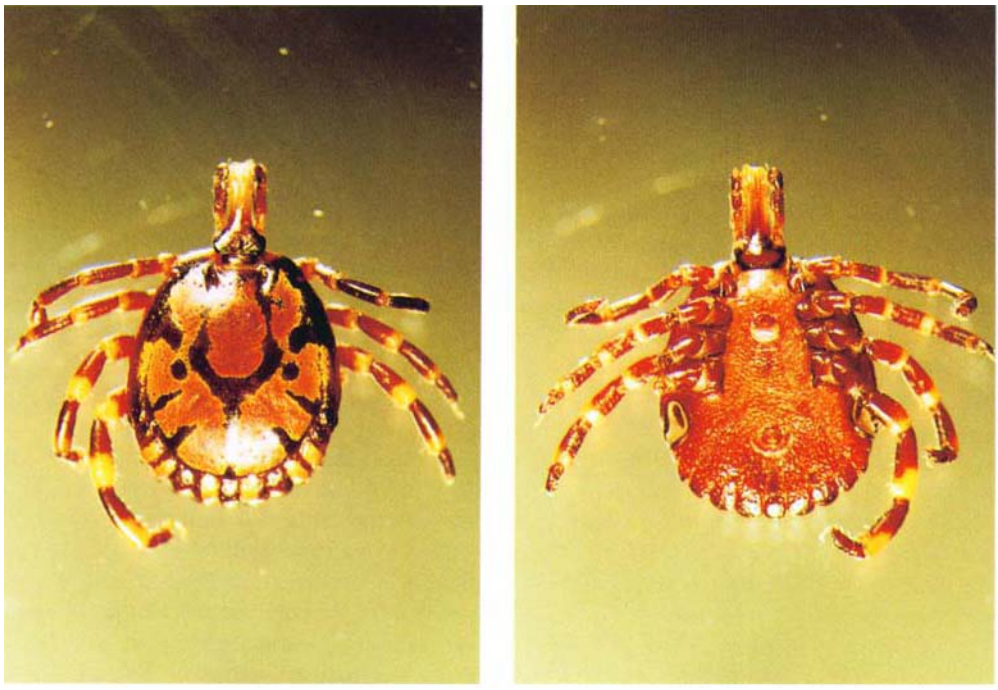
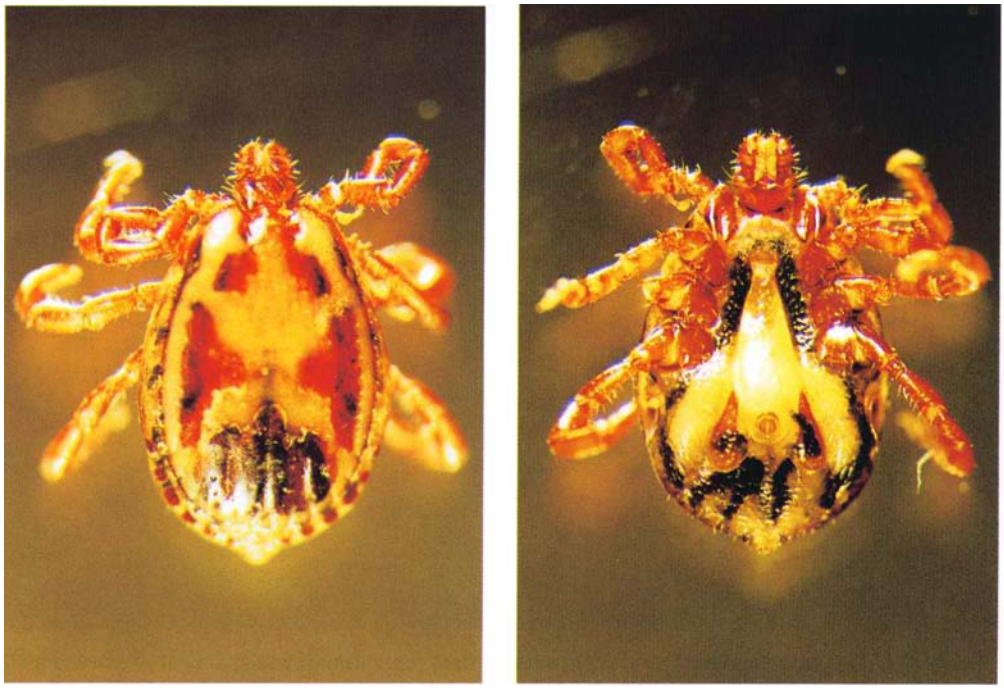


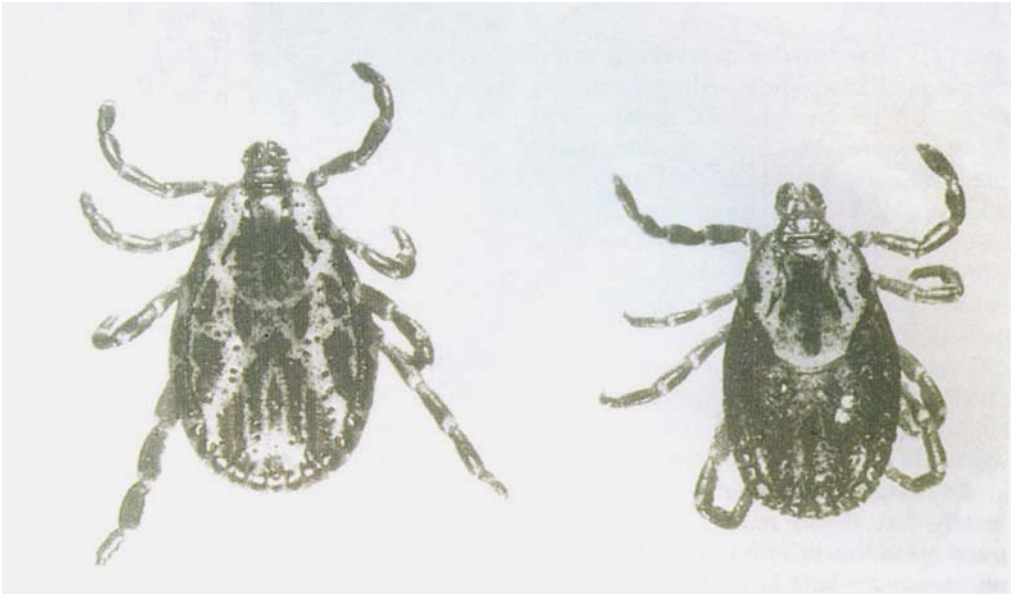
Figure 147a–e Important hard and soft camelid ticks (courtesy of Mr. K. Valsan, Pest Control, Dubai Municipality, UAE) Hard ticks: **(a)** *Hyalomma dromedarii* (male and female from dorsal and ventral)



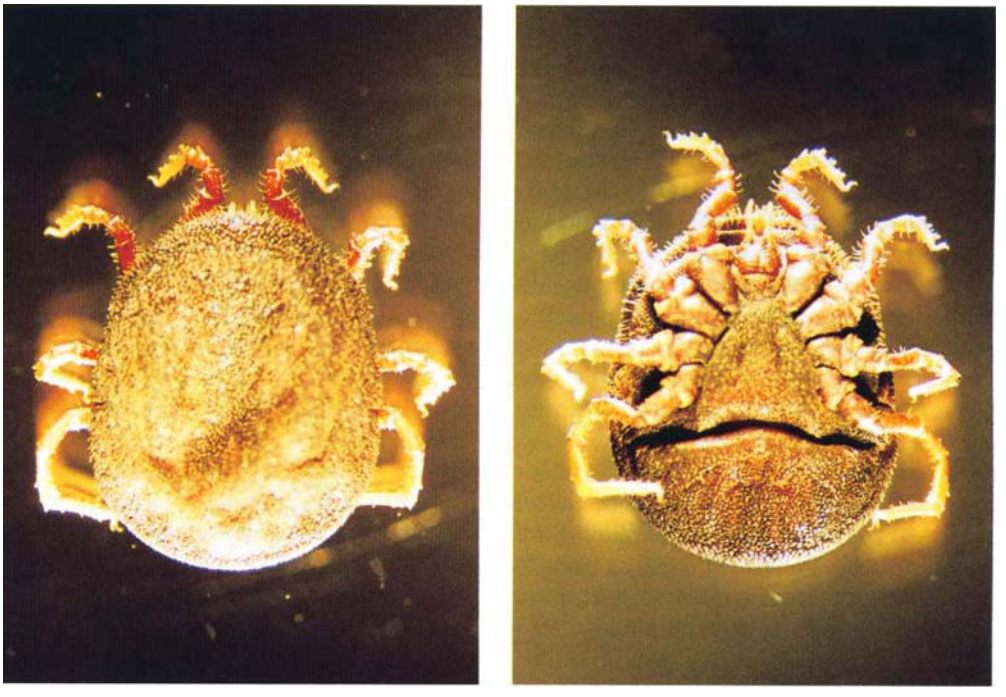
(b) *Amblyomma lepidum* (male dorsal and ventral)



(c) *Rhipicephalus pulchellus* (male dorsal and ventral)



(d) *Dermacentor variabilis* (male and female dorsal)



Soft tick: **(e)** *Ornithodoros savignyi* (dorsal and ventral)

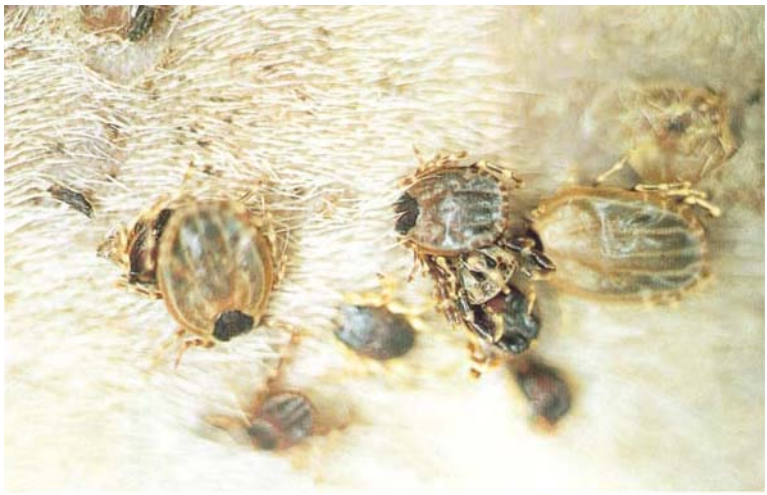


Figure 148 *Hyalomma dromedarii* between the front legs of a young dromedary

numbers of this tick may be seen crawling on the sand where large numbers of animals are kept. The bites of *O. savignyi* may be painful, but it is not considered to be a significant vector of disease to either humans or livestock.

Among the soft ticks (Argasidae), one species mentioned causing disease in llamas is the spinose ear tick *(Otobius megnini)*, which may infest other hosts, including humans. The adults do not feed and may hide for several months in crevices of buildings and feeding troughs where the females lay their eggs. These hatch to larvae, which may attack a host within 10 days seeking out the ears. It is only the larvae and the nymphs which are parasitic, causing excess production of waxy substances and severe inflammation in the outer ear canals. When infestation is heavy, anemia and deconditioning will develop. The parasitic stages may remain on the same host for several months. The larvae may survive up to 4 months without finding a suitable host. The nymph moults twice within the infested ear and drops to the ground, whereafter it moults to the adult stage.

The soft ticks may cause problems in llamas and alpacas in certain localities in the western USA. A preferred tick habitat is around buildings, sheds, wooden fences and trees with rough bark. Animals that are kept on open pastures and ranges are less likely to encounter the spinose ear tick.

Pathology and Pathogenesis Camels may be infested with ticks throughout the year. However, numbers may fluctuate with the climate. On longhaired animals, ticks may go unnoticed, especially during the cold months of the year. The ticks on camels are mostly found in the perineal, inguinal, and axillary regions, around the eyes, lips, in/on the ears, the nostrils and in the nose, between the toes and on the mammary glands (Fig. 148).

Ticks are easily seen on their predilection places, often relatively deeply imbedded in the skin. Numbers may be high, thereby interfering with the well-being of the host, causing irritation and direct injury to the skin. Wounds may become secondarily infected, leading to pyoderma. *Streptococcus agalactiae* was isolated from wounds caused by *Hyalomma* sp. in a herd of dromedaries from Kenya (Younan and Bornstein, pers. com., 2000). The skin may get rough and thickened with scar tissue.

Sores are often seen at dermato-mucosal borders on the nose, lips and vulva. Tick bites may predispose to myiasis. It is not known whether ticks play any part in introducing secondary bacterial infections to the mammary glands. It is suggested that heavy tick loads contribute to reduced growth rates and calf mortality (Dolan et al., 1983). Sizable numbers of ticks may lead to anemia.

Vectors of Disease Pathogens The importance of camel ticks as vectors of disease pathogens for livestock has been described. There is evidence that *Amblyomma lepidum* or *A. gemma* may transmit *Cowdria ruminantium* (Heartwater) to cattle (Karrar et al., 1963) and that *H. dromedarii* is the vector of *Theileria camelensis* (Hoogstraal et al., 1981).

Camel ticks are also vectors of viruses infecting humans: *Hyalomma anatolicum* is an important vector of Crimean-Congo hemorrhagic fever (CCHF) virus, which was reported in the former USSR, Pakistan and Nigeria (Hoogstraal, 1979). This virus has also been isolated from the ticks commonly found on camels, *H. dromedarii* and *H. impeltatum* (see also under 2.2.10 Unusual Arboviruses) (Table 57).

Table 57 Zoonosis associated with camel-infesting ticks (Pegram and Higgins, 1992)

Vector	Agent
H. anatolicum	Thogoto virus
H. excavatum	Rickettsia prowazeki
H. dromedarii	Dhori virus
	Khadam virus
	CCHF virus
	Q-fever *(Coxiella burnetii)*
H. impeltatum	Wanowrie virus
	CCHF virus
H. marginatum	CCHF virus
H. scupense	? virus (Paralysis)
H. truncatum	CCHF virus
	? virus (Paralysis)
R. pulchellus	Rickettsia prowazeki
R. praetextatus	Thogoto virus

5.2.6.2 Tick Paralysis

Many species of ticks have been incriminated in causing tick paralysis, as distinct from tick toxicosis. The latter occurs in susceptible ruminants, pigs and avians through toxins from adult ticks. Toxicosis is characterized by sweating, generalized hyperemia and a severe moist eczema primarily caused by *Hyalomma* spp. (Urquhart et al., 1996).

More than 60 of 869 known tick species are capable of causing paralysis (Hoogstraal, 1985; Gothe and Neitz, 1991). Tick paralysis occurs in OWC as well as NWC. In OWC, it is the larva of *H. dromedarii* that is thought to be the main cause of paralysis. It caused high mortality (above 24%) in calves in the Sudan (Agab and Abbas, 1998). Epidemics of suspected tick paralysis incriminating both *Hyalomma* spp. and *Rhicephalus* spp. have also been reported in Sudan (Musa and Osman, 1990).

Clinical Signs In NWC, individual females of several species of hard ticks, under certain unknown circumstances, produce neurotoxins which are injected by the tick when it ingests a blood meal. A bite from a single tick, e.g. *Dermacentor* spp., may kill an animal. Studies of other animal species have shown that there is variable host susceptibility and most probably also a seasonal or annual variability. *Dermacentor* spp. were identified as causing tick paralysis in two young llamas in the USA (Barrington and Parish, 1995) and also in seven llamas and one alpaca in the USA (Cebra et al., 1996). Seven of the diseased animals showed generalized muscle flaccidity. They recovered following treatment including removal of the ticks. The female llama recovered after 3 days after being clipped to remove all the ticks. A llama in Australia exhibiting typical signs of tick paralysis thought to be caused by *Ixodes holocyclus* did not survive in spite of intensive treatment and removal of the ticks (Jonsson and Rozmanec, 1997).

According to Fowler (1998), the pathogenesis and clinical manifestation of the disease in NWC is similar to that in other animal species. Most cases of tick paralysis in NWC have been reported from non-indigenous regions (Barrington and Parish, 1995). The tick paralysis in North America is most likely due to a salivary neurotoxin, which is thought to act on the end plates of the motor neurons, preventing acetylcholine release into the synapses of the neuromuscular junctions (Gothe et al., 1979).

Signs are usually not apparent until 5 to 7 days after the tick has begun to feed. According to Musa and Osman (1990), the clinical signs may appear earlier and the first deaths may already occur 3 days following the tick invasion. The first signs of paresis and paralysis are seen in the hindquarters, and they progressively increase in severity as they move toward the cranium. The ability to rise is lost in 12 to 36 hours. Loss of all motor functions occurs, preceded by ataxia. Stretch reflexes are also impaired and pain perception remains. The signs may develop rapidly within a few hours or may take 24 to 48 hours until the victim dies of respiratory arrest from involvement of the respiratory centers in the brain.

Diagnosis Diagnosis is based on clinical signs and the finding of ticks known to cause paralysis. Analysis of cerebrospinal fluid may help in distinguishing tick paralysis from other causes of paralytic diseases. There is a strong indication that the diagnosis of tick paralysis is correct if the patient recovers rapidly (within a few days) following removal of the ticks.

5.2.6.3 Tick Control

Chemical Control Routine prophylactic tick control is not practiced in camelids as in cattle. However, control of significant numbers of ticks attacking camels is recommended. This can be done by applying appropriate acaricides to the predilection sites (chlorinated hydrocarbons, organophosphates, carbamates, synthetic pyrethroids or the macrocyclic lactones), as used for cattle. It should be noted whether the ticks in the area have developed resistance to a particular acaricide. A 1% flumethrin (Bayticol® Bayer) pour-on formulation was successfully used (El-Azazy, 1996) in controlling *H. dromedarii* infestations on camels. The drug (1 to 2 mL/10 kg) was poured from the shoulder along the middle of the back over the hump to the tail.

Subcutaneous injections of ivermectin (10 mg/50 kg) are effective in controlling both larvae and nymphs of the spinose ear tick (Fowler, 1998). The ear canals may be cleaned manually and solutions of insecticides or acaricides instilled.

Reinfestation can occur because of the difficulty of eradicating the ticks from the environment. Regular inspection of the outer ear canals followed by treatment is recommended to avoid a build-up of infestation. The recently available endectocides possess an extended period of bioavailability but their pharmacokinetics are unknown in camelids.

There is no effective treatment that can neutralize the tick paralysis toxin. However, *Ixodes holocyclus* canine hyperimmune serum is used in affected small animals and calves (cattle) at a dose rate of 0.5 mL/kg (Jonsson and Rozmanec, 1997) curing about 75% of cases. This hyperimmune serum was used without success in tick paralysis of a llama caused by *I. holocyclus*.

Vaccination The hosts of hematophagous arthropods may stimulate immune defenses that react with tissues and saliva of the parasite. This can disrupt blood meal acquisition, impair physiological responses and/or kill the arthropod (Wikel, 1982, 1996).

Since Trager (1939) showed that guinea pigs immunized with whole larval extract of *Dermacentor variabilis* were resistant to the challenge of the larvae, numerous in-

vestigators have been trying to develop anti-tick vaccines. Today there are two types of tick vaccines available for cattle. One crude vaccine is made from extracts of the partly engorged adult female *B. microplus*. Antibodies produced in the host destroy the cells lining the tick's gut and blood escapes into the hemocele. A certain percentage of the ticks die and the fertility of those remaining may be reduced by up to 70% (Willadsen et al., 1989). The fertility of males is also affected. Recombinant vaccines have also been developed and are commercially available. However, these vaccines have a limited application and have not yet been developed for *Camelidae*.

5.2.7 Insects Found on Camelids

5.2.7.1 Classification of Insects

Among the class Insectea there are several orders of particular veterinary interest: the Anoplurida (sucking lice), the Mallophagida (biting lice), the Siphonapterida (fleas), and the Dipterida (flies).

Phylum Arthropoda
Class Insectea
Order Anoplurida (Sucking lice)
Microthoracius cameli (OWC)
M. mazzai (NWC)
M. minor (NWC)
M. praelongiceps (NWC)

Order Mallophagida (Biting lice)
Damalinia breviceps (NWC)

Order Siphonapterida (Fleas)
Vermipsylla spp. (OWC, NWC)

Order Dipterida (Flies)
Suborder Brachycerina
Family Sarcophagidae (Flesh flies)
Wohlfahrtia magnifica (OWC)
Wohlfahrtia nuba (OWC)
Sarcophaga dux (OWC)

Family Calliphoridae (Blowflies)
Lucilia cuprina (OWC)
Chrysomya bezziana (OWC)
Calliphora spp. (NWC)
Cochliomyia hominivorax (OWC, NWC)
Phormia spp. (NWC)

Family Oestridae (Bot flies)
Cephalopina titillator (OWC)
Oestrus ovis (OWC, NWC)
Cephenomyia spp. (NWC)

Family Muscidae (Flies)
Musca domestica (OWC, NWC)
M. autumnalis (OWC, NWC)
Stomoxys calcitrans (OWC, NWC)
Hydrotea spp.
Haematobia spp.

Family Glossinidae (Tsetse flies)
Glossina spp.

Family Tabanidae (Horse flies)
Tabanus spp. (OWC)
Haematopota spp. (OWC)
Chrysops spp. (OWC)

Suborder Nematocera
Family Ceratopogonidae (Midges)
Culicoides spp.

Phylum Pentastomida
Linguatula serrata

5.2.7.2 Infestation with Lice

There are two orders: the Anoplurida, the sucking lice, and Mallophagida, the biting lice. The latter have not yet been reported on OWC. Llamas may suffer from both biting and sucking lice and both may be found on the same individual. Biting lice have a blunt broad head that is distinctly different from the elongated mouthparts of the sucking lice (Fig. 149).

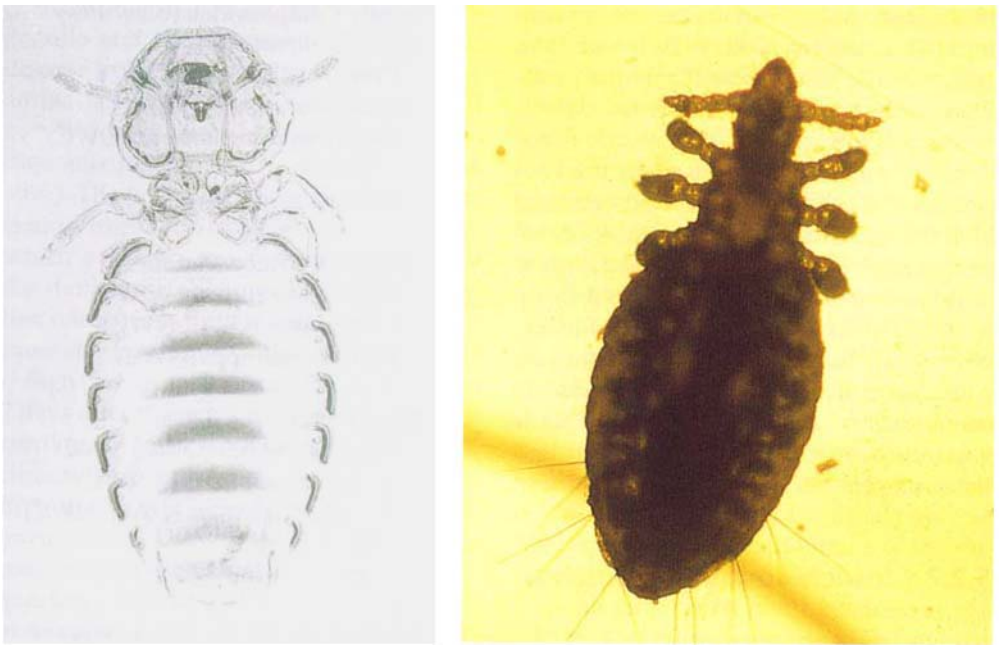


Figure 149 Biting louse (left) (courtesy of Professors Sloss, Kemp and Zajac, Veterinary Clinical Parasitology, 6th ed., 1994, Iowa State University Press, USA) and sucking louse (right)

Anoplurida (Sucking Lice)

The only blood-sucking lice reported to occur both on Bactrians and dromedaries in Asia as well as in Africa is *Microthoracius cameli* (Soulsby, 1982). Lice infestation is characterized by licking, scratching and rubbing. Anemia may follow heavy infestations, particularly in young animals. The coat becomes rough and secondary bacterial infections may follow the pruritus. Infestation may result in damaged hides. Camel lice are generally only a problem in temperate regions where the animals have long winter hair.

The llama and the alpaca may be infested with *M. praelongiceps*, *M. mazzai* and *M. minor*. The same clinical signs of camel lice infection are also seen on NWC. Sucking lice are usually found around the head, neck and withers. They may be quite difficult to see with the naked eye, being smaller than the biting lice (two-thirds their size) and often hidden in the fiber, taking a blood meal.

Mallophagida (Biting Lice)

The biting louse *Damalinia breviceps* is a common llama parasite (Fowler, 1986). Llama wool infested with biting lice lacks luster and the coat is ragged. Heavy infestation may result in matted wool and alopecia. The host experiences pruritus, resulting in self-trauma. The predilection sites are at the base of the tail, the back along the vertebral column and the sides of the neck and body.

The transmission of the parasite is either by direct or close contact between the hosts or indirectly by grooming equipment, blankets, saddles, scratching posts, or dust bath areas.

Treatment Table 58 lists the drugs used against lice.

Table 58 Treatment against lice in camelids

Trade Name	Generic Name	Formulation	Application
Asuntol®	Coumaphos	50% wettable powder	Apply at a concentration of 0.05% (500 ppm) directly onto skin, wet hair coat thoroughly
	Methoxychlor	Dusting powder	50% directly onto skin
Severin®	Severin	Dusting powder	As above
Ivomec®*	Ivermectin	Inj. 0.2 mg/kg	s.c.
Ivomec®*	Ivermectin	0.5 mg/kg	Pour on

* Internationally registration approval for Ivomec injectable exists for *Sarcoptes scabiei* var. *cameli* only. Reports are available for sucking lice and endoparasites. Withdrawal period is 28 days for meat. Camels producing milk for human consumption should not be treated. Ivomex Pour-on is used in camelids (as field reports say successfully) without market authorization.

5.2.7.3 Infestation with Siphonapterida (Fleas)

Vermipsyllidae

Fleas *(Vermipsylla alacurt, V. ioffi)*, like lice, infest camelids in cooler countries (Zedev, 1976). They have also been reported affecting Bactrian camels in zoos (Pegram and Higgins, 1992) and llamas (Fowler, 1998). In addition, several other species may attack camelids, as is the case of *Ctenocephalides felis felis* (Yeruham et al., 1997). Although fleas are important vectors of infectious pathogens, such as Typhus-like rickettsia, *Yersinia pestis*, and as an intermediate host for filarids and cestodes, no instances of pathogen transmission by these insects have been reported in camelids.

Treatment Treatment is the same as for lice.

5.2.7.4 Infestation with Flies

Some *Dipterida* as adults are external parasites, while some parasitize the tissues of the hosts as larvae, causing myiasis. Many members of this order are also important vectors of pathogens. The order is divided into two suborders: Brachycerina, Nematocerina (Table 59).

Myiasis

Myiasis is defined as the invasion of living mammalian tissue by larvae of dipterous flies (Urquhart et al., 1996) that, at least during a certain period of their life, feed on dead or living tissue. There are larvae of six fly species known to cause myiasis in camels. Five of these belong to the blowflies *(Calliphoridae)* and one to the *Oestridae* (Zumpt, 1965; Higgins, 1986). Obligate parasites such as *Chrysomya bezziana* and facultative parasites such as *Lucilia cuprina* may also cause myiasis. *Musca domestica* can also cause myiasis. Myiasis may be cutaneous (e.g. caused by *Lucilia* spp.), nasal (e.g. caused by *Oestrus*), or somatic (e.g. caused by *Hypoderma* spp.).

Sarcophagidae Producing Myiasis (Flesh Flies)
Wohlfahrtia magnifica, Wohlfahrtia nuba, Sarcophaga dux

As an obligate parasite, *Wohlfahrtia magnifica* is the most important fly causing myiasis in camels. It occurs in the Mediterranean basin (James, 1947; Hadani et al., 1971), southern Russia, Turkey, Iran, the Far East (James, 1947), Spain (Ruiz-Martinez et al., 1987), and Mongolia (Yasuda, 1940; Valentin et al., 1997) (Fig. 150). The female fly deposits larvae near any skin

Table 59 The Dipterida associated with myiasis or "nuisance" in camels (after Pegram and Higgins, 1992)

Family (common names)	Species	Vector Capacity
Brachycerina		
Calliphoridae – (Blow flies)	*Cochliomyia hominivorax* – (New World screwworm fly) *Chrysomya bezziana* – (Old World screwworm fly) *Lucilia cuprina* – (Green bottle fly)	
Sarcophagidae – (Flesh flies)	*Sarcophaga dux* *Wohlfahrtia magnifica* – (Old World flesh fly) *Wohlfahrtia nuba*	
Oestridae – (Bot flies)	*Cephalopina titillator* – (Camel nasal bot fly) *Oestrus ovis* – (Sheep nasal bot fly) *Cephenemyia* sp.	
Tabanidae – (Horse flies)	*Tabanus* sp. *Chrysops* sp. *Haematopota coronata*	*T. evansi*
Muscidae – (Muscid flies)	*Stomoxys calcitrans* – (Stable fly) *Haematobia irritans* – (Horn fly) *Haematobia exigua* – (Buffalo fly) *Musca domestica* – (House fly) *Musca autumnalis* – (Face fly) *Hydrotaea irritans* – (Sheep head fly)	*T. evansi* Bacteria and viruses Salmonella *Thelazia leesei*
Hippoboscidae – (Louse flies)	*Hippobosca camelina* – (Camel louse fly) *Hippobosca maculata*	*T. evansi*
Glossinidae – (Tsetse flies)	*Glossina* spp.	*Trypanosoma* spp.
Nematocerina		
Culicidae – (Mosquitoes)	*Aedes* spp.	*Dipetalonema evansi*
Ceratapogindae – (Midges)	*Culicoides* spp.	*Onchocerca fasciata*

wound, mucous membrane or tick bite as well as in the nasal and aural cavities. The fly seems to prefer camels, although other domestic animals and humans are infested (Zumpt, 1965). There have been several reports of the larvae of *W. magnifica* causing severe vaginal myiasis in the Bactrian camels in Mongolia (Schumann et al., 1976; Ribbeck and Beulig, 1977; Ribbeck et al., 1979; Valentin et al., 1997). A case of preputial myiasis was also reported in a camel. Mucous membranes of the female genital organs, the eyes and the nose may be attacked without pre-existing wounds (Zumpt, 1965).

The prevalence of Wohlfahrtian myiasis in thirteen Mongolian Bactrian camel herds ranged between 6.5 to 19% (Schuman et al., 1976). Valentin et al. (1997) found an infestation rate of 8 to 15% among female camels in Mongolia, and Hadani et al. (1989) reported a prevalence of 10% in dromedary camels in the Sinai.

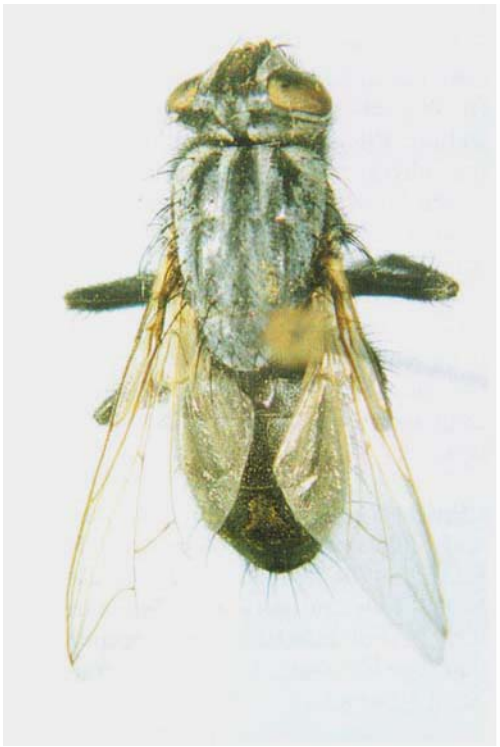


Figure 150 *Wohlfahrtia* spp. fly (flesh fly) from vaginal myiasis of a Bactrian camel from Mongolia (courtesy of Prof. Dr. R. Ribbeck, Germany)

Figure 151 *Lucilia cuprina*

Clinical Signs Ulcerous, blood-oozing lesions, sometimes the size of a tennis ball, may be seen on the vagina and vulval labia. Numerous larvae may be seen in the inflamed wounds, deeply embedded in the sensitive dermis. Valentin et al. (1997) counted an average of 105 larvae per affected Bactrian in Mongolia. The vulval region is usually swollen and the hind legs encrusted with blood. Affected animals often show nervous behavior, tripping with their hind legs and bending their backs (Valentin et al., 1997). These camels often are in bad condition. Some may even be emaciated, with a history of chronically recurring genital myiasis (Valentin et al., 1997).

All three instars may be found concurrently in the wounds suggesting that superinfestations, acute as well as chronic stages, occur simultaneously with various stages of cicatrization. The genital area may become fibrotic and deformed.

Wohlfahrtia nuba causes myiasis in humans and animals particularly in camels in Sudan (Higgins, 1986), Ethiopia and "eastwards to Karachi" (Soulsby, 1982). The larva was reported to be the only facultative parasite in wounds of camels and humans in Sudan (Higgins, 1986).

The larvae of *Sarcophaga dux* have been found in skin lesions of camels, cows and bullocks in India (Alwar and Seshia, 1958).

Calliphoridae Producing Myiasis (Blowflies)

Lucilia cuprina

The most important blowflies belong to the genus *Lucilia*, i.e., the larvae of *L. cuprina*, and are the main cause of blowfly strike in sheep in Australia and South Africa. The larvae of *L. cuprina* have long been known to infest camels (Higgins, 1986). *L. cuprina* is greenish to bronze and is therefore also called the green-bottle fly (Fig. 151).

The green-bottle fly is widely distributed around the world, found not only in Australia but also in the Middle East, India and Africa (Higgins, 1986). The female fly lays clusters of light yellow eggs in carcasses, infected wounds and soiled and matted fur around infected sores and discharges. Attracted by the smell, it even lays eggs onto rotting vegetation. A green-bottle female may lay about 1,000 eggs altogether during her lifespan. Depending on the temperature, it takes between 8 hours to 3 days for the first stage larvae to hatch. The larvae feed on epidermal cells, lymph and necrotic tissue.

Clinical Signs Preferential sites for a fly strike are folds of skin, e.g. in the perineal area where urine and feces attract the ovipositing fly. The larvae may cause considerable stress to the infested camel, which may be seen rubbing and biting the infested parts. Infested wounds may be 10 to 15 cm in diameter (Higgins, 1986).

Chrysomya bezziana

Chrysomya bezziana, the fly of the "old world screwworm", occurs in Africa and in Southern Asia wherever camels are found. It is an obligate parasite. The fly is bluish-green with four black stripes on the prescutum. Its face is orange-yellow. It may lay eggs on the skin of both humans and domestic animals, including camels (Soulsby, 1982). The fly deposits clusters of 150 to 500 eggs at the edge of a wound of a living host. Even small wounds, such as tick bites and injection sites, as well as any discharge from natural orifices will attract the female fly. Wounds resulting from accidents, castration, branding, and scalding by dips may also attract the fly (Fig. 152).

The "new world screwworm" *(Cochliomyia hominivorax)* infested 17 out of 500 dromedaries near Tripoli, Libya (Husni and Elowni, 1992). The infestation was most severe on the legs and umbilical cord, from which second and third instars were collected. Since this finding, the new world screwworm has been eradicated from Libya.

Clinical Signs The maggots penetrate and often liquefy the tissue considerably extending the lesions, which may develop a foul odor and ooze a foul-smelling liquid. Severe infections are common and many cause death. Cattle and camels are often attacked around the ears and under the tail, causing perineal myiasis (Higgins, 1986).

Treatment and Control Insecticides kill the larvae. Once they are destroyed the wound should be cleaned and dressed, and any necrotic tissue should be removed. However, care should be taken to use as little insecticide as possible to avoid further irritation of the lesions. Hydrogen peroxide, ether or chloroform may cause hidden larvae to crawl out from crevices and cavities. Ivermectin may also be used.

Oestridae Infestations (Bot flies)

Three species of bot flies are found in camelids. The camel bot, *Cephalopina titillator* (OWC), the sheep and goat nasal bot, *Oestrus ovis*, and some species of nasopharyngeal deer bot fly found in North America. The latter two species are important in NWC imported into the USA.

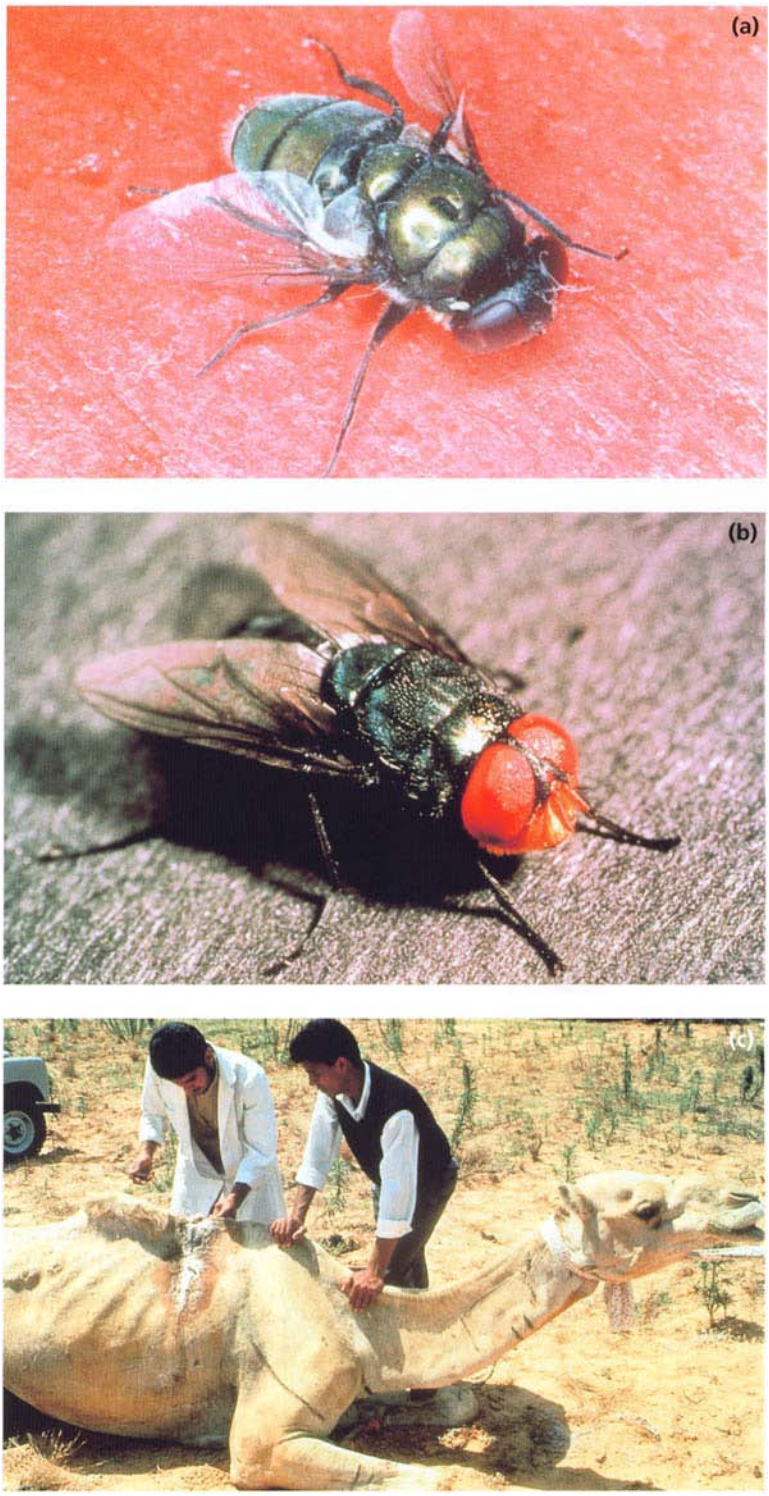


Figure 152a–c
(a) *Chrysomya bezziana* fly, **(b)** *Cochliomyia hominivorax* fly, **(c)** Lesions caused by *C. hominivorax* in a Libyan camel

Figure 153 *Cephalopina titillator*, the camel nasal bot fly (courtesy of Dr. A. Higgins, UK)

Cephalopina titillator

The camel nasal bot fly *Cephalopina titillator*, belonging to the family Oestridae, is an obligate parasite of camels. OWC are commonly infected with *C. titillator* larvae.

The fly has a reddish, dark brown thorax and the head is orange (Fig. 153).

The fly deposits its larvae in the nostrils from which the small, 0.7 mm-long first

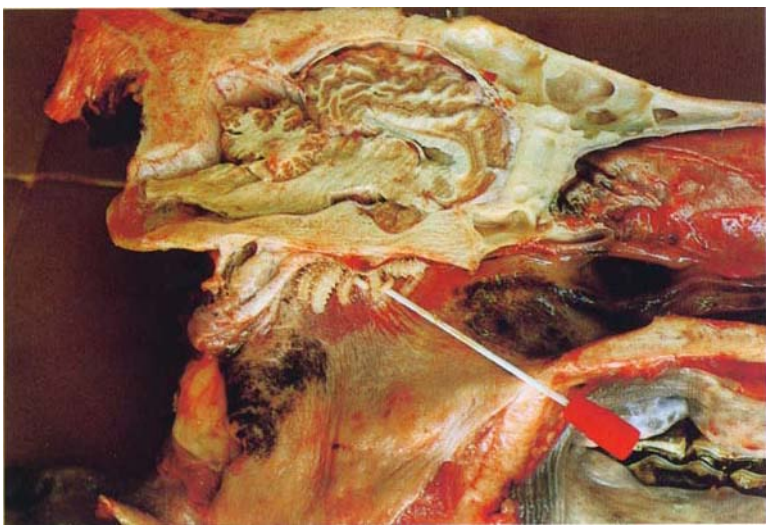


Figure 154 Larvae of *Cephalopina titillator* in the nasopharynx of a racing dromedary near the Eustachian tube

stage larvae migrate to the nasopharynx and nasal sinuses and attach to the mucosa (Fig. 154). The larvae moult twice, spending up to 11 months in the host before leaving to pupate on the ground (Fig. 155).

One generation per year occurs in the former USSR (Zumpt, 1965). In other regions, two generations have been reported (Zumpt, 1965; Higgins, 1986).

Figure 155 Different larval stages of *C. titillator* collected from the nasopharynx of a racing dromedary

According to Zayed (1998) the most common sites of the larvae are the pharyngeal cavity (95.6%), followed by the labyrinth of the ethmoidal bone (71.1%), the turbinates (28.9%) and the lower nasal meatus (6.7%). The first molt of the larvae were only found in the labyrinth of the ethmoidal bone and the second was found to occur in both the labyrinth of the ethmoidal bone and the pharyngeal cavity.

Epidemiology The prevalence of *C. titillator* is very high. In a review of reports from Africa and Asia, including the Middle East, the infestation rates varied between 47 and 100% (Hussein et al., 1983). A 46.7% infestation rate was found in 1250 camels in Iraq (Higgins, 1986). The highest incidence of larval infestation during the year was in January to March, the lowest in November. Similar findings were reported by Patton (1920) cited by Higgins (1986). A survey in Saudi Arabia revealed that 32 out of 35 camels were infested (Hussein et al., 1982). Fatani and Hilali (1994) examined 923 dromedaries for infestation with the second and third instars of *C. titillator* at Al-Asha abattoir in Saudi Arabia; 52% of the camels were infested, peaking in February and September.

In one study, the prevalence in camels from Sudan was 74% (Suliman, 1965) and all 44 dromedaries examined in western Sudan were infested (Musa et al., 1989).

Clinical Signs Unlike many other oestrids, *Cephalopina* flies usually do not make the camels panic. Large numbers of flies may be seen resting on the heads and around the nostrils of the camels.

Often infested camels do not show any clinical signs, but they may be restless or off their feed and may sneeze and snort when infested, particularly during the emergence of mature larvae from the nostrils (Urquhart et al., 1996). The infestation may cause both respiratory and neurological disorders, local inflammation of the pharynx and congestion of the nasal cavity (Hussein et al., 1982). Inflammation of the nasopharyngeal mucosa occurs when the larvae of *C. titillator* hook into the mucous membranes with their two black hooks. Mortalities have reportedly also been associated with heavy infestations, thought to be caused by larvae penetrating the ethmoturbinates leading to meningitis (Burgemeister et al., 1975). Al-Ani et al. (1991) found larvae deep in the turbinate bones and ethmoid area.

Pathology Musa et al. (1989) found 8 to 243 *C. titillator* larvae per animal. Hemorrhagic areas, ulcer-like erosions, nodules containing pus and areas of fibrosis were seen in the mucosa of the nasopharynx. Histopathologic examinations revealed desquamation, hydropic degeneration and hyperplasia of the epithelial cells of the mucosa. Infiltration of lymphocytes, reticuloendothelial cells and fibroblasts and granulomas were seen in the upper part of the submucosa. In addition, the pharyngeal mucus glands showed degenerative atrophy, desquamation of their lining epithelium, lymphocytic infiltration and thickening of the interacinar connective tissue. The isolation of pathogenic bacteria such as *Pasteurella haemolytica, Klebsiella ozaenae, Diplococcus pneumoniae* and *Corynebacterium* spp. from the lesions indicates the risk of secondary infections (Hussein et al., 1982; Al-Ani et al., 1991).

Oryan et al. (1993) reported *C. titillator* larvae in the lungs of 4 camels out of 40 in Iran. The gross pathology in these cases was heavy congestion and hemorrhages. The tissue surrounding the larvae was fibrotic and calcified. Inflammatory reaction infiltration of mononuclear cells was seen in the interstitial lung tissue as well as foci of lymphocytes, eosinophils and plasma cells. Necrosis was also seen around the larvae.

Oestrus ovis

Oestrus ovis has been observed in camels in Egypt (Kaufmann, 1996) and in llamas (Fowler, 1998). Commonly found worldwide, the fly and its larvae are sheep and goat parasites. The female fly produces live larvae that it places around the nostrils. Llamas attacked by flies try to avoid them by pressing their muzzles close to the ground or against other animals. The larvae migrate into the nasal passages where they remain from 2 weeks to 9 months. Then the larvae move into the frontal sinuses where they develop into second and third stage larvae. The mature larvae are evacuated by sneezing onto the ground where they pupate for 3 to 9 weeks. The adult fly only lives for about 2 weeks.

Cephenemyia spp.

Cephenemyia spp. findings are seldom reported in the literature. Several *Cephenemyia* spp. are found in areas of North America where cervids and camelids cohabit pastures. The NWC are aberrant parasite hosts; whether the LC is completed in the llamas is not known. However, according to Fowler (1998), llama breeders in the USA consider these parasites important. *Cephenemyia* spp. were reported in three llamas in California (Fowler and Murphy, 1985). The animals showed sneezing, nasal discharge and coughing. White-tailed deer were common co-habitants of livestock pastures where the three llamas had been grazing, and these deer are commonly infected with *Cephenemyia* spp. In a 9-month-old llama exhibiting inspiratory dyspnea, three *Cephenemyia* bots were found in the nasopharynx (Mattoon et al., 1997). A large soft tissue mass occluding the nasopharynx was observed radiographically.

Clinical Signs Camelids infested with *Oestrus ovis* and *Cephenemyia* spp. show similar signs, such as restlessness, head shaking, sneezing and coughing with or without nasal discharge. The affected animal may be short of breath and consequently fail to keep up with the others when used as a pack animal. Granulomatous swellings may develop in the nasopharynx and nasal cavities. If it becomes obstructive, the animal may be forced to breathe through an open mouth.

Treatment Ivermectin (0.2 mg/kg, s.c.) has been used with some success (about 85% effective against *C. titillator*). Rafoxanide (7.5–10 mg/kg per os) as a drench or bolus and trichlorfon (75 mg/kg, per os) as a drench have been shown to be effective, eliminating the larvae (Kaufmann, 1996).

Muscidae Infestation (House and Stable Flies)

OWC and NWC are pestered by the same fly species that irritate other domestic livestock. The Muscidae family includes many biting and non-biting flies. The most important genera are *Musca* (house fly), *Stomoxys* (stable fly), *Hydrotaea* (sheep-head fly), *Haematobia* (horn fly) and *Fannia* (the lesser house fly). Many of these are responsible for livestock "fly-worry" and are vectors of significant bacterial, helminth and protozoal pathogens causing disease (see Table 59).

Musca autumnalis, the face fly, a very common fly in some temperate and subtropical areas, causes fly-worry to cattle and horses on pasture. It is the intermediate host of several pathogenic parasites, e.g. *Thelazia* spp. and *Parafilaria bovicola*, and may transmit *Moraxella bovis*, causing "pink eye" or infectious bovine keratoconjunctivitis in bovines.

Stomoxys calcitrans is a vector of *T. evansi* and several other pathogens causing severe diseases such as anthrax, brucellosis, leptospirosis and vesicular stomatitis (Higgins, 1986). It was shown in India that the fly preferred to feed on camels rather than horses (Higgins, 1986). Pestered camels may have significant milk reduction.

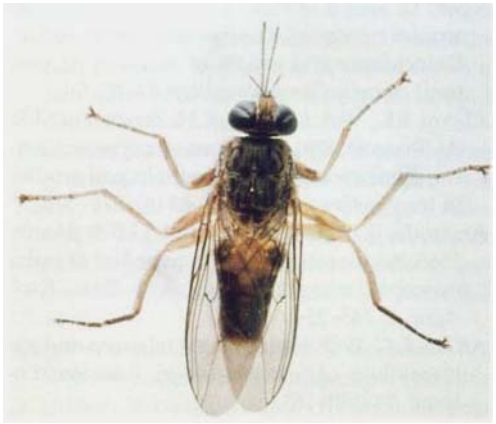


Figure 156 *Glossina* fly (courtesy of Fotoarchiv, Institute for Parasitology, Hanover, Germany)

Glossinidae Infestation (Tsetse Flies)

The genus *Glossina* comprises approximately 30 species and subspecies confined to a large belt of Tropical Africa. Tsetse flies are the common intermediate hosts for *Trypanosoma* of mammals in Central Africa. When taking a blood meal, the flies become infected with salivarian trypanosomes that undergo multiplication. The flies then become infective to other hosts during subsequent feeding (Fig. 156).

Camels may become infected by some Tsetse-transmitted trypanosomes.

5.2.7.5 Tabanidae Infestation (Horse Flies)

There are several species of horse flies or tabanids that are important vectors of *T. evansi* in camels. Two genera particularly: the *Tabanus* and *Haematopota*. Some horse flies are also known to transmit anthrax and other pathogenic bacteria. These ferocious biting flies feed on a variety of animals and humans, attacking anywhere on the body. Their predilection sites are the ventral abdomen, legs and inguinal regions. Afflicted animals usually try to escape from the feeding flies, only causing the flies to move onto another animal in order to complete their blood meals. This is of epidemiological significance as several blood meals increase the risk of transmission of pathogens. In addition, the drops of blood the biting fly leaves at the feeding sites may attract other flies, i.e. Calliphoridae. The irritation and distress caused by the flies may distract the host from feeding and may be so severe that it leads to decreased productivity.

The Tabanidae are large robust flies with powerful wings (wing span of up to 6.5 cm). The coloration of the wings together with the characteristics of their short, stout, three-segmented antennae is useful in differentiating the important genera: *Tabanus*, *Chrysops* and *Haematopota* (Fig. 157).

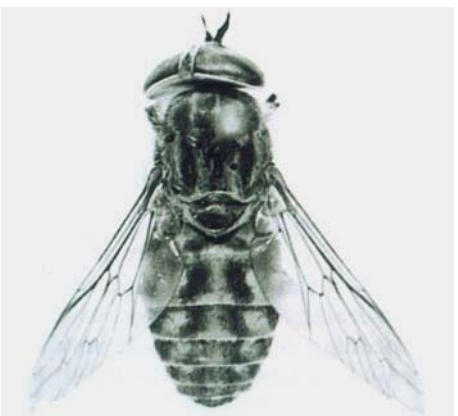


Figure 157 *Tabanus* fly

5.2.7.6 Ceratopogonidae Infestation (Midges)

This family consists of very small flies that are commonly known as biting midges. They belong to the suborder Nematocerina. The females feed on man and animals and are known to transmit various viruses, protozoa and helminths. El Bihari (1985) suggested that *Culicoides* spp. (biting midges) may transmit *Onchocerca fasciata*, a filarid worm of camels.

5.2.8 Linguatula serrata Infection (Tongue Worm)

Linguatula serrata is a cosmopolitan parasite found in the nasal and respiratory passages of canines such as dogs, foxes and wolves. It may also attack humans, horses, goats and sheep. Camels may serve as an intermediate host. The parasite was found in 27% of 11 camels surveyed in Jordan (Sherkov and Rabie, 1976). In a survey of 40 camels in Iran (Oryan et al., 1993), nymphs of *L. serrata* were found in 12.5% of the camels in the portal mesenteric lymph nodes. No adults were seen.

L. serrata is tongue-shaped and the adults of this strange class of arthropods resemble annelid worms rather than arthropods.

Life Cycle The eggs contain larvae that hatch in the intermediate host's intestine. The larvae penetrate the intestinal wall and reach the mesenteric glands via the blood stream and develop into the infective nymphal stage. The nymph lies in a small cyst surrounded by a viscid fluid. Consuming infected viscera completes the cycle.

Parasites in large numbers may cause significant irritation of the host, manifested by sneezing and coughing. Fits of difficult breathing and restlessness may occur and a mucous nasal discharge, often blood-stained, may be observed.

Diagnosis Clinical signs and eggs in the feces or in the nasal discharge help diagnosis. The clinical signs described may be seen in any *Camelidae* respiratory disease, including *C. titillator* infestations.

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5.3 Infection with Nematodes

Introduction

The word helminth is derived from the Greek *helmins* or *helminthes* meaning worm, and usually refers to both parasitic and non-parasitic worms belonging to the phylum Platyhelmintha (flukes and tapeworms) and Nemathelmintha (nematodes or roundworms).

A number of helminths are camelid-specific, but some are also common to other hosts, primarily domestic ruminants and

Table 60 Nematodes of Old World and New World Camels

Disease	Species	Occurrence OWC	Occurrence NWC	Location
Trichostrongylidosis (Gastrointestinal worms)	*Haemonchus contortus*	+	+	C3
	Ostertagia ostertagi	+	+	C3
	Marshallagia marshalli	+	+	C3
	Camelostrongylus mentulatus	+	+	C3
	Spiculopteragia peruviana		+	C3
	Lamanema chavezi		+	Intestine
	Trichostrongylus spp.	+	+	C3
	Cooperia spp.	+	+	Small intestine
	Nematodirus spp.	+	+	Small intestine
	Graphinema aucheniae		+	C3
Dictyocaulosis (Lungworm)	*Dictyocaulus viviparous*		+	Bronchi
	Dictyocaulus filaria	+	+	Bronchi
Parelaphostrongylosis (Meningeal worm)	*Parelaphostrongylus tenuis*		+	Subdural space
Angiostrongylosis	*Angiostrongylus cantonensis*		+	Lung
Oesophagostomosis (Nodular worm)	*Oesophagostomum columbianum*	+		Intestine
Chabertiosis	*Chabertia ovina*	+	+	Intestine
Ancylostomatosis (Hookworm)	*Bunostomum* spp.	+	+	Small intestine
Strongyloidosis	*Strongyloides papillosus*	+		Small intestine
Oxyuridosis (Pinworm)	*Skrjabinema ovis*		+	Colon
Trichuriosis (Whipworm)	*Trichuris* spp.	+	+	Cecum, large intestine
Capillariosis	*Capillaria* spp.	+	+	Small intestine
Gongylonemosis	*Gongylonema* spp.	+	+	Esophagus
Habronematidosis	*Parabronema skrjabini*	+		C3
Thelaziosis	*Thelazia* spp.	+	+	Eye
Onchocercidosis	*Dipetalonema evansi*	+		Blood
	Onchocerca spp.	+		Aorta, Sub-cutaneous tissue

wild animals. Investigations of camelid parasites are fairly recent and veterinarians and parasitologists studying these worms are often not cognizant that the LC of the parasite under study is the same as or similar to that in other animals.

Most investigations of helminthosis in camelids have been surveys of the prevalence of worm eggs in fecal samples or parasites in the intestinal tract of slaughtered camels. Some reports are case histories, but only a few are profound studies of the pathogenesis of particular camelid parasites.

There has been recent interest in defining the parasitic fauna in NWC outside their countries of origin, e.g. in North America and Europe. The species composition of nematodes investigated in these surveys seems to differ between continents. The predominant gastrointestinal nematode species in llamas in North America are different from those found in alpacas in South America (Rickard, 1994).

Dakkak and Ouhelli (1987) have compiled a comprehensive list of helminths found in dromedaries and Fowler (1998) wrote a review of those parasites infecting NWC (Table 60).

5.3.1 Classification of Nematodes

Phylum Nemathelmintha
Class Nematoda
Order Strongylida
Family Trichostrongylidae
(Gastrointestinal worms)
Haemonchus contortus (NWC, OWC)
Haemonchus longistipes (OWC)
Ostertagia ostertagi (NWC, OWC)
Ostertagia spp. (NWC)
Teladorsagia circumcincta (OWC)
Ostertagia trifurcata (OWC)
Marshallagia marshalli (NWC, OWC)
Marshallagia mongolica (OWC)
Camelostrongylus mentulatus (NWC, OWC)
Trichostrongylus spp. (NWC, OWC)
Trichostrongylus axei (NWC, OWC)
T. columbiformis (NWC, OWC)
T. probolurus (OWC)
T. vitrinus (OWC)
T. falculatus (OWC)
T. affinus (OWC)
Cooperia spp. (OWC)
C. oncophora (NWC, OWC)
C. pectinata (OWC)
C. surnabada (NWC, OWC)
Spiculopteragia peruviana (NWC) – only host
Graphinema aucheniae (NWC) – only host
Impalaia tuberculata (OWC) – only host, dromedary
Impalaia nudicollis (OWC) – only host, dromedary
Impalaia aegyptiaca (OWC) – only host

Family Molineidae
Nematodirus spathiger (NWC, OWC)
N. filicollis (NWC)
N. lanceolatus (NWC)
N. mauritanicus (OWC)
N. abnormalis (OWC)
N. dromedarii (OWC) – only host
N. helvetianus (OWC)
N. lamae (NWC) – only host
N. battus (NWC, OWC)
Nematodirella dromedarii (OWC) – only host
Nematodirella cameli (OWC) – only host, Bactrian
Lamanema chavezi (NWC) – only host

5.3.2 Trichostrongylidosis (Gastrointestinal Worm Infection)

These parasites are relatively small and parasitize the gastrointestinal tract (GI). Their LC is direct and the L3 is the infective stage.

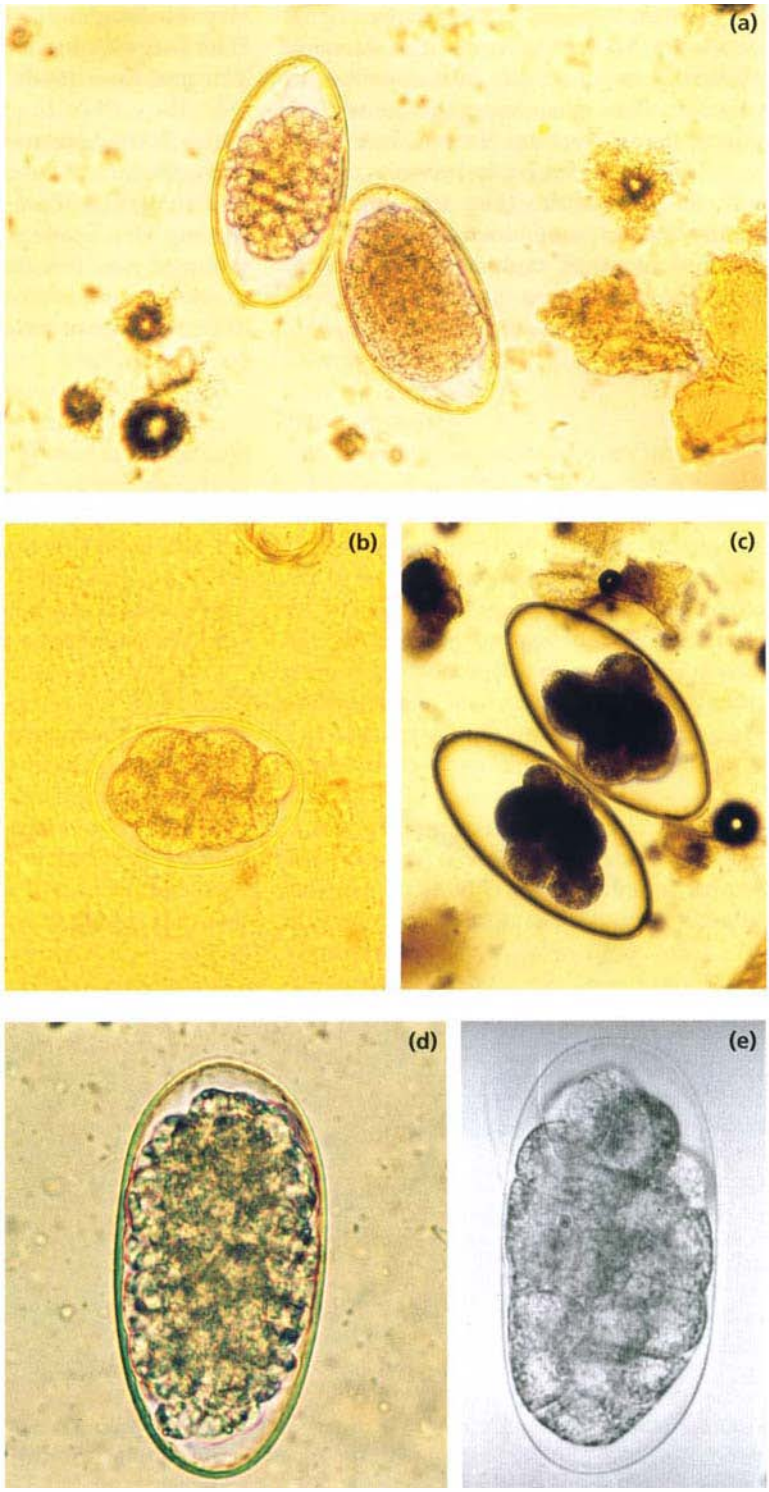

Figure 158a–d Common Trichostrongylidae eggs of camelids: **(a)** *Haemonchus longistipes* (left) and *Trichostrongylus* spp. (right); **(b)** *Ostertagia* spp.; **(c)** *Nematodirus* spp **(d)** *Trichostrongylus* spp.; **(e)** *Cooperia* spp. (Figs. b and e: courtesy of Fotoarchiv, Institute for Parasitology, Hanover, Germany)

The widely spread trichostrongylid parasites are known to cause considerable morbidity and mortality in ruminants and camelids. The most important genera affecting the GI tract are *Haemonchus, Ostertagia, Marshallagia, Trichostrongylus, Cooperia* and *Nematodirus* (Fig. 158). Of these, *Haemonchus* spp. are blood-sucking pathogenic parasites of compartment 3 (C3) of camelids.

Haemonchus
(The Large Stomach Worm or Wire Worm of Ruminants)

Bihari (1985) conveniently grouped helminthic infections of the camelids' GI tract into two categories: common and occasional. Among the common nematodes, one group of very few that have been studied to some extent and known without doubt to be pathogenic are *Haemonchus* spp.

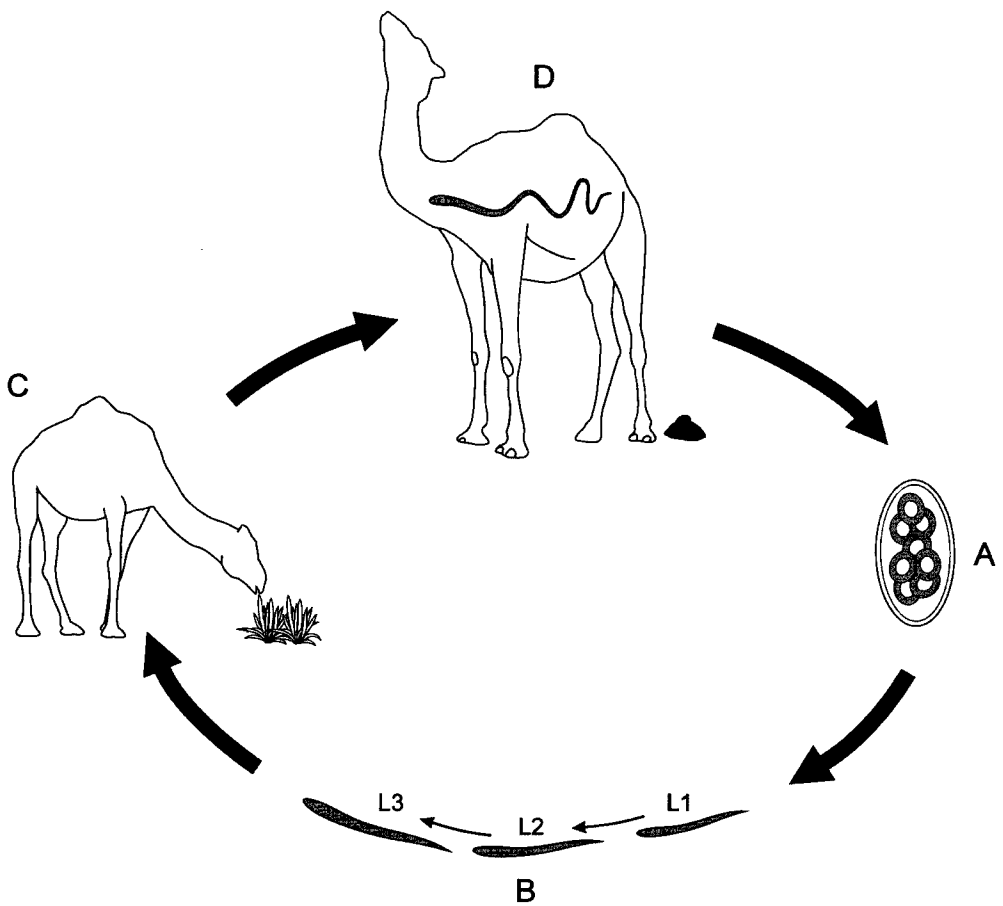

Figure 159 The direct life cycle of Trichostrongylidae (e.g. *Haemonchus, Ostertagia, Trichostrongylus*): A = egg discharged with the feces; B = development of L1 to infective L3 on pasture; C = camel ingests L3 while grazing; D = L3 develops to L4, L5 and to adult parasites

Haemonchus spp. are found most often in dromedaries and *H. longistipes* has been studied by Richard (1989), Jacquiet et al. (1995) and Jacquiet et al. (1996). *Haemonchus longistipes* is considered to be a species adapted solely to camels, but may also infect small stock (Kumar and Yadev, 1993). A high prevalence of *H. longistipes* in dromedaries has been reported from Sudan. Arzoun et al. (1984a) observed a prevalence of 89% during the rainy season and 64% during the dry season. Adult worms are morphologically relatively easy to differentiate from other trichostrongylids such as *Ostertagia* spp. or *Trichostrongylus* spp. They are one of the largest nematodes of the C3. The adult male is often homogeneously red (after a blood meal), and the female has a red and white spiral appearance because the uterus winds around the intestine, giving it the appearance of a barber's pole.

Life Cycle The prepatent period of *Haemonchus* is unknown in camelids. The LC is direct in most of the trichostrongylids as shown in Fig. 159.

The adult female parasites are prolific egg layers, particularly during the rainy season. This was shown to be apparent for *H. longistipes* (Jacquiet et al., 1995). In the pasture, the infective larvae (L3) develop within 4 to 6 days. However, the LC may be delayed for many weeks or even months in cooler conditions. The eggs and infective larvae are sensitive to desiccation and low temperatures. After ingestion, the larva moults twice close to the gastric glands and just before the last moult the "tooth", a piercing lancet, develops, enabling the parasite to draw blood from the mucosal vessels.

Haemonchosis

The most important feature of *Haemonchus* infection is anemia. The parasites ingest blood and are motile, leaving wounds that hemorrhage into the stomach lumen. In sheep, where the disease is well studied, each *H. contortus* may cause a blood loss of 0.05 mL per day by ingestion and seepage from the wounds (Clark et al., 1962). Substantial blood loss may occur, considering the number of L4 and adult worms harbored by the host. Both stages suck blood (Fig. 160).

Haemonchosis in camelids is similar to that described in sheep (Arzoun et al., 1984 a, b). The infection is often accompanied by diarrhea. In heavily infected ani-

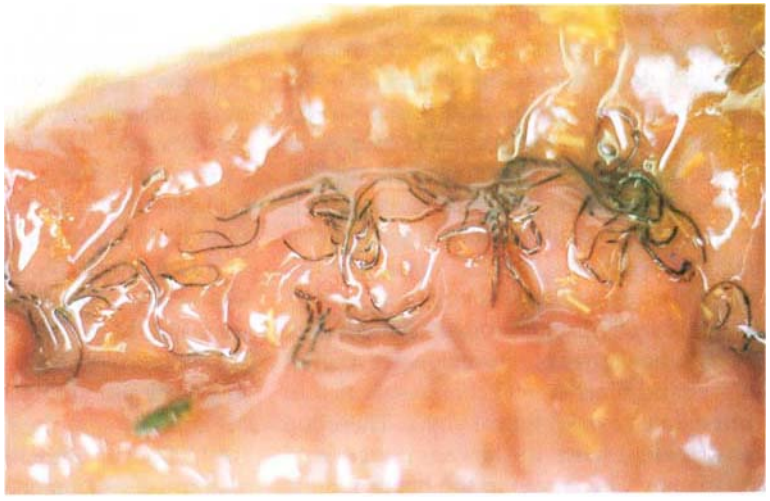

Figure 160
Haemonchus longistipes in a dromedary's C3

mals, progressive deterioration occurs with marked anemia seen in 10 to 45% of cases (Faye, 1997), eventually leading to emaciation and death. The chronic form may be difficult to differentiate from other chronic camel diseases, e.g. trypanosomosis.

Acute haemonchosis in experimentally infected camels induced clinical signs of mucoid diarrhea, anorexia, anemia, loss of body weight, edema of the lower limbs, general malaise and death after 8 to 10 weeks (Arzoun et al., 1984b).

Haemonchosis in the camel is often associated with hypoproteinemia, including hypoalbuminemia and hypoglubulinemia, as well as leucocytosis, including neutrophilia and eosinophilia (Graber et al., 1967; Queval et al., 1967; Richard 1979 and 1989; Arzoun et al., 1984 b; Jacquiet et al., 1995). Low PCV, calcium, phosphate, magnesium and copper levels were also diagnosed by Kaufmann (1996).

Other Common Trichostrongylids

The common stomach worms of *Ostertagia* spp., such as *O. ostertagi, O. lyrata, Teladorsagia circumcincta,* and *O. trifurcata,* are highly adapted to cattle, small stock and wild ruminants. However, they are also found in camelids with an LC similar to *Haemonchus* spp.

Different climates produce differences in the epidemiology of the parasite. In temperate regions, the larvae become arrested (hypobiosis) in early autumn and development starts again in spring. In other areas of the world where the summers are hot, the larvae may survive the hot unfavorable environmental conditions during the summer in hypobiosis. The LC varies according to climate and host species.

Significant numbers of the parasites in the C3 may give rise to extensive pathological and biochemical changes, which in turn create severe clinical signs. These are most evident when the larvae emerge from the glands. The larvae in the host's glands stimulate the formation of grayish white nodules, which are readily seen in the mucosa of C3 at necropsy.

Windsor (1997) reported three cases of ostertagiosis in llamas in northern Scotland south and southwestern England. The affected animals died despite treatment with ivermectin.

There are some other parasites found in the abomasum or C3 of camelids closely resembling Ostertagia: *Marshallagia marshalli, M. mongolica, Teladorsagia* sp., *Camelostrongylus mentulatus,* and *Spiculopteragia peruviana,* which was first described in alpacas, llamas and vicuñas from Titicaca in Peru (Guerrero and Chavez, 1967). At the same time, *Ostertagia lyrata* and *Haemonchus contortus* were first found in alpacas, together with two other species: *Trichostrongylus longispicularis* and *Camelostrongylus mentulatus.* A few years later, *O. ostertagia* and *O. lyrata* were first found in llamas in Peru (Vasques and Marchinares, 1971).

The L3 of *Marshallagia marshalli* penetrate the gastric glands of C3 and are eventually surrounded by a 2 to 4 mm diameter large nodule, each containing two to three larvae that mature in 15 to 18 days. The prepatent period is usually about 3 weeks, but arrested development may occur (Fowler, 1998). The eggs may easily be confused with *Nematodirus* spp. eggs. The parasite has limited distribution. It occurs in llamas in the western USA. It is a common parasite in sheep in the Mediterranean, and has also been reported in camels in India and Russia (Dakkak and Ouhelli, 1987). *Marshallagia mongolica* has only been reported in Mongolia (Dakkak and Ouhelli, 1987), and unidentified *Marshallagia* sp. in guanacos in Argentina (Navone and Merino, 1989).

Camelostrongylus mentulatus is a common camelid stomach worm, particularly in animals sharing grazing with sheep (Dakkak and Ouhelli, 1987). The parasite also infects sheep, goats, antelope and lla-

mas (Soulsby, 1982). *C. mentulatus* is commonly found in the Middle East and in areas north of the African continent (Kaufmann, 1996), but less commonly in South America and the USA. It was first described in llamas in Argentina (Led and Boero, 1972). According to Kaufmann (1996), *C. mentulatus* may cause significant disease in camels. It seldom occurs in single infections.

Trichostrongylus

Trichostrongylus spp. are considered to be one of the most important causes of parasitic gastroenteritis in ruminants. *Trichostrongylus axei* is found in the abomasum of ruminants, in C3 in camelids and in the stomach of horses, donkeys, pigs and humans. Other *Trichostrongylus* spp., such as *T. colubriformis, T. vitrinus, T. probolurus,* are found mainly in the small ruminants' intestines but also frequently in camelids. Occasionally *T. falculatus* and *T. affinus* have been recorded in camelids. The *Trichostrongylus* spp. are small and thin, about 7 mm long and difficult to see with the naked eye.

Cooperia

Cooperia spp. are small nematodes similar in size to *Ostertagia.* They are parasites of the small intestines of ruminants and camelids throughout the world.

C. oncophora and *C. pectinata* are found in OWC and *C. oncophora* and *C. zurnabada* in NWC.

Graphinema aucheniae

This parasite is only found in C3 in NWC. Its LC and epidemiology is similar to trichostrongyles.

5.3.3 Infections with Molineidae

Nematodirus

These parasites are found worldwide, particularly in temperate zones. They are small intestinal parasites. The adults are slender, about 2 mm long, and relatively easy to differentiate from other trichostrongyles. The eggs are large and twice the size of other trichostrongyle eggs (see Fig. 158a–e). *Nematodirus battus* is the most pathogenic species in temperate areas.

Severe damage to the villi and erosion of the mucosa resulting in villous atrophy, coincide with the parasitic phases of the larvae while in the mucosa. Young animals may exhibit rapid progressive dehydration following diarrhea, leading to death. At necropsy, the carcass is dehydrated and enteritis is often evident in the ileum.
N. lamae, N. battus, N. spathiger, N. filicollis and *N. lanceolatus* are species found in NWC (Fowler, 1998).

The following species are reported in OWC: *N. spathiger, N. mauritanicus, N. abnormalis, N. dromedarii* and *N. helvetianus. N. cameli* is reported in the Bactrian camel of the former USSR. In addition, species closely related to *Nematodirus* spp. are found parasitizing dromedaries: *Nematodirella dromedarii, Impalaia tuberculata* and *I. nudicollis.* The latter two species are parasites of the C3, occasionally of the small intestine (Kaufmann, 1996), and are mostly found in camels in Africa (Dakkak and Ouhelli, 1987). Gibbons et al. (1977) discussed the classification of these seldom-mentioned species. *Nematodirella dromedarii* was first reported in India and described by Lodha and Raisinghani (1979). It was found in the districts of Bikaner and Jodhpur in Rajasthan with a prevalence of over 42%.

Lamanema chavezi

One of the most important NWC nematode pathogens is *Lamanema chavezi*. It is thought to be a parasite of the mountain viscacha *Lagidium viscacia boxi*. Llamas and alpacas are believed to be aberrant hosts, in which the infection may be very severe. Particularly vulnerable are recently weaned NWC.

The LC is poorly understood. The infective larvae develop within the eggs, giving them excellent resistance to adverse climatic conditions (Leguia, 1991). Ingested larvae penetrate the intestinal wall and pass to the liver and lungs. When maturation is completed the parasites migrate back to the small intestine via the trachea. The prepatent period is about 30 days (Guerrero et al., 1973).

Heavy infection causes hepatic and respiratory failure and death may follow. This was shown experimentally: a 4-month old alpaca given 200,000 larvae orally died after 20 days, exhibiting severe anemia (Guerrero et al., 1973).

The migration of the larvae causes catarrhal and hemorrhagic enteritis with areas of mucosal necrosis. In acute infections, the liver is congested, showing multiple small foci of coagulative necrosis and petechial hemorrhages. Areas of lung congestion are also seen. When the larvae have returned to the intestine, the liver lesions become fibrotic and may calcify (Fowler, 1998) showing a characteristic mottled appearance (Leguia, 1991). Five young alpaca given 10,000 *L. chavezi* larvae showed increased levels of glutamate-oxalacetate-transaminase 14 days later, indicating liver damage (Guerrero et al., 1973). The liver is often condemned.

5.3.4 Dictyocaulosis (Lungworm Infection) Parelaphostrongylosis (Meningeal Worm Infection) Angiostrongylosis

Family Dictyocaulidae
Dictyocaulus viviparus (NWC)
D. filaria (NWC, OWC)

Family Protostrongylidae
Parelaphostrongylus tenuis
(NWC, llama aberrant host)

Family Angiostrongylidae
Angiostrongylus cantonensis
(NWC, alpaca aberrant host)

Dictyocaulus

The parasite belonging to the family Dictyocaulidae occurs in the respiratory passages of the lungs and is the major cause of parasitic bronchitis in domestic animal species. The parasites are found worldwide, particularly in temperate climates. The adult parasite is long and slender, about 8 cm long, and is found in the trachea and bronchi. Their LC is direct. The females are ovo-viviparous, laying eggs containing fully developed larvae L1. The eggs are coughed up and swallowed. Hatching may already begin in the lungs, but usually occurs while the eggs pass through the gut of the host. Some eggs may be expelled via nasal discharges.

Life Cycle The preparasitic (free) stages feed on food reserves stored in their intestinal cells, unlike those of other trichostrongyle larvae, which actively feed on microorganisms in the environment. The L3 stage is reached in 5 to 7 days. Approximately 4 days following the infection, the ingested L3 penetrate the intestinal mucosa of the host and pass into the mesenteric lymph nodes where they moult into L4. The L4 reach the lungs via the blood and lymph within a week of infection. The

last molt occurs in the bronchioles and the L5 move up the bronchi and mature into the adult form. The prepatent period in cattle is about 3–4 weeks.

Clinical Signs Parasitic bronchitis is a problem, particularly in areas with a mild climate, high rainfall and permanent pastures. The disease is mostly seen in young animals, but it can affect any age group. Affected animals may cough, with dyspnea and nasal discharge. Heavily infected animals may die due to respiratory failure following the development of interstitial emphysema and pulmonary edema. Many animals gradually recover, but this may take months. Superimposed bacterial infections might occur, hindering recovery. Body temperature is usually normal unless secondary pneumonia develops.

Epidemiology In endemic areas with temperate climates, the L3 may hibernate (over winter) on pasture in sufficient numbers to initiate infection the following spring. An infection may also persist from year to year by carrier animals; i.e. small numbers of adult parasites may survive in the bronchi of affected animals.

In Europe generally only calves in their first grazing season show clinical disease. In endemic situations, older animals acquire a strong immunity.

Larvae require a moist environment to survive; thus infections are not considered a problem in hot and dry climates. However, *Dictyocaulus filaria* in OWC has been reported in several African and Asian countries as well as in Europe. *D. cameli* (Boev, 1952) has been described in camels in Asia and Europe. Some authors consider it to be synonymous with *D. viviparus* (Soulsby, 1982). Both *D. viviparus* and *D. filaria* are commonly found parasitizing *Camelidae* in South America (Fowler, 1998).

Diagnosis Diagnosis is based on clinical signs of respiratory distress. It is usually a herd problem. Several young animals may simultaneously show signs of the infection. Coughing may be evident during the prepatent period when no eggs are yet laid. Demonstration of larvae in feces using the Baermann method is an important diagnostic tool. Furthermore, specific antibody tests are commercially available for the diagnosis in cattle.

Parelaphostrongylus tenuis

Adult *P. tenuis* are found in the cranial venous sinuses and subdural spaces of the white-tailed deer *(Odocoileus virginianus)* in which the infection is sub-clinical. The parasite may also infect and cause neurological disease in several other Cervidae and domestic livestock such as sheep, goats and llamas. It is a small, hair-like nematode.

Life Cycle The llama is an aberrant host and it is not known whether the LC of this parasite is the same in the llama as in its natural host. In deer, eggs are laid and eventually hatch on the meninges. The larvae are then carried via the circulation to the lungs. Eggs may also be deposited in the venous circulation and then carried to the lungs, where they embryonate and hatch (Soulsby, 1982). The larvae then reach the bronchi and trachea and are coughed up and swallowed, ending up in the feces. Intermediate hosts such as terrestrial snails and slugs eat the larvae. The final host ingests the infected snails and after the larvae are released into the stomach, they penetrate the wall, reaching the spinal cord in 10 days. They then migrate to the spinal subdural space and further on to the brain where they enter their final location, the venous sinuses, by penetrating the dura mater.

Clinical Signs There are usually no clinical signs of disease but fatal neurological disease may occur in the aberrant hosts,

such as the llama. Migration of the larvae in the spinal cord produces lesions like encephalomalacia, manifested as lameness, ataxia, stiffness, circling, blindness, hypermetria, paraplegia, paralysis and abnormal position of the head (Fowler, 1998). Local hemorrhages in the spinal cord often lead to death (Cheney and Allen, 1989).

Epidemiology Llamas cohabiting with white-tailed deer are at risk. Other ungulates may be aberrant hosts, e.g. elk *(Cervus canadensis)*, moose *(Alces alces)*, caribou *(Rangifer tarandus)* and red deer *(Cervus elaphus)*, as well as sheep and goats. The infection may cause death in llamas, alpacas and guanacos (Rickard, 1994).

Diagnosis At necropsy the thin, slender parasites are difficult to find. Larvae may be found in the feces by the Baermann technique. However, identification of the larvae to species is not possible. A recently developed ELISA has shown promising results in demonstrating meningeal worm infestations in domestic goats (Rickard, 1994).

Angiostrongylus cantonensis

A lungworm, *Angiostrongylus cantonensis*, normally a parasite of rats in the Pacific Basin, was found in an alpaca during quarantine. The alpaca died and the nematodes were found in the lungs at necropsy (Fowler, 1998).

5.3.5 Oesophagostomosis and Chabertiosis (Nodular Worm Infection)

Family Chabertiidae
- *Oesophagostomum* spp. (NWC)
- *O. columbianum* (OWC, NWC)
- *O. venulosum* (OWC)
- *O. vigintimembrum* (OWC)
- *Chabertia ovina* (OWC, NWC)

Oesophagostomum spp. are stout roundworms 1–2 cm long. These nematodes, found in the large intestine of ruminants, camelids, pigs and primates, are often called nodular parasites because many cause nodules in the wall of the intestine.

The parasites are distributed worldwide, but are more important in tropical and subtropical regions. The two species, *O. columbianum* and *O. venulosum*, essentially sheep and goat parasites, have also been reported in camels in Africa and Asia (Kaufmann, 1996). None of the nodular worms in NWC have been identified as to species (Fowler, 1998).

Life Cycle The LC of these nematodes has not been established in camelids. However, it is assumed that the LC in *Camelidae* is similar to that in ruminants. Thin-walled eggs are passed in the feces. The development and bionomics of the preparasitic stages are similar to those of the *Strongylus* spp. The infective stage, the L3, will penetrate the intestinal mucosa where the third ecdysis takes place.

In some species, the development occurs within the nodules. *O. columbianum* and *O. venulosum* can cause severe enteritis. However, the latter species has fewer tendencies to cause nodules, i.e. inflammation in the wall of the intestine, and is less pathogenic. The L3 of *O. columbianum* may migrate deep into the intestinal mucosa, provoking inflammation. The nodules formed are visible to the naked eye.

Chabertia ovina

Another parasite of the cecum and colon, *Chabertia ovina*, also called the "large-mouthed bowel worm" is found in domestic and wild ruminants and in camelids throughout the world. It is particularly common in sheep and goats, but rarely causes clinical disease. The worm is rarely reported in dromedaries and NWC. The

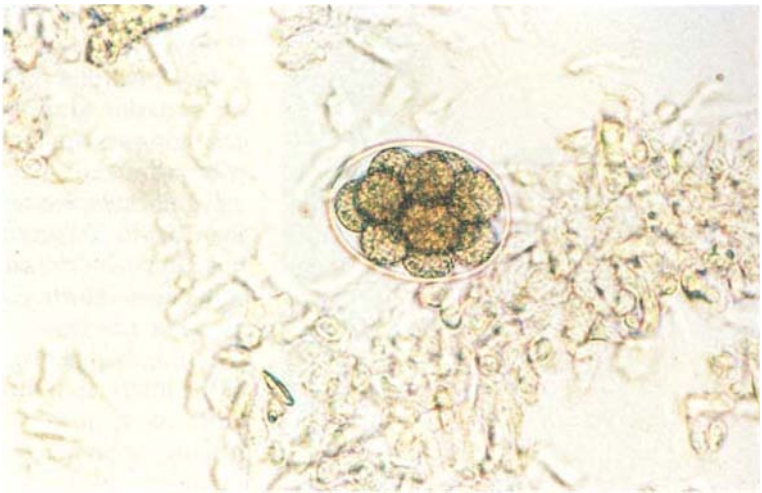

Figure 161 *Chabertia ovina* egg in dromedary feces

first finding of the worm in guanacos was in 1989 (Navone and Merino, 1989).

The adult worm is easily recognized by its 1.5 to 2 cm length and enlarged anterior end, which is ventrally curved with a marked buccal capsule. The oral aperture is surrounded by a double leaf-crown.

Life Cycle The LC is direct. The infection is per os. The L3 enters the mucosa of the small intestine but seldom the cecum or colon. After about a week, the L4 emerges onto the surface of the gut and migrates to the cecum where it develops into the L5. The immature adult then passes to the colon.

Clinical Signs The L5 and adults feed on the intestinal mucosa. This may cause local hemorrhage and loss of proteins. In sheep, the presence of 250–300 worms is considered pathogenic. Clinical signs in heavily infected animals include diarrhea tinged with blood and mucus.

Diagnosis Diagnosis is made by demonstrating the eggs (Fig. 161) in the feces and by identification of the L3 in larval cultures. However, because the pathogenic effect of infection often occurs before the end of the prepatent period, the number of eggs might be low.

5.3.6 Bunostomosis (Hookworm Infection)

Family Ancylostomatidae
Bunostomum spp. (NWC)
Bunostomum trigonocephalum (OWC)

Bunostomum spp. are blood sucking hookworms occurring in small ruminant intestines in many parts of the world. The parasites are seldom reported in camelids. They are mainly found in NWC living in warm tropical climates (Fowler, 1998).

The 1.5 to 2.5 cm long parasites of the small intestine of ruminants belong to the larger nematodes of the ruminants' small intestine. The anterior end of the worm is bent dorsally.

Life Cycle The LC is direct. The L3 infection of the host occurs either orally or through the skin. If the route is through the skin, the larvae migrate to the lungs where the third ecdyces occur. The larvae are then coughed up and swal-

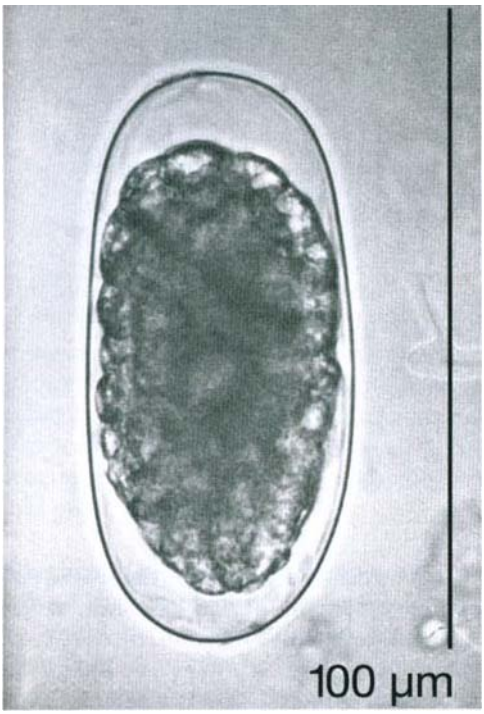

Figure 162 *Bunostomum* spp. egg from dromedary feces (courtesy of Fotoarchiv, Institute for Parasitology, Hanover, Germany)

lowed, reaching the intestine after about 11 days. The prepatent period is between 1 and 2 months. The infective larvae are not resistant to desiccation and therefore can only survive on moist pastures in fairly hot climates.

Significant infections in sheep with more than 100 to 500 worms may produce anemia, hypoalbuminemia, weight loss and sometimes diarrhea with dark feces. Progressive edema may clinically manifest with the characteristic "bottlejaw" (edema of the intermandibular region) that is seen quite often in affected camels. Infection usually occurs together with other gastrointestinal strongyles and thus the hookworms may contribute to the general effect of the parasitism. The egg is shown in Fig. 162.

5.3.7 Strongyloidosis

Order Rhabtitida
Family Strongyloididae
Strongyloides papillosus

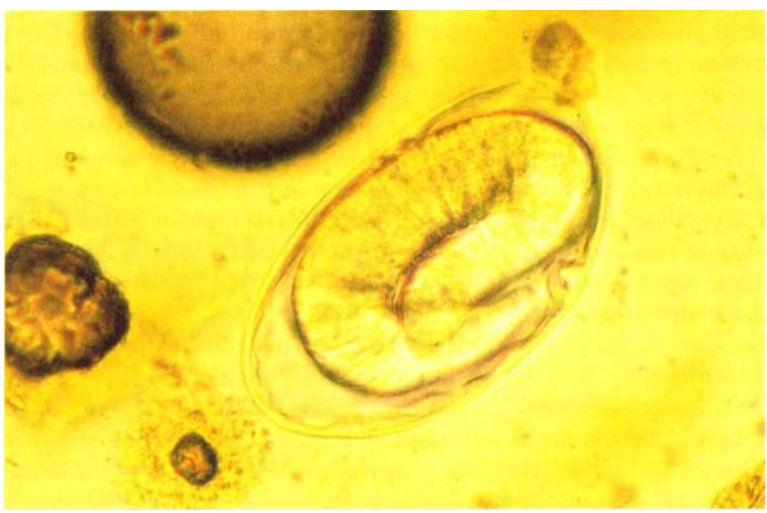

Figure 163 Egg of *Strongyloides papillosus* from a dromedary

Strongyloides papillosus

Strongyloides is the only important species of the Rhabtitida and belongs to a group of parasites of the small intestine, common in very young animals of several host species. Although these worms are generally of little pathogenic significance, they may cause severe enteritis and death.

Neonatal animals acquire the infestation shortly after birth from arrested larvae in the tissues of the dam. Such larvae stimulated by parturition are mobilized and excreted in the dam's milk. Prenatal infections have also been experimentally shown to occur in pigs and cattle. The epidemiology of *S. papillosus* is unknown in camelids. The species is reported to be common in dromedaries, particularly in African countries (Dakkak and Ouhelli, 1987). *Strongyloides* eggs are oval with a thin shell. In camels it is the larvated egg that is passed out in the feces (Fig. 163).

The LC is shown in Fig. 164.

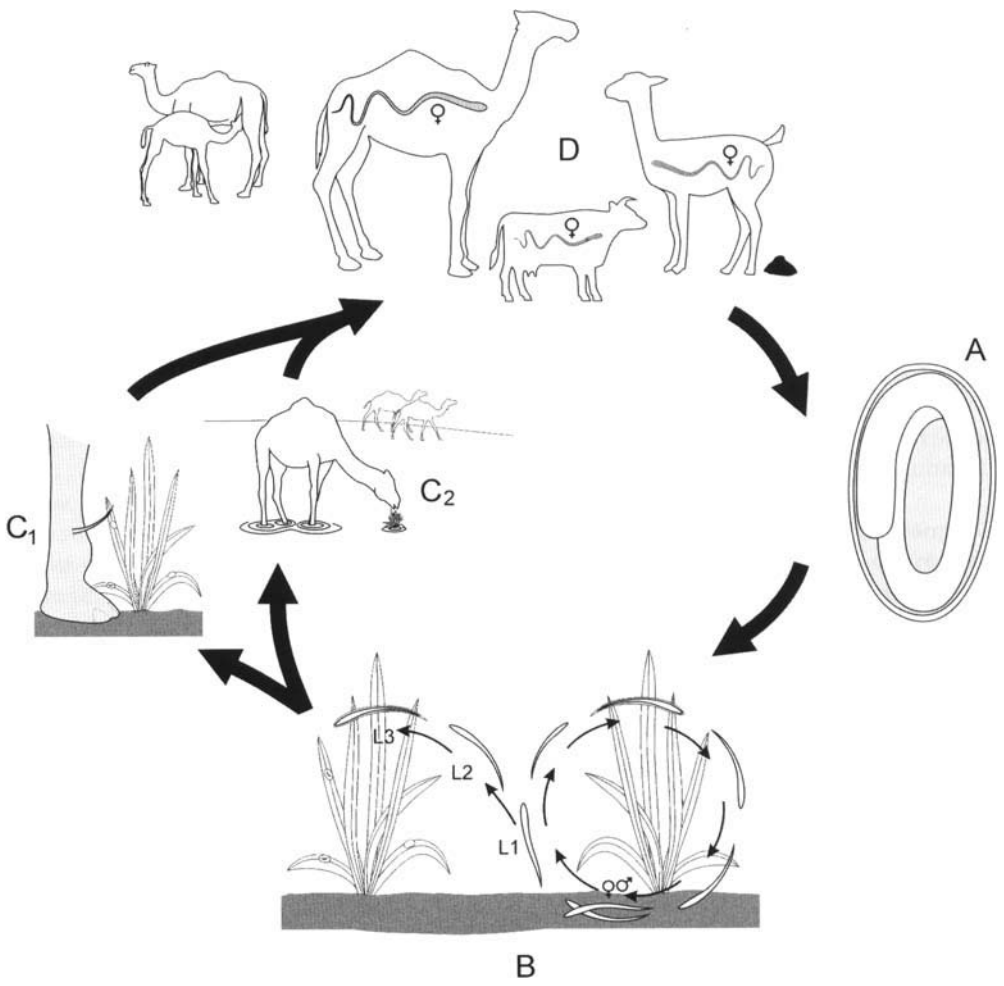

Figure 164 Life cycle of *Strongyloides* sp.: A = egg; B = free-living life cycle (right) and infective L3 (left); C_1 = transcutaneous infection by L3; C_2 = oral infection on pasture; D = final hosts with adult partenogenic female parasites in the gut, infection of calves while suckling

5.3.8 Oxyuridosis (Pinworm Infection)

Order Oxyurida
Family Oxyuridae
Skrjabinema ovis (NWC)

The sheep pinworm, *Skrjabinema ovis*, has been found in the guanaco in Argentina (Fowler, 1998). It is a small parasite measuring between 3 to 8 mm. The eggs are flattened on one side.

Life Cycle The LC is direct. Adults live in the colon and migrate to the rectum from where the female traverses the anal sphincter to deposit her fully embryonated eggs around the anus. The eggs drop off and are ingested with water and food. The L3 hatch in the small intestine and the larvae migrate to the large intestine where the parasites mature within 25 days (Schad, 1959).

Epidemiology The worm has also been found in goats and antelopes. It is considered a benign parasite, although it might cause irritation and pruritus in and around the anus.

Diagnosis The eggs are usually not seen in the regular fecal flotation analysis. Scotch tape applied around the anus and then applied to a glass slide is the recommended diagnostic method.

5.3.9 Trichuriosis (Whipworm Infection) Capillariosis

Order Enoplida
Family Trichuridae
Trichuris tenuis (NWC)
T. ovis (NWC, OWC)
T. globulosa (OWC)
T. affinus (OWC)
T. raoi (OWC)
T. skrjabini (OWC)
T. cameli (OWC)

Family Capillariidae
Capillaria spp. (NWC)

Nematodes belonging to the genus *Trichuris* are common parasites of several mammals, particularly ruminants. Several species are found in camelids and some authors consider *Trichuris* spp. significant parasites in these animal species (Boyce et al., 1984; Hayat et al., 1998; Partani et al., 1998). *T. globulosa* is the most prevalent in dromedaries in Africa and Asia (Kaufmann, 1996). However, as the different species of *Trichuris* are difficult to distinguish, most authors do not identify the particular species. Other *Trichuris* spp. have occasionally been reported to occur in camels: *T. ovis, T. cameli, T. raoi, T. skrjabini* and *T. affinus* (Kaufmann, 1996).

T. ovis is considered to be the species affecting NWC in South America (Fowler, 1998) while *T. tenuis* has been more frequently reported in animals in the northwest Pacific regions (Rickard and Bishop, 1991 a, b). The latter worm was suggested to be the typical whipworm of the llamas (Rickard and Bishop, 1991 b; Rickard, 1994). Recently *T. tenuis* was found during a survey in llamas and vicuñas in northwestern Argentina (Cafrune et al., 1999). Whipworm eggs were found in over 50% of the animals surveyed and *T. tenuis* was demonstrated during necropsy of one llama and one vicuña in each of the herds. The authors were convinced that *T. tenuis* is closely associated with camelids (Cafrune et al., 1999).

Trichuris spp. are between 3 to 8 cm long and are easily identified by the long and much thinner anterior body portion that becomes shorter and thicker posteriorly.

Life Cycle The LC is direct. On pasture, the eggs may reach the infective stage after 3 weeks (Soulsby, 1982). However, devel-

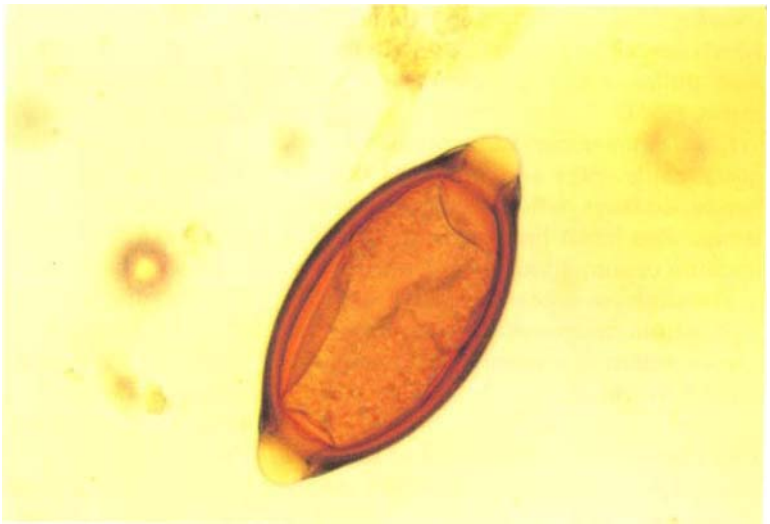

Figure 165 *Trichuris* spp. egg in dromedary feces

opment may be prolonged, depending on the soil moisture and temperature. The ingested eggs hatch and the larvae penetrate the wall of the anterior small intestine of the host and migrate after 2 to 10 days of maturation to the cecum and large intestine, where they develop into adults. The prepatent period varies between species from 50 to 80 days.

Clinical Signs *Trichuris* infections may be benign but high numbers of L5 and adults may cause irritation and inflammation of the cecum and colon, which can result in malfunction of water absorption and dehydration. The parasites traumatize vessels in the mucosa, producing catarrhal enteritis, and can cause hemorrhage. Clinical signs may occasionally be similar to haemonchosis.

Diagnosis Barrel-shaped doubly operculated eggs are characteristic, but may be confused with those of *Capillaria* spp. (Fig. 165).

Treatment Modern anthelmintics are effective against adult *Trichuris* spp. but less so against larval stages.

Capillaria

The capillarids are closely related to *Trichuris*, but are small and thin. The genus contains numerous species found in a variety of hosts. Eggs identified as *Capillaria* spp. have been found in NWC (Fowler, 1998).

5.3.10 Gongylonemosis Parabronemosis Thelaziosis

Order Spirurida

Family Gongylonematidae
Gongylonema pulchrum (NWC, OWC)
G. verrucosum (OWC)

Family Habronematidae
Parabronema skrjabini (OWC)

Family Thelaziidae
Thelazia californiensis (NWC)
T. leesei (OWC)
T. rhodesi (OWC)

Gongylonemosis

The cattle "gullet worm", *Gongylonema pulchrum*, has been reported in alpacas in Peru

(Fowler, 1998) as well as in dromedaries, which are rarely infected also with the rumen gullet worm *G. verrucosum* (Kaufmann, 1996).

G. pulchrum occurs more often in sheep, goats, cattle, pigs and buffaloes than in horses, donkeys, wild boars and camelids. It may also infect humans in the oral epithelium or sometimes subcutaneously.

The adult parasite is found in the esophagus within the mucosa or submucosa in a zigzag pattern. In ruminants, it may also inhabit the rumen wall.

Life Cycle Eggs are passed in the feces and hatch when ingested by an intermediate host, one of 70 different species of beetles, including cockroaches. The larvae are liberated in the stomach of the final host and migrate to the esophagus.

Clinical Signs The infection is usually subclinical and diagnosis is made by chance during necropsy.

Parabronemosis

Parabronema skrjabini is rarely found in dromedaries, sheep, goats and cattle in the abomasum and C3.

Life Cycle *Stomyoxys* and *Haematobia* flies are intermediate hosts that deposit the infective L3 on the final host, which in turn ingests it.

Thelaziosis

Thelazia spp. are 1 to 2 cm long thin parasites mainly found in or around the eyes of numerous animals (as well as humans).

Thelazia californiensis has been found in llamas' conjunctival sac (Fowler, 1998), and *T. leesei* is considered to be the dromedary eye worm (Drobynin, 1974; Kaufmann, 1996). *T. leesi* is reported to occur in Africa and Asia. Also *T. rhodesi*, a species usually found in cattle, has occasionally been found in camels.

The *Thelazia* spp. are ovoviviparous. The adult female parasite deposits eggs containing L1 into the host's lachrymal secretions, which are taken up by the feeding intermediate host: mainly different species of *Musca* flies. Development to L3 occurs in the ovaries of various flies within a month. The infective larvae then migrate to the mouthparts of the fly from which they are transmitted to a new host. This occurs when the intermediate host takes a meal from secretions of the eye of the host.

Life Cycle (Fig. 166) The development of larval stages to maturity takes place in the conjunctival sac. The L3 of *T. leesei* in dromedaries penetrates the conjunctival sac and from there migrates into the lacrimal duct, where the final development to adult worm occurs (Dobrynin, 1974). Adult worms may also be found on the cornea.

Clinical Signs One or both eyes may often be infected without clinical signs. Occasionally the infection may cause irritation, resulting in lacrimation with mild conjunctivitis, which may progress to keratitis. In severe cases the whole cornea may be opaque. Flies may often be seen clustering around affected eyes.

Diagnosis Diagnosis is based on finding the parasites in the eye, usually in the lacrimal duct. Local anesthetics are a useful help in demonstrating the worm. Eggs or L1 may be found in lacrimal secretions.

Treatment The parasites may be physically removed using topical anesthetic drops. Ivermectin drops or diethylcarbamazine (2 mg/L) may also be instilled into the conjunctival sac (Kaufmann, 1996).

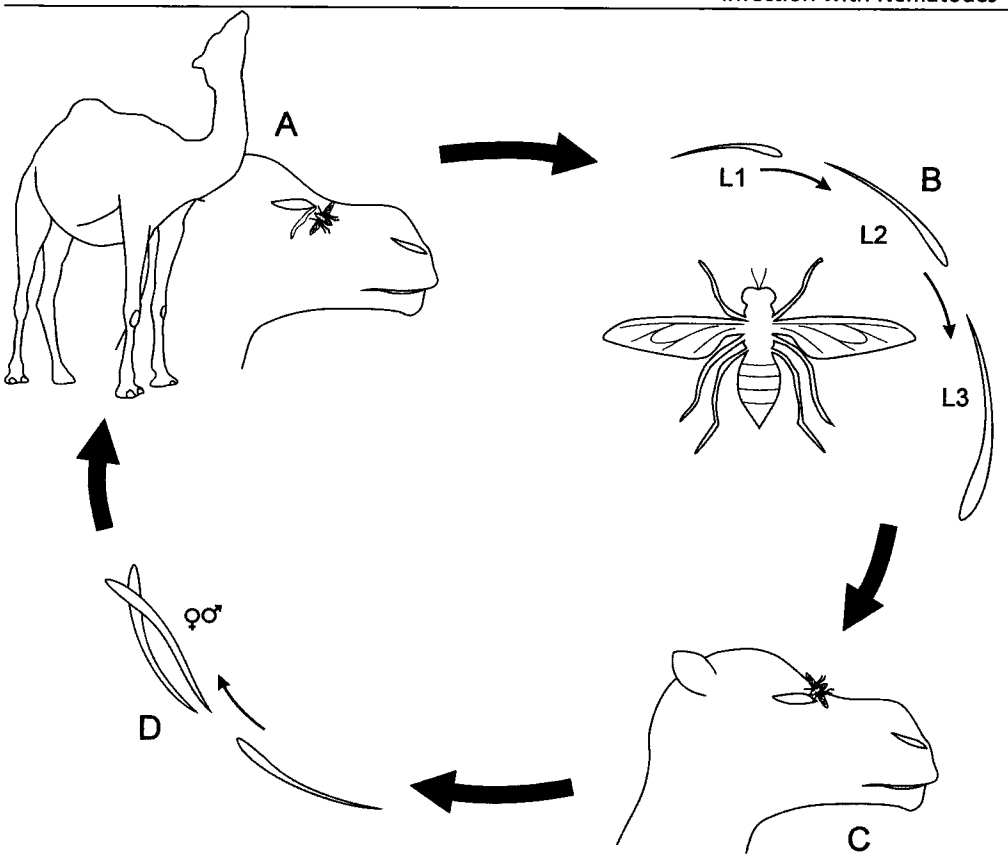

Figure 166 Life cycle of *Thelazia* spp.: A = a fly ingests eye secretions together with eggs and/or L1 of *Thelazia*; B = development of larvae L1 to L3; the infective L3 in the vector fly; C = the infected fly ingests eye secretions and simultaneously infects the animal with L3; D = development of the larvae to adults in the infected host

5.3.11 Onchocercidosis

Order Filariida
Family Onchocercidae
Dipetalonema evansi (OWC)
Onchocerca armillata (OWC)
Onchocerca fasciata (OWC)
Onchocerca gutturosa (OWC, NWC)

Life Cycle Onchocercidae have an indirect LC depending on insect vectors as intermediate hosts. The parasites are long and relatively thin and live in blood or lymph vessels, connective tissues or body cavities of their hosts.

The L1 are called microfilariae. Some are enclosed in a thin membrane, a flexible eggshell. The microfilariae reach the blood or tissue lymph spaces from where the intermediate hosts (mosquitoes and other arthropods) feed and become infected. Microfilariae of some species only appear in the final host's circulation or tissues at certain periods during either the day or the night. They are diurnal or nocturnal. This phenomenon is important in reaching a proper diagnosis.

In the intermediate host, the L1 develops to L3 and migrates to the proboscis of the arthropod vector from where the L3 may be transmitted to the final host.

Dipetalonema evansi

Dipetalonema evansi is a filarid nematode only found in camels. It occurs in the pulmonary and spermatic arteries as well as in the lymph nodes and lymph vessels. The parasites have been reported in dromedaries in Egypt, the Far East and eastern parts of the former USSR (Soulsby, 1982). According to Kaufmann (1996), the parasites are also found in other parts of North and East Africa and in Pakistan and India (Butt et al., 1998). The prevalence of infection may reach 80% in certain areas of Russia and is reported to be approximately 15% in dromedaries of Rajasthan in India (Pathak et al., 1998), and about the same prevalence in Pakistan when direct diagnostic methods are employed (Butt et al., 1998). They only appear in the blood stream around midnight (Michael and Saleh, 1977). The vectors are *Aedes* spp.

Diagnosis Light infections are often clinically not apparent, but heavy infections may cause emaciation, apathy and sometimes orchitis and aneurysms in the spermatic cord as well as arteriosclerosis and heart insufficiency.

Trypanosomosis may be confused with *D. evansi* infection (Kaufmann, 1996). Demonstrating the microfilariae in blood smears or finding the adult parasites during surgery or necropsy confirms diagnosis. For diagnosis, blood samples should only be taken around midnight, considering the nocturnal periodicity of the microfilariae (Fig. 167). The number of microfilariae in circulation is often too small for easy detection and therefore concentration techniques may be used for diagnosis.

Michael and Saleh (1977) described a slide agglutination test for *D. evansi* in camels, and Butt et al. (1998) recommended a formol gel test as being the most reliable of the indirect tests.

Onchocerca

The *Onchocerca* spp. usually cause the formation of nodules in the connective tissue of their final hosts. The parasites are 2 to 6 cm long, thin and slender, and lie tightly coiled in nodules. The intermediate hosts are insects belonging to the families Simuliidae and Ceratopogonidae (*Culicoides* spp.). Most of the parasites are relatively harmless.

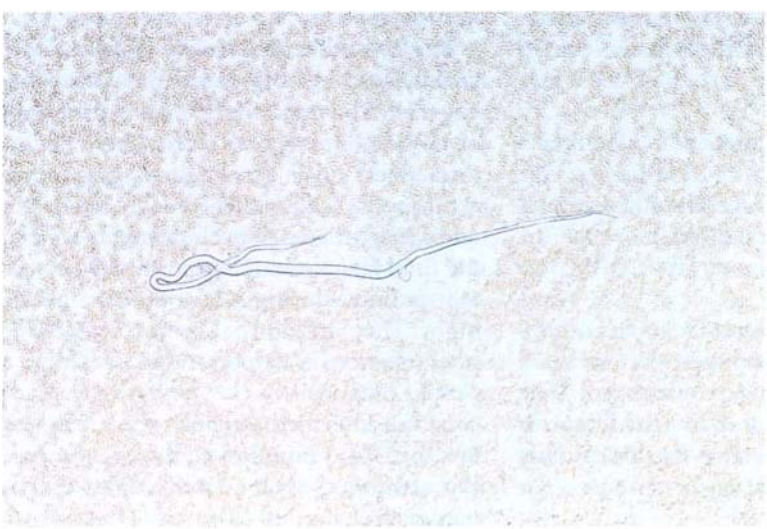

Figure 167 The microfilarian *Dipetalonema evansi* in dromedary blood

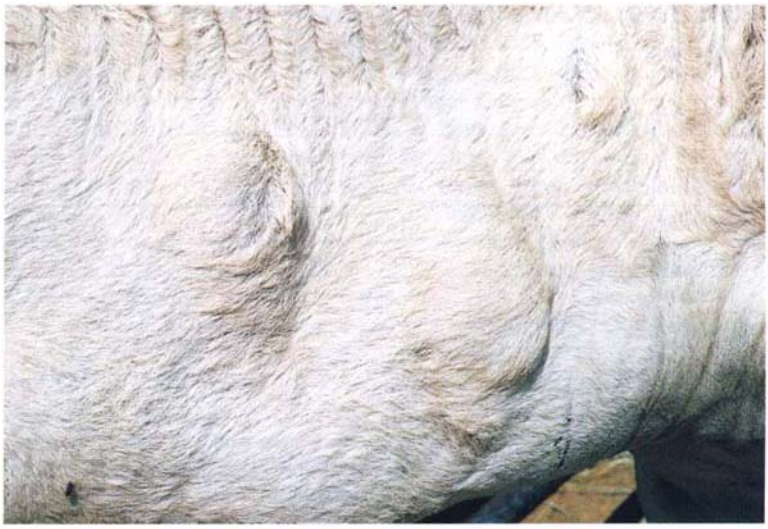

Figure 168 *Onchocerca fasciata* nodules in a dromedary camel

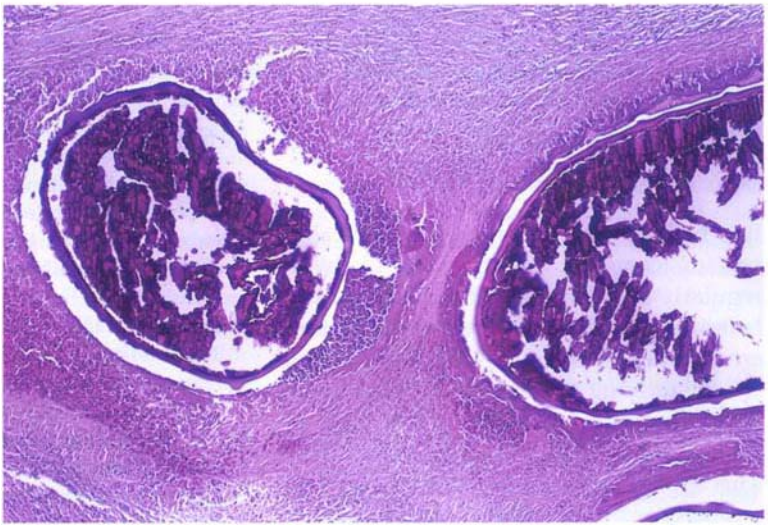

Figure 169 Histological preparation of *O. fasciata* in a dromedary (HE stain)

Onchocerca armillata has been found in the aorta of dromedaries in Nigeria (Kaufmann, 1996). This filariid develops in the aorta particularly in cattle, buffalo, sheep and goats. It has been reported in donkeys in Africa and Asia (Soulsby, 1982). The infection is difficult to diagnose and usually does not cause any clinical signs, although its prevalence in cattle may reach 90%. During slaughter, the worms are often found in nodules in the intima, media and adventitia of the aorta.

Other *Onchocerca* species found in camels are *O. fasciata* and *O. gutturosa*. The former is only found in dromedaries and has been reported in Sudan, Ethiopia, Kenya and Mauritania (Kaufmann, 1996). It occurs in subcutaneous tissue and the nuchal ligament (Figs. 168 and 169). Vectors are *Simulium* spp. flies.

Table 61 Nematocidal anthelmintics for OWC and NWC

Anthelmintic	Administration	Dose mg/kg	
		OWC	NWC
Fenbendazole	orally, 1–3 days	5–7.5	5–8
Febantel	orally, 1 day	5–7.5	5–8
Netobimin	orally, 1 day	12.5	8
Albendazole	orally, 1 day	5–7.5	5–8
Oxfendazole	orally, 1 day	5	5
Thiabendazole	orally, 1–3 days	100–150	50–100
Mebendazole	orally, 1–3 days	5	22
Levamisole	orally, 1 day	7.5	5–8
	s.c.	5	5–8
	spot on	10	–
Pyrantel pamoate	orally, 1 day	25	18
Ivermectin	orally, 1 day	0.2	0.2
	s.c.	0.2	0.2
	spot on	0.5	0.5
Doramectin	s.c. or i.m.	0.2	0.2
Moxidectin	orally, 1 day	0.4	0.4
	spot on	0.5	0.5

5.3.12 Treatment of Nematode Infections

It is believed that of all the domesticated species, camelids are the least likely to regularly suffer from outbreaks of clinical helminthosis due to their natural arid environment. However, one is surprised when studying the extensive list of parasites. Therefore, vigilance in the form of regular monitoring should be maintained. This applies particularly to breeding herds.

There are broad-spectrum antihelmintics that have high efficacy against both larvae and adult nematodes. A single host may be infected by several species of parasites, not all of which have the same sensitivity to the particular anthelmintic drug used. Larvae or immature stages are generally not as sensitive to the drug as adults. Nematocidal anthelmintics are listed in Table 61.

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5.4 Infection with Cestodes (Tapeworms)

Larval and adult cestodes parasitizing in OWC and NWC and their organ localization are listed in Table 62.

Table 62 Cestodes and trematodes of Old World and New World Camelids

Family	Species	Larval stage	Adult parasite	Location of Immature parasite	Occurrence OWC	Occurrence NWC
Taeniidae	*Echinococcus granulosus*	*Echinococcus hydatidosus*	Intestine carnivores	Lung, Liver	+	+
	Taenia multiceps	*Coenurus cerebralis*	Intestine carnivores	Brain Spinal cord	+	
	Taenia hydatigena	*Cysticercus tenuicollis*	Intestine carnivores	Abdominal cavity, Liver	+	
	Taenia saginata	*Cysticercus bovis*	Intestine humans	Muscle	+	
	Taenia hyaenae	*Cysticercus dromedarii*	Intestine carnivores	Muscle, Liver	+	
Anoplocephalidae (Tape worm)	*Moniezia expansa*	–	Small intestine	Small intestine	+	+
	M. benedeni	–	Small intestine	–	+	+
Avitellinidae	*Stilesia* spp.	–	Small intestine	–	+	
	Avitellina woodlandii	–	Small intestine	–	+	
	Thyzaniezia sp.		Small intestine	–	+	+
Fasciolidae (Liver fluke)	*Fasciola hepatica*	–	Bile ducts	Small intestine	+	+
	F. gigantica	–	Bile ducts	Small intestine	+	
	Fascioloides magna	–	Bile ducts	Small intestine	+	+
	Dicrocoelium dendriticum	–	Bile ducts	Peritoneum, liver	+	+
Paramphistomatidae	*Paramphistomum* sp.	–	Forestomachs	Small intestine	+	
Schistomatidae	*Schistosoma* sp.	–	Portal vein in liver, mesenteric vein	Heart, lung	+	

5.4.1 Classification of Cestodes

Phylum Platyhelmintha (Flatworms)
Class Cestodea
Subclass Eucestodia
Order Cyclophyllida
Family Taeniidae
Hydatids of *Echinococcus granulosus* (OWC)
Coenurus cerebralis, cysts of *Taenia multiceps* (OWC)
Cysticercus tenuicollis, cysts of *Taenia hydatigena* (OWC, NWC)
Larval stage of *T. helicometra* (NWC)
Cysticercus bovis, larval stage of *T. saginata* (OWC, NWC)
Cysticercus dromedarii, larval stage of *T. hyaenae* (OWC)

Family Anoplocephalidae
Moniezia expansa (OWC, NWC)
Moniezia benedeni (OWC, NWC)

Family Avitellinidae
Stilesia centripunctata (OWC)
S. globipunctata (OWC)
S. vittata (OWC)
Avitellina woodlandi (OWC)
Thyzaniezia sp. (NWC)
T. ovilla (OWC)

5.4.2 Tapeworm Infection

Tapeworms have an elongated flat body without alimentary canal or body cavity. They are hermaphroditic and the body is segmented. Each segment or proglottid contains one or two sets of male and female reproductive organs, which are formed at the neck – the growth region of the worm. These proglottids mature as they are pushed further away from the head or scolex, and the fully gravid proglottid eventually contains a residual of branched uterus packed with eggs.

The typical LC of the cestodes belonging to the order Cyclophyllidea is indirect, containing one intermediate host. Adult tapeworms are found in the small intestine of the final host, in which the prepatent period lasts between 40 to 50 days. Once the egg is ingested by the intermediate host, it finds its way to its predilection site where it develops to a larval stage named metacestode. The metacestodes have different forms depending on the species. Some of these larval stages are characterized as:

Cysticercus
- A fluid-filled cyst containing one invaginated scolex.

Coenurus
- A metacestode larva similar to cysticercus, but containing numerous invaginated scolices.

Hydatid cyst
- Another metacestode larva consisting of a large cyst filled with fluid. The germinal epithelium lining the cyst produces invaginated scolices, brood capsules.

When the final host ingests the intermediate host containing the metacestode, the scolex attaches to the mucosa of the small intestine. Proglottids start to grow from the base of the scolex. The main families found parasitizing camelids are the Taeniidae and Anoplocephalidae.

5.4.2.1 Cestode Larvae in Internal Organs

Taeniidae

Hydatid disease
Hydatids, caused by the tapeworms belonging to the genus *Echinococcus*, are found worldwide in numerous mammalian species, including humans. Echinococcosis is recognized as one of the world's most important zoonoses. Hydatid cysts are frequently found in OWC as well as in NWC, particularly in lungs and liver. Hydatido-

sis is reported to be common in North and East Africa with a prevalence of 31% in Egypt (Hallawani, 1956), 45.4% in Sudan (Saad et al., 1983), 14.8% in Somalia (Macchioni et al., 1987) and 48% in Libya (Ibrahem and Craig, 1998). A lesser prevalence was reported in Central Africa (Dakkak and Ouhelli, 1987). In Nigeria, a prevalence of >57% was found (Dada, 1978). The infection is also common in Asia (Dakkak and Ouhelli, 1987), in Iraq and Iran with a prevalence of 49.1% and 42.8%, respectively (Barbero et al., 1963; Afshar et al., 1971).

The genus Echinococcus contains four species: *E. granulosus, E. multilocularis, E. oligarthrus* and *E. vogeli.*

Life Cycle (Fig. 170) *E. granulosus* lives in the small intestine of wild and domestic canids. It is a small, 6 mm-long parasite with a scolex and three to four proglottids. The terminal gravid proglottid occupies nearly half of the adult worm and often

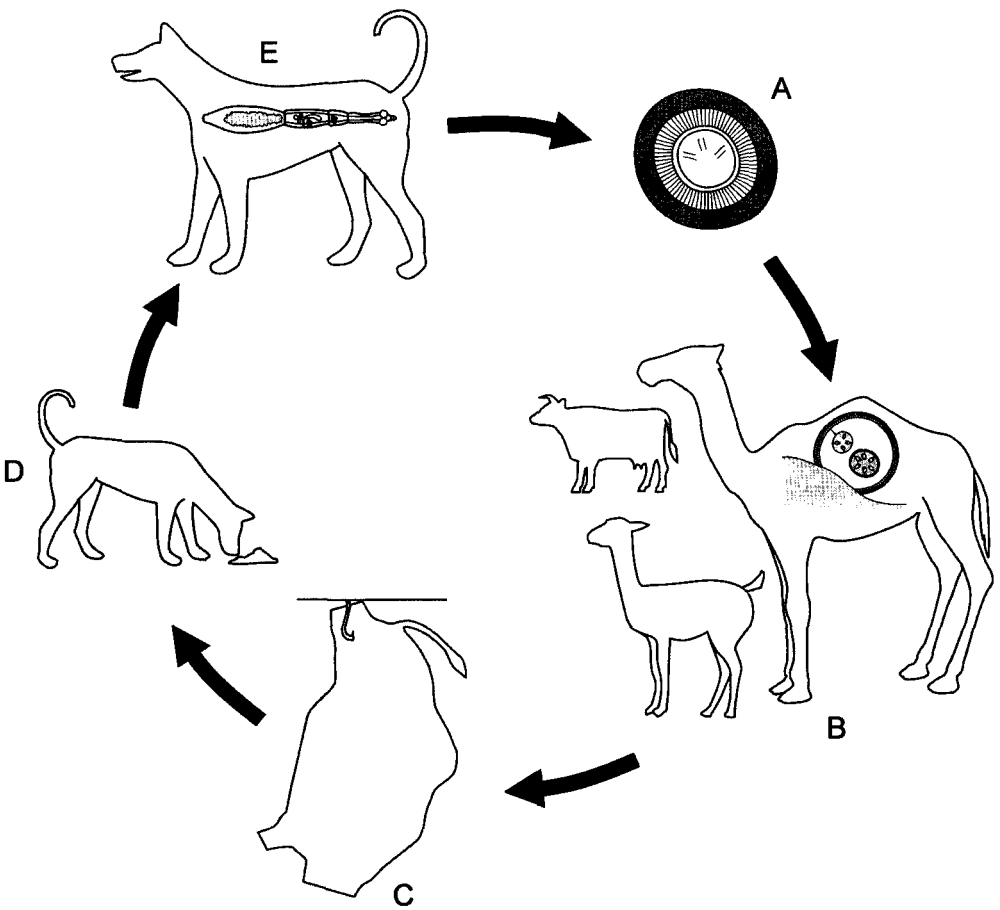

Figure 170 Life cycle of *Echinococcus granulosus*: A = eggs voided in feces; B = intermediate hosts ingest eggs; larval development in the host to hydatid cysts in the lungs and/or liver; C = carcass at slaughter; D = dog becomes infected by eating parasitized offal; E = final host, the dog infected with *Echinococcus granulosus*

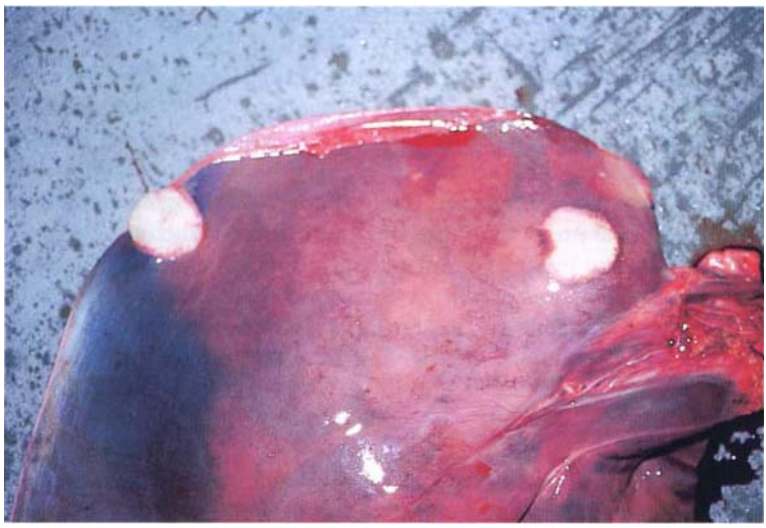

Figure 171 Two hydatid cysts in the liver of a breeding dromedary

disintegrates while still in the gut of the final host, releasing the eggs into the feces. In the canid, adult tapeworms may live up to two years.

The oncospheres within the eggs can survive outside the host for nearly two years. Ingested eggs are immediately infective and hatch in the intestine, releasing the oncospheres that penetrate into venules or lymphatics of the gut wall and migrate to the predilection sites, the liver and lungs. Occasionally oncospheres are found in other organs where they may develop into hydatids.

The cyst, developing slowly and in the lung and liver, may take 6 to 12 months to mature to a diameter of up to 20 cm. In the abdominal cavity, cysts may become very large, sometimes containing several liters of fluid.

The wall of the cyst consists of an outer, relatively thick, concentrically laminated membrane and an inner germinal layer, from which brood capsules each containing a number of scolices or protoscolices (larvae) are budded off. Some of the brood capsules are regularly found floating in the cyst fluid. Complete daughter cysts are sometimes formed and if a cyst ruptures, protoscolices and brood capsules may develop into new external cysts.

The parasite infection in the final host is not pathogenic. In domestic animals, the hydatid cysts in/on the liver and lungs usually cause no clinical signs of disease. Most infections are only revealed during meat inspection at slaughter. However, a variety of clinical signs may occur if cysts have developed at other sites, such as the brain, heart, and kidney. Affected lungs may cause respiratory signs and if one large cyst or several smaller hydatids are present in the liver, abdominal distention may occur. Rupture of hydatid cysts may cause death from anaphylaxis. Additionally, released daughter cysts may spread and develop in other parts of the body. However, some cysts are sterile, depending on the age of the host (Soulsby, 1982).

In camels, the lung is the organ most often affected with hydatids, followed by the liver (Dada and Belino, 1978). Multiple cysts are seen particularly in the lungs. Hydatid cysts are occasionally found in the spleen and are usually solitary (Saad et al., 1983). The number of fertile cysts found is usually higher in the lungs than in other organs. Cysts are often calcified (Fig. 171).

Pathology Gross and histological characteristics of echinococcus cysts in *Camelidae* are similar to those described in other animals (Barker et al., 1993). Some variations in the arrangement of cyst layers were observed histologically by Saad et al. (1983), who found that the cellular infiltration was mainly by lymphocytes, plasma cells and eosinophils (Fig. 172).

Epidemiology There are a number of different strains of *E. granulosus*. They differ in important biological characteristics, including infectivity to humans (Bowles and McManus, 1993). *E. granulosus* of camel origin raised experimentally in dogs was successfully transmitted to goats and sheep, but cattle and donkeys were not susceptible to the infection (Dada et al., 1981).

Various cycles exist between the intermediate and canid final host. These are divided into the pastoral and sylvatic cycles. The dog is always involved in the pastoral cycle, but the domestic intermediate host species may vary: sheep/dog, cattle/dog, OWC/dog, NWC/dog etc. The pastoral cycle is the primary source of hydatid disease in humans. Such infections are caused by accidental ingestion of oncospheres from dog coats or foodstuffs contaminated by dog feces.

In the sylvatic cycle, wild canids are involved: deer/coyote, moose/wolf, wallaby/dingo, NWC/fox and hare/fox. This cycle is less important as a human source of infection. However, in hunting communities the infection may be introduced to domestic dogs by feeding them contaminated viscera from wild animals. The cycle in NWC follows the pattern NWC/dog and NWC/fox (Fowler, 1998).

Public health workers are concerned about the zoonotic risk of hydatidosis in OWC for camel pastoralists. Epidemiological studies have recorded the highest incidence of human hydatid disease in pastoralists in Kenya. However, it has been shown that the *E. granulosus* strain affecting camels in Kenya is different from the sheep and cattle *E. granulosus* strains, and that humans appear resistant to infections by the camel strain (MacPherson and McManus, 1982). However, opinions differ concerning the infectivity of *E. granulosus* from camels to humans (Shaafie et al., 1999).

Diagnosis Hydatids are frequently found during slaughter or necropsy. Infect-

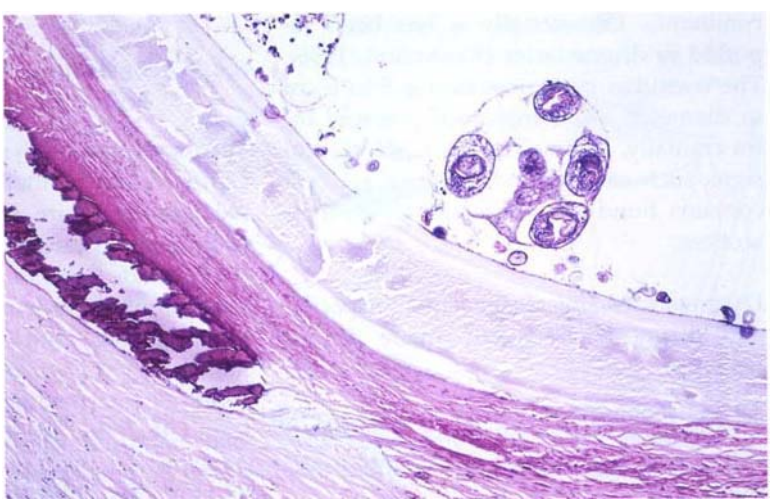

Figure 172 Histology picture (HE staining) of the layers of a hydatid cyst and cut-surface of scolices from a breeding camel

ed dogs pass eggs in their feces that cannot morphologically be distinguished from eggs of *Taenia* spp. The tapeworm may be demonstrated microscopically in the mucous portion of purged material. Immunodiagnostic tests using ELISA are used in human medicine. In addition, radiographic diagnosis is used. Recently, PCR techniques have been available which may identify antigenic material in feces.

Treatment Treatment of domestic animals (intermediate host) is rarely employed in hydatidosis. In endemic areas, anthelmintic treatment of dogs may be used to break the cycle of infection. Praziquantel (5–10 mg/kg per os), bunamidine hydrochloride (25–50 mg/kg per os) and some recent formulations of combinations consisting of both cestocidal and nematocidal components, e.g. febantel/praziquantel/pyrantel, are effective against the adult tapeworms in the dog.

Taenia multiceps – Coenurus cerebralis

The adult tapeworm of *Taenia multiceps* is up to 1 m long and is found in the small intestines of dogs and wild canids. Its larval stage, *Coenurus cerebralis*, is found in the brain or spinal cord of such intermediate hosts as sheep, goats and other ruminants. Occasionally it has been reported in dromedaries (Kaufmann, 1996). The coenurus cysts, measuring 5 to 6 cm in diameter, cause increased pressure intra-cranially, giving rise to CNS clinical signs such as "gid and staggers". A cyst contains hundreds of invaginated protoscolices.

Diagnosis Clinical signs are nonspecific and diagnosis is usually made during necropsy.

Treatment The treatment of *T. multiceps* is the same as described for hydatid infections.

Taenia hydatigena – Cysticercus tenuicollis

Taenia hydatigena, a large tapeworm that may reach 5 m, is found in the small intestine of dogs, wild canids and occasionally other carnivores. The intermediate hosts are ruminants, particularly sheep, but also pigs, alpacas, vicuñas (Fowler, 1998) and dromedaries (Kaufmann, 1996). The oncospheres are carried hematogenously via the blood to the liver. They migrate in the liver parenchyma for about 4 weeks before they emerge to the liver surface and then attach to the peritoneum. Within a further 4 weeks, each larva may develop into a cyst, *Cysticercus tenuicollis*, measuring 6 to 8 cm in diameter.

Each cyst contains one invaginated scolex. During migration in the liver, the larvae cause hemorrhagic tracts that may become fibrotic. If the infection is heavy, the developing cysticerci may cause severe liver damage, with fatal consequences. However, the infection is often asymptomatic. The cysts are occasionally found at slaughter.

Taenia helicometra

Infections with larval stages of *Taenia helicometra* of canids have been reported in alpacas and vicuñas from South America (Fowler, 1998).

5.4.2.2 Cestode Larvae Found in Muscles

Taenia saginata – Cysticercus bovis

Cysticercus bovis, the larval form of the *T. saginata* tapeworm in the small intestine of humans, is rarely found in camels. The metacestode is commonly found in the muscles of cattle worldwide, particularly in Africa and South America. Other ruminants and *Camelidae* may occasionally serve as intermediate hosts. It has been found that the predilection sites for this parasite are the heart, masseter, tongue and muscles of the diaphragm, but the cys-

ticerci may be observed throughout the musculature (Soulsby, 1982).

Life Cycle (in Cattle) The mobile proglottids are shed in the feces. The eggs may stay viable for weeks or months in sewage, in rivers and on pasture. Eggs survive on dry sunny pastures over 14 weeks. When ingested, the eggs release the oncosphere into the small intestine. The oncosphere penetrates the mucosa, reaching the blood circulation, and is disseminated throughout the body into skeletal and heart muscles as well as fat tissue and other organs. The cysticercus *C. bovis* develops and becomes infective in approximately 10 weeks, and will be viable after between 4 and 9 months. Some cysts might stay viable throughout the intermediate host's life, depending upon the degree of infection and the age of the infected animal (Soulsby, 1982).

The cyst is 0.5 cm in size and surrounded by a tissue capsule. Humans, the final hosts, are infected by the ingestion of raw or undercooked infected meat. After approximately 100 days, gravid proglottids will be passed in the stool.

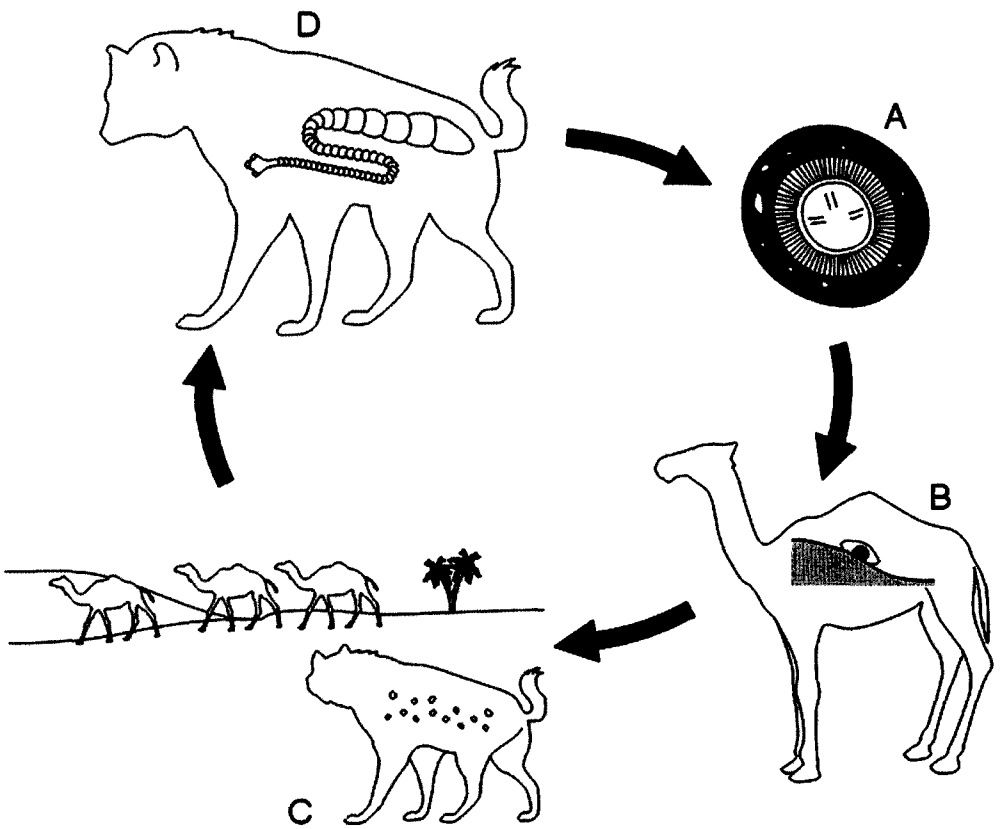

Figure 173 Life cycle of *Taenia hyaenae:* A = eggs voided in feces; B = the intermediate host, the dromedary, has ingested the eggs, which has developed into a *Cysticercus dromedarii;* C = the hyena, the final host, preying on the intermediate host, the dromedary; D = hyena, the final host

Clinical Signs Muscle cysticerci infections are usually not associated with clinical disease.

Public Health Only prevention can break the LC. Rigorous meat inspection should be implemented and the consumption of raw meat should be avoided. In addition, proper disposal of abattoir waste and offal should take place to avoid infestation of carnivores.

Taenia hyaenae – Cysticercus dromedarii

There are a large number of taeniid cestodes with an unknown LC in wild carnivores' small intestine. *Cysticercus dromedarii*, the larvae of *T. hyaenae* (in various species of hyena in Africa), are often found in the muscles of dromedaries, cattle and goats (Kaufmann, 1996). The natural and common intermediate hosts are several species of antelopes. *C. dromedarii* cysts are twice as large as *C. bovis*, 12 to 18 mm in length. Although infected meat is not pathogenic to humans, meat with large numbers of cysts should be destroyed.

Life Cycle The LC is shown in Fig. 173.

5.4.2.3 Cestodes of the Intestine

Anoplocephalidae, Avitellinidae

Seven cestode species are parasites of the small intestine of *Camelidae*. *Moniezia expansa* and *M. benedeni*, both reported in camelids, are common tapeworms in ruminants found worldwide. Both are significant NWC parasites in some areas of South America. *M. expansa* has been found in NWC in the USA and often in OWC in Africa and Asia. *M. benedeni* has only been reported in dromedaries in Africa.

The *Moniezia* spp. are long tapeworms reaching up to 6 m. The heads (scolex) are unarmed with no rostellum, or hooks, but with 4 prominent suckers. The proglottids are broader than they are long. Mature protoglottids release eggs into the feces. The eggs are triangular (Fig. 174). Oribatid mites ingest the oncospheres on the pasture, which develop into cysticercoids within 1 to 4 months. The final host becomes infected by ingestion of the infected mites that contaminate the forage.

Pathogenesis *Moniezia* spp. infections are generally considered to be of little path-

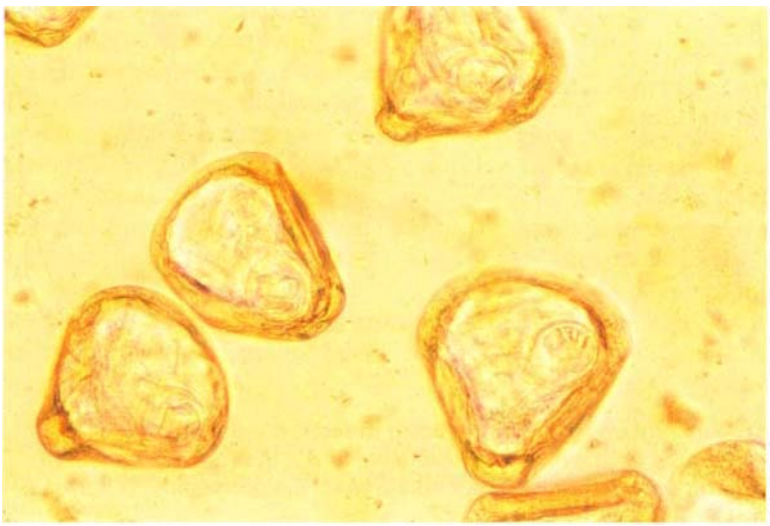

Figure 174 *Moniezia expansa* eggs in dromedary feces

ogenic importance. However, there are indications that heavy infections may impair nutrition and cause diarrhea, debility, and sometimes obstruction of the intestine.

Stilesia

Three *Stilesia* spp. are found in dromedaries. *S. globipunctata* and *S. centripunctata* are widely reported in Africa and Asia, whereas *S. vittata* has only occasionally been observed (Dakkak and Ouhelli, 1987).

S. globipunctata occurs primarily in the small intestine of ruminants in southern Europe, Africa and Asia.

Life Cycle ⁞ Little is known about the LC of *Stilesia* parasites. Oribatid mites may play an important role, as shown by Soulsby (1982) in Chad and India.

The immature worms penetrate the mucosa of the duodenum and jejunum. Nodules are formed with proliferate inflammation and epithelial desquamation (Arnjadi, 1971). The head and the anterior part of the parasite are embedded in the nodule and the posterior proglottids of the worm move freely in the intestinal lumen. The infection may lead to death.

Other tapeworms belonging to Avitellinidae have been reported in camelids. *Avitellina woodlandi* has been found in camels in Africa (Dakkak and Ouhelli, 1987), and according to Kaufmann (1996) *Avitellina centripunctata* is widespread in these animals in Africa and Asia. *Thyzaniezia* species are reported in llamas and *T. ovilla* in camels in Chad (Graber, 1966; Graber et al., 1967). Little is known of the LC and epidemiology of the latter species. They seem to have no pathogenic significance.

Treatment ⁞ Praziquantel has been shown to be effective (15 mg/kg in sheep and goats).

5.5 Infection with Trematodes (Flukes)

Trematodes parasitizing in OWC and NWC and their organ localization are listed in Table 62 (p. 369).

5.5.1 Classification of Trematodes

Superclass Trematoda
Class Digenea
Family Fasciolidae
Fasciola hepatica (OWC, NWC)
F. gigantica (OWC)
Fascioloides magna (NWC)
Eurytrema pancreaticum (OWC)
Dicrocoelium dendriticum (OWC, NWC)

Family Paramphistomatidae
Paramphistomum spp. (OWC)

Family Schistosomatidae
Schistosoma bovis (OWC)
S. mattheei (OWC)

5.5.2 Trematode Infections

5.5.2.1 Trematodes of the Liver

Most of the helminths parasitizing the liver are trematodes, liver flukes. Four species are found in OWC and two are reported in NWC.

Fasciola hepatica, the common liver fluke, is frequently found in dromedaries in Africa and Asia and in Bactrians in Europe (Dakkak and Ouhelli, 1987) as well as in NWC (Leguia, 1991; Cafrune et al., 1996). *Fasciola gigantica,* the giant liver fluke, is also found in camels in Africa and Asia. *Dicrocoelium dendriticum,* the small liver fluke, is reported in dromedaries in Africa, but is less frequent than *F. hepatica. D. dendriticum* is rarely reported in NWC.

Eurytrema pancreaticum, the fluke that occurs in the pancreas and rarely in the biliary ducts, is rarely found in dromedaries. An immature fluke of *Fascioloides magna,* the large American fluke, a deer parasite in North America, has been found in hepatic cysts of one llama (Conboy et al., 1988).

The bodies of the flukes are generally dorsoventrally flattened and unsegmented, and many are leaf-like. The parasites have suckers for attachment. They are hermaphroditic except for some species of Schistosomatidae.

The adult flukes of the Digenea are oviparous. They lay eggs with a lid (operculum) at one pole and, within the egg, the ciliated larva develops (miracidium). The miracidium must find a suitable snail host within a few hours. In the snail, the miracidium develops to a sporocyst, which develops to the cercariae. They emerge in large numbers from the snail and attach themselves to vegetation and encyst to metacercaria. Encysted metacercariae survive for months and, once ingested by a final host, will hatch in the intestine. As juvenile flukes, they then penetrate the gut wall and migrate to the predilection sites of the host.

Fasciola hepatica and *F. gigantica* – The Large and Giant Liver Fluke

The two most important ruminant liver flukes are *Fasciola hepatica* and *F. gigantica.* The former is found in temperate and cool areas of high altitude in the tropics and subtropics. The latter predominates in tropical regions.

Fasciola hepatica
Fasciola hepatica, the common large liver fluke, is found as adults in the bile ducts of a number of mammals, particularly sheep, cattle and other ruminants, but also in several other domestic and wild species, in-

cluding humans and camelids. The fluke is distributed worldwide and causes fasciolosis or liver fluke disease. The disease is characterized by weight loss, anemia and hypoproteinemia. Camelids are sensitive to infections with *F. hepatica*, which may be easily transmitted via wet pastures shared with sheep and cattle.

When young, the 1 to 2 cm-long, lancet-like fluke enters the liver. When fully matured in the bile duct of its final host, it is leaf-shaped and grayish brown in color. Its tegument is armed with backwardly projected sharp spines. An oral and ventral sucker may clearly be seen under a microscope.

Life Cycle The LC is shown in Fig. 175.

There are several factors necessary for outbreaks of fasciolosis. One is the availability of a suitable habitat for the intermediate host, the snail. This host requires a wet environment, including mud or open waters, particularly along banks and edges of small ponds, low-lying swampy areas and continuously irrigated pastures. Spillage from water troughs may also be a suitable habitat for the snail. Another impor-

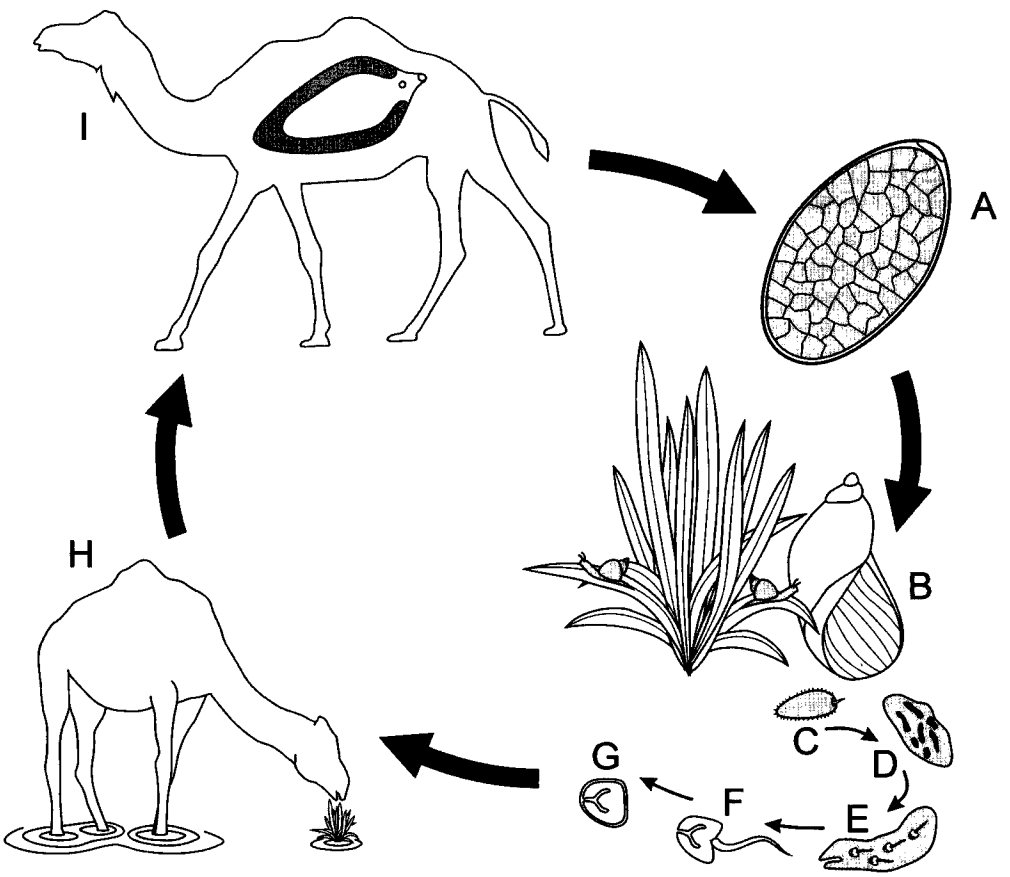

Figure 175 Life cycle of *Fasciola* sp.: A = egg; B = water snail, *Lymnaea* sp.; C = miracidium; D = sporocyst; E = redia; F = cercaria; G = metacercaria, encysted on grass and partly immersed in water; H = final host accidentally ingesting metacercariae; I = final host with adult parasite in liver

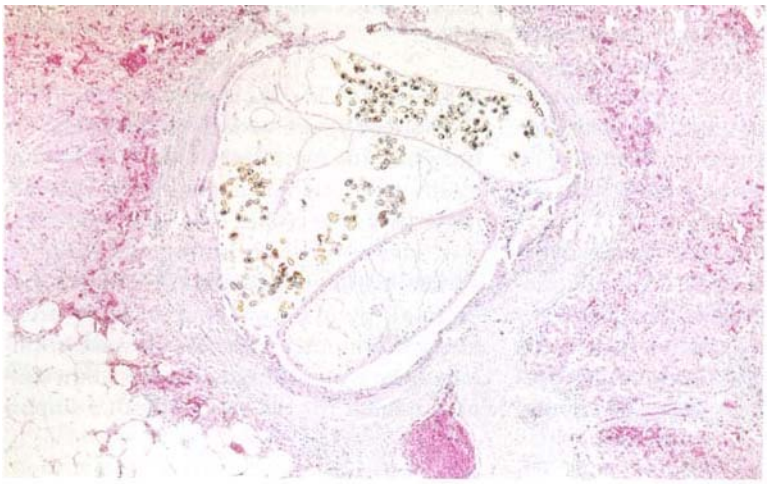

Figure 176 *Fasciola hepatica* infestation in a Bactrian liver (courtesy of Dr. M. Weber, Germany)

tant requirement for the development of the fluke is the optimal temperature. A minimum mean temperature of 10°C is necessary for the snail to breed, for the development of the fluke inside the snail, as well as for the development of the eggs. There is a direct correlation between development time and the temperature.

It is known that llamas and sheep have a low resistance to *F. hepatica* (Rickard and Foreyt, 1992).

Occurrence Several researchers have diagnosed fasciolosis in NWC, but it is rare in OWC.

The prevalence of *F. hepatica* and infections in OWC is relatively low and the infection is benign. Thickening of the bile ducts may occur, resulting in partial or total condemnation of affected livers at meat inspection. Magzoub (1988) reported a fasciolosis prevalence of 92% in camels in Sudan. In Saudi Arabia, Magzoub and Kassim (1978) found a relatively high prevalence of 10.43%, particularly in the Eastern Province. This unexpectedly high prevalence was attributed to either high rainfall or areas with irrigated agriculture. Examination of the feces from 283 dromedaries in Iraq employing a sedimentation method also revealed a relatively high infection rate of *Fasciola* spp. (Al-Khalidi et al., 1990).

The prevalence of fasciolosis in vicuñas was 10 to 18.6% in Argentina (Cafrune et al., 1996) and 8% in alpacas and llamas in Peru (Leguia, 1991), while in alpacas from Bolivia it was over 51% (Cafrune et al., 1996). In llamas in Oregon, USA, a prevalence of 1 to 6% was recorded (Rickard and Bishop, 1991), but a dot-ELISA detected antibodies to *F. hepatica* in 16% of the tested llamas (Rickard, 1995).

Pathology Both the acute and the chronic forms of fasciolosis have been observed in camelids.

The acute disease is associated with liver damage and hemorrhages caused by the parasite's migration to the liver parenchyma. Leguia (1991) has described acute infections in NWC with mortalities reaching 100%.

In the chronic disease, the fluke living in the bile ducts damages the mucosa of the ducts with its cuticular spines (Fig. 176). Continuous stasis of the bile results in hepatic fibrosis, which eventually leads to the elevation of the intrahepatic blood pressure. Leakage of plasma proteins due to cholangitis causes hypoproteinemia. The

compromised fluke-infected liver also may be susceptible to secondary bacterial infections, as is the case in some ruminants.

Fasciolosis is a zoonotic disease and is becoming increasingly important in humans: the Peruvian Sierras have infection rates of 15 to 25% (Leguia, 1991).

Clinical Signs Fasciolosis in camelids is generally subclinical. Acute fasciolosis is less frequent than the chronic manifestation and is associated with liver insufficiency. Animals with the chronic form become anemic and anorectic. Edema may be seen, particularly in the submandibular regions. The milk yield is reduced, and the wool becomes brittle and breaks easily. Diarrhea and/or constipation may occur. Depression and emaciation follow.

Diagnosis Diagnosis is mainly based on clinical signs, seasonal occurrence and climatic conditions. A previous history of fasciolosis on the premises and/or identification of the snail or snail habitat are helpful. Examination of feces for egg identification is also important. However, infections cannot be diagnosed during the period prior to fluke maturation, which lasts for 8 to 12 weeks following infection. The infection is often first recognized at meat inspection after slaughter. Serological tests are employed in research. A dot-ELISA was developed to detect antibodies to *F. hepatica* antigens in llamas (Rickard, 1995). The assay detected specific antibodies during the second week following experimental infections.

Treatment Flukicides are used therapeutically or prophylactically. For the treatment of acute fasciolosis it is important to use a product that is particularly effective against the juvenile parasites that damage the liver parenchyma. For the chronic disease the chosen compound should be effective against adult flukes. The following drugs may be tried against trematodes in camelids:

Triclabendazole is the drug of choice for outbreaks of acute disease (Roberts and Suhardono, 1996); the dose is 10 mg/kg for sheep and 12 mg/kg for cattle given orally. It is effective against all stages of fluke infection. It is also effective in alpacas (Leguia, 1991) and llamas (Duff et al., 1999).

Albendazole has a broad-spectrum activity. It is effective against adult *F. hepatica* in sheep (7.5 mg/kg per os) and in cattle (10 mg/kg per os). It is also ovicidal and kills the eggs present in the bile ducts and in the gut.

Netobimin is metabolized into albendazole and has a similar activity against *F. hepatica*. The dosage for sheep and cattle is 20 mg/kg per os.

Closantel kills most of *F. hepatica* in sheep at a dose of 10 mg/kg per os. It can also be administered s.c. at a dosage of 5 mg/kg.

Clorsulon is available in combination with ivermectin. Clorsulon (2 mg/kg s.c. or 7 mg/kg per os) is effective against adult and 12 to 14-week-old immature flukes in cattle.

Nitroxynil has good effect against adult flukes at a dose of 10 mg/kg, s.c., but the dose must be increased by 50% in acute disease.

Oxyclozanide is used in cattle. It has a shorter milk withholding time than most other flukicides and is only effective against adult flukes. The compound is also available in combination with levamizole. The dosage is 15 mg/kg per os in sheep and 17 mg/kg in cattle.

Fasciola gigantica

The giant liver fluke, *F. gigantica*, is the common liver fluke of African domestic stock. It is frequently found in Asia, the Pa-

cific Islands, southern USA, southern Europe and the Middle East. The epidemiology is similar to *F. hepatica* but it is larger. It can reach 7.5 cm in length.

Life Cycle The intermediate hosts are snails belonging to the genus *Lymnaea* and are primarily aquatic, found in streams, irrigation channels and wet and swampy areas. The LC is similar to *F. hepatica* with the exception that the different stages and the total cycle are longer. The prepatent period is 13 to 16 weeks.

Fascioloides magna – The Large American Liver Fluke

Fascioloides magna, the large American liver fluke, is a large liver fluke mainly parasitizing moose and deer in North America,

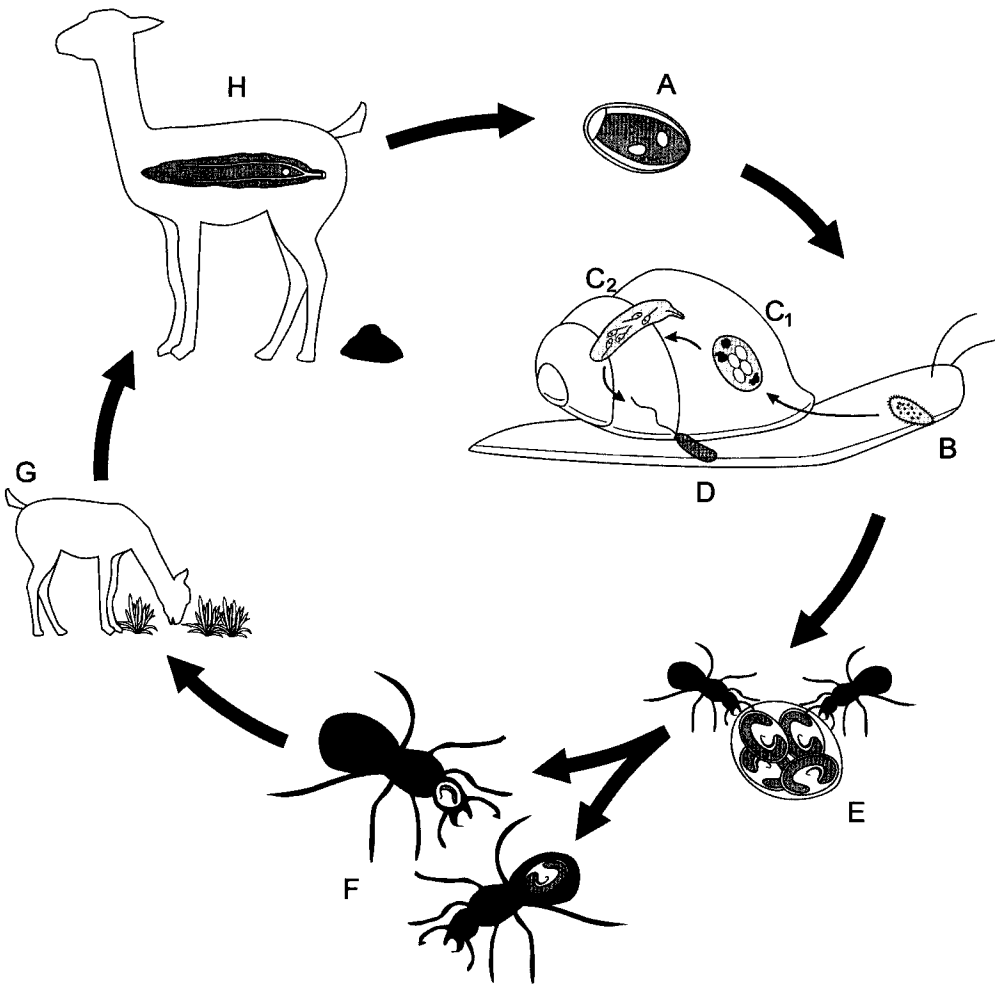

Figure 177 Life cycle of *Dicrocoelium dendriticum:* A = egg; B = intermediate host, land snail ingesting embryonated egg; C_1 = sporocyst; C_2 = daughter sporocyst; D = cercariae released by the snail in clusters; E = cercariae eaten by ants *(Formica)*; F = development of metacercariae in infected ants, second intermediate host; G = final host accidentally ingesting infected ants; H = final host with adult parasite

but also occurring in some European countries. Cattle, sheep, goats and camelids grazing on the same pastures as the infected wild animals may contract the infection.

Life Cycle The LC is similar to that of *F. hepatica*. Several different lymnaeid snails act as intermediate hosts. The final host is infected by ingesting the metacercariae. After 4 weeks the fluke reaches the liver, in which it becomes encapsulated. A free passage between the thin-walled capsule and the bile ducts is maintained through which eggs are passed into the bile and the feces. Each capsule contains one to three flukes. The prepatent period is 30 to 32 weeks.

Dicrocoelium spp. – The Small Liver Flukes or the Lancet Flukes

The small liver flukes or the lancet flukes are rarely found in camelids. However, natural infections with *D. dendriticum* were recently detected in 5 llamas and 2 alpacas in Switzerland southern Germany (Wenker et al., 1998). An experimental infection of the parasite in llamas was described earlier (Gevrey, 1989). *D. dendriticum* is a common parasite of small and large domestic ruminants and is a relatively small fluke, 6 to 10 mm long, with operculate and dark brown eggs. It is particularly widespread in Europe and Asia.

Life Cycle The LC of this small liver fluke differs in significant aspects from that of *F. hepatica*. It involves two intermediate hosts. The eggs are passed in the feces and eaten by land snails. Ants of the genera *Formica* and *Lasius* eat the cercariae, hiding in slime balls. Grazing animals ingest infected ants containing metacercariae. The immature flukes migrate from the gut via the Ductus choledochus into the biliary system where they settle (Fig. 177).

Clinical Signs and Pathology The infection is often subclinical. Heavy infections may manifest as weight loss, general malaise, anemia and hypoproteinemia. An acute decline in general condition followed by recumbency, decreased body temperature, and varying degrees of anemia were observed in seven naturally infected NWC (Wenker et al., 1998). All animals were in a poor nutritional state. At necropsy, liver cirrhosis, liver abscesses and massive infection with *D. dendriticum* were found. Pathogenicity is usually low.

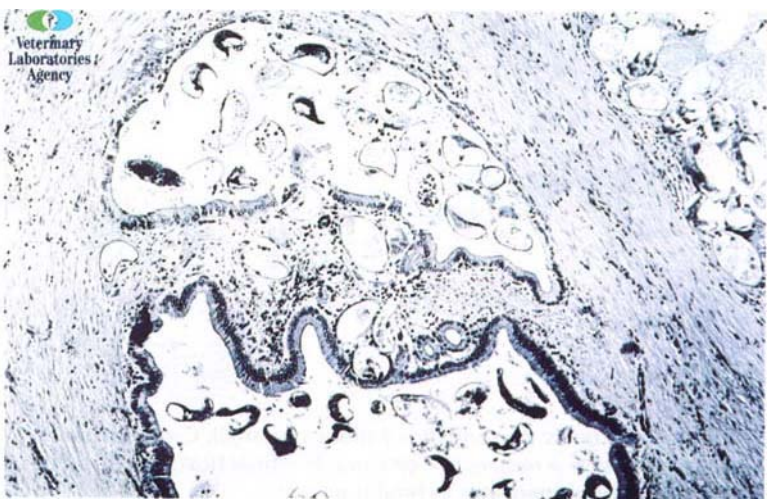

Figure 178 *Dicrocoelium dendriticum* in the biliary system of a llama (© British Crown Copyright. Produced with the permission of the Veterinary Laboratories Agency. Photo – Drs. R. Munro and P. Duff, UK)

However, Gunsser et al. (1999) believe that NWC react more sensitively to this parasite than domestic ruminants. Besides the severe proliferation of the bile ducts, granulomas have been observed in association with *D. dendriticum*. It should be mentioned that NWC show more similarity with the equine bile system than with the bile system of domestic ruminants. The parasites do not migrate through the liver parenchyma like the large liver fluke. However, very heavy infection may cause fibrosis and proliferation and thickening of the small bile ducts. In addition, abscesses and granulomas may be seen in the liver (Wenker et al., 1998) (Fig. 178).

Diagnosis Repeated fecal examinations are necessary to find the characteristic eggs. Clinical signs were associated with the findings of > 1000 eggs/g in feces (Wenker et al., 1998). At necropsy, the small lancet-like flukes are seen in the smaller bile ducts, which may be fibrotic and thickened.

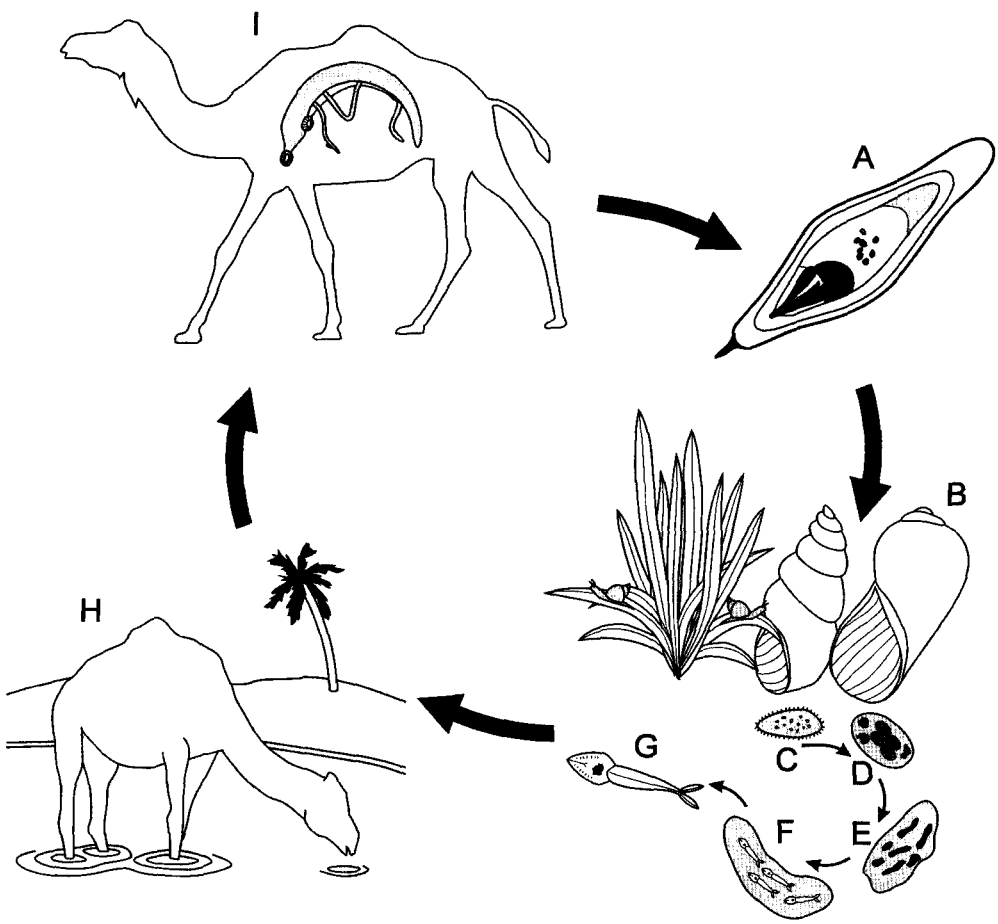

Figure 179 Life cycle of *Schistosoma bovis:* A = egg; B = *Bulinus* spp. snail; C = miracidium; D = sporocyst; E = daughter sporocyst; F = rediae; G = cercaria; H = final hosts are infected while in water; I = development to adult parasites in final host

Treatment Albendazole at a dose rate of 15 mg/kg per os and netobimin at 20 mg/kg per os are effective. Praziquantel at a dose rate of 50 mg/kg per os was well tolerated by a few llamas and alpacas, but achieved only a 90% reduction of eggs in the feces (Wenker et al., 1998).

5.5.2.2 Paramphistomatidae – Rumen Flukes

Different species of rumen flukes, *Paramphistomum*, have been found in camels (Kaufmann, 1996). They are not considered harmful to the host unless there is a massive invasion of immature flukes attached to the intestinal mucosa.

5.5.2.3 Schistosomatidae

Schistosoma bovis and *S. mattheei* causing bilharziosis are rarely found in OWC and are considered occasional parasites. They are not found in NWC.

Schistosomatidae trematodes are dimorphic. They inhabit their hosts' blood vessels. The female worm is slender and in some species longer than the male, which harbors the female in a ventral, gutter-like groove: the gynaecophoric canal. Several species of *Schistosoma* cause severe disease in humans. Animals may act as reservoirs of the infection.

Life Cycle The LC of *Schistosoma bovis* is shown in Fig. 179.

5.6 Infection with Hirudinea (Leeches)

5.6.1 Classification of Hirudinea

Phylum Annelida
Class Hirudinea
Limnatis nilotica

5.6.2 Infection with Leeches

Leeches are occasional parasites feeding on various animals. After their blood meal, they engorge and drop off their host. Leeches have pharyngeal glands from which they secrete an anticoagulant substance when piercing the skin or mucosa for a blood meal. Bleeding may continue for some time after the parasites have engorged.

Eight *Limnatis nilotica* leeches were found attached to the pharyngeal mucosa of a 2-year-old male dromedary in Iraq (Al-Ani and Al-Shareefi, 1995). The camel had difficulty in breathing and exhibited edema of the face and neck. It released snoring sounds, and had difficulty in swallowing food and water.

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Index

Page numbers in **bold type** refer to figures and/or tables.