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CAMEL DERMAL MYCOSES

A DIAGONISTIC PICTORIAL

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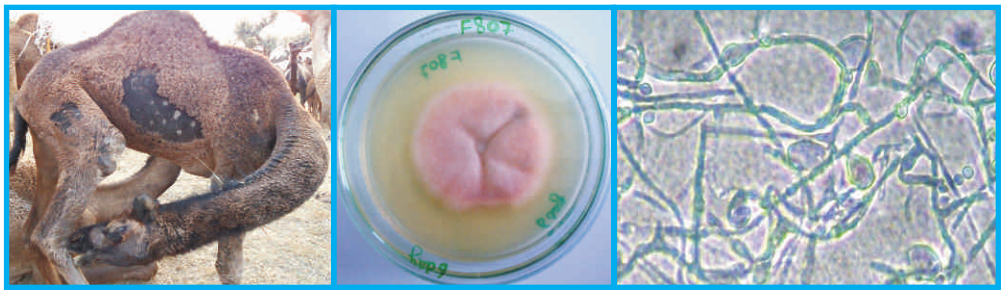

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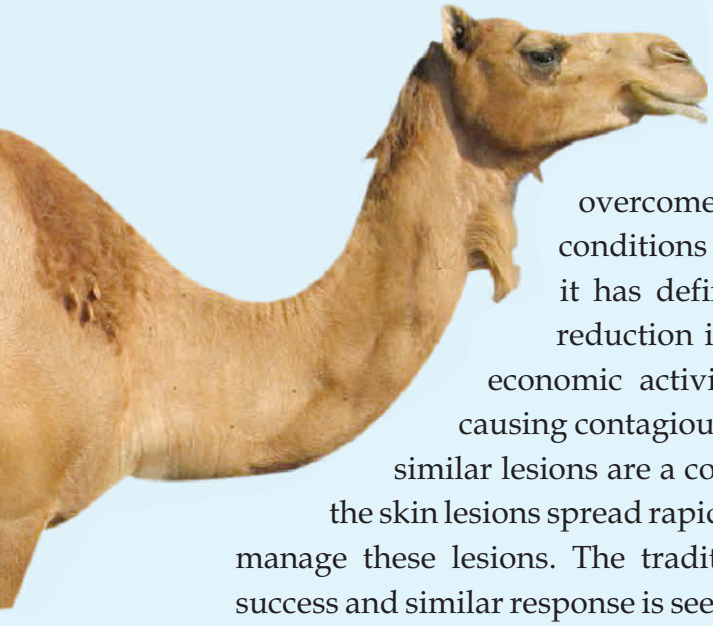
PREFACE



Camel has proven its suitability to not only to survive but also serve as an animal of useful production functions in extreme climatic conditions. Although the Camel has also overcome various biotic stress situations of dry area conditions and do not easily succumb to these situations but it has definitely inflicted a great economical loss due to reduction in draught capacity which still remains a major economic activity for the camel owners. The skin infections causing contagious skin necrosis, dermatitis, wounds, abscesses or similar lesions are a constant problem in camel and at many occasions the skin lesions spread rapidly over the body surface and it is very difficult to manage these lesions. The traditional treatment for this purpose has variable success and similar response is seen in practice of antibiotic therapy. Consequently, the camel becomes useless for any purpose.

The health researches in camel species are few. Further, amongst the various microbes studied the fungal infections are comparatively less studied in this species. Planned long term studies on the type of lesions caused by various type of fungi, remedial measures adopted by the farmers in the remote range land areas and treatments standardized by the scientists of the centre are being presented in this manuscript entitled 'Camel Dermal Mycoses: Diagnostic Pictorial'. Seasonal and age variations in occurrence of various infections are also being covered.

Various fungi of zoonotic significance causing camel skin infections were also isolated and identified. I am hopeful that the researchers, public health personnel's and field veterinarians will find this manuscript to be of use as reference material in dealing specially with camel skin infections.

N.V. Patil

Director

CAMEL DERMAL MYCOSES

Camel and its environment:

Camel belongs to the Kingdom-animalia, Class-mammalia, Family-camelidae, Genus-Camelus and Species-dromedarius. Commonly termed as the dromedary camel or the single-humped camel. They have long-curved neck and deep-narrow chest. The hump is used by the camels as reservoir of fatty tissues. In times of scarcity, the tissues are metabolized and the camel receives energy. The size of the hump is not the same in all the camels. It differs from one camel to another, depending upon its nutritional status. In times of starvation, the hump can get reduced. Indian dromedaries have a heavy growth of hair on throat, shoulder and hump. They are widely used by the people in the dry arid and semi arid regions of the country, especially the state of Rajasthan. They are used for draft purpose and as a means of transportation. Camels do provide milk, leather and fuel or fertilizer in the form of dung. Camels primarily survive on an herbivorous diet, consisting of thorny plants and dry grasses. Indian dromedaries attain maturity at the age of 4 to 5 years. Camels have the ability to endure wide changes in their body temperature as well as water content. Their body temperature may vary as widely as being 103^{0} F in the daytime and being 95^{0} F at night. The thick coat of a camel reflects sunlight and serves as insulation from the heat of the sand.

Rajasthan is the largest state in India situated in the North-West part of India between 23^{0}N to 30^{0}N Latitudes and 69^{0}E to 78^{0}E Longitudes and is characterized with tropical climatic conditions. It has distinct physiographic on account of the existence of oldest mountain ranges *i.e.* Aravalli. The Aravalli hills also demarcate the state into two distinct climatic regions *i.e.* semi arid East of the Aravalli and the arid region West of Aravalli. The Western desert region has extremes of temperature, high velocity of wind and very low humidity. Rajasthan has a tropical desert climate. It is extremely cold from October to February while the scorching sun tortures the land from March to September. There are distinct temp erature range variations diurnal and seasonal, revealing the most typical phenomenon of the warm-dry continental climate. The summer begins in the month of March and the temperature keeps on rising progressively through April, May and June. West of Rajasthan and the eastern side of Aravalli range, in the region of Bikaner, Phalodi, Jaisalmer and Barmer, the maxi mum daily temperature hovers around 40°C to 45°C. Sometimes, it even reaches as high as 49°C during the summer months. Nights of summer have a considerable temperature fall with a minimum daily temperature around 20°C to 30°C. However, Udaipur and Mount Abu have a pleasant climate in summers with a relatively lower daily maximum temp erature that hovers around 30°C to 40°C. The daily minimum temperature at nights for these two stations hovers around 22°C to 25°C. The major portion of the state consists of the arid west and the semi-arid mid-west has an average maximum of 45°C in June. January is the coldest month in the state of Rajasthan. The minimum

temperature sometimes falls to -2°C in the night at places like Sikar, Churu, Pilani and Bikaner. The sandy land gets colder with occasional western winds that cross the western, northern and eastern Rajasthan during winter months and light rainfall may cause chilly winds during this period. Rajasthan being the desert area, its climate varies mostly from arid to sub-humid. To the west of the Aravallis, the climate is marked by low rainfall, extreme diurnal and annual temperature, low humidity and high velocity winds. In the east of the Aravallis, the climate is semi-arid to sub-humid marked by lower wind velocity and higher humidity and better rainfall. The annual rainfall in the state differs significantly. The average annual rainfall ranges from less than 10 centimetre in North-West part of Jaisalmer region (lowest in the state), to 20 to 30 centimetre in the regions of Ganganagar, Bikaner and Barmer; 30 to 40 centimetre in the regions of Nagaur, Jodhpur, Churu and Jalor; more than 40 centimetre in the regions of Sikar, Jhunjhunun, Pali and the western fringes of the Aravalli range. The more fortunate eastern side of the Aravallis receives 55 centimetre rainfall in Ajmer to 100 centimetre rainfall in Jhalawar. Mount Abu in the Sirohi district in the southwest region receives the highest rainfall of more than 160 centimetre. The southwest monsoon begins in the last week of June in the eastern parts and may last till mid-September. There are occasionally pre-monsoon showers in mid-June while post-monsoon rains may occur in October. Winter may also receive a little rainfall with the passing of western distribution over the region. However, Rajasthan receives most of its monthly rainfall during July and August.

Camel is the most suitable mammal for uses in extreme climatic conditions (Wilson, 1984; Yagil, 1985). The skin infections causing contagious skin necrosis, dermatitis, wounds, abscesses or similar lesions are a constant problem in camel. These infections are chronic and difficult to treat medically because of unknown etiology. Though the diseases are not always fatal but an indirect great economical loss is incurred due to reduction in the working efficiency of the animals. At many occasions the skin lesions spread rapidly over the body surface and it is very difficult to manage these lesions. Consequently, the camel becomes useless for any purpose. The in practice antibiotic therapy also does not work satisfactorily. The inability to work or death of the animal result in severe monetary loss which shatters the socio-economic status of camel owner. Since the work on bacterial and fungal diseases in camels have not been given much attention, therefore a project work on 'epidemiology of bacterial and fungal diseases of camels' was carried out in thickly camel populated areas in semi arid and arid climatic conditions of Rajasthan state, covering Bikaner, Hanumangarh, Barmer, Jodhpur, Sirohi, Pali, Nagaur, Udaipur, Jaisalmer, Jhunjhnu and Churu districts. During more than five year-period (from July 2007 to May, 2013), information concerning the occurrence of disease along with age, sex, breed and managemental practices adopted

by the farmers for the treatment and control of camel diseases were recorded. All the relevant samples and photography of the lesions where thought necessary were collected by regular visits in camel rearing villages and by organizing free treatment camps with the help of local veterinary officers and heads of the camel rearing Raika community. Along with these other managemental and preventive health measures practiced by the camel owners were recorded and observed by on farm visits. Here it is pertinent to mention that on farm visits also includes visits in the desert rangelands, since camel husbandry is being managed by zero input system of grazing and large camel herds are kept in the loose areas of the desert for weeks together for browsing, mainly in the arid zone. Findings of camel dermal mycoses are presented.

Collection of samples: In affected camels with clear skin lesions, ointments or other local applications present were first removed with an alcohol wipe. Then using a blunt scalpel lesions were firmly scraped, particularly at the advancing border. If multiple lesions were present then the most recent were chosen for scrapings as old loose scale is often not satisfactory. The tops of any fresh lesions were removed as the fungus is often plentiful in the roof of the lesions. In animals with suspected candidiasis the young 'satellite' lesions which have not undergone exfoliation were scraped if they were present, otherwise the advancing scaly border was scraped. From lesions with abscess formation, swab samples were collected taking due sterile precautions. These samples were collected in sterile vials meant for sterile collection of clinical samples. Then these samples were transferred to the laboratory in thermocol box packed with brine packs.

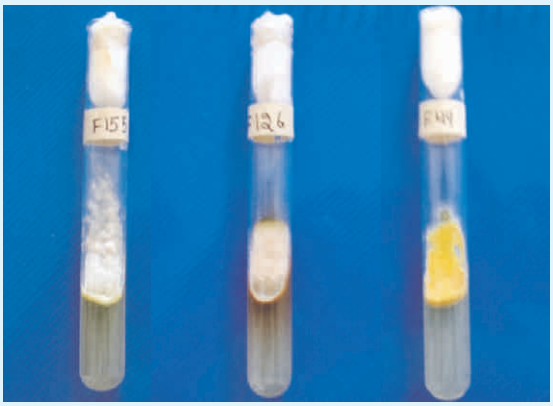
Isolation and identification of fungi:

1. Direct microscopic examination: was performed by placing the scrapings on a glass slide with two or three drops of 20 % potassium hydroxide and placing a cover slip over it. The sample was warmed for five minutes over a flame and was then carefully examined microscopically for the presence of hyphae and/or arthroconidia.

2. Cultural examination: Samples were first mixed with Sabouraud's dextrose chloramphenicol broth and were incubated for up to 24 hours. Then these samples were inoculated onto Sabouraud's dextrose chloramphenicol agar (SDCA) plate and were incubated at 28^{0}C for 3- 4 weeks. For suspected cases of skin candidiasis, 2^{nd} plate was also incubated at 37^{0}C for 4 weeks. In case the growth appeared to be of dimorphic fungi, another plate was sucultured and incubated at 37^{0}C for up to 2-weeks for confirming the yeast stage of the isolate. Where secondary bacterial infection was suspected and separate swabs for routine bacteriology were not collected, the swabs were directly inoculated first onto a blood agar plate, followed by the SDCA plate. These plates were examined daily for the growth of the fungi. The resultant growth was examined for the colony morphology. Microscopic examination was carried out using either lacto phenol cotton

blue or calcofluor white stains using wet mount method (Halley and Standard, 1973).

3. Storage of isolates: All these isolates were stored on SDCA slants for four months at refrigerator temperature and then were again subcultured at four month intervals, specially those isolates where identification of the isolate could not be made immediately.



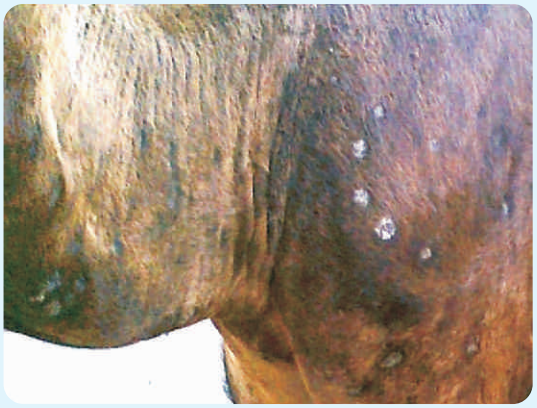
Fungi causing dermal mycoses in camel were dermatophytes and other filamentous fungi including dimorphic fungi. Overall skin infections were observed more in the winter season preceding rainy season. Skin infections are more prevalent in young calves of less than one year of age followed by aged (>15 years), active grower (1-5yrs) and adults. There fore young once or debilitated shall be protected of these infections.

I. Dermatophytes isolated from camel skin infections:

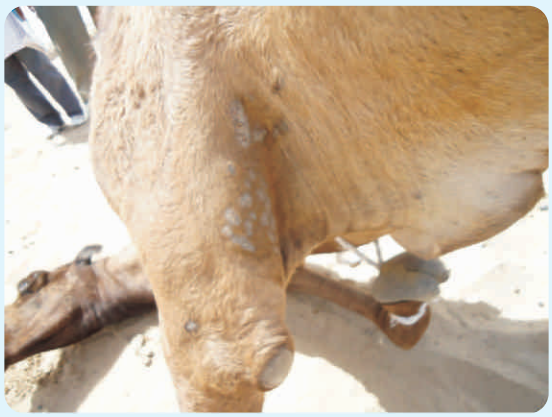
Dermatophytes grow best in warm and humid environments. Therefore rainy season coupled with temperature of the desert is most conducive for the growth of dermatophytes. Dermatophytes are keratinophylic (keratin digesting) fungi are common inhabitants of the soil, where they process the hairs and skin cells shed by animals, as well as all types of keratin products that fall from animals and humans during the natural and continuous cycle of skin and coat shedding. The group of keratinophylic fungi is very large, but only three genera *Microsporum*, *Trichophyton* and *Epidermophyton* are known to cause dermatophytosis and infect the keratinized tissues; hair, skin and nails (Aly *et al,* 2000; Aman *et al,* 2001; Elewski, 2000; Nweze, 2001; Roldan *et al,* 2000; Rubio-Calvo *et al,* 2001) and likewise skin, hair and claws in all the domestic animals worldwide. These organisms are called dermatophytes. *Microsporum* and *Trichophyton* are most frequently found in animals while the *Epidermatophyton* causes problems mainly in humans (Lewis *et al,* 1991). The particular ability of these fungi to be transmissible to animals, as well as to humans, signifies that they are important veterinary and human pathogens worldwide (Chretien and Garagusi, 1990). Georg (1954) proposed a classification for dermatophytes based on their habitat. Based on a large survey of skin samples from animals and humans, dermatophytes were divided into three groups: 1. zoophylic - those found mainly in animals, but transmitted to other animal or to humans; 2. anthropophylic - those found mainly in humans and transmitted amongst humans, but very seldom to animals; 3. geophylic - dermatophytes found mainly in soil that infect both humans and animals. It is now known that practically all dermatophytes have reservoirs in the soil;

however, this classification system is still used to indicate the usual source/ epidemiology of dermatophyte species. In camels *Microsporum* and *Trichophyton* fungi were found causing sporadic cases of skin infections in individually maintained camels as well as affecting many camels in the herds. Whereas Epidermatophyton affected many camels of a single herd.

1. *Microsporum* spp.: (Tuteja *et a*l communicated) Most of the *Microsporum* spp. are widely distributed in the world while some have restricted geographic distribution. Similar to other dermato phytes, microsporum has the ability to degrade keratin and thus can reside on skin and its appendages and remains non-invasive. The keratinases, proteinases and elastases of the fungus may act as virulence factors. These fungi can grow to create distinctive lesions associated with ring worm. *Microsporum* spp. were constant finding from the type of lesions shown in Pic. 1-2, these lesions are small disc-shaped markings and can occur anywhere on the body. Later, the markings develop crusts,



Pic. 1: Ringworm on scapular region

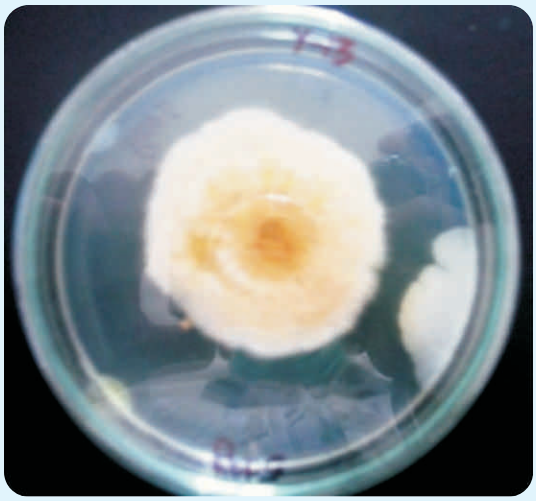


Pic. 2: Ringworm on axillary girth

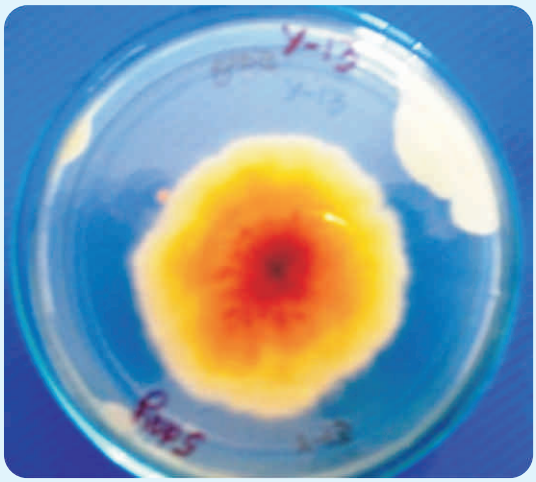
which can ooze and may turn bloody on scratching. Hair above the area of the lesion will fall out, due to damage caused by the fungal colonization.

Microsporum colonies are glabrous, downy, woolly or powdery. The growth on SDCA at 28°C may be slow or rapid and the diameter and colour of the colony varies depending upon the species. Microsporum produce septate hyphae, micro conidia and macro conidia. Conidiophores are hyphae like. Macroscopic morphology along with colour of the colony helps in species differentiation. The isolated species from camel infections were *M. audouinii*, *M. canis, M. nanum* and *M. ferrugineum.*

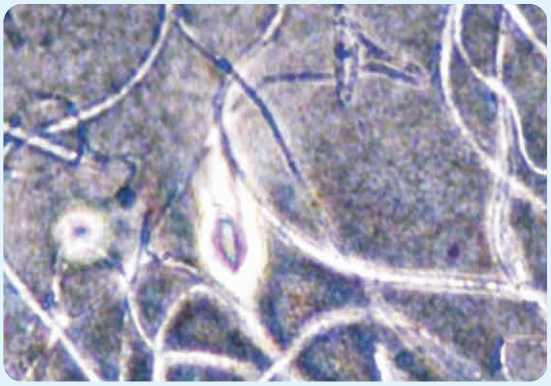
M. audouinii: Colonies on SDCA are flat, spreading, greyish-white to light tan-white in colour and have a dense suede-like to downy surface suggestive of mouse fur in texture. Reverse can be yellow-brown to reddish-brown in colour. Some strains may show no reverse pigment. Macroconidia and microconidia are only rarely produced. Most cultures are sterile or produce only occasional thick-walled terminal or intercalary chlamydoconidia (Pic. 3-5).



Pic. 3: *M. audouinii;* colony (5 days front)



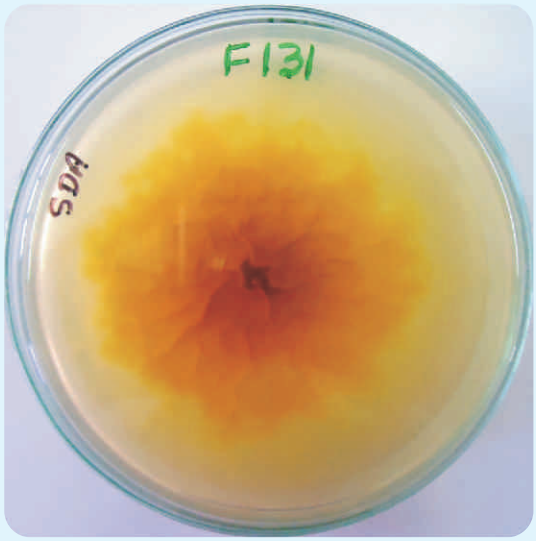
Pic. 4: *M. audouinii;* colony (5 days reverse)



Pic. 5: *M. audouinii;* thick-walled intercalary chlamydoconidium

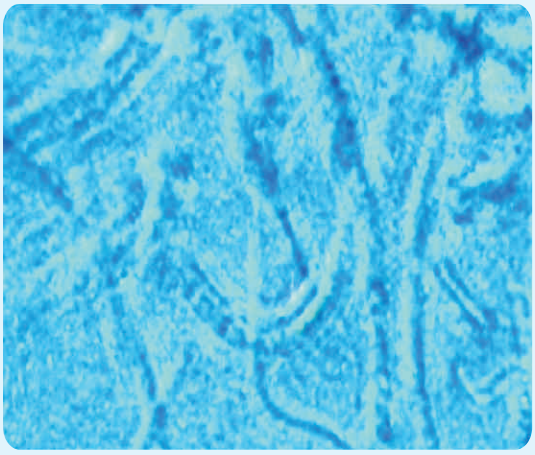
M. canis: Colonies on SDCA are flat, spreading, white to cream-coloured with a dense cottony surface which may show some radial grooves. Colonies usually have a bright golden yellow to brownish yellow reverse pigment, but non-pigmented strains may also occur. Macroconidia are typically spindle-shaped with 5-15 cells, verrucose,

Pic. 6: *M. canis* isolate^{-1}; colony (5 days front)



Pic. 7: *M. canis* isolate^{-1}; colony (5 days reverse)

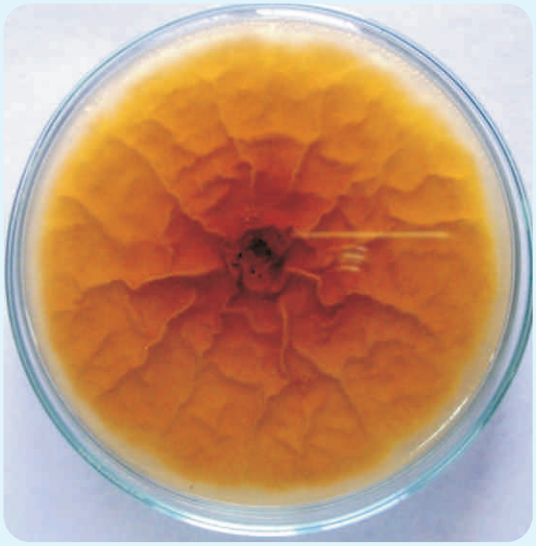
thick-walled and often have a terminal knob. A few pyriform to clavate microconidia are also present (Pic. 6-11).



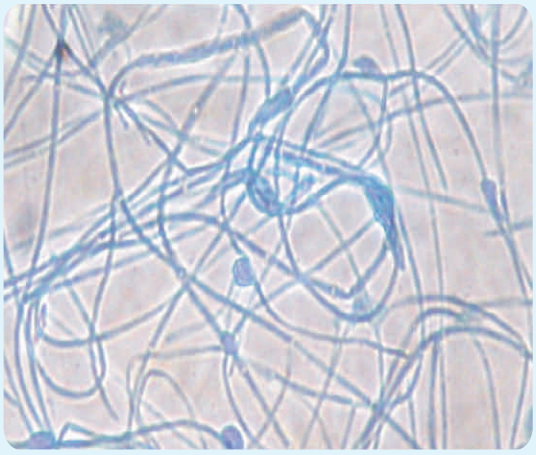
Pic. 8: *M. canis* isolate^{-1}; macroconidium

Pic. 9: *M. canis* isolate^{-2}; colony (10 days front)

M. nanum: Colonies of this species grow moderately on SDCA. The colony is powdery, cottony, thin, spreading, velvety or flat and often has some radial, shallow furrows. The colour is white to dark beige from the front and reddish brown from the



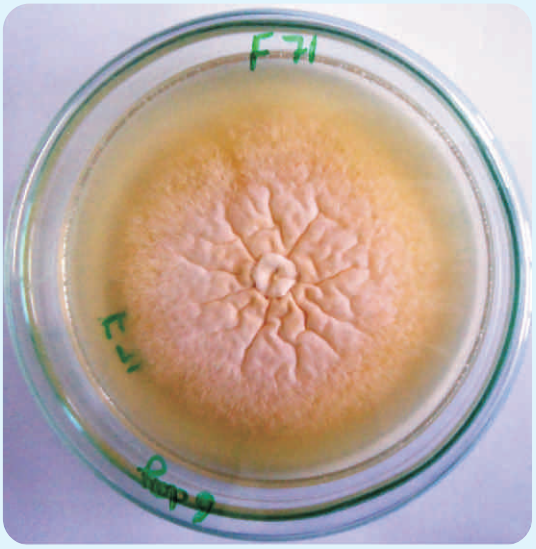
Pic. 10: *M. canis* isolate^{-2}; colony (10 days reverse)



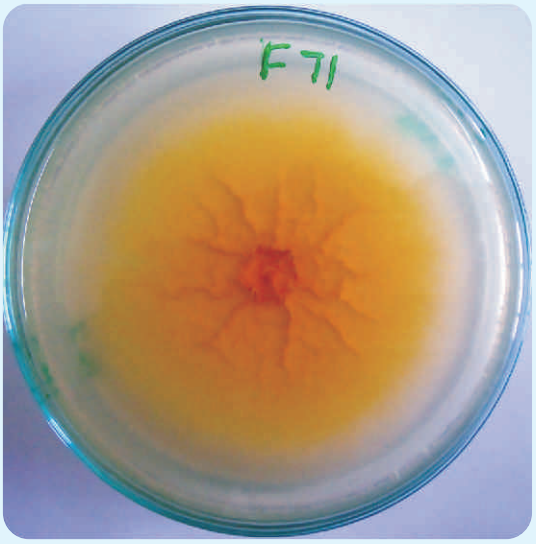
Pic. 11: *M. canis* isolate^{-2}; macroconidium, intercalary chlamydoconidium

reverse. Microscopically it has septate hyphae, macroconidia and microconidia. Macroconidia are 1 to 4 celled (usually 2) thin walled and oval to elliptical in shape. Microconidia are club-shaped and their abundance may vary (Pic. 12-14).

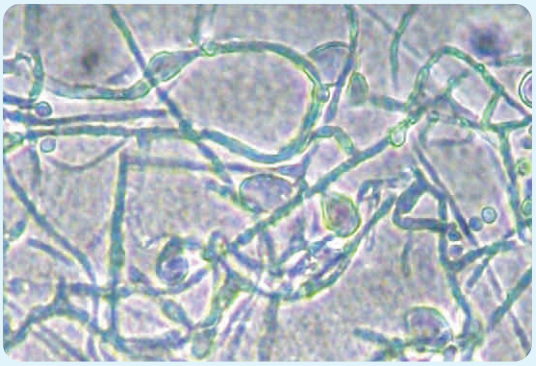
M. ferrugineum: Colonies on SDCA are slow growing, forming a waxy, glabrous,



Pic. 12: *M. nanum;* colony (7 days front)



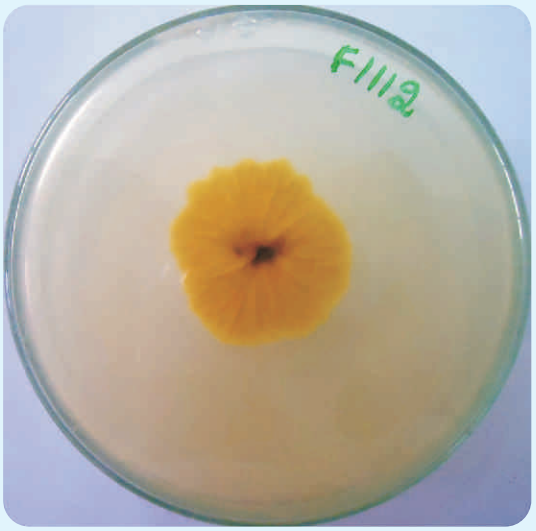
Pic. 13: *M. nanum;* colony (7 days reverse)



Pic. 14: *M. nanum;* macroconidium

convoluted thallus with a cream to buff coloured surface. Cultures rapidly become downy and pleomorphic. Microscopically irregular branching hyphae with prominent cross walls 'bamboo hyphae' which is characteristic of this species and chlamydoconidia are seen (Pic. 15-17).

Pic. 15: *M. ferrugineum;* colony (10 days front)



Pic. 16: *M. ferrugineum;* colony (10 days reverse)

Pic. 17: *M. ferrugineum;* bamboo hyphae

M. gypseum infection has been reported in camels (Boever and Rush, 1975; Mancianti *et al,* 1988; Gitao *et al,* 1998); cattle (Gupta *et al,* 1970; Saxena and Mehra, 1973; Chatterjee and Sen Gupta, 1979; Pal and Singh, 1983; Mitra, 1988; Ranganathan *et al,* 1998); dog (Gugnani *et al,* 1971; Saxena, 1972; Chatterjee and Sen Gupta, 1979; Chittawar and Rao, 1982); sheep (Mitra *et al,* 1989); goats (Thakur and Verma, 1984) and horse (Tewari, 1962; Gupta *et al,* 1970; Saxena and Mehra, 1973; Pal *et al,* 1994; Connole and Pascoe, 1984).

M. canis infection has been reported in cattle (Pal and Singh, 1983) and dog (Khandari and Sethi, 1964; Gupta *et al,* 1968; Singh and Singh, 1970; Saxena, 1972; Pal, 1981; Khan *et al,* 1982; Chittawar and Rao, 1982; Malik *et al,* 1984).

M. distortum infection has been reported in dog (Chatterjee and Sen Gupta, 1979).

M. nanum have been isolated from pigs (Gupta *et al,* 1970; Chatterjee and Sen Gupta, 1979; Sarkar *et al,* 1985).

M. equinum is reported to be main infection causing ring worm in horses and younger horses were most susceptible (Al-Ani *et al,* 2002; Andryushin, 1980). Spores of this fungus were capable of surviving for two years on the walls of a stable, for 17 weeks in stable manure and on the soil surface under snow and for 3.5 years at room temperature. Virulence of individual strains varied considerably and it declined upon continuous culture. The incubation period of experimental infection was 18-30 days. Ringworm lasted for 2-3 months in foals and 1-2 months in adult horses (Andryushin, 1980). In most of the surveys conducted worldwide, *M. canis* is largely predominant in cats with over 90% of the feline isolates, it is less frequent in dogs (70–80%) and in horses (syn. *M. equinum*). It can also be found in rabbits, rodents, all kinds of mammals, including wildlife, depending probably on the intensity and frequency of contact with the domestic fauna (Gallo *et al,* 2005).

M. ferrugineum is a rare isolate in India. *M. ferrugineum*, an anthropophilic dermatophyte is endemic in Africa and Oriental Asia; sporadic cases have been reported from other countries (Seebacher *et al,* 2008). Sahai and Mishra (2011) reported *M. ferrugineum* and *M. audouinii* from human patients from central India. All cases were immunocompetent and neither case had any history of travelling or staying abroad nor had any unusual clinical presentation. Singal *et al* (2001) reported *M. audouinii* and *M. gypseum* from *Tinea capitis* cases from North India.

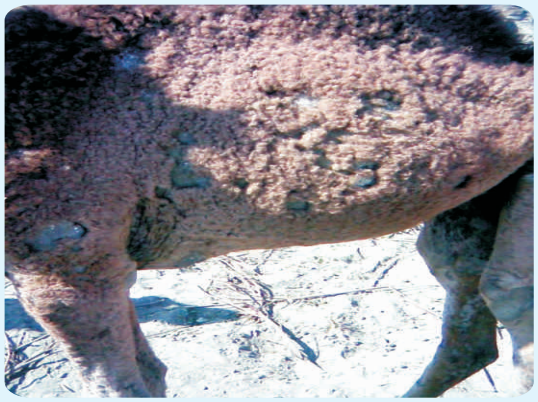
2. *Trichophyton* spp.:(Tuteja *et al.* communicated) Some *Trichophyton* spp. are cosmopolitan while others have a limited geographic distribution. Tricho phyton has the ability to invade keratinized

tissues and possess several enzymes such as acid proteinases, elastases, keratinases and other proteinases which act as virulence factors (Weitzman and Summerbell, 1995).

Lesions of ring worm as reported in *Microsporum* spp. occur in various species of the animals. But lesions of ring worm observed with *Trichophyton* spp. were comparatively dry, hard, crusty, granu lomatous, larger in size, occurring on whole of the body surface. Pic. (18-21) depicts the type of lesions, caused by *Trichophyton* spp.

Pic. 18: Granulomatous lesions on the rump

Pic. 19: Granulomatous lesions on the chest



Pic. 20: Granulomatous lesions on the body

Pic. 21: Granulomatous lesions on the body

The growth rate of trichophyton colonies is slow to moderately rapid. The texture and colour of the colonies varies as per the species. Microscopically septate hyaline hyphae, conidiophores, micro conidia, macroconidia and arthroconidia are observed.

Chlamydospores may also be produced. Conidiophores are poorly differentiated from the hyphae. Microconidia are one-celled and round or pyriform in shape. They are numerous and are solitary or arranged in clusters. Microconidia are often the predominant

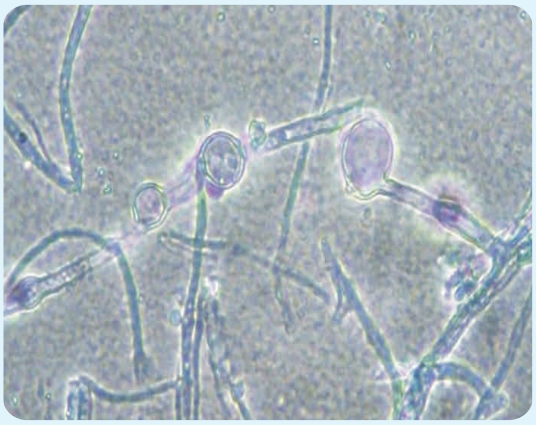
type of conidia produced by trichophyton. Macroconidia are multicellular (2- or more-celled), smooth, thin or thick-walled and cylindrical, clavate or cigar-shaped.

Species of trichophyton isolated from camels were *T. verrucosum, T. menta grophytes, T. schoenleinii, T. equinum, T. concentricum, T. tonsurans, T. violaceum, T. soudanense and T. rubrum* hyper pigmented type.

T. verrucosum: On SDCA colonies are slow growing, small, button-or-disk-shaped, white to cream coloured, with a suede-like to velvety surface, a raised centre with some submerged growth. Reverse pigment may vary from non-pigmented to yellow. Broad, irregular hyphae with many terminal and intercalary chlamydospores. Chlamydos pores are often in chains. The tips of some hyphae are broad and club-shaped and occasionally divided, giving the so-called 'antler' effect. Macroconidia are only rarely produced, but when present has a characteristic tail or string bean shape (Pic. 22-24).

Pic. 22: *T. verrucosum;* colony (5 days front)

Pic. 23: *T. verrucosum;* colony (5 days reverse)

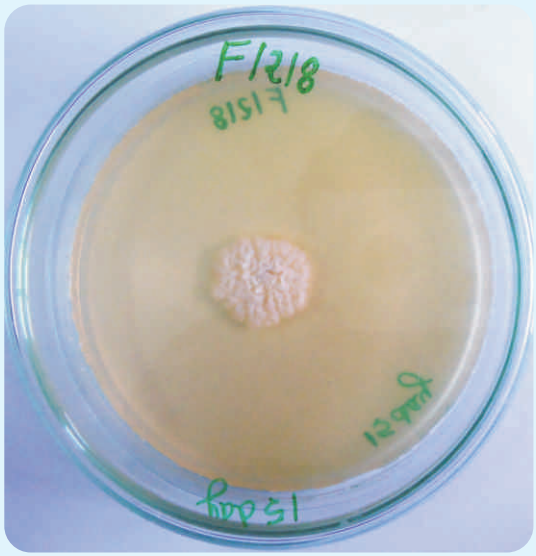


Pic. 24: *T. verrucosum;* chlamydospores in chain, tip of hyphae club-shaped

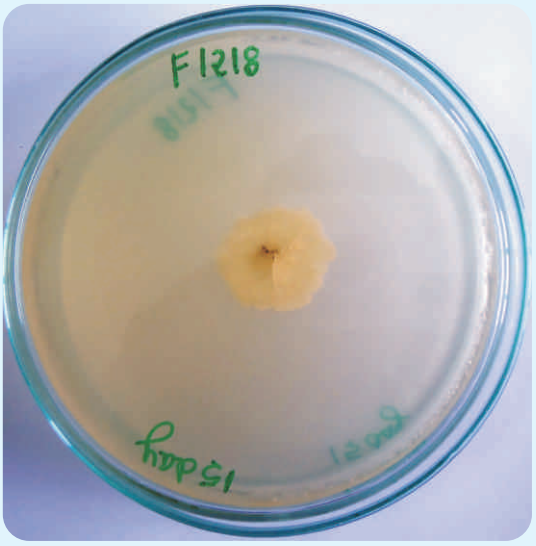
T. mentagrophytes: On SDCA, colonies are generally flat, white to cream in colour, with a powdery to granular surface. Some cultures show central folding or develop raised central tufts or pleomorphic suede-like to downy areas. Reverse pigmentation is usually a yellow-brown to

reddish-brown colour. Numerous single-celled microconidia are formed, often in dense clusters. Microconidia are hyaline, smooth-walled and are predominantly spherical to sub spherical in shape; however occasional clavate to pyriform forms may occur. Varying numbers of spherical chlamydoconidia, spiral hyphae and smooth, thin-walled, clavate shaped, multicelled macroconidia may also be present.

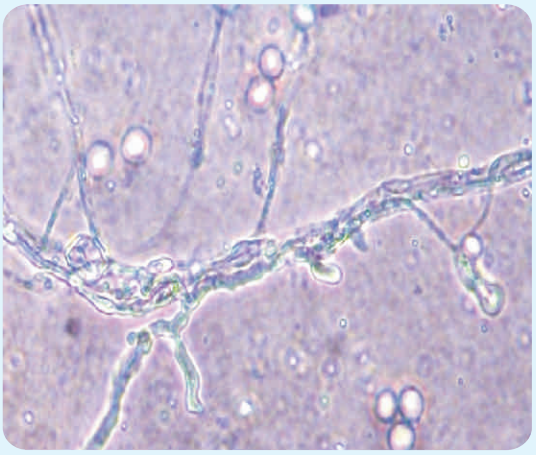
T. schoenleinii: On SDCA, colonies are slow growing, waxy or suede-like with a deeply folded honey-comb-like thallus and some sub-surface growth. The thallus is cream coloured to yellow to orange brown. Cultures are difficult to maintain in their typical convoluted form and rapidly become flat and downy. No reverse pigmentation is present. No macroconidia and microconidia are seen in routine cultures; however numerous chlamydoconidia may be present in older cultures. However, characteristic antler 'nail head' hyphae also known as 'favic chandeliers' may be observed (Pic. 25-27).



Pic. 25: *T. schoenleinii;* colony (15 days front)



Pic. 26: *T. schoenleinii;* colony (15 days reverse)

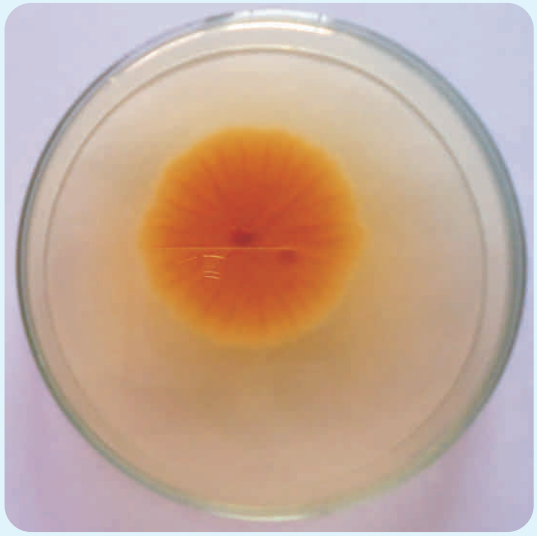


Pic. 27: *T. schoenleinii;* favic chandeliers

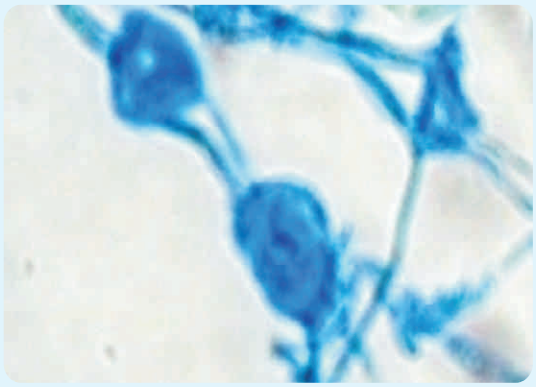
T. equinum: On SDCA, colonies are usually flat, but some may develop gentle folds or radial grooves, white to buff in colour, suede-like to downy in texture. Cultures usually have a deep-yellow sub

merged fringe and reverse which later becomes dark red. Microscopically abundant microconidia which may be clavate to pyriform and sessile or spherical and stalked are formed laterally along the hyphae. Macroconidia are only rarely transformation to produce abundant chlamydoconidia in old cultures (Pic. 28-31).

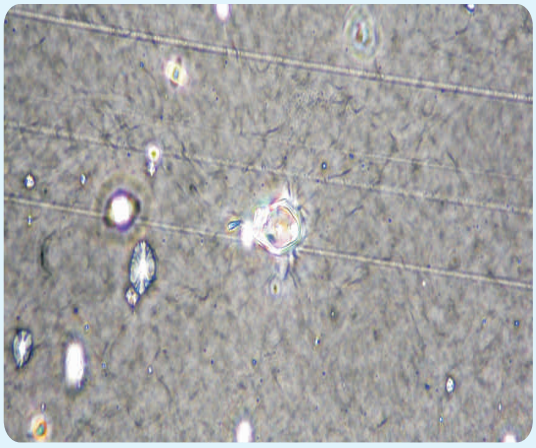
Pic. 28: *T. equinum;* colony (10 days front)



Pic. 29: *T. equinum;* colony (10 days reverse)



Pic. 30: *T. equinum;* nodular organs

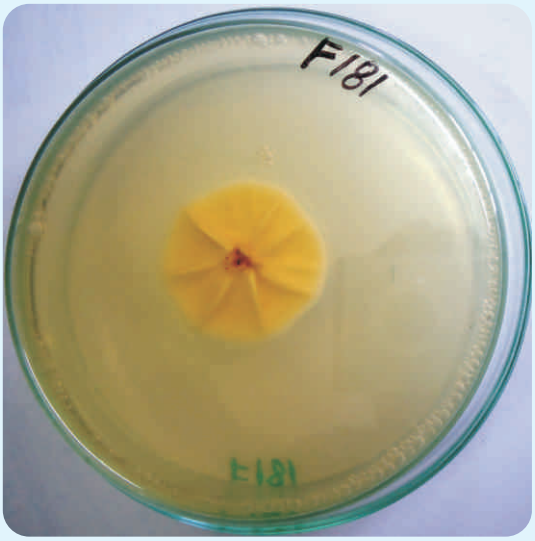


Pic. 31: *T. equinum;* microconidia and chlamydoconidia

T. concentricum: On SDCA colonies are slow growing, raised and folded, glabrous becoming suede-like, mostly white to cream coloured. Reverse is buff to yellow-brown to brown in colour. Cultures consist of broad, much-branched, irregular, often segmented, septate hyphae which may have 'antler' tips. Chlamydoconidia are often present in older cultures. Micro conidia and macroconidia are not usually produced, although some isolates will produce occasional clavate to pyriform

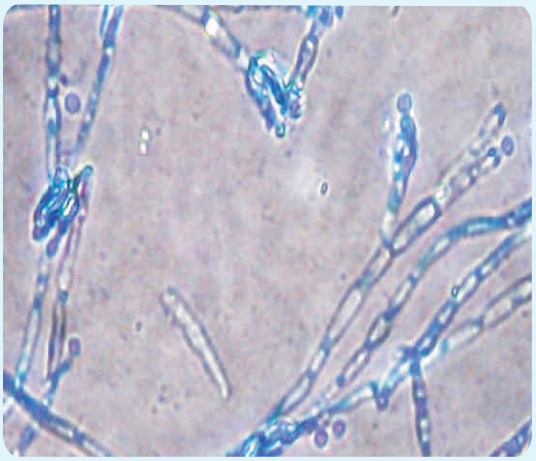
microconidia. Hyphal segments may artificially resemble macroconidia (Pic. 32-34).

Pic. 32: *T. concentricum;* colony (8 days front)



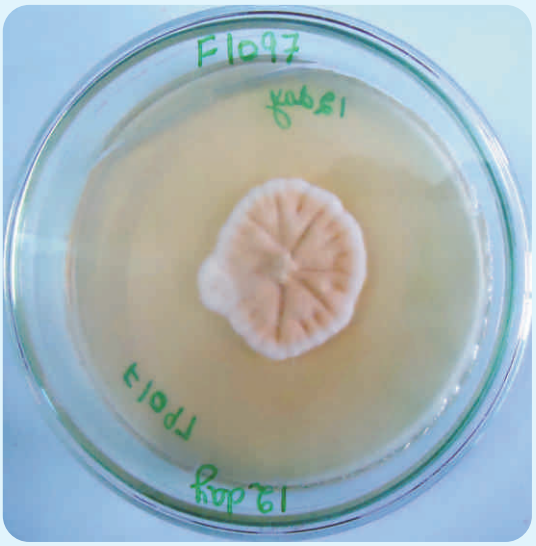
Pic. 33: *T. concentricum;* colony (8 days reverse)

T. tonsurans: On SDCA moderately slow growth, highly variable, suede like, powdery or velvety, flat with a raised centre or folded, often with radial grooves. White, beige, greyish, pale or sulphur yellow, rose or brownish on surface. The reverse colour varies from yellow-brown to reddish-brown to deep mahogany. Hyphae are relatively



Pic.34: *T. concentricum;* much-branched, septate hyphae with 'antler' tips

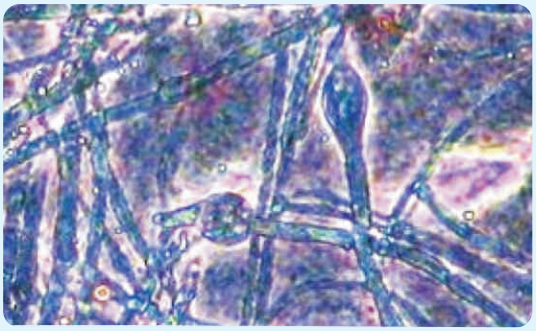
broad, irregular, much branched with numerous septa. Numerous microconidia of various shapes and sizes such as pyriform, tear drop, club shaped or balloon shaped; intercalary and terminal chlamydo



Pic. 35: *T. tonsurans;* colony (12 days front)

spores found in older culture; macroconidia rare, smooth walled and distorted (Pic. 35-37).

Pic. 36: *T. tonsurans;* colony (12 days reverse)

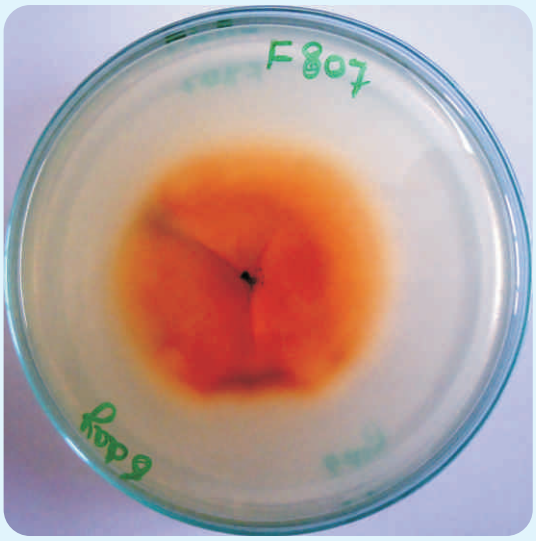


Pic. 37: *T. tonsurans;* balloon shaped intercalary and terminal chlamydospores

T. violaceum: On SDCA colonies are slow growing, glabrous or waxy, heaped and folded and a deep violet in colour. Cultures often become pleomorphic. Hyphae are relatively broad, tortuous, much branched and distorted. Young hyphae usually stain well in lactophenol cotton blue and show small central fat globules and granules.

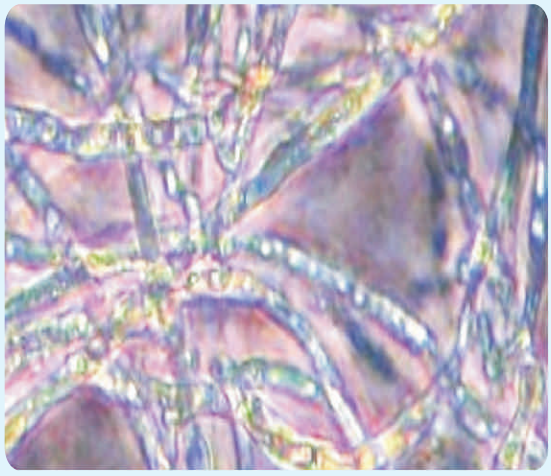
Numerous chlamydoconidia are usually present, especially in older cultures (Pic. 38-40).

Pic. 38: *T. violaceum;* colony (6 days front)



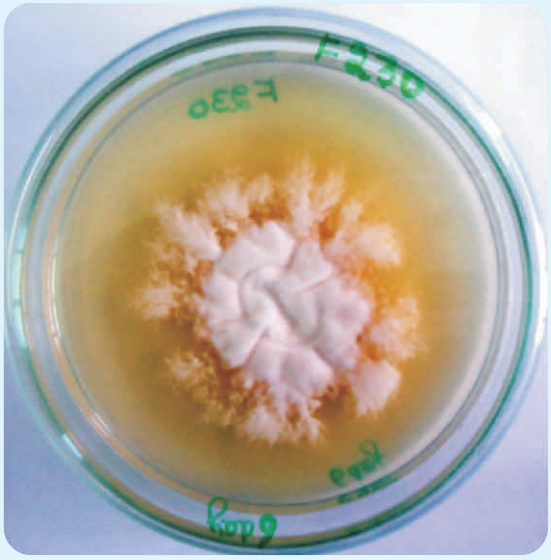
Pic. 39: *T. violaceum;* colony (6 days reverse)

T. soudanense: On SDCA, colonies are slow-growing with a flat to folded, suede-like surface. Often there is a broad fringe of submerged growth. Surface mycelium and reverse pigment is characteristically a deep apricot-orange in colour. Microscopically, the hyphae often show reflexive or right-angle branching.



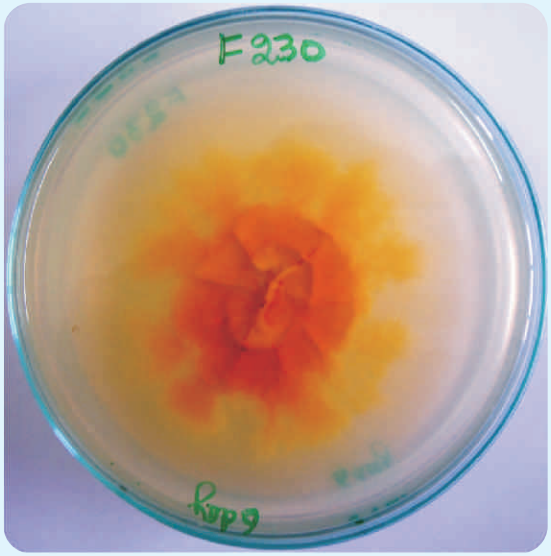
Pic. 40: *T. violaceum;*
hyphae with small
central fat globules and granules

Pyriform microconidia may occasionally be present and numerous chlamydoconidia are often found in older cultures (Pic. 41-43).

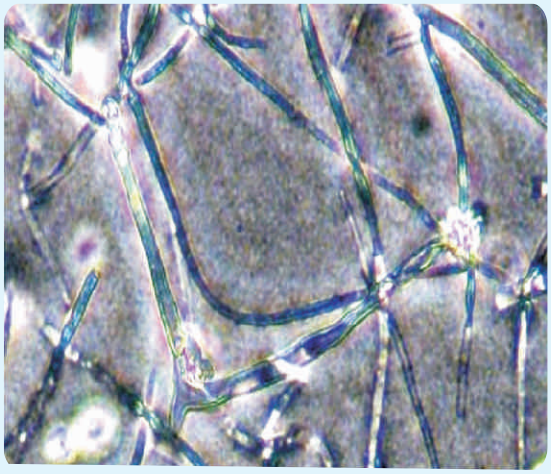


Pic. 41: *T. soudanense;*
colony (10 days front)

***T. rubrum* hyper pigmented type**: On SDCA, growth rate is slow; cultures show a violet to red-violet glabrous surface with



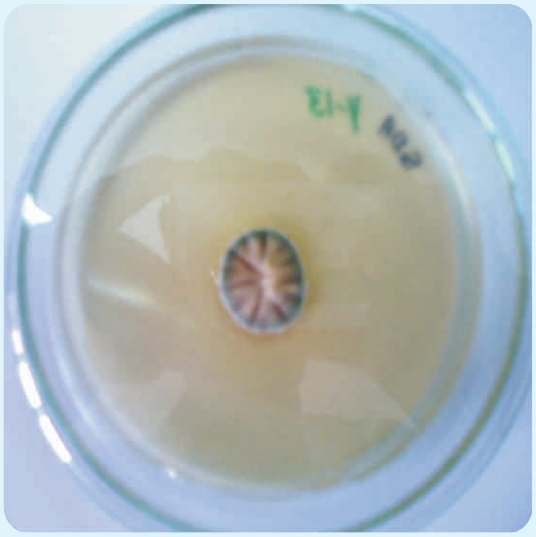
Pic. 42: *T. soudanense;*
colony (10 days reverse)



Pic. 43: *T. soudanense;*
reflex hyphae branching

radial furrows and a deep violet to red-violet reverse. Microscopically few pyri form lateral microconidia, pencil shaped macroconidia, arthroconidia produced from hyphae and macroconidia (Pic. 44-46).

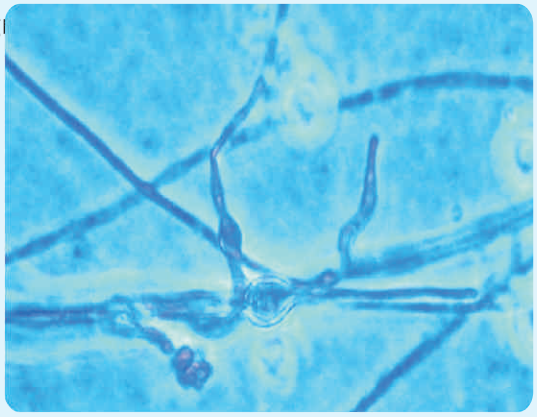
Different species of trichophyton reported in camels include *T. schoenleinii* (Chatterjee *et al,* 1978). Survey of ring worm in camels, showed over 25 % of young animals suffered from *T. verrucosum*



Pic. 44: *T. rubrum;* colony (10 days front)

Pic. 45: *T. rubrum;* colony (10 days reverse)

infection and less than 0.5 % had *T. mentagrophytes* (Kuttin *et al,* 1986). *T. verrucosum* was the primary causal agent in young camels and *T. mentagrophytes* was in older camels (Mahmoud, 1993). The peak incidence of disease was autumn and winter with incidence highest in young



Pic. 46: *T. rubrum;* pencil shaped macroconodia, hyphae showing arthroconidia

observed primarily on head, neck and shoulder with frequent extension to the flanks and limbs. *T. verrucosum* was isolated as the primary causal organism (Fadlelmula *et al,* 1994). Ebrahimi *et al* (2007) reported *T. verrucosum* and *T. tonsurans* from healthy skin coat of camels from Iran. *T. verrucosum* and *T. mentagrophytes* var. *mentagrophytes* was the common cause of dermatophytosis in alpacas and llamas. Spores of *T. verrucosum* and *T. menta grophytes* may remain viable for up to 4-5 years in hair and cellular debris scraped off the animal and left attached to barn walls, fence posts, trees and other fixtures, blankets, leads, grooming apparatus *etc*. Treatment of affected camelids is suggested as iodine 2 % tincture applied directly to lesions daily for two weeks. The less caustic povidone-Iodine preparation diluted 1:4 may be equally effective (Murray, 1998).

T. verrucosum have been isolated from cattle (Tiwari, 1962; Mahajan and Mahapatra, 1968; Singh and Singh, 1970;

Gugnani, 1972; Chatterjee and Sen Gupta, 1979; Sharma *et al,* 1979; Pal and Singh, 1983; Malik *et al,* 1984; Sarkar *et al,* 1985; Pal, 1987; Quinn *et al,* 1994); buffalo (Gupta *et al,* 1970; Singh and Singh, 1970; Thakur *et al,* 1983; Pal and Singh, 1983); sheep (Chatterjee and Sen Gupta, 1979; Thakur *et al,* 1983); goat (Gupta *et al,* 1970; Chatterjee and Sen Gupta, 1979; Pal and Singh, 1983); dog (Chatterjee and Sen Gupta, 1979).

T. mentagrophytes have been isolated from cattle (Tewari, 1962; Gupta *et al,* 1968; 1970; Singh and Singh, 1970; Monga *et al,*1978; Chatterjee and Sen Gupta, 1979; Sharma *et al,* 1979; Thakur *et al,* 1983; Pal and Singh, 1983; Malik *et al,* 1984; Gupta *et al,* 1968; 1970; Mitra, 1998; Quinn *et al,* 1994); buffalo (Gupta *et al,* 1968; Saxena and Mehra, 1973; Chatterjee and Sen Gupta, 1979; Pal and Singh, 1983; Thakur *et al,* 1983); sheep (Thakur *et al,* 1983); goat (Gupta *et al,* 1970; Chatterjee and Sen Gupta, 1979; Pal and Singh, 1983); horse (Gupta *et al,* 1970; Padhye *et al,* 1966; Chatterjee and Sen Gupta, 1979; Connole and Pascoe, 1984); dog (Tewari, 1962; Singh and Singh, 1970; Gugnani *et al,* 1971; Saxena, 1972; Chatterjee and Sen Gupta, 1979; Sharma *et al,* 1979; Chittawar and Rao, 1982; Ranganathan *et al,* 1998).

T. rubrum have been isolated from cattle (Chakraborty *et al,* 1954; Tewari, 1962; Singh and Singh, 1970; Chatterjee *et al,* 1978; Chatterjee and Sen Gupta, 1979; Sharma *et al,* 1979; Sarkar *et al,* 1985; Mitra, 1988); dog (Chakraborty *et al,* 1954; Tewari, 1962; Padhye *et al,* 1966; Yamada *et al,* 1991; Ranganathan *et al,* 1998).

T. terrestre have been isolated from buffalo (Gupta *et al,* 1970).

T. equinum have been isolated from horse (Kulkarni *et al,* 1969; Singh, 1982).

T. simii have been isolated from cattle (Mitra, 1998); dog (Tewari, 1969; Gupta *et al,* 1970; Mohapatra and Mahajan, 1970; Ranganathan *et al,* 1998).

T. megninii have been isolated from cattle (Quinn *et al,* 1994).

T. violaceum have been isolated from dog (Singh and Singh, 1970).

In horses, *Trichophyton* spp. and *Microsporum* spp. are the main causes of ringworm in Saudi Arabia (Bagy and Abdel-Mallek, 1991). In horses, *M. equinum* and *T. equinum* were the most common cause of ring worm (Al-Ani *et al,* 2002; Moretti *et al,* 1998; Stenwig, 1985; Takatoria *et al,* 1981; Weiss *et al,* 1979; Woloszyn, 1987; Connole and Pascoe, 1984).

Abu-Samra and Ibrahim (1988) found that horses were successfully infected with human isolates of *M. canis* and *T. violaceum*. Sahai and Mishra (2011) reported *T. mentagrophytes, T. tonsurans*, *T. verruco sum*, *T. schoenleinii* and *T. rubrum* from human cases from central India. Singal *et al* (2001) reported *T. violaceum*, *T. schoenleinii, T. tonsurans, T. verrucosum* and *T. mentagrophytes* from human cases from North India.

Ringworm is a major public and veterinary health problem reported from different parts of the world and causes great economic loss (Calderone, 1989). The

disease appears to be more common in tropical than temperate climates particularly in countries having hot and humid climatic conditions (Pascoe, 1976). Calves at weaning time are highly susceptible to ringworm infection (Al-Ani *et al,* 2002). This may be in part due to their weak immunity and the high pH of the skin (Radostits *et al,* 1997).

Al-Ani *et al* (2002) reported effective cure of ringworm in cattle calves with topical application of an ointment containing benzoic acid-6 g, salicylic acid-3 g, sulfur-5 g, iodine- 4 g and vaseline-100 g, with two to three applications at 3-4-day intervals. Many topical treatments have been reported to be successful in cattle, because spontaneous recovery is common, claims of efficacy are difficult to substantiate. Valuable individual animals should still be treated because this may well limit both progression of existing lesions and spread to others in the herd. Thick crusts should be removed gently with a brush and the material burned or disinfected with hypochlorite solution. Treatment options depend on the limitations on the use of some agents in animals meant for slaughter. Agents reported to be of use include washes or sprays of 4% lime sulphur, 0.5% sodium hypochlorite (1:10 household bleach), 0.5% chlorhexidine, 1% povidone-iodine, natamycin and enilconazole. Individual lesions can be treated with miconazole or clotrimazole lotions. An attenuated fungal vaccine is in use in some European countries; it prevents development of severe clinical lesions and also has greatly reduced the incidence of zoonotic disease in animal care workers. Unfortunately, vaccinated animals shed fungal spores for a time after vaccination.

Ketoconazole, clotrimazole, itracona zole, terbinafine, naftifine and amorolfine are in general active *in vitro* against trichophyton (Fernandez-Torres *et al,* 2000; Macura, 1993; Regli and Ferrari, 1989). Terbinafine usually appears to be the most effective agent (Fernandez-Torres *et al,* 2000; Jessup *et al,* 2000). Griseofulvin, once the drug of choice for treatment of dermatophytosis is less commonly used due to the availability of more effective and less toxic drugs (Zaias *et al,* 1996). Terbinafine and itraconazole (Arenas, 1995; Zaias *et al,* 1996) are commonly used in treatment of infections due to *Trichophyton* spp. and other dermatophytes. For treatment of tinea capitis and onychomycosis, oral therapy is usually preferred (Zaias *et al,* 1996). Terbinafine, a highly lipophilic allylamine antifungal is a relatively new drug in terms of veterinary use. It is fungicidal and is concentrated in the skin, nails and fat following oral dosing, making it a good choice for dermatological infections, particularly ringworm (Mancianti *et al,* 1999) and side effects are thought to be rare. Intravenous liposomal amphotericin B administration is the mainstay of treatment for human systemic fungal infections (Sorensen *et al,* 2006). This drug has been rarely used in veterinary medicine because it is prohibitively expensive and its cheaper desoxycholate form is highly nephrotoxic. However, reduced nephrotoxicity has been

described using subcutaneous administration making it a more practical option (O'Brien *et al,* 2006).

3. *Epidermophyton* spp.: (Tuteja *et al.* communicated) Man is the primary host of *E. floccosum.* This was isolated from skin infection in a herd of camel at Charanwala (Bajju) with fast spreading lesions. Out of the 50 camels examined 16 were infected with such lesions. Lesions were peculiar as if hairs were burnt with fire leaving behind ash deposit over the skin. Lesions were observed through out the body. All ages of the camel were affected but calves were more severely affected. The general dryness

Pic. 47: Epidermophyton initial lesions on thigh and flank

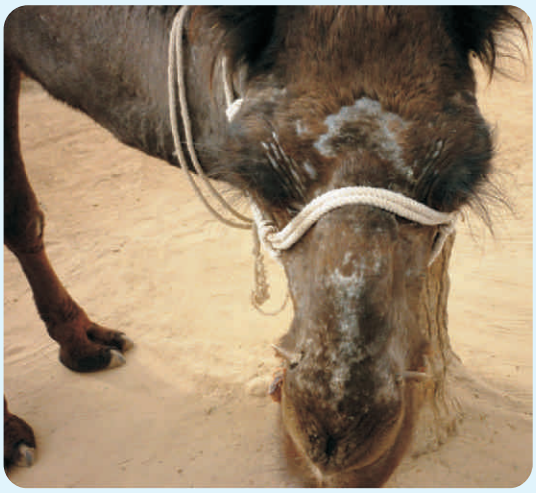
Pic. 48: Epidermophyton mild lesions on the flank and neck

Pic.49: Epidermophyton fast spreading lesions with circular patches

Pic. 50: Epidermophyton lesions giving just burning appearance

Pic. 51: Epidermophyton lesions on the neck

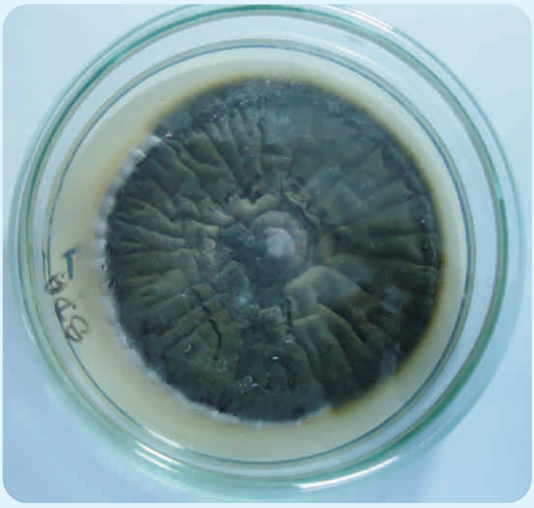
of the skin coat was more pronounced in such cases. During development of the lesions necrosis follows alopecia. It caused itching, uneasiness and resulted in weakness and debility of the animals (Pic. 47-52).



Pic. 52: Epidermophyton lesions on the forehead

This infection occurred after heavy rains in the year 2010, leading to high humidity in the environment. During this year average rainfall was high compared to previous years and the whole Rajasthan state received 114 % rainfall of its normal rainfall. Along with these infections, incidence of many other diseases was high and the major one was upper respiratory tract infection, which affected majority of the camel population and this infection was restricted only to the camels and no other species of the livestock were observed to be infected by such infection.

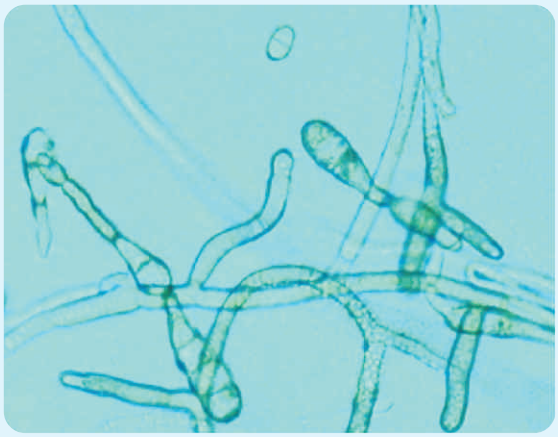
Cultural examination of skin scrapings revealed for the isolation of this fungi from all the affected animals with such lesions. *E. floccosum* grow moderately rapidly and mature within 10 days. Following incubation at 28°C. The colour of the colonies was brownish yellow to olive gray or khaki from the front. Orange to brown with an occasional yellow border from the reverse. Surface was flat and grainy initially and became radially grooved and velvety by aging (Pic. 53-54).



Pic. 53: *E. floccosum;* colony (10 days front)

Pic. 54: *E. floccosum;* colony (10 days reverse)

Microscopically septate, hyaline hyphae. Thin walled macroconidia, 3- 5 celled, smooth and clavate shaped with rounded ends, single or in clusters (Pic. 55). Chlamydoconidium like cells, as well as arthroconidia, are common in older cultures (de Hoog *et al,* 2000; Larone, 1995; St-Germain and Summerbell, 1996; Sutton *et al,* 1998).



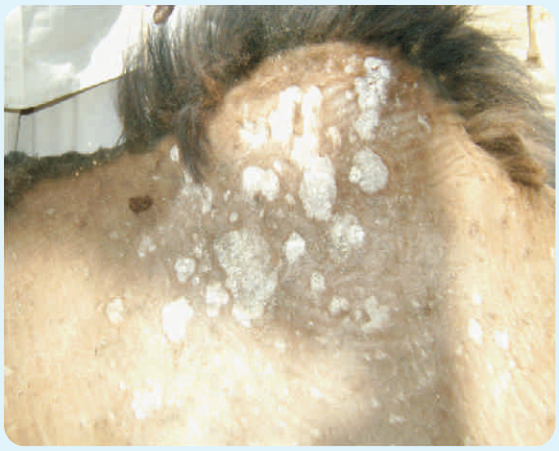
Pic. 55: *E. floccosum;* hyphae, macroconidia

E. floccosum has been reported from mule, dog and goat (Boro *et al,* 1980). Terreni *et al* (1985) isolated *E. floccosum* from a lesion of dermatophytosis on a dog with hyperadrenocorticism in the United States. The infection is restricted to the nonliving cornified layers of epidermis since the fungus lacks the ability to penetrate the viable tissues of the immunocompetent host (Aman *et al,* 2001; Ogawa *et al,* 1998; Weitzman and Summerbell, 1995). However, invasive infection has been reported in an immunocompromised patient with Behcet's syndrome (Seddon and Thomas, 1997). Terbinafine, itracona zole and ketoconazole are being practised for treatment of *E. floccosum* infections (Boonk *et al,* 1998; Degreef and DeDoncker, 1994; Hay, 2000; Van Cutsem, 1983).

II. Filamentous fungi including dimorphic fungi isolated from camel skin infections.

1. *Candida* spp.: As described by Tuteja *et al* (2010; 2012) Skin candidiasis is an acute and contagious fungal infection of camel calves of less than one year of age. Infection affects almost every young calf in that particular herd. It is also called '*thikria*' by camel keepers. This vernacular word means broken piece of an earthen pot or pitcher, its shape resembles with lesion.

Lesions of the disease are initially observed on the back near the hump; later on the lesions extend towards the abdomen and may cover the whole body (Pic. 56). Lesions are initially round in shape and measure less than one centimetre in size which may enlarge to more than 10 centimetres in size and may co-alsace. The lesions are hard and fibrous crusts with papules accompanying alopecia. Scraping the lesion with scalpel reveals foul smelling



Pic.56: Initial stage of lesions of candidiasis

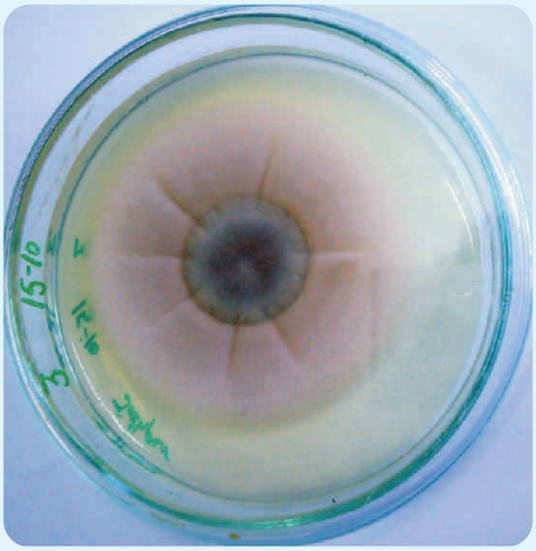
blackish brown dry crusts bunched with hairs along with roots. In prolonged cases it causes itching, uneasiness and may lead to bleeding and ulceration of skin (Pic. 57) and may result in weakness and debility of calves (Pic.58).

Pic. 57: Initiation of bleeding from lesions in candidiasis

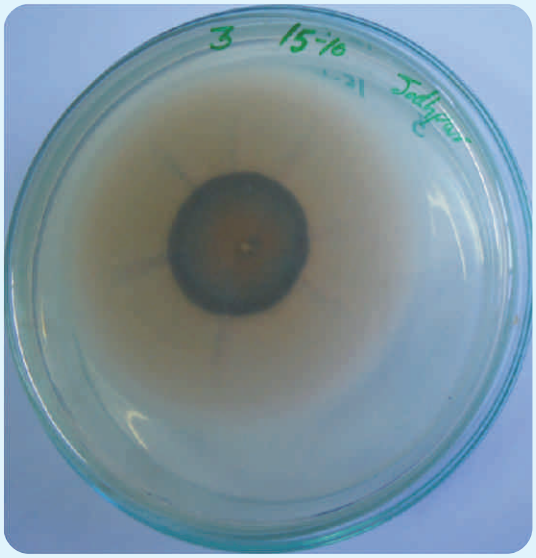
Pic. 58: Weakness and debility of calf

Survey in thickly camel populated areas revealed the prevalence of this disease everywhere in Rajasthan. Moreover in the herds surveyed none of the calves born in previous year had this infection, whereas in the same herd recently born calves had this infection. Lactating camels living in close contact with their infected suckling off springs were not infected.

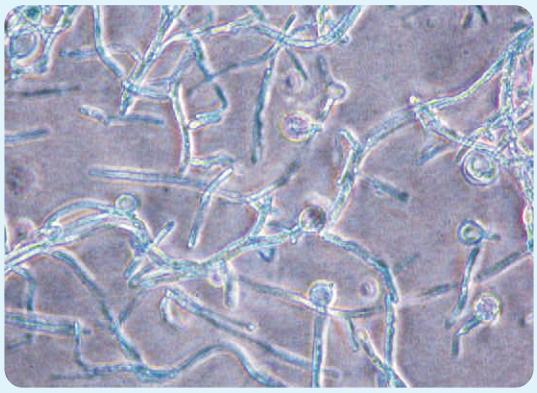
Repeated **c**ulture of skin scrapings on SDCA and germ tube formation in horse serum from all infected calves led to isolation and conformation of *Candida albicans* (Pic. 59-62).



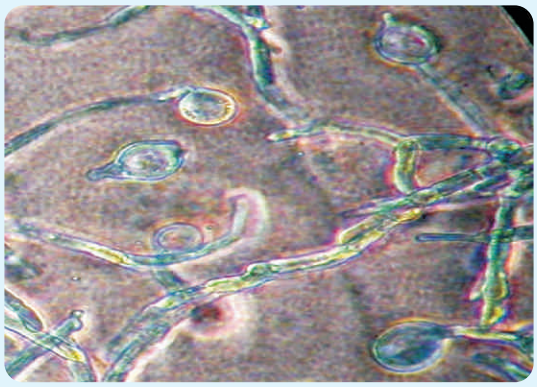
Pic. 59: Candida; colony at 28^{0}C (6 days front)



Pic. 60: Candida; colony at 28^{0}C (6 days reverse)



Pic. 61: Candida; microscopy



Pic. 62: Germ tube formation of *C. albicans*

Farmers believed it causes only morbidity in young suckling calves. At well managed camel farm, comparison of growth of five infected calves with seven healthy calves at one year of age revealed that physical condition of calves was severely affected. Taking into consideration the age, sex and breed parity of calves, average weight gain was less in infected calves (256.6±12.84 kg) compared to healthy calves (301.149±6.09 kg) under same managemental conditions.

The following line of treatment was given in five severely affected calves of approximately six month of age. Initially entire dead tissue was removed by scraping and spray bath was made with 10 percent sodium thiosulphate. Next day an ointment made of sulphur- 6gm + salicylic acid- 3gm + mustard oil- 100 ml was applied on the affected skin daily for five days. On the seventh day again spray bath was done with 10 percent sodium thiosulphate and the same ointment was applied daily for next six days. Then skin scrap was removed and 10 percent sodium thiosulphate was applied daily for next two days. Along with this treatment mineral mixture feeding was done daily for 30 days at the rate of 20 gm per calf per day. This treatment schedule resulted in complete recovery of lesions in all the five cases. Recurrence of the condition in these calves was not observed up to one year of age.

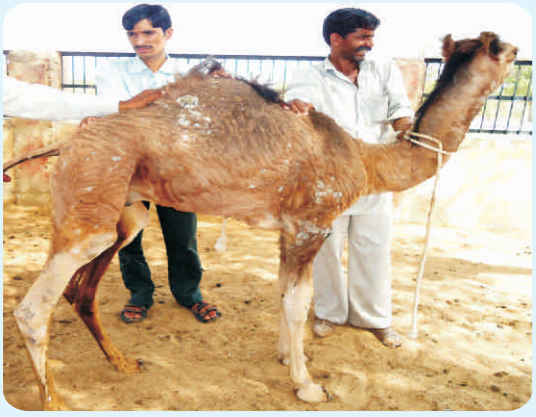
For simplified treatment options for skin candidiasis**:** Therapeutic potential of three formulations consisting of 2% potassium iodide; 6% sulphur in mustard oil; and 6% sulphur and 3% salicylic acid in mustard oil were evaluated topically in naturally occurring cases of skin candidiasis in camel calves.

Fifteen naturally infected camel calves of an organized herd were divided into three groups of five calves each, in such a way that each group should have the calves with varying degree and severity of the lesions (Pic.63-65).

In calves of all the groups initially entire dead tissue was removed by traditional method of scrapping with a piece of brick stone as adopted by the many camel owners. This method was very rapid

Pic. 63: Skin candidiasis in calf herd

Pic. 64: heavily infected camel calf



Pic. 65: low infected camel calf

and less troublesome to the animal. Aseptically collected skin scrapings of these calves were examined mycologically using SDCA plates. Following treatments regimens were given in these calves.

Gp.1: 2% potassium iodide in distilled water was applied topically with a duster cloth on alternate days till complete recovery of the lesions.

Gp.2: 6% sulphur (80% sulphur; contact fungicide used in agricultural operations) in mustard oil (*Brassica* spp.) was applied topically with a duster cloth on alternate days till complete recovery of the lesions.

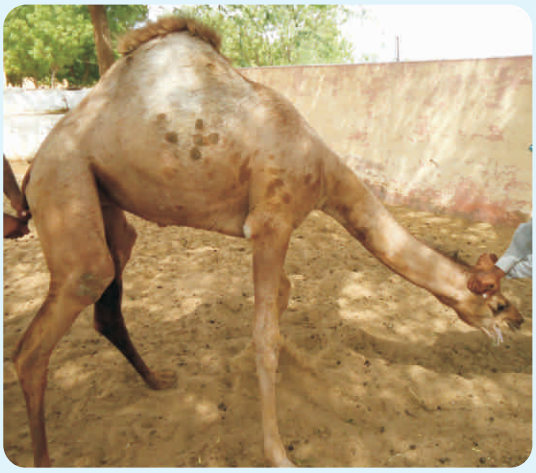
Gp.3: on the 1st day lesions were washed with sodium thiosulphate (10%) solution. Starting the next day 6% sulphur (80% sulphur; contact fungicide used in agricultural operations) and 3% salicylic acid in mustard oil (*Brassica* spp.) was applied topically with a duster cloth on alternate days till complete recovery of the lesions.

Lesions were observed before each application for growth of the fungus in terms of debris formation or visibility of mycelium, healing of the lesions in terms of healthy appearance of the skin and growth of the hairs. Finally skin scrapings from these calves were examined mycologically with in three days of the discontinuation of the therapy.

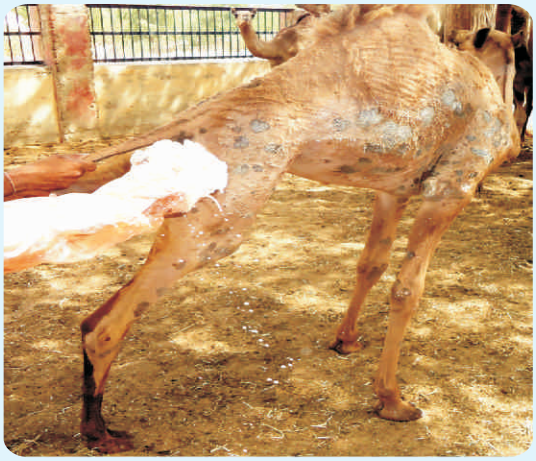
All the three treatments were found effective with almost similar application schedule but with variable duration of treatment (table 1; Pic. 66-71) and were safe without any adverse reactions noticed.

Sulphur, a yellow, non-metallic element, possesses medicinal properties, making it useful in treating many skin conditions. For nearly 70 years, sulphur has been used as a therapeutic agent, according

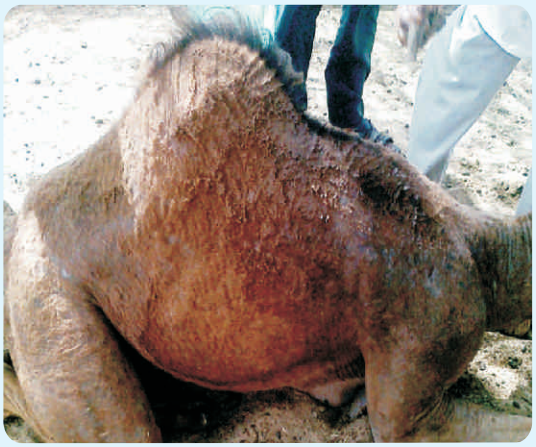
Pic. 66: calf after debris removal



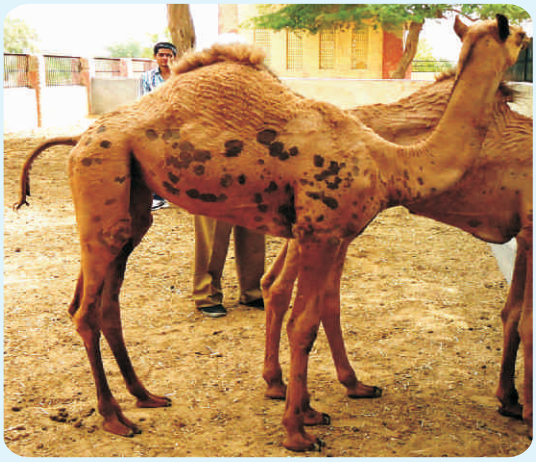
Pic. 69: calf showing lesions before recovery phase



Pic. 67: calf of gp.1 under KI application



Pic. 70: calf under last dose of oily base application



Pic. 68: calf of gp. 1 after 72 hrs of 1st treatment

Pic. 71: completely recovered calf

Table 1: Efficacy of treatment regimens against skin candidiasis (Tuteja *et al*, 2012)

Treatment groups	Duration of treatment	Gross recovery from lesions	Mycological recovery	Recovery of hair growth at the end of the treatment
Gp. 1	10 treatments (19 days)	5/5	5/5	2/5
Gp.2	8 treatments (15 days)	5/5	5/5	2/5
Gp.3	7 treatments (14 days)	5/5	5/5	3/5

to the 'Journal of Drugs in Dermatology'. Although the exact mechanism of how sulphur works is unknown, it is believed to help in part by breaking down keratin, by being toxic to fungi and by inhibiting the growth of acne. Batra (2003) evaluated mustard oil as health oil in rat model, reported glucosinolate, the pungent principle in mustard oil, to possess anti-bacterial and anti-fungal properties. Some antifungal activity has also been observed with mustard oil (Nielsen and Rios, 2000; Dhingra *et al,* 2004; Sitara *et al,* 2008). In cats the combination of oral itraconazole and topical lime sulphur rinses were effective and safe for the treatment of dermatophytosis (Newbury *et al*, 2007).

The salicylic acid may play a central role in plant disease resistance, particularly during systemic acquired resistance (Ross, 1961). Thus, the level of salicylic acid increases several fold in tobacco and cucumber after pathogen infection (Malamy *et al,* 1990; Rasmussen *et al,* 1991) and this increase is correlated with systemic acquired resistance (Malamy *et al,* 1990; Metraux *et al,* 1990). Further more, transgenic tobacco and arabidopsis plants that are unable to accumulate salicylic acid due to the expression of the bacterial nahG gene (NahG plants) fail to develop systemic acquired resistance and exhibit increased susceptibility to an infection with virulent and avirulent pathogens (Delaney *et al,* 1994; Gaffney *et al,* 1993).

Wernery *et al* (2007) repeatedly cultured *C. albicans* from skin scrapings of very young camel calves. Despite intensive treatment, the lesions did not heal until a year later, when the dromedary calves changed their fur. The physical condition of dromedary calves was affected; they were smaller and weighed less than calves in the same age group. Pal *et al* (2007) reported a case of otitis in camel due to *C. albicans*. Khosravi *et al* (2008) recorded 18.6 % of external ear canal of camel harbouring *C. albicans.* Khosravi *et al* (2009) isolated *C. albicans* from 5.8 % of healthy eye and 10.9 % of healthy nose samples of camels.

It has been reported that *C. albicans* is a commensal of the gastrointestinal tract and is an opportunistic fungus. Hence, any predisposing factors such as poor hygiene and insanitary conditions in animals including poultry may offer a chance for *C. albicans* to become pathogenic. The organisms may produce systemic candidiasis and may affect the skin. Bhoyar *et al* (2010) reported *C. albicans* in association with *Escherichia coli* to cause severe enteric infection in an Asian elephant. *C. albicans* has been isolated from stomach ulcers of country swine (Rao and

Sambamurthy, 1972); *C. parakrusei* from a nodular lesion in the intestine of goat (Singh and Singh, 1972). Cutaneous candidiasis in 5-30 weeks old Japanese quails (*Coturnix coturnix japonica*) has been reported (Shah *et al,* 1982). *C. tropicalis* from deep skin scrapings of one month old male lion cub (*Panther leo*) showed PAS positive septate branching filaments together with yeast-like structures both in degenerated epidermis and in dermal layers (Verma *et al,* 1983). Sikdar and Uppal (1985) isolated *C. guillermondi* from suspected case of bovine lymphangitis. *C. tropicalis* has been isolated from a lymph node from buffalo slaughtered for food (Pal and Ragi, 1989). Sikdar *et al* (1972) isolated *C. pseudo tropicalis* from cases of abortion among mares. Various fungi including *C. albicans* were cultured from stomach contents from aborted foetuses of horses (Monga *et al,* 1983). *C. albicans*, *C. stellatoidea*, *C. parapsilosis*, *C. guilliermondii* were isolated from swabs taken from anterior vagina and cervix of sheep and goats (Jand *et al,* 1978). Kodagali (1979) isolated *C. tropicalis* and *C. stellatoidea* from semen samples of buffalo bulls.

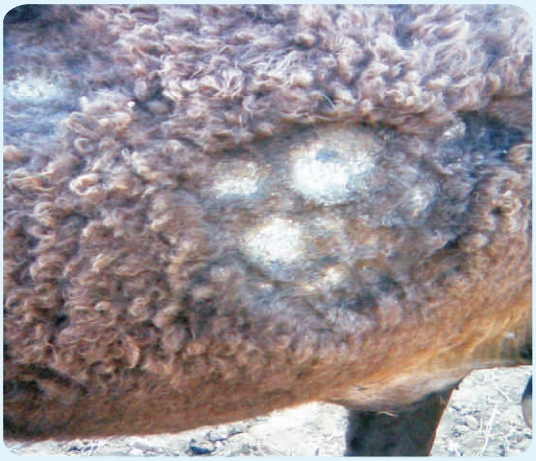
From cases of mastitis in animals *C. albicans* (Jand and Dhillon, 1975; Rahman and Baxi, 1983; Singh *et al,* 1992), *C. paraapsilosis* (Jand and Dhillon, 1975; Singh *et al,* 1992), *C. tropicallis* (Singh and Singh, 1968; Jand and Dhillon, 1975; Verma, 1988), *C. guillermondi* (Singh and Singh, 1968; Jand and Dhillon, 1975). *C. krusei* (Singh *et al,* 1992) and *C. stellatoidea* (Jand and Dhillon, 1975) have been isolated.

In human beings, the fungus that often causes cutaneous candidiasis is *C. albicans*. Candida is the most common cause of diaper rash in infants. Candida infection is common in obese and diabetic individuals. Antibiotics and oral contraceptives increase the risk of cutaneous candidiasis. Candida can also cause onychomycosis and angular cheilitis. Oral thrush, a form of candida infection of the mucous membranes of the mouth, is usually associated with taking antibiotics. It may also be a sign of immunodeficiency disorders when it occurs in adults. Individuals with candida infections are not usually contagious, though in some settings immuno compromised people can catch the infection. Candida is also the most frequent cause of vaginal yeast infections, which are extremely common and often associated with antibiotics use (Kauffman, 2007).

2. *Alternaria* spp.: As described by Tuteja *et al* (2010). This is an infectious disease mainly of camel calves of approximately one year of age and the incidence decreases with the advancement of age. Disease is present in all seasons with increase in rate towards the end of autumn and early winter and occurs more frequently in semi arid than arid region.

Vernacular word *tat* means a 'fibre carpet' *ki* means 'of' and *bimari* means 'disease'. Since during early winter when camel start growing fur and this disease starts, in which skin resembles *tat* that is why farmers call it as '*tat ki bimari*'.

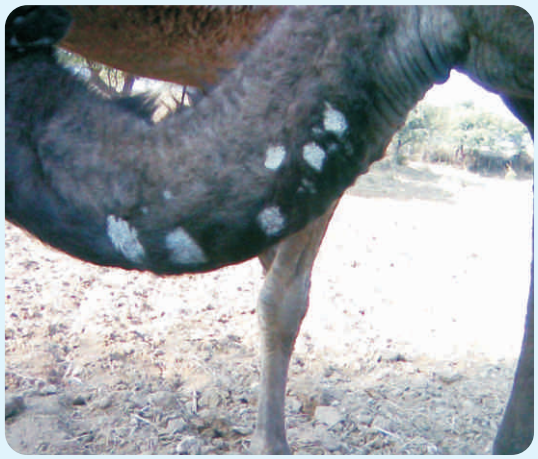
Lesions of the disease were observed throughout the body including the lips and udder (Pic. 72- 76). Lesions initially starts as small raised areas which gives roughness of the affected skin then there appears a slight whiteness at the top of the raised area.



Pic. 72: Lesions of alternariasis on abdomen

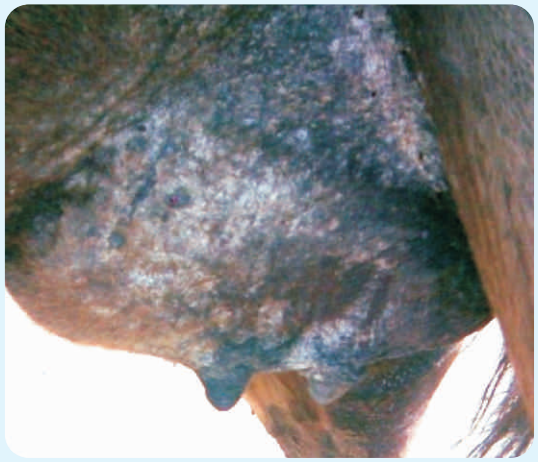
Pic. 73: Lesions of alternariasis on thighs

Lesions may enlarge to more than 10 centimetre in size, enlargement of the lesions occur in centrifugal manner and later the lesions may coalesce. During development of the lesions necrosis follows alopecia. The general presentation is a



Pic. 74: Lesions of alternariasis on neck

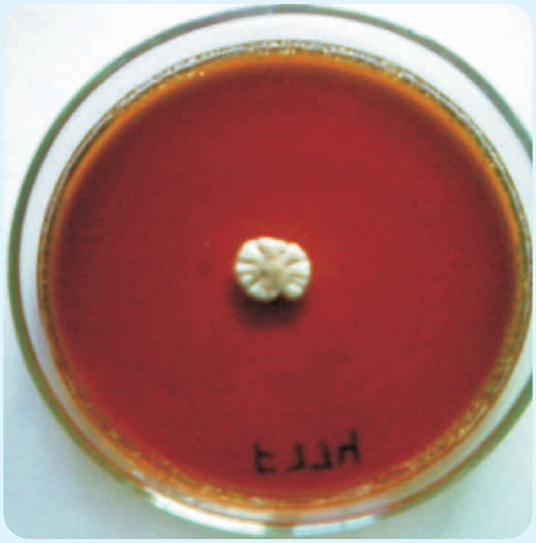
Pic. 75: Lesions of alternariasis on lips



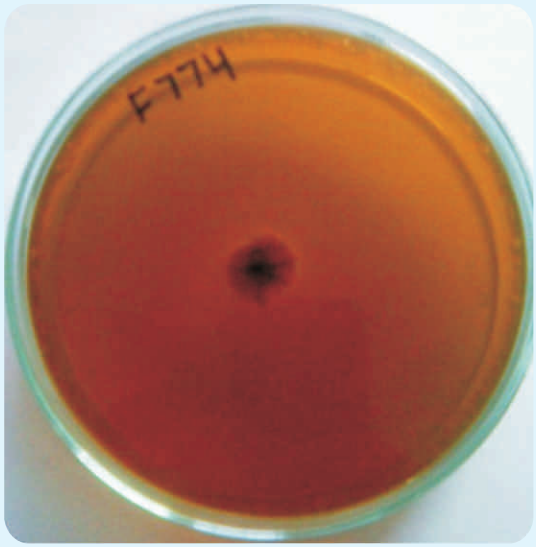
Pic. 76: Lesions of alternariasis on udder

regular and circular alopecia with erythematous margin and a thick desquamation. Finally the lesions are observed as white dry areas. Scrapping the lesion with scalpel reveals skin necrosis just like a layer of granular lime deposit about half centimetre in thickness. In untreated cases it causes itching, uneasiness and may leads to bleeding and ulceration which results in weakness and debility of calves.

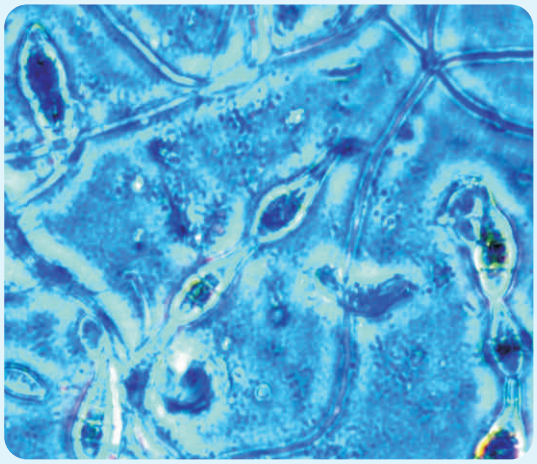
Repeated culture of skin scrapings on SDCA led to isolation of *A. alternata* (Pic. 77-79). Alternaria species are ubiquitous in nature and are commonly considered saprophytic contaminants. Thus, cultural examination of scrapings requires cautious evaluation. Grossly alternaria grows rapidly and matures within five days. The colony of *A. alternaria* is flat, downy to cottony and may eventually be covered by greyish, short, aerial hyphae. The reverse side is typically brown to black due to pigment production. Microscopically alternaria has septate, dark hyphae. Conidiophores are also septate and sometimes have a zigzag appearance. They bear simple or branched large conidia (8-16 x 23-50 µm) which have both transverse and longitudinal septations. These conidia may be observed singly or in acropetal chains and may produce germ tubes. They are ovoid to obclavate, darkly pigmented, muriform and smooth or roughened. The end of the conidia nearest the conidiophores is round



Pic. 77: Alternaria;
colony (5 days front)



Pic. 78: Alternaria;
colony (5 days reverse)



Pic. 79: *A. alternata;* conidiophores

while it tapers towards the apex. This gives the typical beak or club-like appearance of the conidia.

Incorporating the ethno veterinary knowledge of the farmers for the treatment of this condition, four severely affected cases of calves of approximately one year of age were selected at the farmer's field. Initially entire dead tissue was removed by scraping and then an ointment containing 6% sulphur and 3% salicylic acid in mustard oil was applied on the affected skin daily for seven days. This treatment resulted in complete recovery of the lesions.

Khosravi *et al* (2008) recorded 6.8 % of external ear canal of camel harbouring *A. alternata*. Cutaneous infections caused by alternaria are often associated with debilitating diseases or conditions. Cabanes *et al* (1988) reported a phaeohyphomycotic infection caused by *A. alternata* in a 5-month-old Spanish mare. Salkin and Stone (1974) reported isolation of *A. alternata* from a subcutaneous infection in the ear of a white-tailed deer. Reddy *et al* (1974) experimentally exposed rabbits and guinea pigs to *A. alternata* by intradermal and intraperitoneal injection and by scari fication. It caused superficial mycosis when inoculated by scarification. Direct intraperitoneal inoculation of *A. alternata* into animals has been reported to be either lethal (Ohashi, 1960) or harmless (Reddy *et al,* 1974). Dye *et al* (2005) developed an indirect ELISA for the detection of anti-alternaria IgG antibodies in serum to determine the prevalence of alternaria exposure in domestic cats. The cats with disease caused by alternaria infections did not have significantly higher concentrations of antibody than the healthy cats or cats with other diseases. Dovgich (1981) observed zoonotic significance of *A. alternata*. In one case a horse was infected when eating clover infected with this fungus. The horse showed symptoms of dermatomycosis on the chest and belly. The same horse transmitted infection to his owner who showed similar symptoms on his hands.

A. alternata have been isolated from human cutaneous or subcutaneous infections (Singh *et al,* 1990; De Hoog and Vitale, 2007; Mbata and Nwajagu, 2007). It is among the causative agents of otitis media in agricultural field workers (Wadhwani and Srivastava, 1984). *Alternaria* spp. has emerged as opportunistic pathogens particularly in patients with immuno suppression such as the bone marrow transplant patients (Morrison *et al,* 1993; Vartivarian *et al,* 1993). Cases of onycho mycosis, sinusitis, ulcerative cutaneous infections and keratitis as well as visceral infections and osteomyelitis have been reported (Anaissie *et al,* 1989; Garau *et al,* 1977; Manning *et al,* 1991; Schell, 2000). In immunocompetent patients, alternaria colonizes the paranasal sinuses, leading to chronic hypertrophic sinusitis. In immuno compromised patients the colonization may end up with development of invasive disease (Vennewald *et al,* 1999).

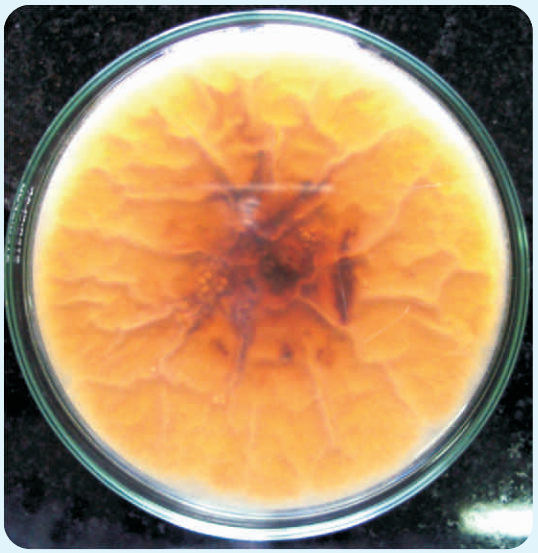
3. *Scopulariopsis* spp.: (Tuteja *et al.* communicted) *Scopulariopsis brevicaulis* is basically a fungus causing human infections.

It was isolated and identified as cause of skin infection in a herd of camel at Jhunjhnu. This infection occurred after heavy rains in the year 2010, leading to high humidity in the environment. In a herd of 147 camels, several hyperkeratotic nodules were detected on the back of few animals. Lesions were generalized in distribution but occurred particularly on the abdomen. Lesions were observed more under the hairy portion of the skin. Cutting the hairs revealed more clear visibility of the lesions. After about 15 days there occurred incrustation of the nodules, which gave appearance of patchy skin necrosis. These lesions measured up to five centimetre in diameter (Pic. 82-83). A total of 40 camels in this herd had this infection. All ages of the camel were affected but calves up to two years of age were more severely affected.

Pic. 80: Scopulariopsis hyperkeratotic nodules

Pic. 81: Scopulariopsis lesions of large patchy skin necrosis

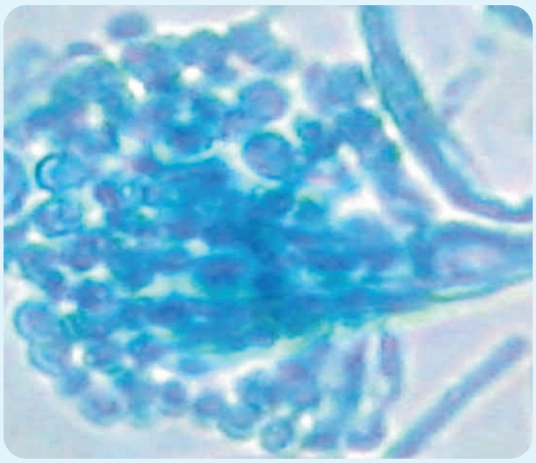
Pic. 82: *S. brevicaulis;* colony (10 days front)



Pic. 83: *S. brevicaulis;* colony (10 days reverse)

Scopulariopsis colonies grow moderately rapidly and are granular to powdery. Front colour is white initially and

lbecomes light brown or buff tan in time. Reverse colour is usually tan with brownish centre (Pic.85-86). Microscopically septate hyphae, conidiophores are hyphae-like and simple or branched. Lemon-shaped, roughened conidia with truncated bases produced from the tips of annellidic conidiogenous cells. The annellides were produced singly or in penicillate heads they were cylindrical and slightly swollen (Pic. 84).



Pic. 84: *S. brevicaulis;* annellides in penicillate heads

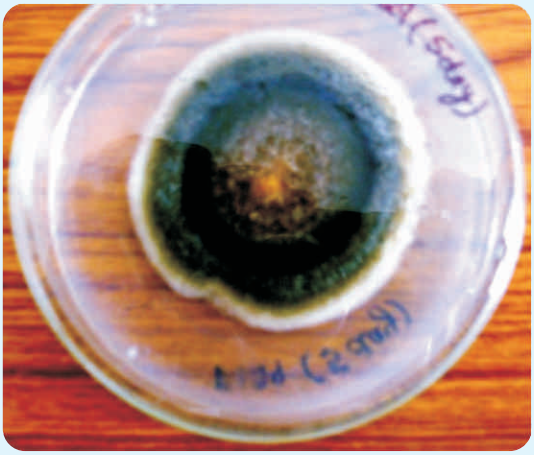
Scopulariopsis has been recovered from 53.3 % of bovine claw samples (Abdel-Gawad, 1989) and from 1.3 % of hair samples (Bagy, 1986). Ogawa *et al* (2008) isolated *S. brevicaulis* from the skin of a 6-month-old Japanese black female calf with hyperkeratotic nodules spread over almost the entire body surface. They first reported the disease caused by *S. brevicaulis* in animals. The fungus has also been isolated from equine hooves (Keller *et al,* 2000), buffalo claws (Abdel-Gawad, 1989), canine hair (Bagy, 1986) and ducks claws (Abdel-Gawad and Moharram, 1989).

In human, various cutaneous lesions include subcutaneous granulomas on the cheek (Bruynzeel and Starink, 1998) and forearm (Sellier *et al,* 2000); ulcerative granulomatous cheilitis (Creus *et al,* 1994); neutrophilic follicular inflammation on the legs (Cox and Irving, 1993) and dermal spongiosis on the sole (Ginarte *et al,* 1996). This fungus do causes onychomycosis (Tosti *et al,* 1996; Goettmann-Bonvallot, 2003; Romano *et al,* 2005) and keratitis (Malecha, 2004). Dhar and Carey (1993) reported the area of the skin lesions in the AIDS patient was much greater than in the other patients. In the herd animals described here, the cutaneous lesions were generalized and thus distinct from the localized ones found in immunocompitent human patients. In the herd examined, in this particular season almost 90 percent of the camel population suffered with respiratory problem with mucopurulent nasal discharge, anorexia *etc*. The etiology of this disease is still unclear but thought to be immuno suppressive disease for camels. This infection was species specific to camels. Other apparent clinical abnorm alities observed in the herd under investigation were pica in 20 animals, mastitis 14 animals and weakness in 33 animals. In view of the clinical findings (anorexia and emaciation), camels may have undergone an immunosuppressive stage, which might have increased its susceptibility to the fungal dermatitis.

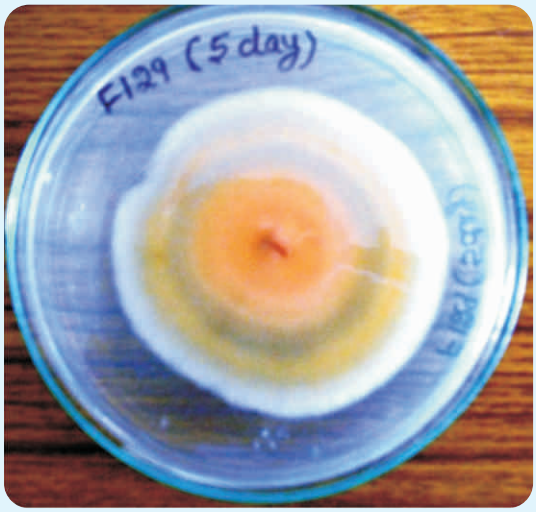
Some of the authors have suggested that *S. brevicaulis* is resistant *in vitro* to

amphotericin-B, flucytosine and azole compounds (Aguilar *et al,* 1999; Johnson *et al,* 1999). Because of its resistance, invasive infections due to *S. brevicaulis* are unlikely to respond to particular antifungal treatment and other therapeutic approaches should be considered (*e.g.* combined therapy and immunotherapy), particularly in immuno suppressed patients with disseminated mycoses (Estrella *et al,* 2003).

4. *Sporothrix* spp.: (Tuteja *et al*. communicated) *Sporothrix schenckii* is a thermally dimorphic fungus and the macroscopic morphology varies depending on the temperature of growth. At 28°C, colonies are slow growing, moist and glabrous with a wrinkled and folded surface. Some strains may produce short aerial hyphae and pigmentation may vary from white to cream to black (Pic. 85-86). Conidiophores arise at right angles from the thin septate hyphae and are usually solitary, erect and tapered towards the apex. Conidia are formed in clusters on tiny denticles by sympodial proliferation of the conidiophore,

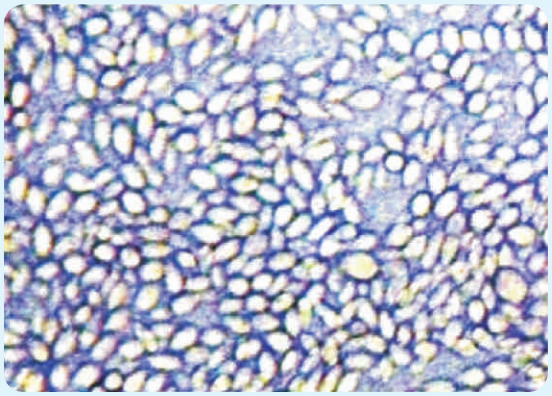


Pic. 85: *S. schenckii;*
colony at 28°C (5 days front)



Pic. 86: *S. schenckii;*
colony at 28°C (5 days reverse)

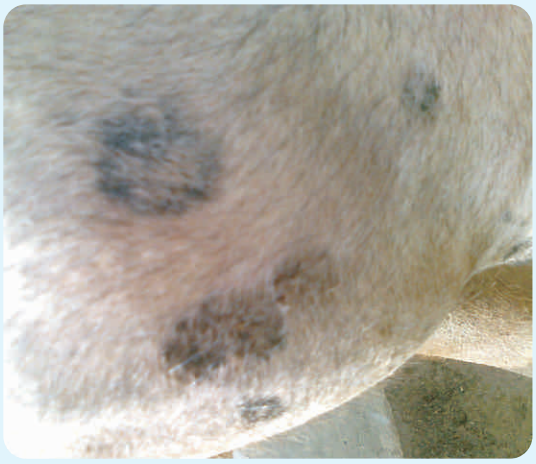
their arrangement often suggestive of a flower. As the culture ages, conidia are subsequently formed singly along the sides of both conidiophores and undifferentiated hyphae. Conidia are ovoid or elongated, hyaline, one-celled and smooth-walled. In some isolates, solitary, darkly pigmented, thick-walled, one-celled, obovate to angular conidia may also be observed along the hyphae. At 37°C, colonies are glabrous, white to greyish yellow and yeast-like, consisting of spherical or oval budding yeast cells (Pic. 87).



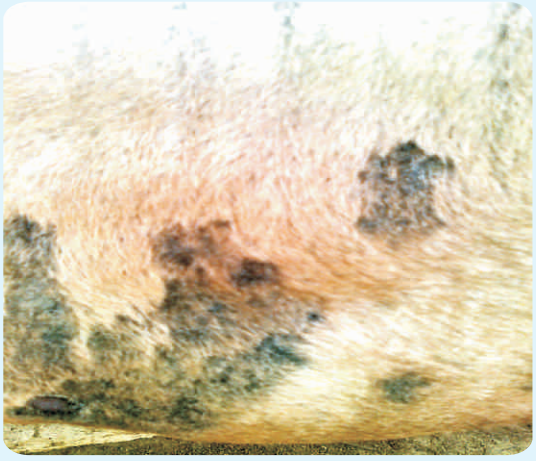
Pic. 87: *S. schenckii;*
yeast cells at 37°C

As shown in Pic. 88-89, the type of moist, multicentric lesions from such lesions, *S. schenckii* was isolated.

S. schenckii, has two important mechanisms through which its potential to infect the mammalian host is maximized. First, *S. schenckii* has the ability to change phases to an ascomycete telemorph that survives on living or decaying plant material. This fungus has been isolated from decaying vegetation such as thorns, straw, hay, wood, moss and soil. Second, after entering the skin via puncture, bite or scratch, the fungus converts to a yeast phase, thereby causing lesions locally and possibly systemically in the mammalian host. *S. schenckii* can be found worldwide. Sporotrichosis is particularly common in the tropics where high humidity and temperatures promote fungal growth.



Pic. 88: *S. schenckii;* moist, multicentric lesions on the belly



Pic. 89: *S. schenckii;* fast spreading moist lesions on the belly

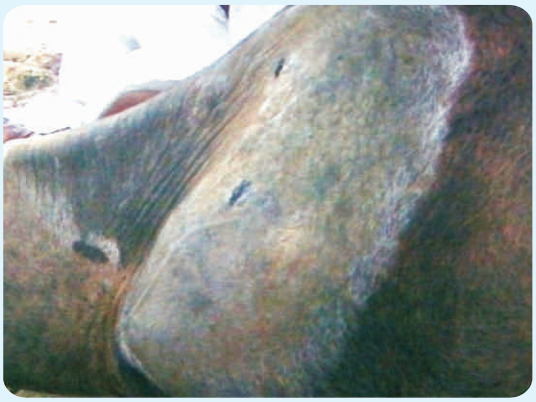
Clinically sporotrichosis may be grouped into 3 forms: lymphocutaneous, cutaneous and disseminated. In lympho cutaneous form small, firm dermal to subcutaneous nodules, develop at the site of inoculation. As infection ascends along the lymphatic vessels, cording and new nodules develop. Lesions ulcerate and discharge a sero hemorrhagic exudate. Although systemic illness is not seen initially, chronic illness may result in fever, listlessness and depression. The cutaneous form remains localized to the site of inoculation, although lesions may be multicentric. Disseminated sporotrichosis is rare but potentially fatal and may develop with neglect of cutaneous and lympho cutaneous forms.

S. schenckii is the causative agent of sporotrichosis or rose handler's disease (Rex and Okhuysen, 2000). Sporotrichosis is a subcutaneous infection with a common chronic and a rare progressive course. Following entry of the infecting fungus through the skin via a minor trauma infection may spread via the lymphatic route and nodular lymphangitis may develop (Kostman and DiNubile, 1993; Tomimori-Yamashita *et al,* 1998). Patients infected

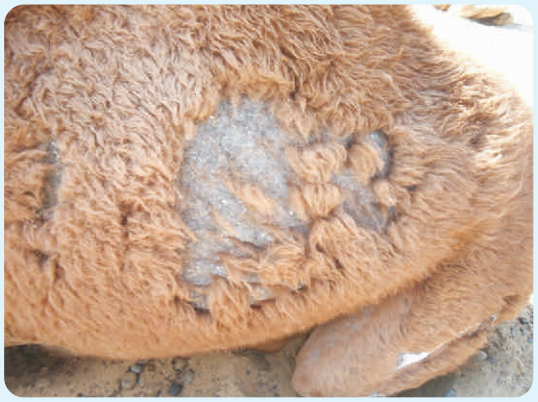
with *S. schenckii* may be misdiagnosed as pyoderma gangrenosum due to the large ulcerations observed during the course of sporotrichosis (Byrd *et al,* 2001).

Pulmonary (England and Hochholzer, 1987; Gori *et al,* 1997; Kauffman, 1999) and osteoarticular infections (Edwards *et al,* 2000; Lesperance *et al,* 1988), granulama tous tenosynovitis and carpal tunnel syndrome (Stratton *et al,* 1981), bursal infection (Wang *et al,* 2000), endoph thalmitis (Witherspoon *et al,* 1990), meningitis (Penn *et al,* 1992), invasive sinusitis (Morgan and Reves, 1996) and disseminated (Morgan *et al,* 1984; Ware *et al,* 1999) sporotrichosis have been described. The infection remains localized in immunocompetent individuals while fungemia and disseminated infection may be observed in immunocompromised patients, such as those with AIDS (Al-Tawfiq and Wools, 1998; Edwards *et al,* 2000; Kurosawa *et al,* 1988; Morgan and Reves, 1996; Ware *et al,* 1999). Fatal fungemia may develop also in patients with diabetes mellitus and alcoholism (Castrejon *et al,* 1995). Primary granulamatous pneumonia without any cutaneous disease may develop in alcoholics (England and Hochholzer, 1987).

Itraconazole is generally used for the treatment of lymphocutaneous infection (Conti *et al,* 1992; Lortholary *et al,* 1999; Sharkey-Mathis *et al,* 1993), while amphotericin-B is indicated for severe infections or when itraconazole therapy fails (Kauffman *et al,* 2000). Potassium iodide is one of the oldest therapeutic modalities used for treatment of sporotrichosis (Tomimori-Yamashita *et al,* 1998).

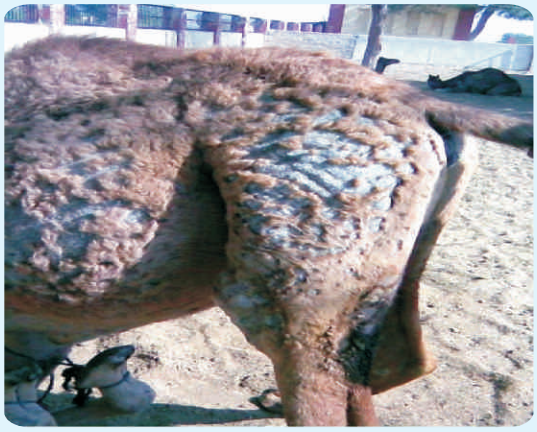


Pic. 90: Acid burn like lesion dry and fast spreading

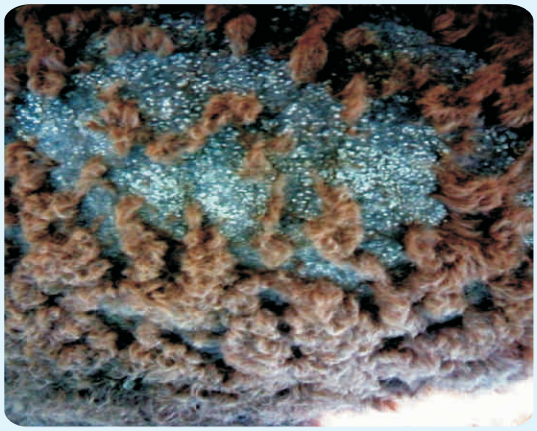


Pic. 91: Dry rapidly spreading lesion on the flank

Pic. 92: Semi dry rapidly spreading lesion on the flank



Pic. 93: Semi dry rapidly spreading lesion on the thigh

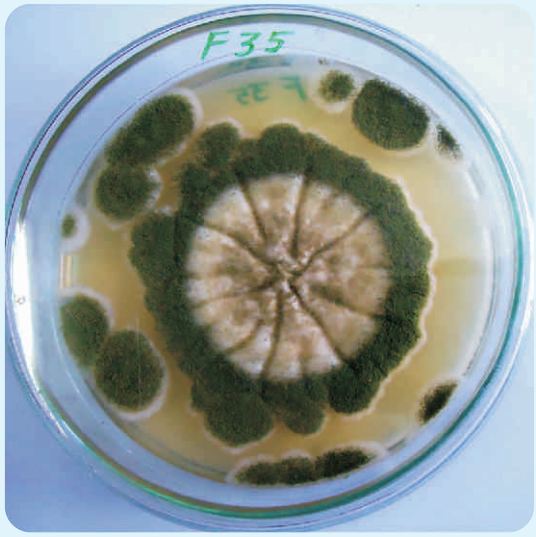


Pic. 94: Moist lesion on the flank with lot of granular debris deposit

Pic. 95: Moist foul smelling lesion on the flank with lot of thick debris deposit

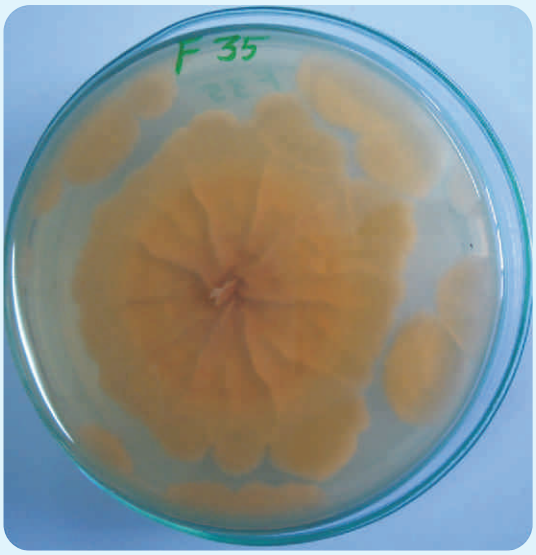
From advanced complicated cases of dermatophytosis (Pic. 90-95) along with dermatophytes (*Microsporum* and *Trichophyton* spp.) other filamentous fungi including dimorphic fungi were also isolated. These fungi included *Basidiobolus* spp., *Coccidioides* spp., *Penicillium* spp., *Curvularia* spp., *Exserohilum* spp., *Absidia* spp., *Rhizopus* spp. and *Aspergillus* spp.

5. *Basidiobolus* spp.: (Tuteja *et al.* communicated) *Basidiobolus* is a filamentous fungus. Although it is cosmopolitan, the human infections due to basidiobolus are reported mostly from Africa, South America and tropical Asia. In the past, clinical isolates of basidiobolus were classified as *B. ranarum, B. meristosporus and B. haptosporus*. But recent taxonomic studies based on antigenic analysis, isoenzyme banding and restriction enzyme analysis of rDNA indicate that all human pathogens belong to *B. ranarum*.



Pic. 96: *B. ranarum;* colony with satellite colonies (10 days front)

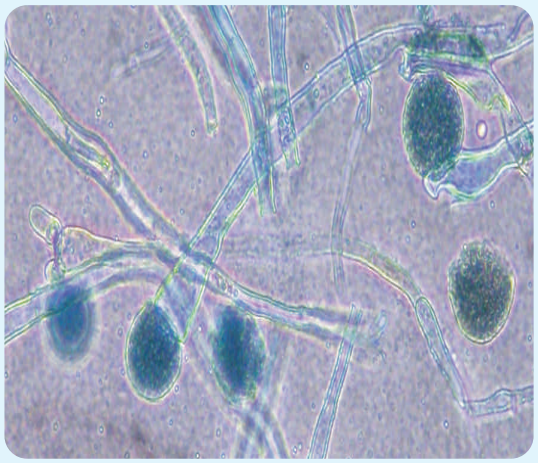
Colonies are moderately fast growing at 28^{0}C, flat, yellowish-grey to creamy-grey, glabrous, becoming radially folded and covered by a fine, powdery, white surface mycelium. Satellite colonies are often formed by germinating conidia ejected from the primary colony (Pic. 96-97).



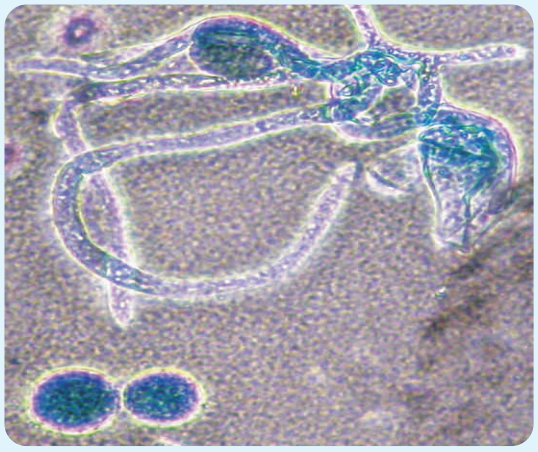
Pic. 97: *B. ranarum;* colony (10 days reverse)

Microscopic examination usually shows the presence of large vegetative hyphae forming numerous round, smooth, thick-walled zygospores that have two closely appressed beak-like appendages.

Two types of asexual spores are formed: primary and secondary. Primary spores are globose, one-celled, solitary and are forcibly discharged from a sporophore. The sporophore has a distinct swollen area just below the spore that actively participates in the discharge of the spore. Secondary spores are clavate, one-celled and are passively released from a sporophore. Their sporophores are not swollen at their bases. The apex of the passively released spore has a knob-like adhesive tip. These spores may function as sporangia, producing several sporangio spores (Pic. 98-99).



Pic. 98: *B. ranarum;* primary spore with swollen sporophore



Pic. 99: *B. ranarum;* zygospores along with beaked one

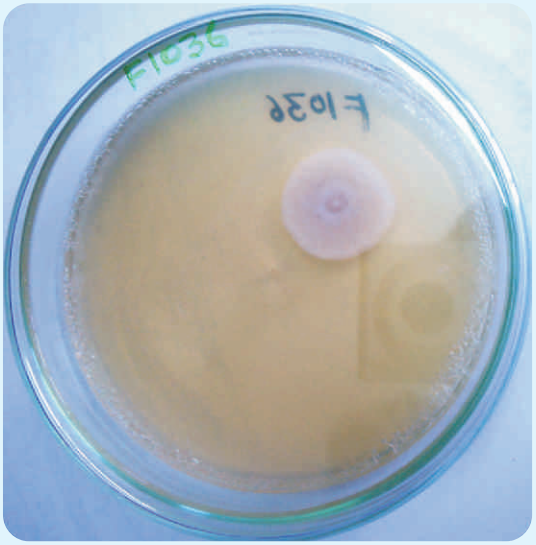
B. ranarum causes subcutaneous chronic zygomycosis in man. This infection is also called entomophthoromycosis basidiobolae. Entomophthoromycosis is characterized by the formation of firm and non tender swellings, generally on the extremities, trunk and rarely other parts of

the body (Naniwadekar *et al,* 2009). The mode of infection is not known but it is assumed that traumatic implantation may play a role (Khan *et al,* 2001). Basidiobolus is a true pathogen, causing infections in immuno competent host. It is also causing angioinvasive infections in immuno compromised patients (Ribes *et al,* 2000) and gastrointestinal infections (Zavasky *et al,* 1999). Most patients with entomoph thoromycosis respond very well to oral potassium iodide therapy as well as to azoles, particularly itraconazole (Sujatha *et al,* 2003), oral ketoconazole and fluconazole may be of help in some cases (Restrepo, 1994).

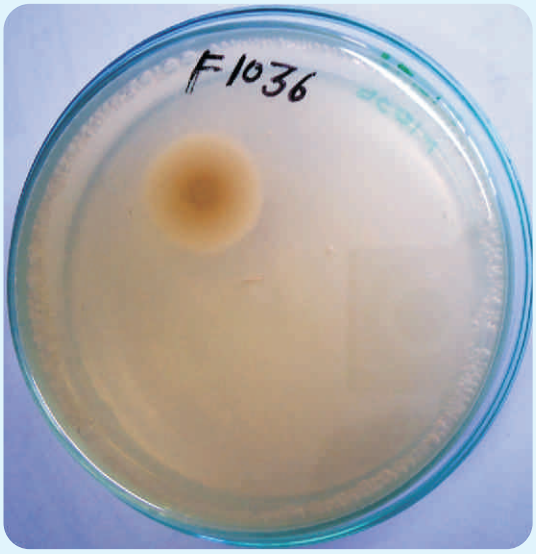
B. ranarum/ haptosporus causes infection of the subcutaneous tissues located in anatomical areas other than the face in horses (Connole, 1973; Miller and Pott, 1980; Miller and Campbell, 1984; Owens *et al,* 1985; Speare and Thomas, 1985) and dogs (Miller and Turnwald 1984; Greene *et al,* 2002; Grooters, 2003). Two cases of cutaneous phycomycosis of horses were successfully treated by surgery and potassium iodide therapy (Owens *et al,* 1985).

6. *Coccidioides* spp.: (Tuteja *et al*. communicated) Coccidioides are thermally dimorphic fungi found in soil particularly at warm and dry areas with low rain fall, high summer temperatures and low altitude. The two species *Coccidioides immitis* and *C. posadasii* are morphologically identical but genetically and epidemiologically distinct (Fisher *et al,* 2001; Fisher *et al,* 2002). The two species can be distinguished by genetic analysis and different rates of growth in the presence of high salt concentrations (*C. posadasii* grows more slowly). *C. immitis* is geographically limited to California's San Joaquin valley region, whereas *C. posadasii* is found in the desert southwest of the United States, Mexico and South America. The two species appear to co-exist in the desert southwest and Mexico specifically inhabits alkaline soil. *C. immitis/posadasii* is a pathogenic fungus and is among the causative agents of true systemic (endemic) mycoses. Imported cases may occur following travel to endemic areas (Cairns *et al,* 2000).

On SDCA, *C. immitis/posadasii* colonies grow rapidly. At 25 or 37°C, the colonies are moist, glabrous, membranous, and greyish initially, later producing white and cottony aerial mycelium. With age, colonies become tan to brown in colour (Pic. 100-102).



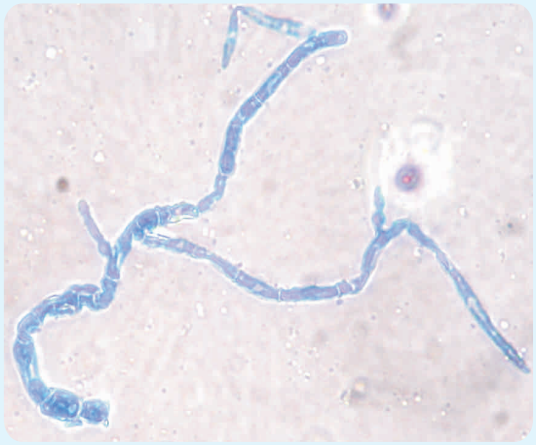
Pic. 100: *C. immitis;*
colony at 25°C (5 days front)



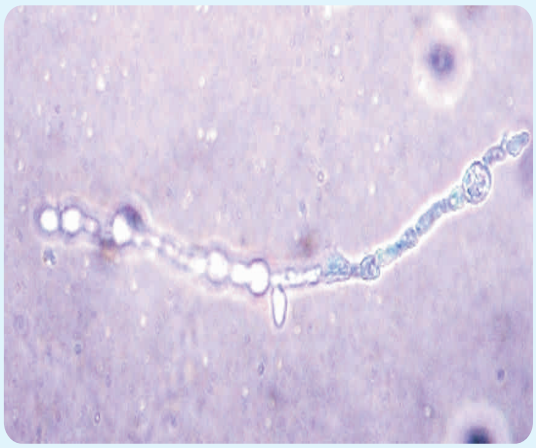
Pic. 101: *C. immitis;* colony at 25°C (5 days reverse)

Pic. 102: *C. immitis;* colony at 37°C (7 days front)

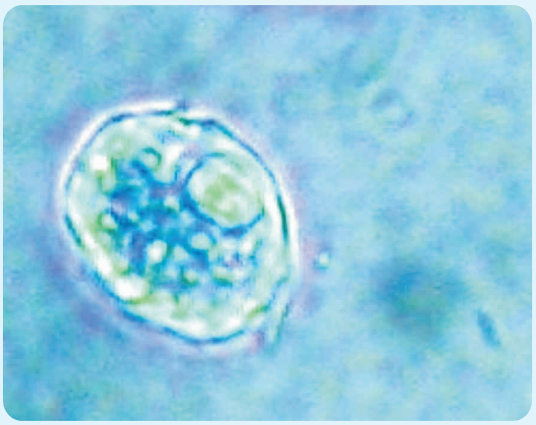
Microscopic morphology varies depending upon the incubation temperature. At 25°C, hyphae and arthroconidia are produced (Pic. 103-104). Arthroconidia are thick-walled, barrel-shaped. These arthroconidia alternate with empty disjuncture cells. At 37°C, large, round, thick-walled spherules filled with endospores are observed (Pic. 105). The definitive identification of an isolated *C. immitis/posadasii* strain requires demonstration of spherule production *in vitro*, use of DNA probes, application of exoantigen tests or demonstration of spherule production *in vivo* by animal experiments (Larone, 1995; Lindsley *et al,* 2001). Molecular typing studies have also been initiated and appear useful in identification (McEwen *et al,* 2000).



Pic.103: *C. immitis;* arthrospore formation in the hyphae



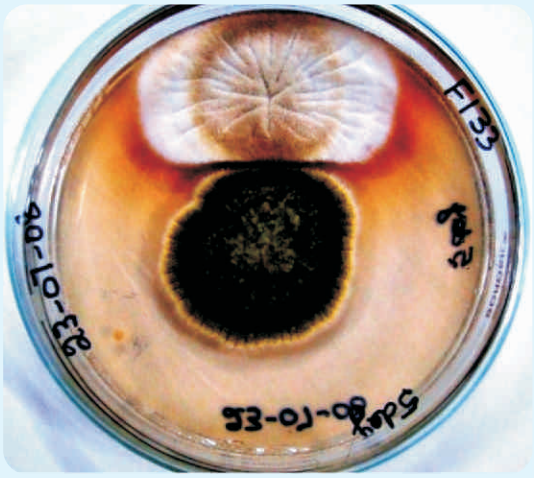
Pic. 104: *C. immitis;* arthroconidia at 25°C



Pic. 105: *C. immitis;* spherule with endospores

C. immitis/posadasii is the causative agent of coccidioidomycosis in humans. Coccidioidomycosis is one of the true systemic mycoses (Galgiani, 1999). Inhalation of the dry arthroconidia of *C. immitis/posadasii*, which are carried by dust storms, initiates the infection. The infection remains as an acute and self-limited respiratory infection in most exposed hosts. Spontaneous healing is observed in as high as 95% of the otherwise healthy hosts. It may progress to a chronic and sometimes fatal disease in others. Airway coccidioidomycosis involving the endotracheal and endobronchial tissues may develop (Polesky *et al,* 1999). Hematogenous spread of the organism results in infection of skin, bones, joints, lymph nodes, adrenal glands and central nervous system (Ampel *et al,* 1986; Bayer and Guze, 1979; Blair and Logan, 2001). Dissemination may occur particularly during pregnany and carries a high risk of mortality (Powell *et al,* 1983). Although coccidioidomycosis basically affects otherwise healthy immunocompetent hosts due to the true pathogenic nature of the fungus, it may also develop in immuno compromised patients, such as patients with AIDS and organ transplant recipients (Blair and Logan, 2001; Medoff *et al,* 1992). Coccidioidomycosis has also been described in warm-blooded water animals such as bottlenose dolphins (Reidarson *et al,* 1998) and horses (Ziemer *et al,* 1992).

Amphotericin B, itraconazole (Li *et al,* 2000) and voriconazole (Kappe, 1999; Li *et*



Pic. 106: *P. marneffei;* colony with green saprophytic spp. (5 day front)

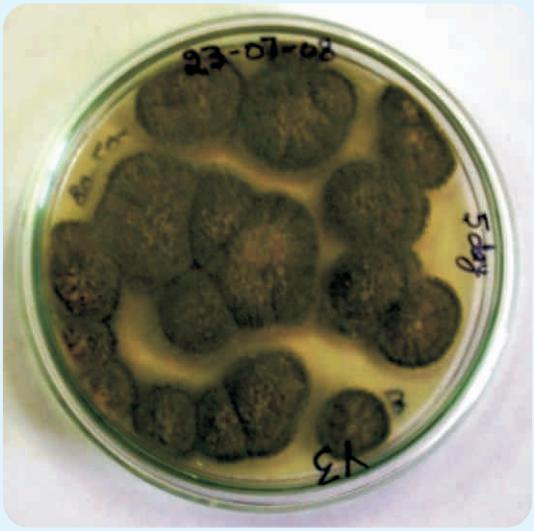
Pic. 107: *P. marneffei;* colony producing red diffusible pigment (5 day reverse)

al, 2000) are active *in vitro.* Nikkomycins are additive to synergistic *in vitro* with fluconazole or itraconazole against this fungus (Li and Rinaldi, 1999). Amphotericin - B (Drutz, 1983) and azoles, such as fluconazole, itraconazole and ketoconazole are used for the treatment of coccidioido mycosis (Blair and Logan, 2001; Galgiani *et al,* 2000; Medoff *et al,* 1992). Animal experiments suggest that caspofungin (Gonzalez *et al,* 2001), sordarins (Aviles *et al,* 2001; Clemons and Stevens, 2000; Odds, 2001) and nikkomycins (Hector *et al,* 1990) are also promising in treatment of coccidioido mycosis.

Researches focused on vaccine development for prevention of coccidioido mycosis are in progress (Dixon *et al,* 1998; Jiang *et al,* 1999; Pappagianis, 1993; Peng *et al,* 1999; Zimmermann *et al,* 1998).

7. *Penicillium* spp.: (Tuteja *et al*. communicated) The most common species of *Penicillium are: P. chrysogenum*, *P. citrinum*, *P. janthinellum*, *P. marneffei* and *P. purpurogenum*. Only one species *P. marneffei is* thermally dimorphic and other species are filamentous fungi.

Identification to species level is based on macroscopic morphology and microscopic features (de Hoog *et al,* 2000). The colonies of penicillium are rapid growing, flat, filamentous, velvety, woolly or cottony. Colours of the colonies are initially white and become blue green, gray green, olive gray, yellow or pinkish in time. Reverse of colony is usually pale to yellowish (Pic. 108-109).

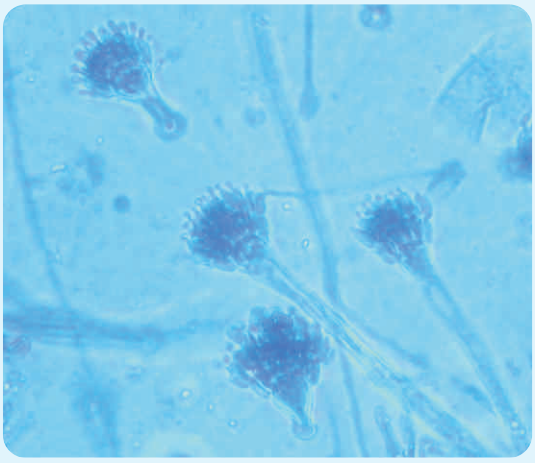


Pic. 108: *Penicillium* spp.; colony (5 days front)

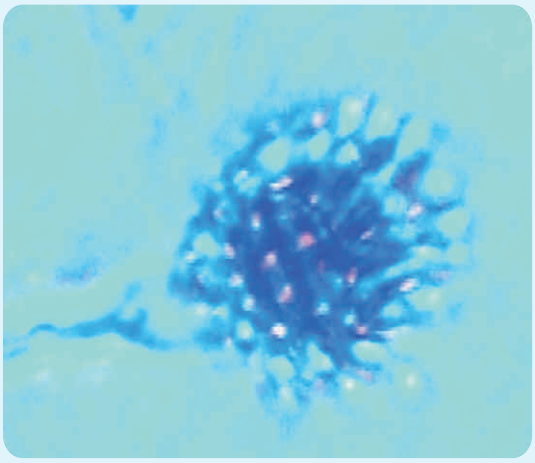
Pic. 109: *Penicillium* spp.; colony (5 days reverse)

Microscopically septate hyaline hyphae (1.5 to 5 µm in diameter), simple or branched conidiophores, metulae, phialides and conidia are observed. Metulae are secondary branches that form on conidiophores. The metulae carry the

flask-shaped phialides. The organization of the phialides at the tips of the conidiophores is very typical. They form brush-like clusters which are also referred to as 'penicilli'. The conidia (2.5- 5 µm in diameter) are round, unicellular and visualized as unbranching chains at the tips of the phialides (Pic. 110-111).



Pic. 110: Penicillium; hyphae, conidiophores, phialides



Pic. 111: Penicilli; phialides and conidia

P. marneffei is thermally dimorphic produces filamentous, flat, radially sulcate colonies at 25°C. These colonies are bluish-gray-green at centre and white at the periphery. The red, rapidly diffusing, soluble pigment observed from the reverse is very typical (Pic. 106-107), at 37°C colonies are cream to slightly pink in colour and glabrous to convolute. Microscopically the yeast phase is visualized as globose to elongated sausage-shaped cells (3 to 5 µm) that multiply by fission. Microscopically the filamentous stage of *P. marneffei* is similar to other species.

P. marneffei is pathogenic and endemic specifically in Southeast Asia where it infects bamboo rats which serve as epidemiological markers and reservoirs for human infections. *P. marneffei* is pathogenic particularly in patients with AIDS and its isolation from blood is considered as an HIV marker in endemic areas (Deng *et al,* 1988; Singh *et al,* 1999; Supparatpinyuo *et al,* 1992). *P. marneffei* infections have also been reported in patients with haematological malignancies and those receiving immunosuppressive therapy (Wong *et al,* 2001). Infection is acquired via inhalation and results in initial pulmonary infection followed by fungemia and dissemination of the infection (Cheng *et al,* 1998; Garbino *et al,* 2001; Rimek *et al,* 1999; Singh *et al,* 1999). The lymphatic system, liver, spleen and bones are usually involved. Acne-like skin papules on face, trunk and extremities are observed during the course of the disease. *P. marneffei* infection is often fatal.

Penicillium spp. are known to produce mycotoxins (Pitt *et al,* 2000). Penicillium has been isolated from patients

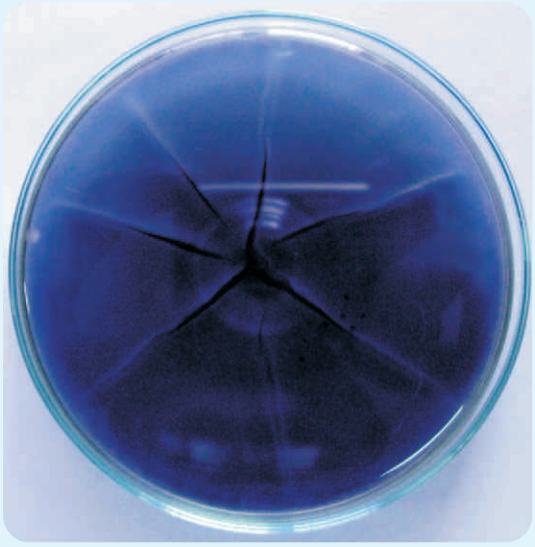
with keratitis (Deshpande and Koppikar, 1999), endophtalmitis, otomycosis, necrotizing esophagitis, pneumonia, endocarditis, peritonitis and urinary tract infections. Most penicillium infections are encountered in immunosuppressed hosts. Corneal infections are usually post-traumatic (Deshpande and Koppikar, 1999). In addition to its infectious potential, *P. verrucosum* produces a mycotoxin, ochratoxin-A, which is nephrotoxic and carcinogenic. The production of the toxin usually occurs in cereal grains at cold climates (Pitt, 2000).

Amphotericin B, oral itraconazole and oral fluconazole have so far been used in treatment of penicilliosis marneffei (Cheng *et al,* 1998; Lortholary *et al,* 1999; Rimek *et al,* 1999). Oral itraconazole was found to be efficient when used prophylactically against penicilliosis marneffei in patients with HIV infection (Chariyalertsak *et al,* 2001).

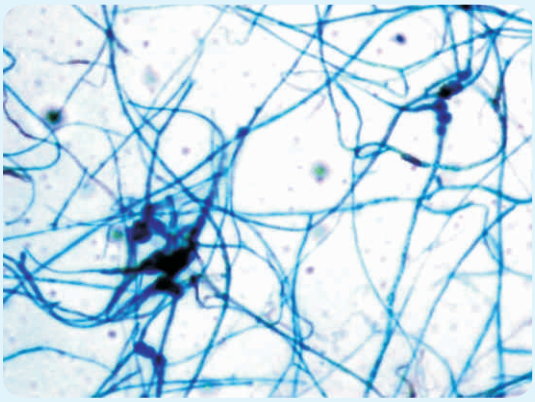
8. *Curvularia* spp: (Tuteja *et al.* communicated) Curvularia is a demati aceous filamentous fungus. Amongst various species, *Curvularia lunata* is the most prevalent cause of disease in humans and animals (Knudtson and Kirkbride, 1992; Larone, 1995; St-Germain and Summerbell, 1996).

On SDCA curvularia produces rapidly growing, woolly colonies at 28°C and may fill the plate in 10 days. From the front, the colour of the colony is white to pinkish gray initially and turns to olive brown or black as the colony matures. From the reverse, it is dark brown to blue or black (Pic. 112-113). Microscopically septate, brown hyphae, brown conidiophores and conidia are visualized (Pic. 114-115). Conidiophores are simple or branched and are bent at the points where the conidia originate. This bending pattern is called sympodial

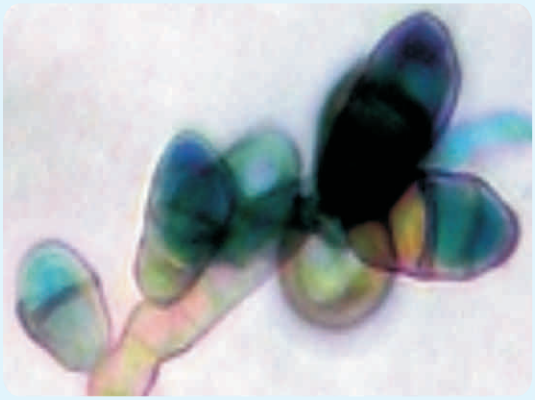
Pic. 112: *C. lunata;* colony (10 days front)



Pic. 113: *C. lunata;* colony (0 days reverse)



Pic. 114: Curvularia; septate hyphae and conidiophores



Pic. 115: *C. lunata;* conidia

geniculate growth. The conidia (8-14 x 21-35 µm), which are also called the poroconidia are straight or pyriform, brown, multi septate and have dark basal protuberant hila. The septa are transverse and divide each conidium into multiple cells. The central cell is typically darker and enlarged compared to the end cells in the conidium. The central septum may also appear darker than the others. The swelling of the central cell usually gives the conidium a curved appearance (de Hoog *et al,* 2000; Larone, 1995; St-Germain and Summerbell, 1996; Sutton *et al,* 1998). The conidia of *C. lunata* are pale brown have 3 septa and 4 cells, conidium of curvularia is usually curved, has an enlarged, darker central cell, thinner cell wall and narrower septations between the cells (Larone, 1995; St-Germain and Summerbell, 1996). Germination is bipolar and some species may have a prominent hilum.

Curvularia species are relatively common pathogens of animals and humans (Thomas *et al,* 1988). *Curvularia* spp. have been associated with allergic sinusitis, popularly known as helminthosporiosis in older literature (Bartynski *et al,* 1990) and dermatomycosis in cattle (Qureshi *et al,* 2006), subcutaneous tumefactions/ mycetomas in horses (Boomker *et al,* 1977), osteomyelitis in dogs (Coyle *et al,* 1984) and central nervous infection in parrots (Clark *et al,* 1986). Qureshi *et al* (2006) reported complete recovery of dermatomycosis in cattle with clotrimazole therapy (1% cream topically) for 10 days.

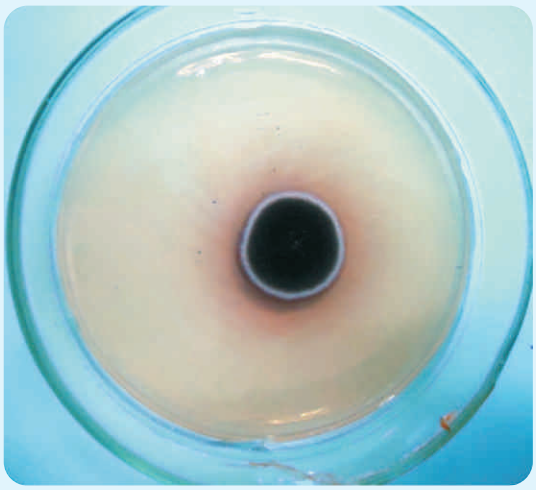
Curvularia species are reported to cause fatal cerebral phaeohyphomycosis (Carter and Boudreaux, 2004), mycetoma (Baylet *et al,* 1959), disease in the upper respiratory tract (Berry *et al,* 1984; Loveless *et al,* 1981; Rinaldi *et al,* 1987), lower respiratory tract (McAleer *et al,* 1981; Rohwedder *et al,* 1979), skin (Maghoub, 1973), endocardium (Kaufman, 1971) and cornea (Anderson *et al,* 1959). Central nervous system inolvement (Friedman *et al,* 1981; Lampert *et al,* 1977; Pierce *et al,* 1986; Rohwedder *et al,* 1979) and cerebral infection (Lampert *et al,* 1977; Friedman *et al,* 1981). Infections with curvularia do not appear to require an immunosuppressed

host. In a review of human curvularia infections, Rinaldi *et al* (1987) found that of the 24 patients, only two were systemically immunosuppressed. In curvularia sinusitis, infections can produce extensive compression and destruction of the bony walls of the sinuses as well as adjacent cranial bone (Ebright *et al,* 1999; Schroeder *et al,* 2002). Keratomycosis in a pet rabbit due to *C. lunata* has been reported (Pal *et al,* 1995).

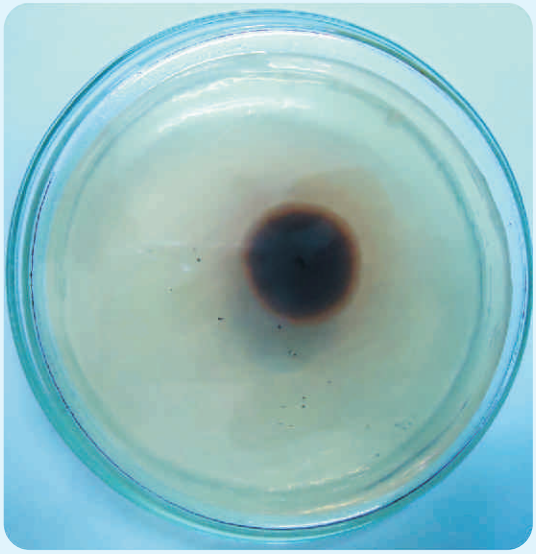
Amphotericin B, ketoconazole, miconazole, itraconazole and voriconazole showed favourable activity and low minimum inhibitory concentrations for most of the curvularia isolates (Fung-Tomc *et al,* 1995; Guarro *et al,* 1999; McGinnis and Pasarell, 1998). Caspofungin also appeared active *in vitro* against *C. lunata* (Del Poeta *et al,* 1997). For the treatment of allergic sinusitis, surgical treatment and administration of steroids are usually recommended along with antifungal therapy. Surgery may also be required in other infections, such as keratitis and localized cutaneous infections (Collier *et al,* 1998; Kuhn and Javer, 2000; Ujhelyi *et al,* 1990; Vartivarian *et al,* 1993).

9. *Exserohilum* spp.: (Tuteja *et al.* communicated) *Exserohilum* species are common environmental moulds found in soil and on plants, especially grasses. Several species have been reported as agents of phaeohyphomycosis especially *E. rostratum*, *E. meginnisii* and *E. longi rostratum.*

E. rostratum colonies on SDCA are dark gray to olivaceous black with a black reverse (Pic. 116-117). Rapid growth and woolly to cottony. Hyphae are septate and dark. Conidiophores are long, septate, non branched, geniculate and become pale near the apex. Conidia are olivaceous brown, straight to slightly curved, ellipsoidal to rostrate, contain 4-14 but typically 7-9 disto- or pseudosepta, have prominent, dark basal

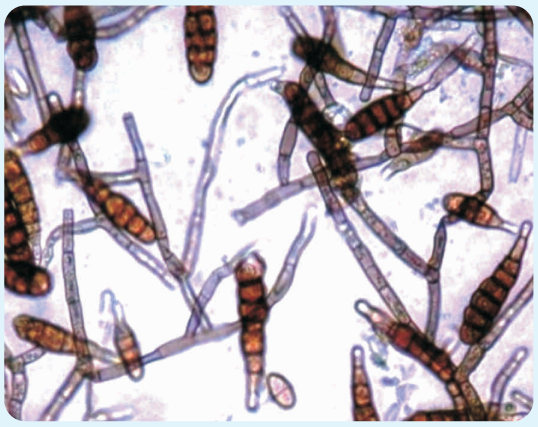


Pic. 116: *E. rostratum;* colony (7 days front)



Pic. 117: *E. rostratum;* colony (7 days reverse)

and distal septa, a strongly protruding truncate hilum (Pic. 118).

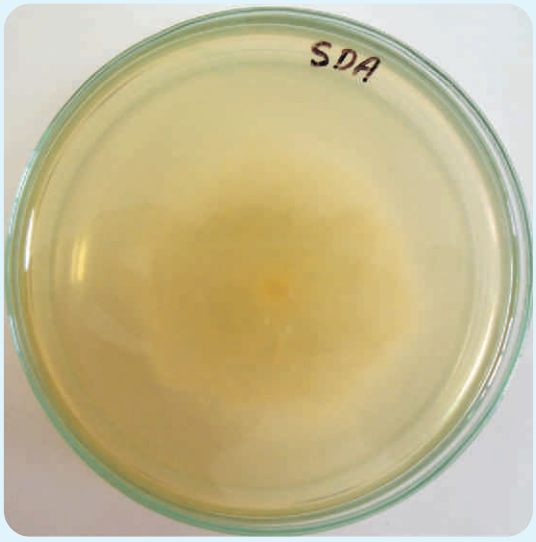


Pic. 118: *E. rostratum;* conidia and conidiophores

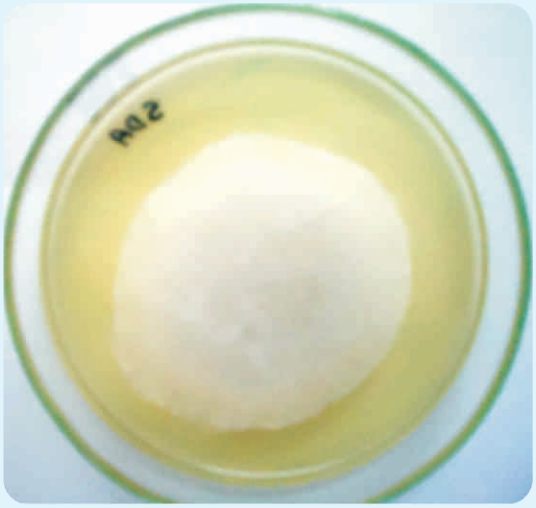
It has been isolated from cutaneous phaeohyphomycosis (Agarwal and Singh, 1995), in a child with leukemia (Money maker *et al,* 1986), subcutaneous phaeohyphomycoses (Burges *et al,* 1987; Hsu and Lee, 1993), keratitis (Anandi *et al,* 1991; Mathews and Maharajan, 1999) and fatal disseminated infection in a patient with aplastic anemia (Aquino *et al,* 1995). Alder *et al* (2006) in his review reported impaired immunity in the majority of patients with invasive and skin infections. Human exserohilum infections occurred mainly in warm, tropical and subtropical areas such as the southern USA, India and Israel. *E. rostratum* has also been isolated from equine dermatitis (Pal and Lee, 1994). Amphotericin B was the initial single antifungal agent used in all cases of invasive disease; the response rate was low but improved with the addition of triazole agents.

10. *Absidia* spp.: (Tuteja *et al*. communicated) Absidia are filamentous fungi that are cosmopolitan and ubiquitous in nature as common environmental contaminants. They often cause food spoilage. Absidia currently contains 21 mostly soil-borne species. *Absidia corymbifera* is the only species known to cause disease in man and animals. Absidia is characterized by a differentiation of the hyphae into arched stolons bearing more or less verticillate sporangiophores at the internode and rhizoids formed at the point of contact with the substrate. This feature separates species of *Absidia* from the genus *Rhizopus* where the sporangia arise from the nodes and are therefore found opposite the rhizoids.

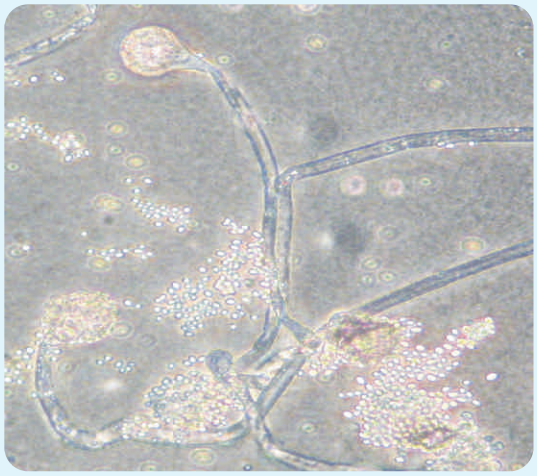
A. corymbifera grows rapidly. The colony is flat, woolly to cottony and olive gray and mature within 4 days. The texture of the colony is typically woolly to cottony (Pic. 119-120). Microscopically it has wide non septate hyphae. A few septa may



Pic. 119: *A. corymbifera;* colony (3 days front)



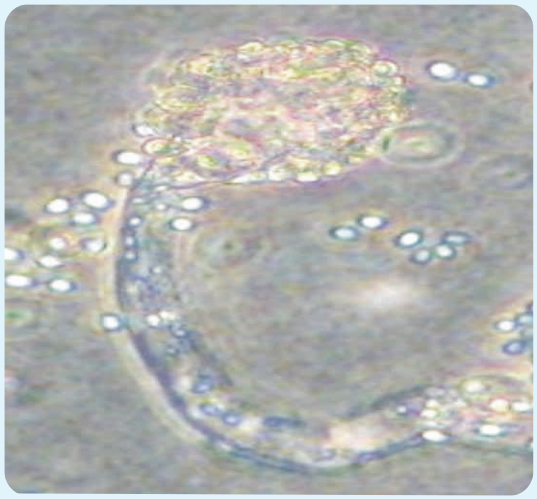
Pic. 120: *A. corymbifera;* colony (3 days reverse)



Pic. 121: *A. corymbifera;* non septate hyphae, sporangiophores, sporangia, apophysis

occasionally be present. The sporangio phores are branched and arise in groups of 2-5 at the internodes. Sporangiophores carry pyriform, relatively small sporangia. A septum is usually present just below the sporangium in the sporangiophore. The sporangiophore widens to produce the funnel shaped apophysis beneath the sporangium. The apophysis is very well developed and typical. The columella, the tip of the sporangiophore that extends into the sporangium, is semicircular in shape and has a small projection on the top. The sporangiospores are one-celled, hyaline to light black, round to oval in shape, smoothor rarely echinulate on surface. They are found in the sporangium and are released to the surrounding when the sporangium ruptures (Pic.121-122).

A. corymbifera may cause mycotic abortion in the cow (Knudtson and Kirkbride, 1992). Spontaneous occurrence of granulomatous lesions of zygomycosis has been reported in different tissues like



Pic. 122: *A. Corymbifera;* sporangium filled with sporangiospores

the fore stomach (Pohlenz *et al,* 1973), lymph nodes (Davis *et al,* 1955; Sadana and Kalara, 1973), lungs (Sadana and Kalara, 1973), intestine (Vitovec *et al,* 1976) and skin (Lopez *et al,* 2000) of cattle, pigs and horse. It is relatively rare cause of human opportunistic mycoses that manifests with pulmonary, rhinocerebral, cutaneous, gastrointestinal, renal or meningeal involvement. It is very rarely observed in

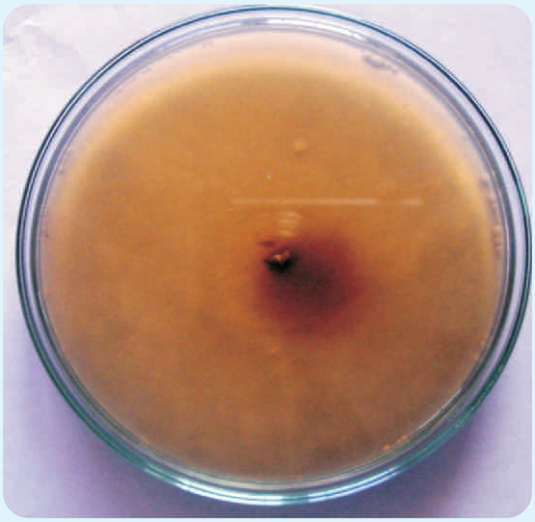
immunocompetent host (Hagensee *et al,* 1994; Ribes *et al,* 2000). Kindo *et al* (2010) isolated *A. corymbifera* with unusual presentation (as multiple discharging sinuses) from a diabetic patient.

Cutaneous infection can be success fully treated in early stages (Belfiori *et al,* 2007; Thami *et al,* 2003). Amphotericin-B appears as the sole antifungal drug which is consistently active against *A. corymbifera*. In general, it is resistant to azoles, including the newer derivatives such as variconazole (Wildfeuer *et al,* 1998). *In vivo* response largely depends on administration of full-dose amphotericin B therapy as well as extensive surgical debridement and correction of the underlying predisposing factors such as immunosuppression and diabetic acidosis (Ribes *et al,* 2000).

11. *Rhizopus* spp.: (Tuteja *et al.* communicated) Fungal infections caused by *Rhizopus* and *Mucor* species are commonly termed mucormycosis (Nathan *et al,* 1982). *Rhizopus oryzae* is the most common etiologic agent of mucormycosis (Fu *et al,* 2004). This fungus is a saprophytic agent of the nasal cavity and paranasal sinuses (Hoffman *et al,* 1993). *R. oryzae* has a world-wide distribution with a high prevalence in tropical and subtropical regions. It has been isolated from many substrates, including a wide variety of soils, decaying vegetation, foodstuffs, animal and bird dung. *R. oryzae* is often used in the production of fermented foods and alcoholic beverages in Indonesia, China and Japan. However, it also produces the ergot alkaloid agroclavine which is toxic to humans and animals.

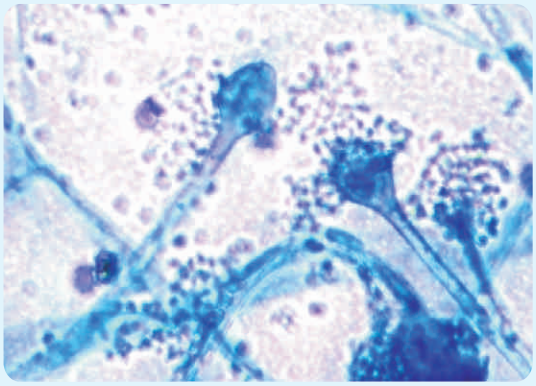
On SDCA colonies are very fast growing at 28°C, with some tendency to collapse, white cottony at first, becoming brownish grey to blackish-grey depending on the amount of sporulation (Pic. 123-124). Sporangiophores smooth walled, non-septate, simple or branched, arising

Pic. 123: *R. oryzae;* colony (5 days front)



Pic. 124: *R. oryzae;* colony (5 days reverse)

from stolons opposite rhizoids usually in groups of three or more. Sporangia are globose, often with a flattened base, greyish black. Columella and apophysis together are globose, subglobose or oval, up to 130 µm in height and soon collapse to an umbrella-like form after spore release. Sporangiospores are angular, subglobose to ellipsoidal with ridges on the surface (Pic. 125).



Pic. 125: *R. oryzae;* sporangiophores, rhizoids, sporangia and sporangium with collapsed columella

Mucormycosis is a severe fungal disease, which is observed as localized or disseminated forms (Neri *et al,* 2002). In recent years, the clinical importance of mucormycosis has significantly increased (Eucker *et al,* 2001). The most frequent form of the disease in human begins from the nose and the paranasal sinuses and can extend into the brain. It is a fulminant and often fatal disease, which is not well known by many specialists (Sanabria *et al,* 1992). Shirani *et al* (2008) reported an unusual case of nasal mucormycosis in dog.

Mucormycosis usually occurs in immunocompromised hosts and in trauma or burn victims as well (Chaney *et al,* 2004). There is also a close relationship between diabetes mellitus and mucormycosis in human and animals (Ossent, 1987). The most common clinical forms which is reported in dogs and cattle, affects the lymph nodes of respiratory and intestinal tracts characterized by caseous necrosis. The involvement of internal organs can occur (Quinn *et al,* 1994).

Iron is required by virtually all microbial pathogens for growth and virulence (Howard, 1999) and sequestration of serum iron is a major host defense mechanism against *R. oryzae* infection (Artis *et al,* 1982). *R. oryzae* lacks genes for non-ribosomal peptide synthesis, the enzymes that produce the hydroxamate sidero phores. These siderophores are used by other microbes to acquire iron. *R. oryzae* relies solely on rhizoferrin, which is ineffective in acquiring serum-bound iron and dependent on free iron for pathogenic growth (De Locht *et al,* 1994). Therefore iron chelators during early *R. oryzae* infection explain the strategy of treating infections which cannot be utilized by this organism as a source of iron (Sugar, 1995).

12. *Aspergillus* spp.: (Tuteja *et al.* communicated) Aspergillus is a filamentous and ubiquitous fungus. Aspergillus includes over 185 species. Around 20 species have so far been reported as causative agents of opportunistic infections in man. Among these *A. fumigatus* is the most commonly isolated species, followed by *A. flavus* and *A. niger, A. clavatus*, *A. glaucus* group, *A. nidulans*, *A. oryzae*, *A. terreus*, *A. ustus* and

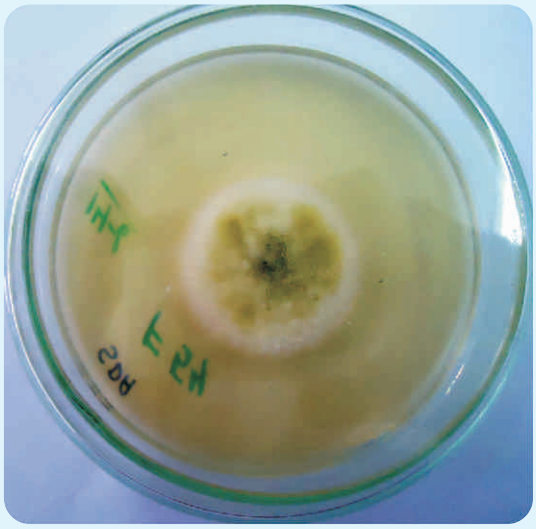
A. versicolor are among the other species less commonly isolated as opportunistic pathogens.

The major macroscopic features remarkable in species identification are the growth rate, colour of the colony and thermo tolerance. Aspergillus colonies are downy to powdery. The surface colour may vary depending on the species. The reverse is uncoloured to pale yellow in most of the isolates. However, reverse colour may be orange to purple in *A. versicolor. A. fumigatus* is a thermo tolerant fungus can grow at a temperature range of 20 to 50°C. The basic microscopic morphology of aspergilli is hyphae are septate and hyaline. The conidiophores originate from the basal foot cell located on the supporting hyphae and terminate in a vesicle at the apex. Vesicle is the typical feature for the aspergillus. The morphology and colour of the conidiophore vary from one species to another. Covering the surface of the vesicle entirely 'radiate head' or partially only at the upper surface 'columnar head' are the flask-shaped phialides which are both uniseriate and attached to the vesicle directly or are biseriate and attached to the vesicle via a supporting cell metula. Over the phialides are the round conidia (2-5 µm in diameter) forming radial chains (Pic. 126-155).

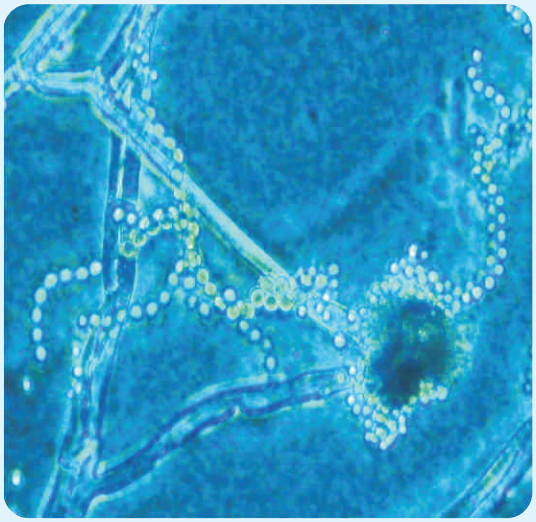
Some other microscopic features are unique to certain species and constitute the key features for species identification together with the surface colour of the colony. These microscopic structures include sclerotia, cleistothecia, aleuric onidia and Hulle cells. These structures are of key importance in identification of some of the species. Cleistothecium is a round, closed structure enclosing the asci which carry the ascospores. The asci are spread to the surrounding when the cleistothecium bursts. Cleistothecium is produced during the sexual reproduction stage of some species. Aleuriconidium is a type of conidium produced by lysis of the cell that supports it. The base is usually truncate and carries remnants of the lysed supporting cell. These remnants form annular frills at its base. Hulle cell is a large sterile cell bearing a small lumen. Similar to cleistothecium, it is associated with the sexual stage of some species (Collier *et al,* 1998; Larone, 1995; St-Germain and Summerbell, 1996).

A. flavus: Colonies on SDCA at 25°C are olive to lime green with a cream reverse. Growth is rapid and texture is woolly to cottony to somewhat granular. A clear to pale brown exudate may be present in some isolates. Hyphae are septate and hyaline.Conidial heads are radiate to loosely columnar with age. Conidiophores are coarsely roughened, uncoloured, vesicles globose to subglobose, metulae covering nearly the entire vesicle in biseriate species. Some isolates may remain uniseriate, producing only phialides covering the vesicle. Conidia are smooth to very finely roughened, globose to subglobose (Pic. 126-128).

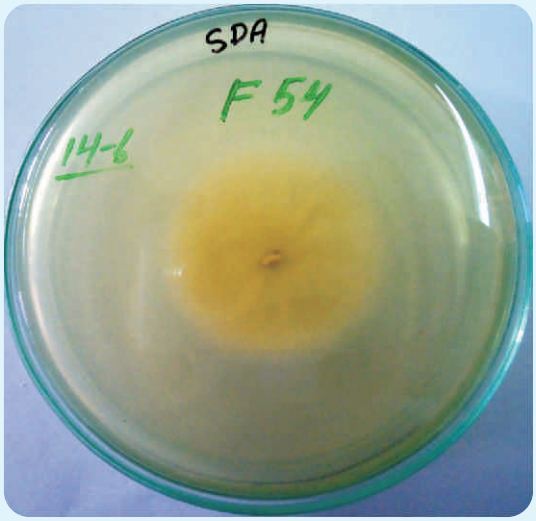
A. fumigatus: Colonies on SDCA at 25°C are smoky gray-green with a slight yellow reverse. Some isolates may display a lavender diffusible pigment. Very mature colonies turn slate gray. Growth is rapid and



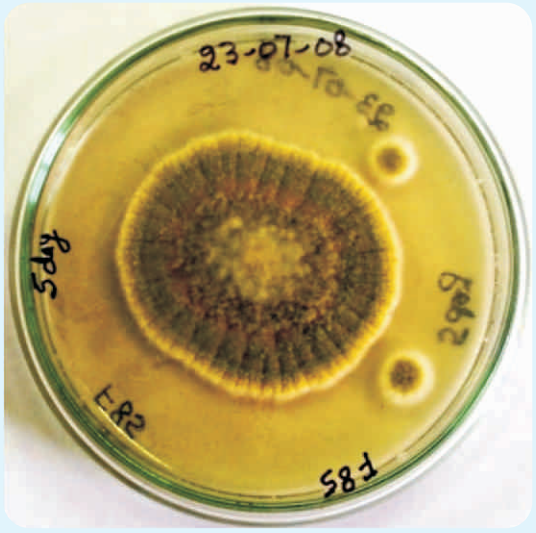
Pic. 126: *A. flavus;* colony (5 days front)



Pic. 128: *A. flavus;* microscopy



Pic. 127: *A. flavus;*
colony (5 days reverse)

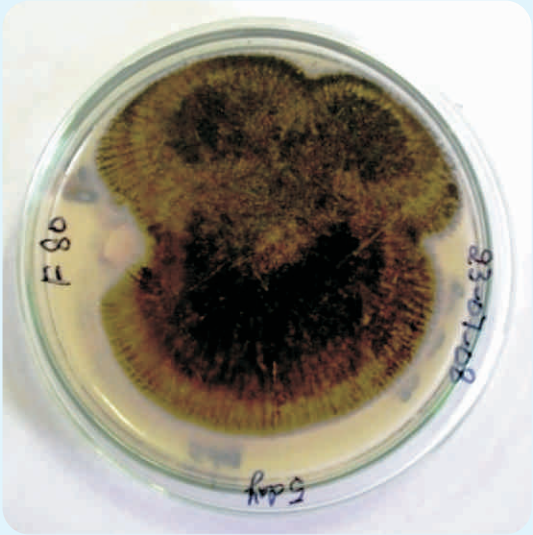


Pic. 129: *A. fumigatus;*
colony (5 days front)

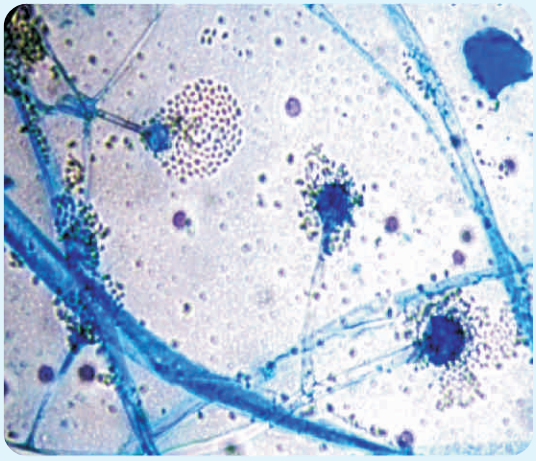
texture is woolly to cottony to somewhat granular. Atypical isolates may remain white with little conidiation (de Hoog *et al,* 2000; Sutton *et al,* 1998). Hyphae are septate and hyaline. Conidial heads are strongly columnar. Conidiophores are smooth-walled, uncoloured and terminate in a dome-shaped vesicle. This species is uniseriate with closely compacted phialides occurring only on the upper portion of the vesicle. Conidia are smooth to finely roughened, subglobose (Pic. 129- 131).

A. niger: Macroscopically colour of the colony is black and reverse colour is white to yellow. Microscopically conidio phore are long, smooth, colourless or brown, biseriate phialides and vesicle is round with radiate head (Pic. 132-134).

Pic. 130: *A. fumigatus;* colony (5 days reverse)



Pic. 132: *A. niger;* colony (5 days front)

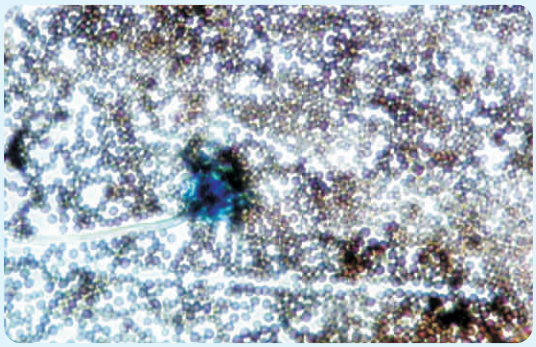


Pic. 131: *A. fumigatus;* microscopy

Pic. 133: *A. niger;* colony (5 days reverse)

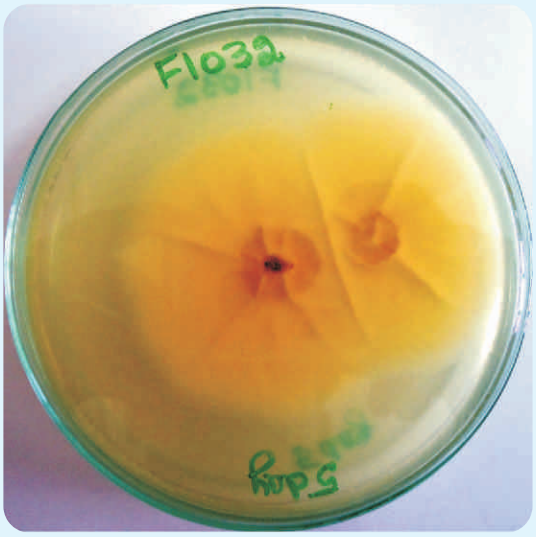
A. terreus*:* Macroscopically colour of the colony is cinnamon to brown and reverse colour is white to brown. Microscopically conidiophore are short (<250 µm), smooth, colourless, biseriate phialides and vesicle is round with compact columnar head (Pic. 135- 137).

A. versicolor: Macroscopically colour of the colony is white at the beginning, turns to yellow, tan, pale green or pink and reverse

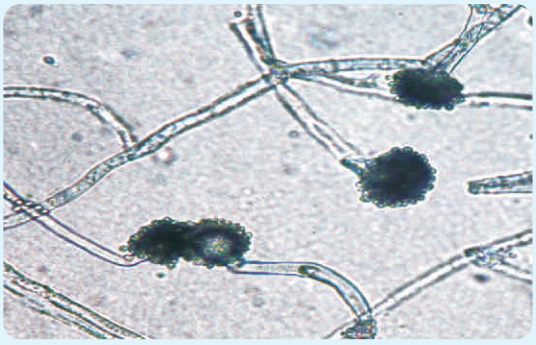


Pic. 134: *A. niger;* microscopy

Pic. 135: *A. terreus;* colony (5 days front)



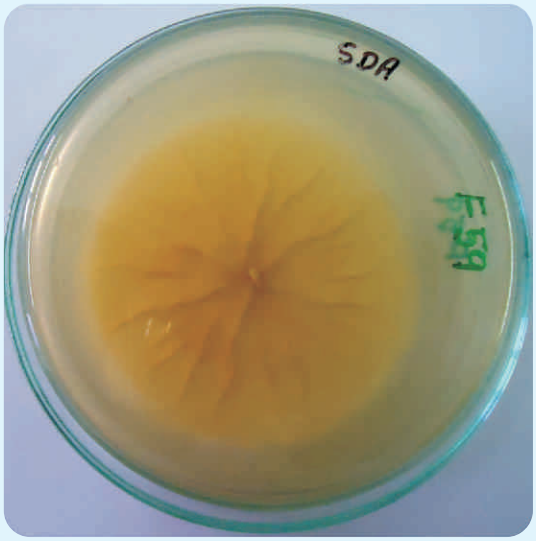
Pic. 136: *A. terreus;* colony (5 days reverse)



Pic. 137: *A. terreus;* microscopy

colour is white to yellow or purplish red. Microscopically conidiophores are long, smooth, colourless, biseriate phialides and vesicle is round with loosely radiate head (Pic. 138- 140).

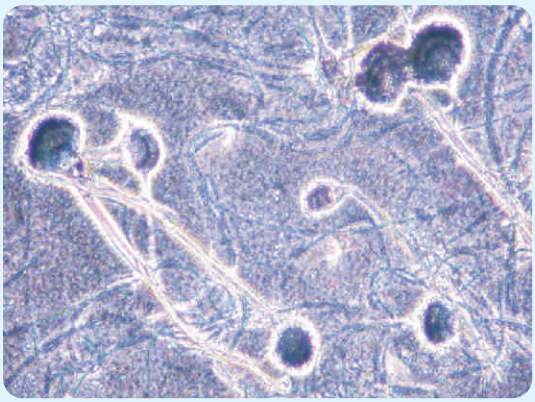
Pic. 138: *A. versicolor;* colony (5 days front)



Pic. 139: *A. versicolor;* colony (5 days reverse)

Aspergillus is well-known to play a role in three different clinical settings in man by opportunistic infections, allergic states and

toxicoses. Immunosuppression is the major factor predisposing to development of opportunistic infections (Ho and Yuen, 2000).



Pic. 140: *A. versicolor;* microscopy

These infections may present in a wide spectrum, varying from local involvement to dissemination and as a whole called aspergillosis. Almost any organ or system in the human body may be involved like cutaneous aspergillosis (Arikan *et al,* 1998), osteomyelitis (Flynn *et al,* 1990), pulmonary aspergillosis (Gefter, 1992), fungemia (Duthie and Denning, 1995), rhinosinusitis (Gillespie and O'Malley, 2000), otitis (Gordon and Giddings, 1994; Harley *et al,* 1995), endocarditis (Gumbo *et al,* 2000), onychomycosis (Gupta and Summerbell, 1999), myocarditis (Rouby *et al,* 1998), meningitis (Mikolich *et al,* 1996), sinusitis (Drakos *et al,* 1993).

Aspergillosis has been reported in calf affecting the skin (Pal, 1956) and causing generalised infection (Nag and Malik, 1961). The occurrence of fungi in lymph nodes of domestic buffaloes (*Bubalis bubalis*) subjected to slaughter was investigated. *A. fumigatus*, *A. flavus*, *A. niger* and *A. terreus* were isolated (Pal and Ragi, 1989). *A. terreus*, *A. nidulans* and *A. flavus* were isolated from the respiratory tract of buffaloes, goats and sheep (Singh and Singh, 1970). *A. fumigatus* isolated from corneal ulcer in a 7-month old buffalo calf was pathogenic to mice (Pal, 1983). In another report of mycotic keratitis in a buffalo calf, *A. flavus* was isolated (Pal, 1997). Pal and Mehrotra (1984) isolated *A. fumigatus* pathogenic to mice form nasal exudate of 1 of 23 mules and 1 of 7 camel dromedaries but none from 9 horses and 2 pigs with a history of rhinitis. *A. nidulans* has been reported from the guttural pouch of a horse having a history of epistaxis (Pal, 1996). *A. niger* has been isolated from otitis in a dog (Pal, 1982). *A. niger* has also been associated with dermatitis (Pal *et al,* 1987). *A. terreus* was isolated from a mycetoma case in a dog (Pal and Verma, 1987). *A. fumigatus* was isolated from 2 out of 17 dogs and 1 of 6 cats with sinusitis. The isolated *A. fumigatus* was pathogenic to Swiss mice (Pal *et al,* 1986). *A. flavus* has been reported to be associated with cancerous tissue of the horn core in a cow (Pal, 1989).

Some *Aspergillus* spp. especially *A. flavus* produces various mycotoxins. These mycotoxins by chronic ingestion have proven to possess carcinogenic potential particularly in animals. Among these mycotoxins, aflatoxin is well-known and may induce hepatocellular carcinoma. It is mostly produced by *A. flavus* and

contaminates foodstuff, such as peanuts (Mori *et al,* 1998). It may induce mycotic abortion in the cattle and the sheep (St-Germain and Summerbell, 1996). Ingestion of high amounts of aflatoxin may induce lethal effects in poultry fed with grain contaminated with the toxin. Pal *et al* (2007) reported a case of otitis in camel due to *A. flavus*.

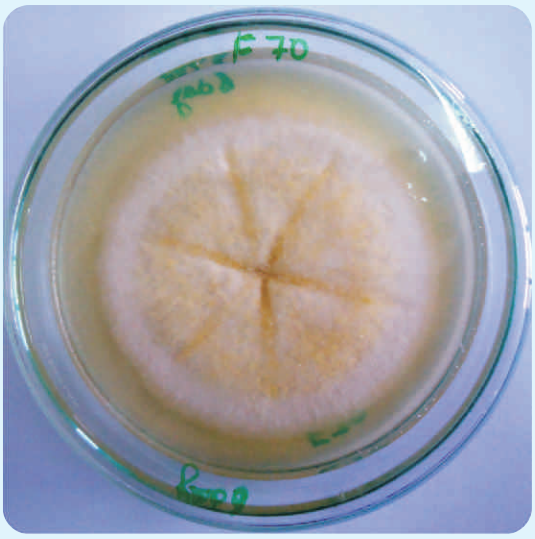
Cutaneous aspergillosis may be primary or secondary. Primary cutaneous aspergillosis refers to cases in which the initial infection begins in the skin due to direct inoculation, whereas secondary cutaneous aspergillosis is due to haematogenous seeding or spread from contiguous structures. The clinical manifestations of primary cutaneous aspergillosis are protean and non-specific. Typically, the lesions begin as erythematous, violaceous indurated plaques which progress to necrotic ulcers with central eschar formation (Prystowsky *et al,* 1976; Magid *et al,* 1988). Less commonly, hemorrhagic vesicles or bullae, subcutaneous nodules, granulomas, pustular lesions and vegetating plaques are observed (Jones *et al,* 1986; Van Burik *et al,* 1998; Isaac, 1996; Miele *et al,* 2002). Secondary cutaneous aspergillosis refers to cases in which skin involvement arising due to hematogenous seeding is a well recognized feature of disseminated aspergillosis and usually occurs in the setting of significant immunological impairment. Typically there are multiple skin lesions in anatomically unrelated sites.

A variety of clinical appearances similar to primary cutaneous aspergillosis are described, including erythematous to violacaeous nodules, plaques and papules with necrotic ulcerative and suppurative tendencies (Findlay *et al,* 1971; Gercovich *et al,* 1975).

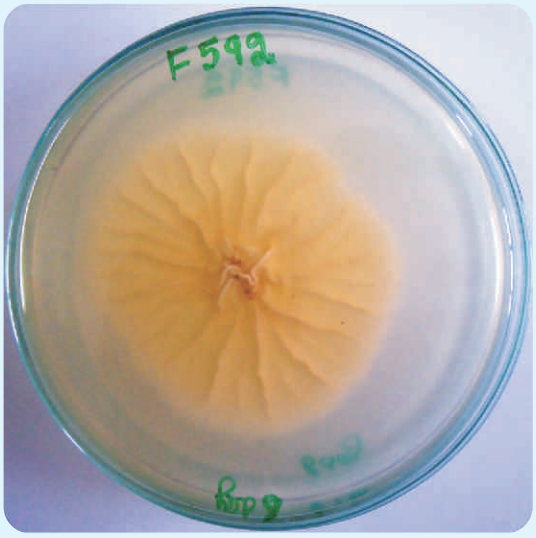
Correlation of the *in vitro* suscepti bility test results with the clinical outcome has been documented for itraconazole and aspergillus (Denning *et al,* 1997). Amphotericin B and itraconazole are the currently available therapeutic options, the success rate is still unsatisfactory due both to the low efficacy and or high toxicity of the drugs and existence of unfavourable immune status of the host (Denning, 1996; Denning, 1994; Denning *et al,* 1998; Dornbusch *et al,* 1995; Elgamal and Murshid, 2000; Fisher *et al,* 1999; Gerson *et al,* 1984; Grossi *et al,* 2000; Gurwith, 1999). The concomitant use of colony stimulating factors may activate the macrophages, enhance their fungicidal activity and prevent dissemination of the infection (Fujita *et al,* 1995).

Although the visualization of the *in vitro* effect of echinocandins requires distinctive parameters. They are active against aspergillus both *in vitro* and *in vivo* (Arikan *et al,* 2001; Maertens *et al,* 2000; Oakley *et al,* 1998). The demonstration of the synergistic effect of amphotericin B with echinocandins against aspergillus *in vitro* and in animal models is noteworthy and exciting (Stevens, 1999; Arikan *et al,* 2002).

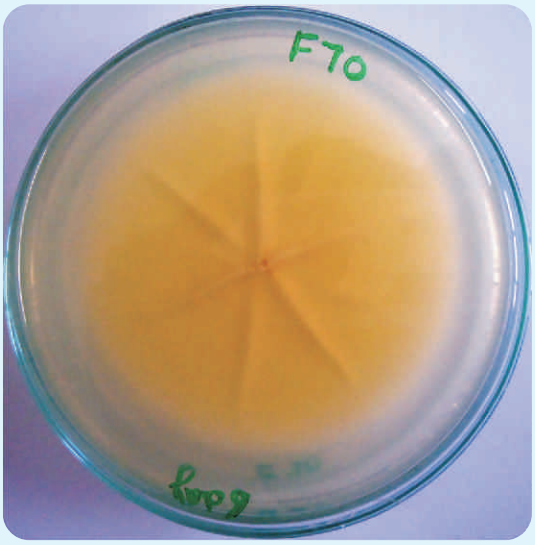
Pic. 141: Aspergillus isolate[1]; colony (6 days front)



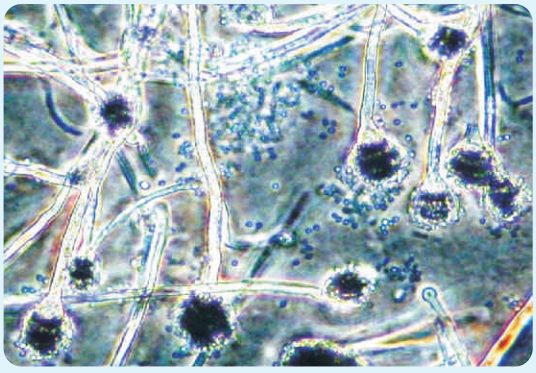
Pic. 144: Aspergillus isolate[2]; colony (6 days front)



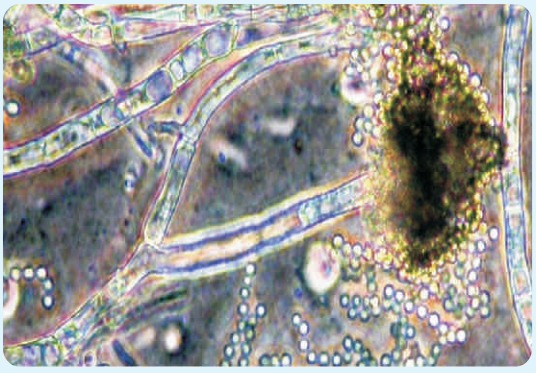
Pic. 142: Aspergillus isolate[1]; colony (6 days reverse)



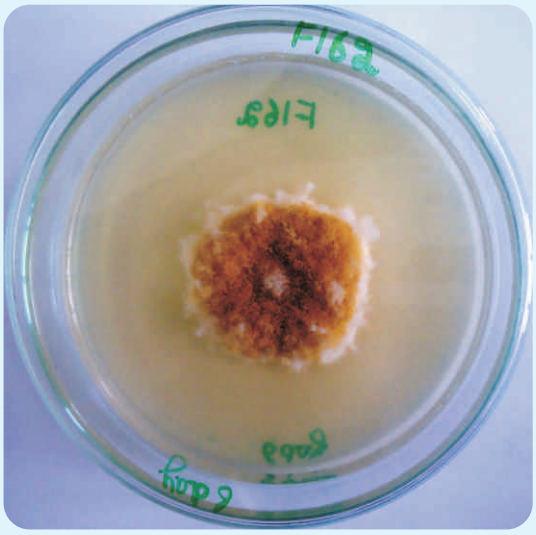
Pic. 145: Aspergillus isolate[2]; colony (6 days reverse)



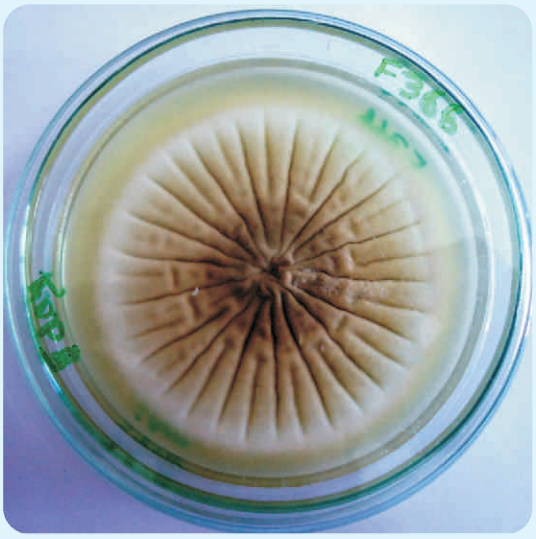
Pic. 143: Aspergillus isolate[1]; microscopy



Pic. 146: Aspergillus isolate[2]; microscopy

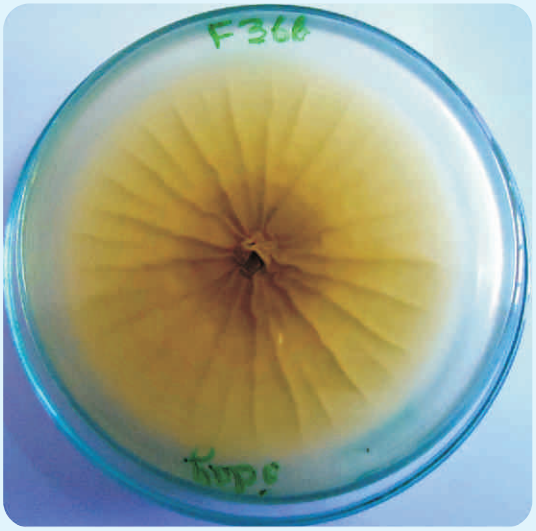


Pic. 147: Aspergillus isolate[3]; colony (6 days front)

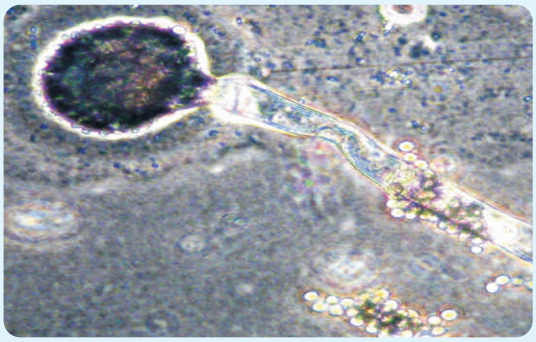


Pic. 150: Aspergillus isolate[4]; colony (6 days front)

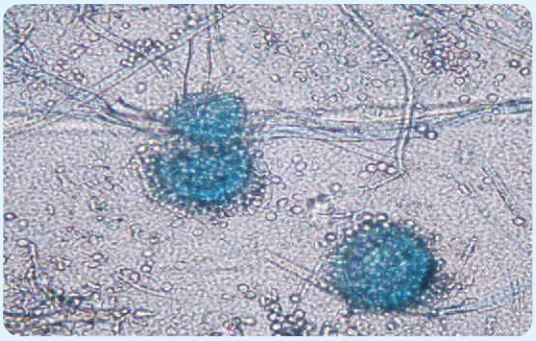
Pic. 148: Aspergillus isolate[3]; colony (6 days reverse)



Pic. 151: Aspergillus isolate[4]; colony (6 day reverse)

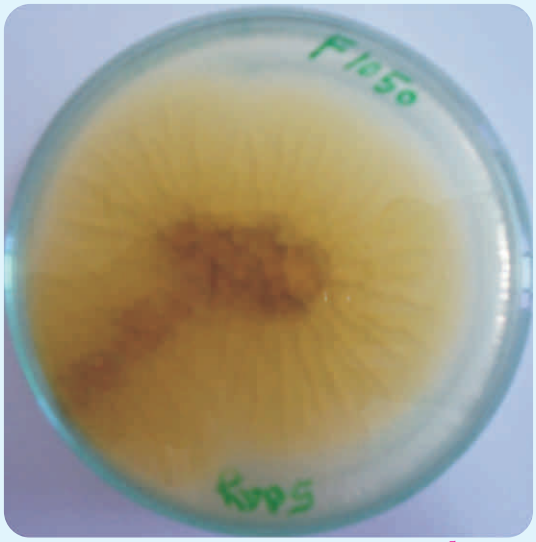


Pic. 149: Aspergillus isolate[3]; microscopy

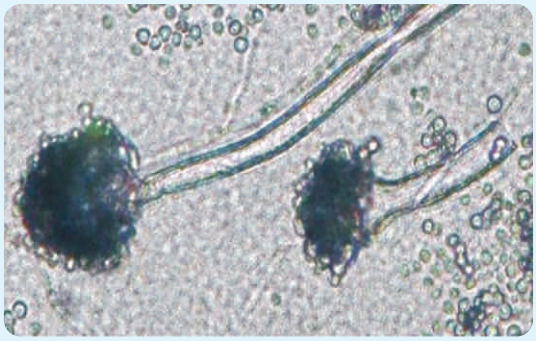


Pic. 152: Aspergillus isolate[4]; microscopy

Pic. 153: Aspergillus isolate[5]; colony (6 days front)



Pic. 154: Aspergillus isolate[5]; colony (6 days reverse)



Pic. 155: Aspergillus isolate[5]; microscopy

High incidence of skin infections in camels: Overall picture of study of skin fungi in camels revealed, high incidence of skin infections in camels, compared to other domesticated animals, this may be due to;

1. More keratin deposition on the skin of camels (Pic.156), because camel don't get showering like cattle and buffalo. Dipping like that of sheep and grooming like equines is not practised. Extremes of temperature variations in the desert especially in summer coupled with lot of sweating leads to lot of debris formation and deposition on the skin of the camels which favours growth of keratinophilic fungi. Increase in humidity during rainy season further favours growth of such fungi.

Pic. 156: High amount of keratin deposit on the skin

2. Injury caused by high population of biting flies and mosquitoes especially during the rainy season (Pic.157)

3. Injury caused by ticks since tick infestation is common in camels (Pic.158).

4. Grazing habit of the camels, camels mostly browse on thorny bushes and plants like Acacia, Zizypus, Prosopis *etc.* Thorn injuries/ scratches are common on any

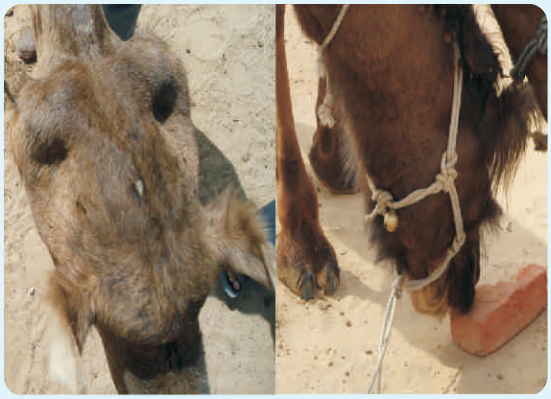
portion of the body (Pic.159).

Pic. 157: Mosquito sting bites along with mosquitoes sitting on the camel

Pic. 158: Tick and lice infestation on the skin

Pic. 159: Browsing of camels

5. Totally unbalanced ration: camels survive mostly on unbalanced diet because of low availability of feeds and fodders for camels. During drought camels are even left free in the jungle for leftover browsing on rangelands. This low plan of nutrition ultimately lowers the disease resistance capabilities of the camels. Other parameter to judge this situation is reflected by high incidence of PICA in camels (Pic.160).



Pic. 160: Pica affected camel eating brick along with weak body condition as reflected by deep temporal fosse

The variable composition and structure of the keratins. The specific requirements, enzyme system, genes responsible for pathogencity of dermato phyte species, the various defence mechanisms of the hosts may affect incidence and severity of dermatophytosis among host species and the particular distribution of lesions on the host.

Susceptibility to dermatophytosis depends also on the general health status of the animals. Young animals are more infected than adults, which could be related to the development of a stronger immunity in older animals due to the

multiplicity of contacts with the fungus rather than an intrinsic role of age. For example, the strong immune response elicited by *T. verrucosum* may explain the common occurrence of ringworm in calves and young cattle and its rarity in adults. On the contrary, *M. canis* is known to induce a weaker immune response and is frequently found in adult carnivores. As the cell-mediated immune response is required for the resolution of clinical ringworm and for resistance, the assessment of major *M. canis* fungal antigens may be important in order to explore the immune status of cats chronically infected and to detect a possible hyporesponsiveness (Descamps *et al,* 2003; Moriello *et al,* 2003).

As transmission of ringworm occurs through direct contact with infected animals or indirectly from contaminated fomites, all circumstances that favour those contacts act as predisposing factors. It explains a higher occurrence of ringworm in animals which are confined in catteries, kennels, stables, cowshed or intensive breeding units. In addition, the high resistance of the dermatophyte conidia for months or years in the environment explains why the use of material that is shared between animals for grooming, harnessing or transportation favours the contamination. Interestingly, the prevalence of equine ringworm was higher in training farms than among breeding horses and a majority of lesions were located on the girth area (Pascoe, 1976). For geophilic dermatophytes, the soil is the reservoir in which the fungi multiply.

Thus, the risk of contamination is higher for animals with outdoor contacts.

Zoonotic encounter: Most of the fungi isolated are reported to be of zoonotic significance as discussed in this manuscript. During survey work it was observed that camel handlers who remain mostly with camel herds were not found to have skin lesions to be caused by dermatophytes/ skin fungi. Possible reasons for this could be: (1) by constant exposure to such fungi they develop immunity against such fungi; (2) they daily use mustard oil as hair tonic on the scalp and face after bath and also they use this oil on the skin when so ever the skin is dry, some persons daily use mustard oil on the whole body before taking bath. Batra (2003) evaluated mustard oil as health oil in rat model, reported glucosinolate, the pungent principle in mustard oil, to possess anti-bacterial and anti-fungal properties; (3) pearl millet (*Pennisetum glaucum)* is the main cereal crop of the camel inhibiting region and they use this cereal grain food as main part of their diet. Joshi *et al* (1998) found pearl millet seeds contain a cysteine protease inhibitor as an anti-fungal protein; (4) they almost daily eat raw onion (*Allium cepa)* as part of their food. *Allium cepa* is having antibacterial and antifungal properties (Augusti, 1996; Kim, 1997).

Treatment and control of animal dermatophytosis: Dermatophytosis is considered to be self-limiting disease in immunocompetent animals. Immune response may be sufficient to control the spread of the cutaneous lesions of dermatophytoses. However, antifungal

therapy should be systematically recommended in order to shorten the course of the infection and to reduce dissemination of arthroconidia to other animals and into the environment. Systemic antifungal drugs contribute to speed the resolution of the infection whereas topical antifungals reduce the risk of transmission and environmental contamination. Current treatment recommendations are generally based on both *in vivo* and *in vitro* studies concerning dermatophyte species commonly found in animals. An appropriate duration of treatment must be respected. In dogs and cats, combined systemic and topical treatment should be maintained for at least 10 weeks. In large animals, the cost of antifungal drugs may lead to reduce the duration of treatment. Treatment failures may be due to drugs resistance, improper administration or misdiagnosis. Environ mental disinfection and the complete separation of infected animals from non-infected ones is required. Camel breeders usually consider dermatophytosis a benign infection that does not deserve specific and expensive treatment. They usually go for their ethno veterinary treatment methods.

a. Systemic antifungal treatments: Griseofulvin remains the principal drug for the systemic treatment of animal dermatophytosis. This drug has been successfully used in pet carnivores, cattle, sheep, goats, horses, rodents and rabbits (Scott, 1988; Hill *et al,* 1995). In dogs and cats, the micronised formulation of griseofulvin is administered orally at 25 mg per kg body weight twice daily and with fatty meals. In large animals, a dosage of 7.5–10 mg per kg body weight once daily is usually recommended (Scott, 1988). However, this dosage has not been validated and the use of griseofulvin is now prohibited in European countries for food animals. Griseofulvin is contraindicated in pregnant animals as it is a teratogenic drug. Adverse reactions include vomiting, diarrhoea and anorexia. Few reports suggest that feline immuno deficiency virus infection predisposes cats to griseofulvin-induced bone marrow suppression (Shelton *et al,* 1990). Azole derivatives have been proposed as an alternative therapy of animal dermatophytosis. Ketoconazole is the first orally administered azole derivative that has been used in veterinary medicine. A dosage of 2.5–5 mg per kg body weight twice daily is usually recommended in the treatment of ringworm. Adverse effects of ketoconazole may occur in cats which include anorexia, vomiting and diarrhoea. A dose dependent elevation in liver enzymes may also occur and it is recommended to monitor these enzymes at a monthly interval during therapy. Ketoconazole is contraindicated in pregnant animals. It interferes with steroid hormones synthesis and may decrease serum concentrations of estosterone and cortisol. Itraconazole is the latest systemic active azole derivative to become available for veterinary medicine. The drug is proved to have a very broad spectrum of activity and is labelled for use in cats. The recommended dosage may vary according to the animal species and the fungal diseases. In dogs and cats,

itraconazole seems to be much better tolerated than ketoconazole and anorexia is the only problem that could be occasionally detected. Although itraconazole is not teratogenic at 10 mg per kg body weight, embryo toxicity and teratogenicity were observed at very high dosages (160 mg per kg body weight) in rats (Van Cauteren *et al,* 1987) and the use of this drug in pregnant animals is not suggested. Terbinafine is not labelled for use in veterinary medicine. However, a few investigations demons trated the efficacy of this allylamine drug in cases of dermatophytosis in dogs and cats (Mancianti *et al,* 1999; Moriello, 2004). The dosage of 20-30 mg per kg body weight daily is usually suggested. Another form of systemic treatment may be immuno therapy using an anti-dermatophyte vaccine. This option has been suggested in cattle and in cats with the LTF 130 and Fel-O-Vax MC-K- vaccines, respectively.

b. Topical antifungal treatments: Many products have been proposed for the topical treatment of animal derma tophytosis. A solution of enilconazole at 0.2% is approved for use in dogs, cats, horses and cattle in most countries. Enilconazole, first described in 1969, can be given orally, but its poor solubility limits its use to topical applications on the skin. Local or general side effects are very seldom reported, even in cats. A combination of 2% miconazole and 2% chlorhexidine administered topically twice weekly was proved to be efficient for the treatment of dermatophytosis in cats (Moriello, 2004; Paterson, 1999) and horses (Paterson, 1997).

Lime sulphur may be used in dogs and cats in combination with griseofulvin or itraconazole (Newbury *et al,* 2007) but it has an offensive odour and may stain light-coloured hair. As oral ulcerations have been sometimes reported in cats, an Elizabethan collar must be used to prevent cats from licking the solution. Sodium hypochlorite solution has been used as topical treatment of ringworm in cats. However, this product should not be recommended because it dries and irritates the skin and bleaches the hair coat.

The decision to use topical therapy should be based upon the owner's or breeder's ability and willingness to pour or sponge the product over the entire surface of the body of the infected animals, whatever the distribution of the lesions could be. Spot treatment of lesions is not recommended. The frequency of topical treatment should be at least twice a week. The clipping of the hair coat is sometimes useful, especially in severely infected animals, long-haired cats or dogs or in multi-animal households. Clipping makes topical application easier and allows for better penetration of the drug. However, clipping must be performed carefully. Moreover, the unavoidable contamination of the material such as clipper, tables *etc.* with infected hairs requires a meticulous disinfection after clipping. In cattle, crusts should be removed and further destroyed, before the application of topical antifungal drugs.

Management of skin infections by the camel farmers: Camel farmers or traditional healers are adopting certain ethno veterinary practices, for the management of skin infections.

I. In cases of skin candidiasis (thikria): First they scrap the lesions with knife to remove skin debris then they apply, any of the following (Tuteja *et al.* 2011).

1. Pearl millet (*Pennisetum glaucum* L.) flour and common salt (1:1), by topical rubbing on alternate days for 1-2 weeks.

2. Sulphur in mustard (*Brassica* spp.) oil (1:10), topical application, alternate days for 1-2 week.

3. Any oil (sesame, mustard, sump), topical application, alternate days for 1-2 week.

4. Upper fatty foam of camel milk formed during milking, topical application, daily for one week.

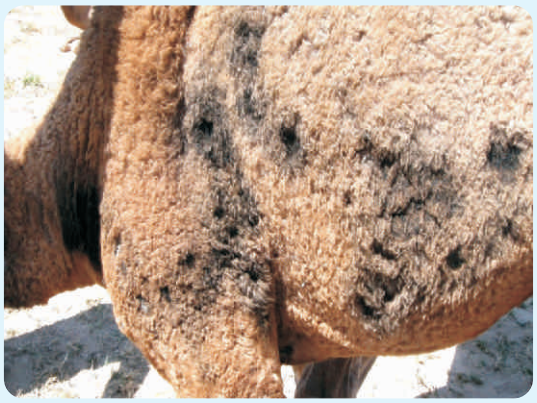
II. In cases of other skin infections: First they scrap the lesions to remove skin debris then they apply, any of the following (Tuteja *et al.* 2011).

1. Leather ash mixed with butter fat India (1:1), topical application, alternate days for 1-2 week.

2. Sulphur in mustard (*Brassica* spp.) oil (1:10), topical application, alternate days for 1-2 week.

3. Sulphur and karanj {*Pongamia pinnata (L.) Pierre*} oil (1:10), topical application, weekly for 3-4 weeks.

4. Topical application of sump oil, alternate days for 1-2 week (Pic. 161)



Pic. 161: Application of sump oil on the affected skin

There is urgent need for the development of low cost topical antifungal cream for the veterinary uses and low cost antifungal systemic therapy for large animal practice. The use of disinfectant like iodophores *etc* for animal bedding may not be effective in case of camels, because camel mostly remain loose for browsing in rangelands. Few antifungal agents are available for use in veterinary practice and the use of systemic drugs is limited in livestock due to the problems of residues in products intended for human consumption and very high cost of treatment. The multiplicity of host species and the confinement of animals are cause of an enzootic situation in many cases.

Collection and mailing of samples: It is obvious that certain essential steps in collection and handing of specimens be taken to ensure that pathogenic fungi are successfully isolated, particularly from specimens contaminated with rapidly growing bacteria and non-pathogenic fungi. Prompt transportation of freshly collected

clinical specimens with the addition of antibiotics to combat unwanted conta minants may help in the isolation of pathogenic fungi. It is necessary to plate each specimen on selective, enriched and simple mycological media as no single medium is adequate for isolation of all species of fungi. For best results, if possible, agar cultures should be mailed to a laboratory for mycological examination.

Liquid samples *e.g*. urine, sputum, exudates *etc*. from lesions may be sent in properly labelled screw capped vials, preferably under ice. Test tubes of standard size are recommended. Petri dish cultures should never be sent through the mail as they almost invariably break in transit; neither should cotton swabs be mailed since they usually become dry before arrival to the laboratory. Only pure cultures should be mailed to a reference laboratory.

From the affected area of skin and hair, material may be collected after sterilizing the part with 70% alcohol. Scrapings should be collected from the margins of lesions with the help of a blunt scalpel. Few hairs may also be pulled out to be included in the specimen which should be packed in non- absorbent paper and despatched to the laboratory. Similarly, scrapings of infected nails or clippings of the nails may also be collected.

Specimens collected for histo pathology studies should be sent in 10% neutral formol saline.

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References:

Abdel-Gawad, K.M. (1989). Fungi on the claws of buffalo and cow in Egypt. *Journal of Basic Microbiology*. **29**: 323-328.

Abdel-Gawad, K.M. and Moharram, A.M. (1989). Keratinophilic fungi from the duck nails in Egypt. *Journal of Basic Microbiology*. **29**: 259-263.

Abu-Samra, M.T. and Ibrahim, K.E. (1988). The effect of 9-a fluorprednisolone on the pathogenicity of *Microsporum canis* and *Trichophyton violaceum* to horses. *Mycoses*. **31**: 71-79.

Adler, A., Yaniv, I., Samra, Z., Yacobovich, J., Fisher, S., Avrahami, G. and Levy, I. (2006). Exserohilum: an emerging human pathogen. *European Journal of Clinical Microbiology and Infectious Diseases*. **25**: 247-253.

Agarwal, A. and Singh, S.M. (1995). A case of cutaneous phaeohyphomycosis caused by *Exserohilum rostratum*, its *in vitro* sensitivity and review of literature. *Mycopathologia*. **131**: 9-12.

Aguilar, C., Pujol, I. and Guarro, J. (1999). *In vitro* antifungal susceptibilities of scopulariopsis isolates. *Antimicrobial Agents and Chemotherapy*. **43**: 1520-1522.

Al-Ani, F.K., Younes, F.A. and Al-Rawashdeh, O.F. (2002). Ringworm infection in cattle and

horses in Jordan. *Acta Veterinaria Brno*.**71**: 55-60.

Al-Tawfiq, J.A. and Wools, K.K. (1998). Disseminated sporotrichosis and *Sporothrix schenckii* fungemia as the initial presentation of human immunodeficiency virus infection. *Clinical Infectious Diseases*. **26**: 1403-1406.

Aly, R., Hay, R.J., Del Palacio, A. and Galimberti, R. (2000). Epidemiology of tinea capitis. *Medical Mycology*. **38**: 183-188.

Aman, S., Haroon, T.S., Hussain, I., Bokhari, M.A. and Khurshid, K. (2001). Tinea unguium in Lahore, Pakistan. *Medical Mycology*. **39**: 177-180.

Ampel, N. M., Ryan, K.J., Carry, P.J., Wieden, M. A. and Schifman, R.B. (1986). Fungemia due to *Coccidioides immitis*. *Medicine*. **65**: 312-321.

Anaissie, E.J., Bodey, G.P. and Rinaldi, M.G. (1989). Emerging fungal pathogens. *European Journal of Clinical Microbiology and Infectious Diseases*. **8**: 323-330.

Anaissie, E., Bodey, G.P., Kantarjian, H., Ro, J., Vartivarian, S.E., Hopfer, R., Hoy, J. and Rolston, K. (1989). New spectrum of fungal infections in patients with cancer. *Review Infectious Disease*. **11**: 369-378.

Anandi, V., George, J.A., Thomas, R., Brahmadathan, K.N. and John, T.J. (1991). Phaeohyphomycosis of the eye caused by *Exserohilum rostratum* in India. *Mycoses*. **34**: 489-491.

Anderson, B., Roberts, Jr. S.S., Gonzalez, C. and Chick, E.W. (1959). Mycotic ulcerative keratitis. *Archives of Ophthalmology*. **62**: 169-197.

Andryushin, V.V. (1980). *Microsporum equinum* infection (ringworm) in horses. *Veterinariya*, Moscow. (6) 40-42.

Aquino, V.M., Norvell, J.M., Krisher, K. and Mustafa, M.M. (1995). Fatal disseminated infection due to *Exserohilum rostratum* in a patient with aplastic anemia: case report and review. *Clinical Infectious Diseases*. **20**: 176-178.

Arenas, R., Dominguez-Cherit, J. and Fernandez, L.M. (1995). Open randomized comparison of itraconazole versus terbinafine in onychomycosis. *International Journal of Dermatology*. **34**: 138-143.

Arikan, S., Lozano-Chiu, M., Paetznick, V. and Rex, J.H. (2001). *In vitro* susceptibility testing methods for caspofungin against aspergillus and fusarium isolates. *Antimicrobial Agents and Chemotherapy*. **45**: 327-330.

Arikan, S., Lozano-Chiu, M., Paetznick, V. and Rex, J.H. (2002). *In vitro* synergy of caspofungin and amphotericin B against *Aspergillus* and *Fusarium* spp. *Antimicrobial Agents and Chemotherapy*. **46**: 245-247.

Arikan, S., Uzun, O., Cetinkaya, Y., Kocagoz, S., Akova, M. and Unal, S. (1998). Primary cutaneous aspergillosis in human immunodeficiency virus- infected patients: Two cases and review. *Clinical Infectious Diseases*. **27**: 641-643.

Artis, W.M., Fountain, J.A., Delcher, H.K. and Jones, H.E. (1982). A mechanism of susceptibility to mucormycosis in diabetic ketoacidosis: transferrin and iron availability. *Diabetes*. **31**: 1109 -1114.

Augusti, K.T. (1996). Therapeutic values of onion (*Allium cepa* L.) and garlic (*Allium sativum* L.). *Indian Journal of experimental Biology*. **34**: 634-640.

Aviles, P., Pateman, A., Roman, R.S., Guillen, M.J., De las Heras, F.G. and Gargallo-Viola, D. (2001). Animal pharmacokinetics and interspecies scaling of sordarin derivatives following intravenous administration. *Antimicrobial Agents and Chemotherapy*. **45**: 2787-2792.

Bagy, M.M. (1986). Fungi on the hair of large mammals in Egypt. *Mycopathologia*. **93**: 73-75.

Bagy, M.M. and Abdel-Mallek, A.Y. (1991). Saprophytic and keratogenic fungi associated with animal's hair from Riyadh, Saudi Arabia. *Zbl. Mikrobiol*. **146**: 305-310.

Bartynski, J.M., McCaffrey, T.V. and Frigas, E.

(1990). Allergic fungal sinusitis secondary to dematiaceous fungi-*Curvularia lunata* and *Alternaria*. *Otolaryngology*. **103**: 32-39.

Batra, P. (2003). Evaluation of mustard oil as health oil in rat model. Thesis in toxicology. Department of medical elementology and toxicology, Faculty of science. Jamia Hamdard, Hamdard Nagar, N. Delhi.

Bayer, A.S. and Guze, L.B. (1979). Fungal arthritis. II. Coccidioidal synovitis: clinical, diagnostic, therapeutic and prognostic considerations. Seminars in Arthritis & Rheumatism. **8**: 200-211.

Baylet, J., Camain, R. and Segretain, G. (1959). Identification of the agents of maduromycoses of Senegal and Mauritania. Description of a new species. *Bulletin of the Exotic Pathology Society*.**52**: 448-477.

Belfiori, R., Terenzi, A., Marchesini, L. and Repetto, A. (2007). *Absidia corymbifera* in an immune competent accident victim with multiple abdominal injuries: case report. *BMC Infectious Diseases*. **7**: 46.

Berry, A.J., Kerkering, T.M., Giordano, A.M. and Chiancone, J. (1984). Phaeohyphomycotic sinusitis. *Pediatrics Infectious Disease Journal*. **3**: 150-152.

Bhoyar, R., Karuna, I., Anilkumar, M.C., Filia, G. and Patil, P.K. (2010). Moniliasis in an Asian elephant- A case study. Scientific abstracts National Symposium on 'Recent Developments in Diagnostic and Therapeutic Approaches for Economically Important Diseases of Livestock and Companion Animals' and 28th Annual convention of ISVM. Feb.17-19, Hyderabad (Andhra Pardesh) P.142.

Blair, J.E. and Logan, J.L. (2001). Coccidioidomycosis in solid organ transplantation. *Clinical Infectious Diseases*. **33**: 1536-1544.

Boever, W.J. and Rush D.M. (1975). *Microsporum gypseum* infection in a dromedary camel. *Veterinary Medicine Small Animal Clinician*.**70**: 1190-1192.

Boomker, J., Coetzer, J.A.W. and Scott, D.B. (1977). Black grain mycetoma (maduromycoses) in horses. *Onderstepoort Journal of Veterinary Research*. **44**: 249-252.

Boonk, W., de Geer, D., de Kreek, E., Remme, J. and van Huystee, B. (1998). Itraconazole in the treatment of tinea corporis and tinea cruris: comparison of two treatment schedules. *Mycoses*. **41**: 509-514.

Boro, B.R., Sharma, G, Sharma, A.K. and Chakraborty, A.K. (1980). Ringworm in animals due to *Epidermatohpyton floccosum*. *Veteterinary Record*. **107**: 491-492.

Bruynzeel, I. and Starink, T.M. (1998). Granulomatous skin infection caused by *Scopulariopsis brevicaulis*. *Journal of the American Academy of Dermatology*. **39**: 365-367.

Burges, G.E., Walls, C.T. and Maize. J.C. (1987). Subcutaneous phaeohyphomycosis caused by *Exserohilum rostratum* in an immunocompetent host. *Archives of Dermatology*. **123**: 1346-1350.

Byrd, D.R., el-Azhary, R.A., Gibson, L.E. and Roberts, G.D. (2001). Sporotrichosis masquerading as pyoderma gangrenosum: case report and review of 19 cases of sporotrichosis. *Journal of the European Academy of Dermatology and Venereology*. **15**: 581-584.

Cabanes, F.J., Abarca, L., Bragulet, M.R. and Brugara, T. (1988). Phaeohyhomycosis caused by *Alternaria alternata* in a mare. *Journal of Medical and Veterinary Mycology*. **26**: 359-365.

Cairns, L., Blythe, D., Kao, A., Pappagianis, D., Kaufman, L., Kobayashi, J. and Hajjeh, R. (2000). Outbreak of coccidioidomycosis in Washington State residents returning from Mexico. *Clinical Infectious Diseases*. **30**: 61-64.

Calderone, R.A. (1989). Immunoregulation of dermatophytosis. *Critical Reviews in Microbiology*. **16**: 339-368.

Carter, E. and Boudreaux, C. (2004). Fatal cerebral phaeohyphomycosis due to *Curvularia lunata* in an immunocompetent patient. *Journal of Clinical Microbiology*. **42**: 5419-5423.

Castrejon, O.V., Robles, M. and Zubieta Arroyo, O.E. (1995). Fatal fungaemia due to *Sporothrix schenckii*. *Mycoses*. **38**: 373-376.

Chakraborty, A.N. Ghosh, S. and Blank, F. (1954). lsolation of *Trichophyton rubrum* (castollani) Sabourauds 1911 from animal. *Canadian Index of Comparative Medicine*. **18**: 436-438.

Chaney, S., Gopalan, R. and Berggren, R.E. (2004). Pulmonary *Pseudallescheria boydii* infection with cutaneous zygomycosis after near drowning. *Southern Medical Journal*. **97**: 683-687.

Chariyalertsak, S., Supparatpinyo, K., Sirisanthana, T. and Nelson, K.E. (2001). A controlled trial of itraconazole as primary prophylaxis for systemic fungal infections in patients with advanced human immunodeficiency virus infection in Thailand. *Clinical Infectious Diseases*. **34**: 277-284.

Chatterjee, A. and Sengupta, D.N. (1979). Ringworm in domestic animals. *Indian Journal of Animal Health*. **18**: 38-46.

Chatterjee, A., Banerjee, P.C., Chattopadhaya, D., Dutta, A.K. and Sengupta, D.N. (1978). Isolation of *Trichophyton rubrum* from a cow. *Indian Journal of Animal Health*. **17**: 167 -168.

Chatterjee, A., Chakraborty, P., Chattopadhyay, D. and Sengupta, D.N. (1978). Isolation of *Trichophyton schoenleinii* from a camel. *Indian Journal of Animal Health*. **17**: 79-81.

Cheng, N.C., Wong, W.W., Fung, C.P. and Liu. C.Y. (1998). Unusual pulmonary manifestations of disseminated *Penicillium marneffei* infection in three AIDS patients. *Medical Mycology*. **36**: 429-432.

Chittawar, D.R. and Rao, K.N.P. (1982) Incidence of canine dermatitis of mycotic origin in central India. *Indian Veterinary Journal*. **59**: 675- 677.

Chretien, J.H. and Garagusi, V.F. (1990). Infections associated with pets. *American Family Physician*. **41**: 831-845.

Clarke, F.D., Jones, L.P. and Panigrahy, B. (1986). Mycetoma in a grand Eclectus (*Eclectus rotatus*) parrot. *Avian Diseases*. **30**: 441-442.

Clemons, K.V. and Stevens, D.A. (2000). Efficacies of sordarin derivatives GM193663, GM211676, and GM237354 in a murine model of systemic coccidioidomycosis. *Antimicrobial Agents and Chemotherapy*. **44**: 1874-1877.

Collier, L., Balows, A. and Sussman, M. (1998). Topley and Wilson's Microbiology and Microbial Infections, 9th Ed, vol. 4. Arnold, London, Sydney, Auckland, New York.

Connole, M.D. (1973). Equine phycomycosis. *Australian Veterinary Journal*. **49**: 215-216.

Connole, M.D. and Pascoe, R.R. (1984). Recognition of *Trichophyton equinum* var. *equinum* infection of horses. *Australian Veterinary Journal*. **61**: 94.

Conti, I.A., Civila, E. and Gezuele, E. (1992). Treatment of human cutaneous sporotrichosis with itraconazole. *Mycoses*. **35**: 153-156.

Cox, N.H. and Irving, B. (1993). Cutaneous 'ringworm' lesions of *Scopulariopsis brevicaulis*. *British Journal of Dermatology*. **129**: 726 -728.

Coyle, V., Isaacs, J.P. and O'Boyle D.A. (1984). Canine mycetoma: a case report and review of the literature. *Journal of Small Animal Practice*. **25**: 261-268.

Creus, L., Umbert, P., Torres-Rodriguez, J.M. and Lopez-Gil, F. (1994). Ulcerous granulomatous cheilitis with lymphatic invasion caused by *Scopulariopsis brevicaulis* infection. *Journal of the American Academy of Dermatology*. **31**: 881-883.

Davis, C.L., Anderson, W.A. and McCrory, B.R. (1955). Mucormycosis in food producing

animals: a report of twelve cases. *Journal of American Veterinary Medical Association*. **126**: 261-267.

De Hoog, G.S. and Vitale, R.G. (2007). Bipolaris, Exophiala, Scedosporium, Sporothrix and other dematiaceous fungi. In PR Murray *et al* (eds.) Manual of Clinical Microbiology. Ch. 125. 9th ed. ASM Press. Washington, DC. P. 1898-191.

De Hoog, G.S., Guarro J., Gene, J. and Figueras, M.J. (2000). Atlas of Clinical Fungi, 2nd ed, vol. 1. Central bureau voor Schimmelcultures, Utrecht, The Netherlands.

De Locht, M., Boelaert, J.R. and Schneider, Y.J. (1994) Iron uptake from ferrioxamine and from ferrirhizoferrin by germinating spores of *Rhizopus microsporus*. *Biochemical Pharmacology*. **47**: 1843-1850.

DeGreef, J.H. and DeDoncker, P.R.G. (1994). Current therapy of dermatophytosis. *Journal of the American Academy of Dermatology*. **31**: S25-S30.

Del Poeta, M., Schell, W.A. and Perfect, J.R. (1997). *In vitro* antifungal activity of pneumocandin L-743, 872 against a variety of clinically important moulds. *Antimicrobial Agents and Chemotherapy*. **41**: 1835-1836.

Delaney, T.P., Uknes, S., Vernooij, B., Friedrich, L., Weymann, K., Negrotto, D., Gaffney, T., Gut-Rella, M., Kessmann, H., Ward, E. and Ryals, J. (1994). A central role of salicylic acid in plant disease resistance. *Science.* **266**: 1247-1250.

Deng, Z., Ribas, J.L. Gibson, D.W. and Connor, D.H. (1988). Infections caused by *Penicillium marneffei* in China and Southeast Asia: Review of eighteen published cases and report of four more Chinese cases. *Reviews of Infectious Diseases*. **10**: 640-652.

Denning, D.W. (1994). Treatment of invasive aspergillosis. *Journal of Infection*. **28** (Suppl. 1): 25-33.

Denning, D.W. (1996). Therapeutic outcome in invasive aspergillosis. *Clinical Infectious Diseases*. **23**: 608-615.

Denning, D.W., Marinus, A., Cohen, J., Spence, D., Herbrecht, R., Pagano, L., Kibbler, C., Kcrmery, V., Offner, F., Cordonnier, C., Jehn, U., Ellis, M., Collette, L. and Sylvester, R. (1998). An EORTC multicentre prospective survey of invasive aspergillosis in haematological patients: diagnosis and therapeutic outcome. EORTC Invasive Fungal Infections Cooperative Group. *Journal of Infection*. **37**: 173-180.

Denning, D.W., Radford, S.A. Oakley, K.L., Hall, L. Johnson, E.M. and Warnock, D.W. (1997). Correlation between *in-vitro* susceptibility testing to itraconazole and *in-vivo* outcome of *Aspergillus fumigatus* infection. *Journal of Antimicrobial Chemotherapy*. **40**: 401-414.

Descamps, F., Brouta, F., Vermout, S., Monod, M., Losson, B. and Mignon, B. (2003). Recombinant expression and antigenic properties of a 31.5-kDa keratinolytic subtilisin-like protease from *Microsporum canis*. FEMS *Immunology and Medical Microbiology*. **38**: 29-34.

Deshpande, S.D. and Koppikar, G.V. (1999). A study of mycotic keratitis in Mumbai. *Indian Journal of Pathology and Microbiology*. **42**: 81-87.

Dhar, J. and Carey, P.B. (1993). *Scopulariopsis brevicaulis* skin lesions in an AIDS patient. *AIDS*. **7**: 1283 -1284.

Dhingra, O.D., Costa, M.L.N., Silva, G.J. and Mizubuti, E.S.G. (2004). Essential oil of mustard to control *Rhizoctonia solani* causing seedling damping off and seedling blight in nursery. *Fitopatologia Brasileira*. 24: 683-686.

Dixon, D.M., Casadevall, A., Klein, B., Mendoza, L., Travassos, L. and Deepe, G.S. (1998). Development of vaccines and their use in the prevention of fungal infections. *Medical Mycology*. **36**: 57-67.

Dornbusch, H.J., Urban, C.E., Pinter, H., Ginter, G., Fotter, R., Becker, H., Miorini, T. and Berghold, C. (1995). Treatment of invasive

pulmonary aspergillosis in severely neutropenic children with malignant disorders using liposomal amphotericin B (AmBisome), granulocyte colony-stimulating factor and surgery: report of five cases. *Pediatric Hematology and Oncology*. **12**: 577-586.

Dovgich, N.A. (1981). Dermatomycoses of animals and man due to *Alternaria alternata* (Fr.) Keissler. *Mikrobiologicheskii Zhurnal*. **43**: 382-384.

Dr. Fungus: Fungi, Fungus, Fungal http. //www.doctorfungus.org / loc.cit.

Drakos, P.E., Nagler, A., Or, R., Naparstek, E., Kapelushnik, J., Engelhard, D., Rahav, G., Ne'emean, D. and Slavin, S. (1993). Invasive fungal sinusitis in patients undergoing bone marrow transplantation. *Bone Marrow Transplantation*. **12**: 203-208.

Drutz, D.J. (1983). Amphotericin B in the treatment of coccidioidomycosis. *Drugs*. **26**: 337-346.

Duthie, R. and Denning, D.W. (1995). Aspergillus fungemia: Report of two cases and review. *Clinical Infectious Diseases*. **20**: 598-605.

Dye, C., Peters, I., Taker, S., Caney, S.M.A., Dye, S., Gruffydd-Jones, T.J. and Johnson, E. (2005). Preliminary study using an indirect ELISA for the detection of serum antibodies to alternaria in domestic cats. *The Veterinary Record*. **156**: 633-635.

Ebrahimi, A., Montazeri, B. and Lolfalian, Sh. (2007). Dermatophytes isolated from the hair coat/ skin scrapping of healthy dromedaries in Iran. *Journal of Camel Practice and Research*. **14**: 133-134.

Ebright, J.R., Chandrasekar, P.H., Marks, S., Fairfax, M.R., Aneziokoro, A. and McGinnis, M.R. (1999). Invasive sinusitis and cerebritis due to *Curvularia clavata* in an immunocompetent adult. *Clinical Infectious Diseases*. **28**: 687-689.

Edwards, C., Reuther, W.L. and Greer, D.L. (2000). Disseminated osteoarticular sporotrichosis: Treatment in a patient with acquired immunodeficiency. *Southern Medical Journal*. **93**: 803-806.

Elewski, B.E. (2000). Tinea capitis: a current perspective. *Journal of the American Academy of Dermatology*. **42**: 1-20; quiz 21-24.

Elgamal, E.A. and Murshid, W.R. (2000). Intracavitary administration of amphotericin B in the treatment of cerebral aspergillosis in a non immune-compromised patient: case report and review of the literature. *British Journal of Neurosurgery*. **14**: 137-141.

England, D.M. and Hochholzer, L. (1987). Sporothrix infection of the lung without cutaneous disease: Primary pulmonary sporotrichosis. *Archives of Pathology and Laboratory Medicine*. **111**: 298-300.

Estrella, M.C., Lopez, A.G., Mellado, E., Buitrago, M.J., Monzon, A. and Tudela, J.L.R (2003). *Scopulariopsis brevicaulis*, a fungal pathogen resistant to broad-spectrum antifungal agents. *Antimicrobial Agents and Chemotherapy*. **47**: 2339-2341.

Eucker, J., Sezer, O., Graf, B. and Possinger, K. (2001). Mucormycoses. *Mycoses*. **44**: 253-260.

Fadlelmula, A., Agab, H., Le Horgne, J.M., Abbas, B. and Abdalla, A.E. (1994). First isolation of *Trichophyton verrucosum* as the aetiology of ringworm in the Sudanese camels (*Camelus dromedarius*). *Revue d Elevage et de Medecine Veterinaire des Pays Tropicaux* (Paris). **47**: 184-187.

Fernandez-Torres, B., Vazquez-Veiga, H., Llovo, X., Pereiro, Jr.M. and Guarro, J. (2000). *In vitro* susceptibility to itraconazole, clotrimazole, ketoconazole and terbinafine of 100 isolates of *Trichophyton rubrum*. *Chemotherapy*. **46**: 390-394.

Findlay, G.H., Roux, H.F. and Simson, I.W. (1971). Skin manifestations in disseminated aspergillosis. *British Journal of Dermatology*. **85**: 94-97.

Fisher, M.C., Koenig, G.L., White, T.J. and

Taylor, J.W. (2002). Molecular and phenotypic description of *Coccidioides posadasii* sp. nov., previously recognized as the non-California population of *Coccidioides immitis*. *Mycologia*. **94**: 73-84.

Fisher, M.C., Koenig, G.L., White, T.J., San-Blas, G., Negroni, R., Alvarez, I.G., Wanke, B. and Taylor, J.W. (2001). Biogeographic range expansion into South America by *Coccidioides immitis* mirrors New World patterns of human migration. *Proc National Academy of Sciences*. U S A. **98**: 4558-4562.

Fisher, N.C., Singhal, S., Miller, S.J., Hastings, J.G.M. and Mutimer. D.J. (1999). Fungal infection and liposomal amphotericin B (AmBisome) therapy in liver transplantation: a 2 year review. *Journal of Antimicrobial Chemotherapy*. **43**: 597-600.

Flynn, P.M., Magill, H.L., Jenkins, J.J., Pearson, T., Crist, W.M. and Hughes. W.T. (1990). Aspergillus osteomyelitis in a child treated for acute lymphoblastic leukemia. *Pediatric Infectious Disease Journal*. **9**: 733-736.

Friedman, A.D., Campos, J.M., Rorke, L.B., Bruce, D.A. and Arbeter, A.M. (1981). Fatal recurrent *Curvularia* brain abscess. *Journal of Pediatrics*. **99**: 413-415.

Fu, Y., Lee, H., Collins, M., Tsai, H.F., Spellberg, B., Edwards, J.E., Kwon-Chung, K.J, and Ibrahim, A.S. (2004). Cloning and functional characterization of the *Rhizopus oryzae* high affinity iron permease (rFTR1) gene. *FEMS Microbiology Letters*. **235**: 169-176.

Fujita, H., Masuda, H., Nakajima, T., Yada, K., Watanabe, M. and Kagitani, Y. (1995). Protective effect of human macrophage colony-stimulating factor on fungal infection (2). *In vitro* effect of human macrophage colony-stimulating factor on systemic aspergillosis and *in vitro* effect on the activities of macrophage. *Kansenshogaku Zasshi - Journal of the Japanese Association for Infectious Diseases*. **69**: 582-589.

Fung-Tomc, J.C., Minassian, B., Huczko, E., Kolek, B., Bonner, D.P. and Kessler. R.E. (1995). *In vitro* antifungal and fungicidal spectra of a new pradimicin derivative, BMS-181184. *Antimicrobial Agents and Chemotherapy*. **39**: 295-300.

Gaffney, T., Friedrich, L., Vernooij, B., Negrotto, D., Nye, G., Uknes, S., Ward, E., Kessmann, H. and Ryals, J. (1993). Requirement of salicylic acid for the induction of systemic acquired resistance. *Science*. **261**: 754-756.

Galgiani, J.N. (1999). Coccidioidomycosis: A regional disease of national importance - Rethinking approaches for control. *Annals of Internal Medicine*. **130**: 293-300.

Galgiani, J.N., Ampel, N.M., Catanzaro, A., Johnson, R.H., Stevens, D.A. and Williams, P.L. (2000). Practice guidelines for the treatment of coccidioidomycosis. *Clinical Infectious Diseases*. **30**: 658-661.

Gallo, M.G., Lanfranchi, P., Poglayen, G., Calderola, S., Menzano, A., Ferroglio, E. and Peano, A. (2005). Seasonal 4-year investigation into the role of the alpine marmot (*Marmota marmota*) as a carrier of zoophilic dermatophytes. *Medical Mycology*. **43**: 373-379.

Garau, J., Diamond, R.D., Lagrotteria, L.B. and Kabins, S.A. (1977). Alternaria osteomyelitis [letter]. *Annals of Internal Medicine*. **86**: 747-748.

Garbino, J., Kolarova, L., Lew, D., Hirschel, B. and Rohner, P. (2001). Fungemia in HIV-infected patients: A 12-year study in a tertiary care hospital. *AIDS Patient Care and STDs*. **15**: 407-410.

Gefter, W.B. (1992). The spectrum of pulmonary aspergillosis. *Journal of Thoracic Imaging*. **7**: 56-74.

Georg, L.K. (1954). Animal ringworm in public health. Dermatophytes: New methods in classification. Atlanta, GA, US. Public Health Service.

Gercovich, F., Richman, S., Odriguez, V., Una, M., Mccrediem, K. and Bodey, G. (1975).

Successful control of systemic *Aspergillus niger* infections in two patients with acute leukemia. *Cancer*. **36**: 2271-2276.

Gerson, S.L., Talbot, G.H., Hurwitz, S., Strom, B.L., Lusk, E.J. and Cassileth, P.A. (1984). Prolonged granulocytopenia: the major risk factor for invasive pulmonary aspergillosis in patients with acute leukemia. *Annals of Internal Medicine*. **100**: 345-351.

Gillespie, M.B. and O'Malley, B.W. (2000). An algorithmic approach to the diagnosis and management of invasive fungal rhino sinusitis in the immunocompromised patient. *Otolaryngologic Clinics of North America*. **33**: 323-334.

Ginarte, M., Pereiro, Jr. M., Fernandez-Redondo, V. and Toribio, J. (1996). Plantar infection by *Scopulariopsis brevicaulis*. *Dermatology*. **193**: 149-151.

Gitao, C.G., Agab, H. and Khalifalla, A.J. (1998). An outbreak of a mixed infection of *Dermatophilus congolensis* and *Microsporum gypseum* in camels (*Camelus dromedarius*) in Saudi Arabia. *Revue scientifique et technique International Office of Epizootics*. **17**: 749-755.

Goettmann-Bonvallot, S. (2003). Clinical types of onychomycosis. *Annales de Dermatologie et de Venereologie*. **130**: 1237-1243.

Gonzalez, G.M., Tijerina, R., Najvar, L.K., Bocanegra, R., Luther, M., Rinaldi, M.G. and Graybill, J.R. (2001). Correlation between antifungal susceptibilities of *Coccidioides immitis in vitro* and antifungal treatment with caspofungin in a mouse model. *Antimicrobial Agents and Chemotherapy*. **45**:1854-1859.

Gordon, G. and Giddings, N.A. (1994). Invasive otitis externa due to *Aspergillus* species: Case report and review. *Clinical Infectious Diseases*. **19**: 866-870.

Gori, S., Lupetti, A., Moscato, G., Parenti, M. and Lofaro, A. (1997). Pulmonary sporotrichosis with hyphae in a human immunodeficiency virus-infected patient. A case report. *Acta Cytologica*. **41**: 519-521.

Greene, C.E., Brockus, C.W., Currin, M.P. and Jones, C.J. (2002). Infection by *Basidiobolus ranarum* in two dogs. *Journal of American Veterinary Medical Association*. **221**: 528-532.

Grooters, A.M. (2003). Pythiosis, lagenidiosis and zygomicosis in small animals. *Veterinary Clinics of North America: Small Animal Practice*. **33**: 695-720.

Grossi, P., Farina, C., Fiocchi, R. and Dalla Gasperina, D. (2000). Prevalence and outcome of invasive fungal infections in 1,963 thoracic organ transplant recipients-A multicenter retrospective study. *Transplantation*. **70**: 112-116.

Guarro, J., Akiti, T., Almada-Horta, R., Leite, L.A. M., Gene, J., Ferreira-Gomes, S., Aguilar, C. and Ortoneda, M. (1999). Mycotic keratitis due to *Curvularia senegalensis* and *in vitro* antifungal susceptibilities *of Curvularia* spp. *Journal of Clinical Microbiology*. **37**: 4170-4173.

Gugani, H.C. (1972). *Trichophytobn verrucosum* infection in cattle in India. *Mykosen*. **15**: 285-288.

Gugnani, H.C., Randhawa, H.S. and Srivastava, B. (1971). Isolation of dermatophytes and other kerationophilic fungi from apparently healthy skin coats of domestic animals. *Indian Journal of Medical Research*. **59**: 1699-1702.

Gumbo, T., Taege, A.J., Mawhorter, S., McHenry M. C., Lytle, B.H., Cosgrove, D.M. and Gordon, S.M. (2000). Aspergillus valve endocarditis in patients without prior cardiac surgery. *Medicine*. **79**: 261-268.

Gupta, A.K. and Summerbell, R.C. (1999). Combined distal and lateral subungual and white superficial onychomycosis in the toe nails. *Journal of the American Academy of Dermatology*. **41**: 938-944.

Gupta, P.K., Singh, R.P. and Singh, I.P. (1968). Dermatophytes from man, dogs, pigs with special reference to *Trichophyton simii* and *Microsporum nanum*. *Indian Journal of Animal Health*. **11**: 1-3.

Gupta, P.K., Singh, R.P. and Singh, I.P. (1970). A

study of dermatomycoses (ringworm) in domestic animals and fowls. *Indian Journal of Animal Health*. **9**: 85-89.

Gurwith, M. (1999). Clinical efficacy of amphotericin B colloidal dispersion against infections caused by *Aspergillus* spp. *Chemotherapy*. **45**: 34-38.

Hagensee, M.E., Bauwens, J.E., Kjos, B. and Bowden, R.A. (1994). Brain abscess following marrow transplantation: experience at the Fred Hutchinson Cancer Research Center, 1984-1992. *Clinical Infectious Diseases*. **19**: 402-408.

Halley, L.D. and Standard, P.G. (1973). Laboratory Methods in Medical Mycology, 3rd Ed. U S Department of Health, Education and Welfare, Center of Disease Control, Atlanta, pp. 41- 57.

Harley, W.B., Dummer, J.S., Anderson, T.L. and Goodman, S. (1995). Malignant external otitis due to *Aspergillus flavus* with fulminant dissemination to the lungs. *Clinical Infectious Diseases*. **20**: 1052-1054.

Hay, R.J. (2000). Dermatophytosis and other superficial mycoses. Pp. 2757-2768. In Mandell, G.L. Bennett, J.E. and Dolin, R. (ed.), Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases, 5th ed. vol. 2. Churchill Livingstone, London.

Hector, R.F., Zimmer, B.L. and Pappagianis, D. (1990). Evaluation of nikkomycins X and Z in murine models of coccidioidomycosis, histoplasmosis and blastomycosis. *Antimicrobial Agents and Chemotherapy*. **34**: 587-593.

Hill, P.B., Moriello, K.A. and Shaw, S.E. (1995). A review of systemic antifungal agents. *Veterinary Dermatology*. **6**: 59-66.

Ho, P.L. and Yuen, K.Y. (2000). Aspergillosis in bone marrow transplant recipients. *Critical Reviews in Oncology/Hematology*. **34**: 55-69.

Hoffman, P., Gari-Toussaint, M., De Bievre, C., Michiels, J.F., d'Horpock, F.A. and Loubiere, R. (1993). Rhino-orbito-cerebral mucormycosis caused by *Rhizopus oryzae*. A typical case in a cirrhotic patient. *Annals of Pathology*. **13**: 180-183.

Howard, D.H. (1999). Acquisition, transport, and storage of iron by pathogenic fungi. *Clinical Microbiology Review*. **12**: 394-404.

Hsu, M.M. and Lee, J.Y. (1993). Cutaneous and subcutaneous phaeohyphomycosis caused by *Exserohilum rostratum*. *Journal of the American Academy of Dermatology*. **28**: 340-344.

Isaac, M. (1996). Cutaneous aspergillosis. *Dermatologic Clinics*. **14**: 137-140.

Jand, S.K and Dhillon, S.S (1975). Mastitis caused by fungi. *Indian Veterinary Journal*. **52**: 123-128.

Jand, S.K., Singh, K.B. and Narula, A.S. (1978). Preliminary studies on mycoflora of the vulvo-vaginal tract of sheep and goat. *Indian Veterinary Journal*. **55**: 518-520.

Jessup, C.J., Ryder, N.S. and Ghannoum, M.A. (2000). An evaluation of the *in vitro* activity of terbinafine. *Medical Mycology*. **38**: 155-159.

Jiang, C., Magee, D.M., Quitugua, T.N. and Cox, R.A. (1999). Genetic vaccination against *Coccidioides immitis:* Comparison of vaccine efficacy of recombinant antigen 2 and antigen 2 cDNA. *Infection and Immunity*. **67**:630-635.

Johnson, E.M., Szekely, A. and Warnock, D.W. (1999). *In vitro* activity of Syn-2869, a novel triazole agent, against emerging and less common mold pathogens. *Antimicrobial Agents and Chemotherapy*. **43**: 1260-1263.

Jones, N.F, Conklin, W.T. and Albo, V.C. (1986). Primary invasive aspergillosis of the hand. *Journal of Hand Surgery* (America). **11**: 425-428.

Joshi, B.N., Sainani, M.N., Bastawade, K.B., Gupta, V.S. and Ranjekar, P.K. (1998). Cysteine protease inhibitor from pearl millet: a new class of antifungal protein. *Biochemical and Biophysical Research Communications*. **246**: 382-387.

Kappe, R. (1999). Antifungal activity of the new azole UK-109, 496 (voriconazole). *Mycoses*. **42**: 83-86.

Kauffman, C.A. (1999). Sporotrichosis. *Clinical Infectious Diseases*. **29**: 231-237.

Kauffman, C.A. (2007). Candidiasis. In: Goldman L, Ausiello D, eds. *Cecil Medicine*. 23rd ed. Philadelphia, Pa: Saunders Elsevier. Chap. 359.

Kauffman, C.A., Hajjeh, R. and Chapman, S.W. (2000). Practice guidelines for the management of patients with sporotrichosis. *Clinical Infectious Diseases*. **30**: 684-687.

Kaufman, S.M. (1971). *Curvularia* endocarditis following cardiac surgery. *Journal of Clinical Pathology*. **56**: 466-470.

Keller, M., Krehon, S., Stanek, C. and Rosengarten, R. (2000). Keratinopathogenic mould fungi and dermatophytes in healthy and diseased hooves of horses. *Veterinary Record*. **147**: 619 -622.

Khan, Z.U., Khoursheed, M., Makar, R., Al-Waheeb, S., Al-Bader, I., Al-Muzaini, A., Chandy, R. and Mustafa, A.S. (2001). *Basidiobolus ranarum* as an etiologic agent of gastrointestinal zygomycosis. *Journal of Clinical Microbiology*. **39**: 2360-2363.

Khan, Z.U., Randhawa, H.S. and Lulla, M. (1982). Isolation of *Blastomyces dermatidis*- from the lungs of a bat *Rhinopoma hardwickei gray* in Delhi. *Sabouraudia*. **20**: 137-144.

Khandhari, K.C. and Sethi, K.K (1964). Dermatophytosis in Delhi area. *Indian Journal of Medical Research*. **42**:324 -326.

Khosravi, A.R., Shokri, H. and Sharifzadeh, A. (2009). Fungal flora of the eye and nose of healthy dromedary camels (*Camelus dromedaries*) in Iran. *Journal of Camel Practice and Research*. **16**: 63-67.

Khosravi, A.R., Shokri, H., Ziglari, T. and Niasari-Naslaji, A. (2008). A study of mycoflora of the external ear canals in dromedary camels in Iran. *Journal of Camel Practice and Research*. **15**: 153-157.

Kim, J.H. (1997). Antibacterial action of onion (*Allium cepa* L.) extracts against oral pathogenic bacteria. *Journal of Nihon University School of Dentistry*. **39**: 136-141.

Kindo, A.J., Rana, N.S., Rekha, A. and Kalyani, J. (2010). Fungal infections in the soft tissue: A study from a tertiary care center. *Indian Journal of Medical Microbiology*. **28**: 164-166.

Knudtson, W.U. and Kirkbride, C.A. (1992). Fungi associated with bovine abortion in the northern plains states (USA). *Journal of Veterinary Diagnostic Investigation*. **4**: 181-185.

Kodagali, S.B. (1979). Prevalence of fungi in buffalo semen. *Indian Veterinary Journal*. **56**: 807-809.

Kostman, J.R. and DiNubile, M.J. (1993). Nodular lymphangitis: a distinctive but often unrecognized syndrome. *Annals of Internal Medicine*. **118**: 883-888.

Kuhn, F.A. and Javer, A.R. (2000). Allergic fungal rhinosinusitis - perioperative management, prevention of recurrence and role of steroids and antifungal agents. *Otolaryngologic Clinics of North America*. **33**: 419-432.

Kulkarni, V.B., Chaudhari, P.G., Kulkarni, M.P. and Sane, H.D. (1969). Equine ringworm caused by *Trichophyton tonsurans var sufureum*. *Indian Veterinary Journal*. **46**: 215-218.

Kurosawa, A., Pollock, S.C., Collins, M.P., Kraff, C.R. and Tso, M.O.M. (1988). *Sporothrix schenckii* endophthalmitis in a patient with human immunodeficiency virus infection. *Archives of Ophthalmology*. **106**: 376-380.

Kuttin, E.S., Alhanaty, E., Feldman, M., Chaimovits, M. and Muller, J. (1986). Dermatophytosis of camels. *Journal of Medical and Veterinary Mycology*. **24**: 341-344.

Lampert, H.B., Hutto, J.H., Donnelly, W.H. and Shulman, S.T. (1977). Pulmonary and cerebral mycetoma caused by *Curvularia pallescens*. *Journal of Pediatrics*. **91**: 603-605.

Larone, D.H. (1995). Medically important fungi - A guide to identification, 3rd ed. ASM Press,

Washington, D.C.

Lesperance, M., Baumgartner, D. and Kauffman, C.A. (1988). Polyarticular arthritis due to *Sporothrix schenckii*. *Mycoses*. **31**: 599-603.

Lewis, D.T., Foil, C.S. and Hosgood, G. (1991). Epidemiology and clinical features of dermatophytosis in dogs and cats at Louisiana State University: 1981-1990. *Veterinary Dermatology*. **2**: 53-58.

Li, R.K. and Rinaldi, M.G. (1999). *In vitro* antifungal activity of nikkomycin Z in combination with fluconazole or itraconazole. *Antimicrobial Agents and Chemotherapy*. **43**: 1401-1405.

Li, R.K., Ciblak, M.A., Nordoff, N., Pasarell, L., Warnock, D.W. and McGinnis, M.R. (2000). *In vitro* activities of voriconazole, itraconazole and amphotericin B against *Blastomyces dermatitidis*, *Coccidioides immitis* and *Histoplasma capsulatum*. *Antimicrobial Agents and Chemotherapy*. **44**: 1734-1736.

Lindsley, M.D., Hurst, S.F., Iqbal, N.J. and Morrison, C.J. (2001). Rapid identification of dimorphic and yeast-like fungal pathogens using specific DNA probes. *Journal of Clinical Microbiology*. **39**: 3505-3511.

Lopez-sanranan, J., Paya, M.J., Cutuli, M.T. and Gonzalez, J.L. (2000). Cutaneous mucormycosis caused by *Absidia corymbifera* in a horse. *Veterinary Dermatology*. **11**: 151-155.

Lortholary, O., Denning, D.W. and Dupont, B. (1999). Endemic mycoses: a treatment update. *Journal of Antimicrobial Chemotherapy*. **43**: 321-331.

Loveless, M.O., Winn, R.E., Campbell, M. and Jones, S.R. (1981). Mixed invasive infection with *Alternaria* species and *Curvularia* species. *American Journal of Clinical Pathology*. **76**: 491-493.

Macura, A.B. (1993). *In vitro* susceptibility of dermatophytes to antifungal drugs: a comparison of two methods. *International Journal of Dermatology*. **32**: 533-536.

Maertens, J., Raad, I., Sable, C.A., Ngai, A., Berman, R., Patterson, T.F., Denning, D. and Walsh, T. (2000). Multicenter, non comparative study to evaluate safety and efficacy of caspofungin in adults with aspergillosis refractory or intolerant to amphotericin B, amphotericin B lipid formulations or azoles. 40th Interscience Conference on Antimicrobial Agents and Chemotherapy, Abstract No. 1103.

Maghoub, E.S. (1973). Mycetomas caused by *Curvularia lunata*, *Madurella grisea*, *Aspergillus nidulans* and *Nocardia brasiliensis* in Sudan. *Sabouraudia*. **11**: 179-182.

Magid, M.L., Prendiville, J.S. and Esterly, N.B. (1988). Violaceous nodules on the arm of a child with acute lymphocytic leukemia. Primary cutaneous aspergillosis (*Aspergillus flavus*). *Archives of Dermatology*. **124**: 122, 125-126.

Mahajan, V.M. and Mohapatra, L.N. (1968). Study of human and animal dermatophytes in rural areas. ll. *Mykosen*. **11**: 293-789.

Mahmoud, A.L.E. (1993). Dermatophytes and other associated fungi isolated from ringworm lesions of camels. *Folia Microbiologica* (Praha). **38**: 505-508.

Malamy, J., Carr, J.P., Klessig, D.F. and Raskin, I. (1990). Salicylic acid - a likely endogenous signal in the resistance response of tobacco to tobacco mosaic virus infection. *Science*. 250: 1002-1004.

Malecha, M.A. (2004). Fungal keratitis caused by *Scopulariopsis brevicaulis* treated successfully with natamycin. *Cornea*. **23**: 201-203.

Malik, A.K., Arora, D.R. and Prakash, K. (1984). Animal dermatophytosis in North India. *Indian Veterinary Medical Journal*. **8**: 93-96.

Mancianti, F., Papini, R. and Cavicchio, P. (1988). Dermatofizia da *Microsporum gypseum* in un camello (*Camelus dromedarius*). *Annali della Facolta di Medicina Veterinaria di University Pisa*. **4**: 233-237.

Mancianti, F., Pedonese, F., Millanta, F. and Guarnieri, L. (1999). Efficacy of oral terbinafine in feline dermatophytosis due to *Microsporum canis*. *Journal of Feline Medicine and Surgery*. **1**: 37-41.

Manning, S.C., Schaefer, S.D., Close, L.G. and Vuitch, F. (1991). Culture-positive allergic fungal sinusitis. *Archives of Otolaryngology-Head and Neck Surgery*. **117**:174-178.

Mathews, M.S. and Maharajan, S.V. (1999). *Exserohilum rostratum* causing keratitis in India. *Medical Mycology*. **37**: 131-132.

Mbata, T.I. and Nwajagu, C.C. (2007). Dermatophytes and other fungi associated with hair-scalp of nursery and primary school children in Awka, Nigeria. *Sudanese Journal of Dermatology*. **5**: 15-21.

McAleer, R., Kroenert, D.B., Elder, J.L. and Froudist, J.H. (1981). Allergic bronchopulmonary disease caused by *Curvularia lunata* and *Drechslera hawaiiensis*. *Thorax*. **36**: 338-344.

McEwen, J.G., Taylor, J.W., Carter, D., Xu, J., Felipe, M.S.S., Vilgalys, R., Mitchell, T.G., Kasuga, T., White, T., Bui, T. and Soares, C.M.A. (2000). Molecular typing of pathogenic fungi. *Medical Mycology*. **38**: 189-197.

McGinnis, M.R. and Pasarell, L. (1998). *In vitro* testing of susceptibilities of filamentous ascomycetes to voriconazole, itraconazole and amphotericin B, with consideration of phylogenetic implication. *Antimicrobial Agents and Chemotherapy*. **36**: 2353-2355.

Medoff, G., Dismukes, W.E., Pappagianis, D., Diamond, R., Gallis, H.A. and Drutz, D. (1992). Evaluation of new antifungal drugs for the treatment of systemic fungal infections. Infectious Diseases Society of America and the Food and Drug Administration. *Clinical Infectious Diseases*. 15 Suppl 1: S274-281.

Metraux, J.P., Signer, H., Ryals, J., Ward, E., Wyss-Benz, M., Gaudin, J., Raschdorf, K., Schmid, E., Blum, W. and Inverard, B. (1990). Increase in salicylic acid at the onset of systemic acquired resistance in cucumber. *Science*. **250**: 1004-1006.

Miele, P.S., Levy, C.S., Smith, M.A., Dugan, E.M., Cooke, R.H., Light, J.A. and Lucey, D.R. (2002). Primary cutaneous fungal infections in solid organ transplantation: a case series. *American Journal of Transplantation*. **2**: 678-683.

Mikolich, D.J., Kinsella, L.J., Skowron, G., Friedman, J. and Sugar, A.M. (1996). Aspergillus meningitis in an immunocompetent adult successfully treated with itraconazole. *Clinical Infectious Diseases*. **23**: 1318-1319.

Miller, R. and Campbell, R.S.F. (1984). The comparative pathology of equine cutaneous phycomycosis. *Veterinary Pathology*. **21**: 325-332.

Miller, R.I. and Pott, B. (1980). Phycomycosis of the horse caused by *Basidiobolus haptosporus*. *Australian Veterinary Journal*. **56**: 224-227.

Miller, R.I. and Turnwald, G. (1984). Disseminated basidiobolus mycosis in a dog. *Veterinary Pathology*. **21**: 117-119.

Mitra, S.K. (1998). Dermatophytes isolated from selected ruminants in India. *Mycopathologia*. **142**: 13-16.

Mitra, S.K., Sikdar, A. and Harbola, P.C. (1989). Prospective survey on bovine derma tophytes in Uttar Pradesh. *Indian Journal of Comparative Microbiology Immunology and Infectious Diseases*. **10**: 97-99.

Mohapatra, L.N. and Mahajan, V.M. (1970). *Trichophyton simii* infection in man and animals. *Mycopathologia et Mycologia Applicata*. **41**: 357-362.

Moneymaker, C.S., Shenep, J.L., Pearson, T.A., Field, M.L. and Jenkins, J.J. (1986). Primary cutaneous phaeohyphomycosis due to *Exserohilum rostratum* (*Drechslera rostrata*) in a child with leukemia. *Pediatric Infectious Disease Journal*. **5**: 380-382.

Monga, D.P., Kapur, M.P. and Dixit, S.N. (1978). Bovine abortion caused by *Nocardia asteroides*. *Mykosen*. **21**: 152-153.

Monga, D.P., Tewari, S.C. and Parased, S. (1983). Mycotic abortions in equines. *Mykosen.* **26**: 612-614.

Moretti, A., Boncio, L., Pasquali, P. and Fioretti, D.P. (1998). Epidemiological aspects of dermatophyte infections in horses and cattle. *Zentbl. Veterinary Medicine*. **B45**: 205-258.

Morgan, M. and Reves, R. (1996). Invasive sinusitis due to *Sporothrix schenckii* in a patient with AIDS. *Clinical Infectious Diseases*. **23**: 1319-1320.

Morgan, M.A., Cockerill, F.R., Cortese, D.A. and Roberts, G.D. (1984). Disseminated sporotrichosis with *Sporothrix schenckii* fungemia. *Diagnostic Microbiology and Infectious Disease*. **2**: 151-155.

Mori, T., Matsumura, M., Yamada, K., Irie, S., Oshimi, K., Suda, K., Oguri, T. and Ichinoe, M. (1998). Systemic aspergillosis caused by an aflatoxin-producing strain of *Aspergillus flavus*. *Medical Mycology*. **36**: 107-112.

Moriello, K.A. (2004). Treatment of dermatophytosis in dogs and cats: review of published studies. *Veterinary Dermatology*. **15**: 99-107.

Moriello, K.A., DeBoer, D.J., Greek, J., Kuhi, K. and Fintelman, M. (2003). The prevalence of immediate and delayed type hypersensitivity reactions to *Microsporum canis* antigens in cats. *Journal of Feline Medicine and Surgery*. **5**: 161-166.

Morrison, V.A., Haake, R.J. and Weisdorf, D.J. (1993). The spectrum of non- candida fungal infections following bone marrow transplantation. *Medicine (Baltimore)*. **72**: 78-89.

Murray, E. Fowler (1998). Medicine and surgery of South American Camelids. 2nd Edition. Blackwell Publishing, pp. 156-158.

Mycology Online | http. //www.mycology. adelaide.edu.au / loc.cit.

Nag, P.C. and Mallik, B.S. (1961). Aspergillosis in a calf. *Canadian Veterinary Journal*. **2**: 30-32.

Naniwadekar, M.R., Jagtap, S.V., Nikam, B.P. and Sanghavi, K.D. (2009). Subcutaneous phycomycosis in a child. *Online Journal of Health and Allied Sciences*. **8**: 14.

Nathan, M.D., Keller, A.P., Lerner, C.J. and Davis, J.C. (1982). Entomophthorales infection of the maxillofacial region. *Laryngoscope*. **92**: 767-769.

Neri, G., Ciardo, M.G. and Croce, A. (2002). Rhinocerebral mucormycosis: report of a rare case in the head-neck and chest area. *Acta otorhinolaryngologica Italica*. **22**: 28-33.

Newbury, S., Moriello, K., Verbrugge, M. and Thomas, C. (2007). Use of lime sulphur and itraconazole to treat shelter cats naturally infected with *Microsporum canis* in an annex facility: an open field trial. *Veterinary Dermatology*. **18**: 324- 331.

Nielsen, P.V. and Rios, R. (2000). Inhibition of fungal growth on bread by volatile components from spices and herbs and the possible application in active packaging, with special emphases on mustard essential oil. *International Journal of Food Microbiology*. **60**: 219-229.

Nweze, E.I. (2001). Etiology of dermatophytoses amongst children in northeastern Nigeria. *Medical Mycology*. **39**: 181-184.

O'Brien, C.R., Krockenberger, M.B., Martin, P., Wigney, D.I. and Malik, R. (2006). Long-term outcome of therapy for 59 cats and 11 dogs with cryptococcosis. *Australian Veterinary Journal*. **84**: 384-392.

Oakley, K.L., Moore, C.B. and. Denning. D.W. (1998). *In vitro* activity of the echinocandin antifungal agent LY303, 366 in comparison with itraconazole and amphotericin-B against *Aspergillus* spp. *Antimicrobial Agents and Chemotherapy*. **42**: 2726-2730.

Odds, F.C. (2001). Sordarin antifungal agents. *Expert Opinion on Therapeutic Patents*. **11**: 283-294.

Ogawa, H., Summerbell, R.C., Clemons, K.V., Koga, T., Ran, Y.P., Rashid, A., Sohnle, P.G., Stevens, D.A. and Tsuboi, R. (1998). Dermatophytes and host defence in cutaneous mycoses. *Medical Mycology*. **36**: 166-173.

Ogawa, S., Shibahara, T., Sano, A., Kadota, K. and Kubo, M. (2008). Generalized hyperkeratosis caused by *Scopulariopsis brevicaulis* in a Japanese black calf. *Journal of Comparative Pathology*. **138**: 145-150.

Ohashi, Y. (1960). On a rare disease due to *Alternaria tenuis* nees (Altemariasis). *The Tohoku Journal of Experimental Medicine*. **72**: 78-82.

Ossent, P. (1987). Systemic aspergillosis and mucormycosis in 23 cats. *Veterinary Record*. **120**: 330-333.

Owens, W.R., Miller, R.I., Haynes, P.F. and Snider, T.G. (1985). Phycomycosis caused by *Basidiobolus haptosporus* in two horses. *Journal of American Veterinary Medical Association*. **186**: 703-705.

Padhya, A.A., Radhakrishnan, C.V. and Thirumalachar, M.J. (1966). Studies on dermatomycosis in Poona. I. *Hindustan Antibiotic Bulletin*. **9**: 23-26.

Pal, K.N. (1956). Aspergillosis skin infection in a calf. *Indian Veterinary Journal*. **33**: 150-151.

Pal, M. (1981). Isolation of *Microsporum canis* from man and dog. *Arogya Journal of Health Sciences*. **7**: 125-127.

Pal, M. (1982). Isolation of *Aspergillus niger* from otitis in dog. *Veterinary Research Journal*. **5**: 62-63.

Pal, M. (1983). Keratomycosis in a buffalo calf (*Bubalis bubalis*) caused by *Aspergillus fumigatus*. *Veterinary Record*. **113**: 67.

Pal, M. (1987). Dermatophytosis in cattle, clinical and mycological studies. *Indian Journal of Animal Sciences*. **57**: 856-857.

Pal, M. (1989). Colonization of a squamous cell carcinoma in the bovine horn core by *Aspergillus terreus*. *Mycoses*. **32**:197-199.

Pal, M. (1996) Guttural pouch mycosis in a horse: the first reported case in India. *Revista-due to Aspergillus flavus*. *Buffalo Bulletin*.**16**: 20-22.

Pal, M. and Lee, C.W. (1994). Pulmonary zygomycosois in chicks due to *Absidia Corymbifera*. *Korean Journal of Veterinary Clinical Medicine*. **11**: 1-3.

Pal, M. and Mehrotra, B.S. (1984). Association of *Aspergillus fumigatus* with rhinitis. *Veterinary Record*. **115**: 167.

Pal, M. and Ragi, A.S. (1989). Fungi isolated from lymph nodes of buffaloes. *Mycoses*. **32**: 578-580.

Pal, M. and Singh, D.K. (1983). Studies on dermatomycoses in dairy animals. *Mycoses*. **26**: 317-323.

Pal, M. and Verma, J.D. (1987). *Aspergillus terreus* as a possible cause of mycetoma on the foot of a dog. *Mykosen*. **30**: 172-174.

Pal, M., Dholakia, P.M. and Anjaria, J.M. (1987). *A. niger* as a causative agent of dermatitis. *Indian Veterinary Medical Journal*. **11**: 46-49.

Pal, M., Dube, G.D. and Mehrotra, B.S. (1986). Some observations on sinusitis in dogs and cats due to *Aspergillus fumigatus*. *Indian Veterinary Medical Journal*. **10**: 120-124.

Pal, M., Lee, C., Pal, M. and Lee, C.W. (1995). Keratomycosis in a pet rabbit due to *Curvularia lunata*. *Korean Journal of Veterinary Clinical Medicine*. **12**: 61-64.

Pal, M., Mattsusaka, N. and Lee, C.W. (1994). Clinical and mycological observations on equine ringworn due to *Microsporum gypseum*. *Korean Journal of Veterinary Clinical Medicine*. **11**: 5 - 8.

Pal, M., Patil, D.B., Kelawala, N.H., Parikh, P.V., Barvalia, D.R. and Patel, D.M. (2007). Occurrence of fungal otitis in camels. *Journal of Camel Practice and Research*. **14**: 73-74.

Pappagianis, D. (1993). Evaluation of the protective efficacy of the killed *Coccidioides immitis* spherule vaccine in humans. The Valley Fever Vaccine Study Group. *American*

Review of Respiratory Disease. **148**: 656-660.

Pascoe, R.R. (1976). Studies on the prevalence of ringworm among horses in racing and breeding stables. *Australian Veterinary Journal*. **52**: 419-421.

Paterson, S. (1997). Dermatophytosis in 25 horses-a protocol of treatment using topical therapy. *Equine Veterinary Education*. **9**: 171-173.

Paterson, S. (1999). Miconazole/chlorhexidine shampoo as an adjunct to systemic therapy in controlling dermatophytosis in cats. *Journal of Small Animal Practice*. **40**: 163 -166.

Peng, T., Orsborn, K.I., Orbach, M.J. and Galgiani, J.N. (1999). Proline-rich vaccine candidate antigen of *Coccidioides immitis*: Conservation among isolates and differential expression with spherule maturation. *Journal of Infectious Diseases*. **179**: 518-521.

Penn, C.C., Goldstein, E. and Bartholomew, W.R. (1992). *Sporothrix schenckii* meningitis in a patient with AIDS. *Clinical Infectious Diseases*. **15**: 741-743.

Pierce, N.F., Millan, J.C. Bender, B.S. and Curtis, J.L. (1986). Disseminated curvularia infection. Additional therapeutic and clinical considerations with evidence of medical cure. *Archives of Pathology and Laboratory Medicine*. **110**: 959-961.

Pitt, J.I. (2000). Toxigenic fungi: which are important? *Medical Mycology*. **38**: 17-22.

Pitt, J.I., Basilico, J.C., Abarca, M.L. and Lopez, C. (2000). Mycotoxins and toxigenic fungi. *Medical Mycology*. **38**: 41-46.

Pohlenz, J., Ehrensperger, F. and Breer, C. (1973). Spontaneous death in cattle as a result of phycomycosis of the fore stomach. *Schweizer Arch Tierheilk*. **115**: 161-168.

Polesky, A., Kirsch, C.M., Snyder, L.S., LoBue, P., Kagawa, F.T., Dykstra, B.J., Wehner, J.H., Catanzaro, A., Ampel, N.M. and Stevens, D.A. (1999). Airway coccidioidomycosis - Report of cases and review. *Clinical Infectious Diseases*. **28**: 1273-1280.

Powell, B.L., Drutz, D.J., Huppert, M. and Sun, *lberoamericana-Micologia*. **13**: 31-32.

Pal, M. (1997). Mycotic keratitis in a buffalo calf S.H. (1983). Relationship of progesterone- and estradiol-binding proteins in *Coccidioides immitis* to coccidioidal dissemination in pregnancy. *Infection and Immunity*. **40**: 478-485.

Prystowsky, S.D., Vogelstein, B., Ettinger, D.S., Merz, W.G., Kaizer, H., Sulika, V.I. and, Zinkham, W.H. (1976). Invasive aspergillosis. *New England Journal of Medicine*. **295**: 655-658.

Quinn, P.J., Carter, M.E., Markey, B. and Carter, G.R. (1994). Clinical Veterinary Microbiology, 1st Ed, Wolfe Publishing, London, PP: 116-119; 1164-1167.

Qureshi, S. Wani, S.A. and Beg, S. (2006). Curvularia dermatomycosis in a Jersey heifer: A case Report. *Pakistan Veterinary Journal*. **26**: 149-150.

Radostits, O.M., Blood, D.C. and Gay, C.C. (1997). Veterinary Medicine, 8th Ed, Bailliere Tindall, London, pp. 381-390.

Rahman, H. and Baxi, K.K. (1983). Prevalence of *Candida albicans* in bovine mastitis. *Indian Journal of Comparative Microbiology, Immunology and Infectious Diseases*. **4**: 49-50.

Ranganathan, S., Balajee, A.M.S. and Raja, S.M. (1998). A survey of dermatophytosis in animals in Madras, India. *Mycopathologia*. **140**: 137-140.

Rao, K.S. and Sambamurthy, B. (1972). *Candida albicans* gastric ulcers of country swine. *Indian Veterinary Journal*. **49**: 457.

Rasmussen, J.B., Hammerschmidt, R. and Zook, M.N. (1991). Systemic induction of salicylic acid accumulation in cucumber after inoculation with *Pseudomonas syringae* pv *syringae*. *Plant Physiology*. **97**:1342-1347.

Reddy, B.D., Kelley, D.C., Minocha, H.C. and Anthony, H.D. (1974). Pathogenicity of *Alternaria alternata* and its antibody

production in experimental animals. *Mycopathologia et mycologia applicata*. **54**: 385-390.

Regli, P. and Ferrari, H. (1989). *In vitro* action spectrum of a new antifungal agent derived from morpholine: amorolfin. *Pathologie Biologie* (Paris). **37**: 617-620.

Reidarson, T.H., Griner, L.A., Pappagiansis, D. and McBain, J. (1998). Coccidioidomycosis in a bottlenose dolphin. *Journal of Wildlife Diseases*. **34**: 629-631.

Restrepo, A. (1994). Treatment of tropical mycoses. *Journal of the American Academy of Dermatology*. **31**: S91-102.

Rex, J.H. and Okhuysen, P.C. (2000). *Sporothrix schenckii*, p. 2695-2698. In G. L. Mandell, J. E. Bennett, and R. Dolin (ed.), Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases, 5th ed. Churchill Livingstone, New York.

Ribes, J.A., Vanover-Sams, C.L. and Baker, D.J. (2000). Zygomycetes in human disease. *Clinical Microbiology Reviews*. **13**: 236-301.

Rimek, D., Zimmermann, T., Hartmann, M., Prariyachatigul, C. and Kappe, R. (1999). Disseminated *Penicillium marneffei* infection in an HIV-positive female from Thailand in Germany. *Mycoses*. **42**: 25-28.

Rinaldi, M.G., Phillips, P., Schwartz, J.G., Winn, R.E., Holt, G.R., Shagets, F.W., Elrod, J., Nishioka, G. and Aufdemorte, T.B. (1987). Human curvularia infections. Report of five cases and review of the literature. *Diagnostic Microbiology and Infectious Diseases*. **6**: 27-39.

Rohwedder, J.J., Simmons, J.L., Colfer, H. and Gatmaitan, B. (1979). Disseminated *Curvularia lunata* infection in a football player. *Archives of Internal Medicine*. **139**: 940-941.

Roldan, Y.B., Mata-Essayag, S. and Hartung, C. (2000). Erysipelas and tinea pedis. *Mycoses*. **43**: 181-183.

Romano, C., Papini, M., Ghilardi, A. and Gianni, C. (2005). Onychomycosis in children: a survey of 46 cases. *Mycoses*. **48**: 430 -437.

Ross, A.F. (1961). Systemic acquired resistance induced by localized virus infections in plants. *Virology*. 14: 340-358.

Rouby, Y., Combourieu, E., Perrier-Gros-Claude, J.D., Saccharin, C. and Huerre, M. (1998). A case of Aspergillus myocarditis associated with septic shock. *Journal of Infection*. **37**: 295-297.

Rubio-Calvo, C., Gil-Tomas, J., Rezusta-Lopez, A. and Benito-Ruesca, R. (2001). The aetiological agents of tinea capitis in Zaragoza (Spain). *Mycoses*. **44**: 55-58.

Sadana, J.R. and Kalra, D.S. (1973). A note on pulmonary phycomycosis in a pig. *Indian Journal of Animal Science*. **43**: 961-964.

Sahai, S. and Mishra, D. (2011). Change in spectrum of dermatophytes isolated from superficial mycoses cases: First report from Central India. *Indian Journal of Dermatology Venereology Leprology*. **77**: 335-336.

Salkin, F. and Stone, W.B. (1974). Subcutaneous mycotic infection of a white tailed deer. *Journal of Wildlife Diseases*. **10**: 34-38.

Sanabria Gomez, F., Cenjor Espanol, C., Marquez Dorsch, F.J. and Sarasa Corral, J.L.A. (1992). Severe rhinological mycosis: mucormycosis. A report of 3 cases. *ACTA Otorrinolaringologica Espanola*. **43**: 273-278.

Sarkar, S., Sinham, R.V. and Thakur, D.K. (1985). Epidemiology of dermatophytosis in domestic animals. *Indian Veterinary Journal*. **62**:107-112.

Saxena, S.P. (1972). Incidence of dermato mycoses in dogs and cats. *JNKVV Research Journal*. **6**: 106-108.

Saxena, S.P. and Mehra, K.N. (1973). A note on the occurrence of dermatophyte infection in domestic animals. *Indian Journal of Animal Sciences*. **43**: 347-350.

Schell, W.A. (2000). Unusual fungal pathogens in fungal rhinosinusitis. *Otolaryngologic Clinics of North America*. **33**: 367-373.

Schroeder, Jr., W.A., Yingling, D.G., Horn, P.C.

and Stahr, W.D. (2002). Frontal sinus destruction from allergic eosinophilic fungal rhinosinusitis. *Molecular Medicine*. **99**: 197-199.

Scott, D.W. (1988). Large animal dermatology. Philadelphia: WB Saunders; 487 pp. 64. Lefe`vre, P.C., Blancou, J., Chermette, R. eds. Dermatophilose. In: Principales maladies infectieuses et parasitaires du be´tail, Tome 2. Paris: Lavoisier; 2003. p. 977–92.

Seddon, M.E. and Thomas, M.G. (1997). Invasive disease due to *Epidermophyton floccosum* in an immunocompromised patient with Behcet's Syndrome. *Clinical Infectious Diseases*. **25**: 153-154.

Seebacher, C., Bouchara, J.P. and Mignon, B. (2008). Updates on the epidemiology of dermatophyte infections. *Mycopathologia*. **166**: 335-352.

Sellier, P., Monsuez, J.J., Lacroix, C., Feray, C., Evans, J., Minozzi, C., Vayre, F., Del Giudice, P., Feuilhade, M., Pinel, C., Vittecoq, D. and Passeron, J. (2000). Recurrent subcutaneous infection due to *Scopulariopsis brevicaulis* in a liver transplant recipient. *Clinical Infectious Diseases*. **30**: 820-823.

Shah, R.L., Mall, M.P. and Mohanty, G.C. (1982). Cutaneous candidiasis in Japanese quail (*Coturnix coturnix japonica*). *Mycopathologia*. **80**: 33-37.

Sharkey-Mathis, P.K., Kauffman, C.A. Graybill, J.R. Stevens, D.A., Hostetler, T.S., Cloud, G. and Dismukes, W.E. (1993). Treatment of sporotrichosis with itraconazole. *American Journal of Medicine*. **95**: 279-285.

Sharma, V.D., Saxena, O.P. and Singh, S. (1979). Prevalence of dermatomycoses in man and animals in the vicinity of Pantnagar. *Indian Journal of Animal Research*. **13**: 61-62.

Shelton, G.H., Grant, C.K., Linenberger, M.L. and Albokovitz, J.L. (1990). Severe neutropenia associated with griseofulvin in cats. *Journal of Veterinary Internal Medicine*. **4**: 317-319.

Shirani, D., Selk Ghaffari, M., Khosravi, A.R., Vajhi, A.R., Khorami, N., Zahraei Salehi, T., Shokri, H. and Abarkar, M. (2008). An unusual case of nasal mucormycosis caused by *Rhizopus oryzae* in a German shepherd dog. *Iranian Journal of Veterinary Research*, Shiraz University. **9**: 378.

Sikdar, A. and Uppal, P.K. (1985). Allergic test in the diagnosis of epizootic lymphangitis. XIX convention. Indian College of Allergy and Applied Immunology, Calcutta.

Sikdar, A., Singh, G., Banerjee, M.C. and Sharma, R.N. (1972). Isolation of *Candida pseudotropicalis* from cases of abortion among mares: A note. *Indian Journal of Animal Science*. **42**: 737-738.

Singal, A., Rawat, S., Bhattacharya, S.N., Mohanty, S. and Baruah, M.C. (2001). Clinico-mycological profile of tinea capitis in North India and response to griseofulvin. *Journal of Dermatology*. **28**: 22-26.

Singh, M.P. and Singh, C.M. (1970). Aspergilli associated with respiratory tract of poultry. *Indian Journal of Animal Sciences*. **44**: 218-219.

Singh, M.P. and Singh, C.M. (1970). Fungi associated with ringworm in dogs. *Indian Journal of Animal Health*. **9**: 109-110.

Singh, M.P. and Singh, C.M. (1970). Fungi associated with superficial mycoses of cattle and sheep in India. *Indian Journal of Animal Health*. **9**: 75-77.

Singh, M.P. and Singh, V.M. (1968). Mycotic dermatitis in camels. Paper presented in National Seminar in Zoonosis in India. National Institute of Communicable Disease, Delhi.

Singh, M.P. and Singh. C.M. (1972). *Trichophyton simii* infection in poultry. *Veterinary Record*. **90**: 218.

Singh, P.N., Ranjana, K., Singh, Y.I., Singh, K.P., Sharma, S.S., Kulachandra, M., Nabakumar, Y., Chakrabarti, A., Padhye, A.A., Kaufman, L. and Ajello, L. (1999). Indigenous disseminated *Penicillium marneffei* infection in the state of Manipur, India: Report of four

autochthonous cases. *Journal of Clinical Microbiology*. **37**: 2699-2702.

Singh, R.P. (1982). Equine ringworm a case study. *Journal of Remount Veterinary Corps*. **20**: 91-93.

Singh, S.D., Thakur, D.K., Sudhan, N.A. and Verma, B.B. (1992). Incidence of mycotic mastitis in cows and buffaloes. *Indian Veterinary Journal*. **69**: 86-87.

Singh, S.M., Naidu, J. and Pouranik, M. (1990). Ungual and cutaneous phaeohyphomycosis caused by *Alternaria alternata* and *Alternaria chlamydospora*. *Medical Mycology*. **28**: 275-278.

Sitara, U., Niaz, I., Naseem, J. and Sultana, N. (2008). Antifungal effect of essential oils on *in vitro* growth of pathogenic fungi. *Pakistan Journal of Botany*. **40**: 409-414.

Sorensen, J., Becker, M., Porto, L., Lambrecht, E., Schuster, T., Beske, F., Rickerts, V., Klingebiel, T. and Lehrnbecher, T. (2006). Rhinocerebral zygomycosis in a young girl undergoing allogeneic stem cell transplantation for severe aplastic anaemia. *Mycoses*. **49**: 31-36.

Speare, R. and Thomas, A.D. (1985). Kangaroos and wallabies as carriers of *Basidiobolus haptosporus*. *Australian Veterinary Journal*. **62**: 209-210.

Stenwig, H. (1985). Isolated dermatophytes from domestic animals in Norway. *Nordisk Veterinaer Medicine*. **37**: 161-169.

Stevens, D.A. (1999). Drug interactions *in vitro* between a polyene (Ambisome) and an echinocandin (FK463) vs. *Aspergillus species*. 39th Interscience Conference on Antimicrobial Agents and Chemotherapy, Abstract No. 151.

St-Germain, G. and Summerbell, R. (1996). Identifying Filamentous Fungi - A Clinical Laboratory Handbook, 1st ed. Star Publishing Company, Belmont, California.

Stratton, C.W., Lichtenstein, K. A., Lowenstein, S.R., Phelps, D.B. and Reller, L.B. (1981). Granulomatous tenosynovitis and carpal tunnel syndrome caused by *Sporothrix schenckii*. *American Journal of Medicine*. **71**: 161-164.

Sugar, A.M. (1995). Agent of mucormycosis and related species. In: Mandell, G, Bennett J, Dolin R. (Eds.) Principles and practices of infectious diseases. 4th Ed. New York: Churchill Livingstone. pp. 2311-2321.

Sujatha, S., Sheeladevi, C., Khyriem, A.B., Parija, S.C. and Thappa, D.M. (2003). Subcutaneous zygomycosis caused by *Basidiobolus ranarum* -a case report. *Indian Journal of Medical Microbiology*. **21**: 205-206.

Supparatpinyuo, K., Chiewchanvit, S., Hirunsri, P., Uthammachai, C., Nelson, K.E. and Sirisanthana, T. (1992). *Penicillium marneffei* infection in patients infected with human immunodeficiency virus. *Clinical Infectious Diseases*. **14**: 871-874.

Sutton, D.A., Fothergill, A.W. and Rinaldi, M.G. (eds.). (1998). Guide to clinically significant fungi, 1st ed. Williams & Wilkins, Baltimore.

Takatoria, K., Ichijo, S., Konishi, T. and Tanaka, I. (1981). Occurrence of equine dermatophytosis in Hokkaido. *Journal of Veterinary Science*. **43**: 307-313.

Terreni, A.A., Gregg, W.B., Jr, Morris, P.R. and Di Salvo, A.F. (1985). *Epidermophyton floccosum* infection in a dog from the United States. *Sabouraudia*. **23**: 141-142.

Tewari, R.P. (1962). Studies on some aspects of mycoses. MVSc thesis submitted to Agra University.

Tewari, R.P. (1969). *Trichophyton simii* infections in chickens, dogs and man in India. *Mycopath. Mycol. Appl.* **39**: 233-238.

Thakur, D.K., Misra, S.K. and Singh, K.B. (1983). A preliminary study on dermatomycosis in water buffalo in Pubjab. *Indian Journal of Veterinary Medicine*. **3**: 57-60.

Thakur, S.K. and Verma, B.B. (1984). Dermatomycosis due to *M. gypseum* in goats. *Indian Veterinary Journal*. **61**: 1083.

Thami, G.P., Kaur, S., Bawa, A.S., Chander, J.,

Mohan, H., Bedi, M.S. (2003). Post-surgical zygomycotic necrotizing subcutaneous infection caused by *Absidia corymbifera*. *Clinical and Experimental Dermatology*. **28**: 251-253.

Thomas, P.A., Abraham, D.J. and Kalawathy, C.M. (1988). Oral itraconizole therapy for mycotic keratitis. *Mykosen*. **31**: 271-279.

Tomimori-Yamashita, J., Takahashi, C.H., Fichman, O., Costa, E.B., Michalany, N.S. and Alchorne, M.M.A. (1998). Lymphangitic sporotrichosis: An uncommon bilateral localization. *Mycopathologia*. **141**: 69-71.

Tosti, A., Piraccini, B.M., Stinchi, C. and Lorenzi, S. (1996). Onychomycosis due to *Scopulariopsis brevicaulis*: clinical features and response to systemic antifungals. *British Journal of Dermatology*. **135**: 799 -802.

Tuteja, F.C., Ghorui, S.K. and Narnaware, S.D. (2010). Cutaneous alternariosis in dromedary camel. Journal of Camel Practice and Research. 17: 225-228.

Tuteja, F.C., Nath, K. Chirania, B.L., Patil, N.V. and Sena, D.S. (2012). Skin candidiasis (Thikria) in dromedary calves. Journal of Camel Practice and Research. 19: 243-247.

Tuteja, F.C., Pathak, K.M.L., Ghorui, S.K., Chirania, B.L. and Kumar, S. (2010). Skin candidiasis in dromedary camel calves. Journal of Camel Practice and Research. 17: 59-61.

Tuteja, F.C., Dixit, S.K., Kumar, S., Patil, N.V. and Singh, J.P. (2011). Traditional treatment practices against camel diseases in Rajasthan. Journal of Camel Practice and Research. 18: 231-242.

Tuteja, F.C., Patil, N.V., Narnaware, S.D., Nagarajan, G. and Dahiya, S.S. Primarily human pathogenic fungi causing dermatophytosis in camel. Journal of Camel Practice and Research. Communicated.

Tuteja, F.C., Patil, N.V., Dahiya, S.S., Narnaware, S.D. and Nagarajan, G. Dimorphic fungi isolated from camel dermal mycoses. Journal of Camel Practice and Research. Communicated.

Tuteja, F.C., Patil, N.V. Nagarajan, G., Dahiya, S.S. and Narnaware, S.D. Camel dermal mycoses caused by dermatophytes. Journal of Camel Practice and Research. Communicated.

Tuteja, F.C., Patil, N.V., Nagarajan, G., Narnaware, S.D. and Dahiya, S.S. Filamentous fungi isolated from camel dermal mycoses. Journal of Camel Practice and Research. Communicated.

Ujhelyi, M.R., Raasch, R.H., van der Horst, C.M. and Mattern, W.D. (1990). Treatment of peritonitis due to curvularia and trichosporon with amphotericin B. *Review Infectious Diseases*. **12**: 621-627.

Van Burik, J.A., Colven, R., Spach, D.H. (1998). Cutaneous aspergillosis. *Journal of Clinical Microbiology*. **36**: 3115-3121.

Van Cauteren, H., Vanparys, P., de Meester, C., Lambotte-Vandepaer, M., Vandenberghe, J. and Marsboom, R. (1987). Mutagenic and leukemogenic activity of haloperidol: A negative study. *Drug Chemical Toxicology*. **10**: 311-327.

Van Cutsem, J. (1983). The antifungal activity of ketoconazole. *American Journal of Medicine*. **74**: 9-15.

Vartivarian, S.E., Anaissie, E.J. and Bodey, G.P. (1993). Emerging fungal pathogens in immunocompromised patients: classification, diagnosis and management. *Clinical Infectious Diseases*. **17**: S487-491.

Vennewald, I., Henker, M., Klemm, E. and Seebacher, C. (1999). Fungal colonization of the paranasal sinuses. *Mycoses*. **42**: 33-36.

Verma, R. (1988). Studies on clinical and subclinical mastitis. *Indian Journal of Comparative Microbiology, Immunology and Infectious Diseases*. **9**: 28-33.

Verma, R., Arora, B.M. and lyer, P.K.R. (1983). Cutaneous mycosis in a lion cub (*Panther leo*) caused by *Candida tropicalis*. *Indian Journal of Veterinary Pathology*. **7**: 63-65.

Vitovec, J., Vladik, P. and Fragner, P. (1976). Visceral mucormycosis in cattle and pig.

Veterinarstvi. **26**: 459-461.

Wadhwani, K. and Srivastava, A.K. (1984). Fungi from otitis media of agricultural field workers. *Mycopathologia*. **88**: 155-159.

Wang, J.P., Granlund, K.F., Bozzette, S.A., Botte, M.J. and Fierer, J. (2000). Bursal sporotrichosis: Case report and review. *Clinical Infectious Diseases*. **31**: 615-616.

Ware, A.J., Cockerell, C.J., Skiest, D.J. and Kussman, H.M. (1999). Disseminated sporotrichosis with extensive cutaneous involvement in a patient with AIDS. *Journal of the American Academy of Dermatology*. **40**: 350-355.

Weiss, R., Bohm, K.H., Mumme, J. and Nicklas, W. (1979). Dermatophytennachweise in den Jahren 1965 bis1977. *Sabouraudia*. **17**: 345-353.

Weitzman, I. and Summerbell, R.C. (1995). The dermatophytes. *Clinical Microbiology Reviews*. **8**: 240-259.

Wernery, U., Kinne, J. and Nagy, P. (2007). *Candida dermatitis* in camel calves- A case report. Proceedings of the International Camel Conference. Recent trends in camelids research and future strategies for saving camels, Bikaner, Feb. 16-17.P.226.

Wildfeuer, A., Seidl, H.P., Paule, I. and Haberreiter, A. (1998). *In vitro* evaluation of voriconazole against clinical isolates of yeasts, moulds and dermatophytes in comparison with itraconazole, ketoconazole, amphotericin B and griseofulvin. *Mycoses*. **41**: 309-319.

Wilson, R.T. (1984). In: The Camel, Longman Group limited, Essex, U.K.

Witherspoon, C.D., Kuhn, F., Owens, S.D., White, M.F. and Kimble, J.A. (1990). Endophthalmitis due to *Sporothrix schenckii* after penetrating ocular injury. *Annals of Ophthalmology*. **22**: 385-388.

Woloszyn, S. (1987). Fungicidal activity of iodophores in the arthrospores of dermatophytes. *Polskie Archiwum Weterynaryjne*. **27**: 25-33.

Wong, S.S.Y., Wong, K.H., Hui, W.T., Lee, S.S., Lo, J.Y.C., Cao, L. and Yuen, K.Y. (2001). Differences in clinical and laboratory diagnostic characteristics of penicilliosis marneffei in human immunodeficiency virus (HIV) - and non-HIV-infected patients. *Journal of Clinical Microbiology*. **39**: 4535-4540.

Yagil, R. (1985). In: The Desert Camel. Verlag Karger, Basel.

Yamada, C., Hasegawa, A., Ono, K., Pal, M., Kitamura, C. and Takahashi, H. (1991). *Trichophyton rubrum* infection in a dog. *Japaness Journal of Medical Mycology*. **32**: 67-71.

Zaias, N., Glick, B. and Rebell, G. (1996). Diagnosing and treating onychomycosis (see comments). *Journal of Family Practice*. **42**: 513-518.

Zavasky, D.M., Samowitz, W., Loftus, T., Segal, H. and Carroll, K. (1999). Gastrointestinal zygomycotic infection caused by *Basidiobolus ranarum*: Case report and review. *Clinical Infectious Diseases*. **28**: 1244-1248.

Ziemer, E.L., Pappagianis, D., Madigan, J.E., Mansmann, R.A. and Hoffman, K.D. (1992). Coccidioidomycosis in horses: 15 cases (1975-1984). *Journal of the American Veterinary Medical Association*. **201**: 910- 916.

Zimmermann, C.R., Johnson, S.M., Martens, G.W., White, A.G., Zimmer, B.L. and Pappagianis, D. (1998). Protection against lethal murine coccidioidomycosis by a soluble vaccine from spherules. *Infection and Immunity*. **66**: 2342-2345.

Chatterjee, A. and Sengupta, D.N. (1979). Ringworm in domestic animals. *Indian Journal of Animal Health*. **18**: 38 - 46.