Chapter 6
Mycotic Infections in Bovines: Recent Trends and Insights on Pathogenicity After Post-Industrial Temperature Rise

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6.1 Introduction

According to the Intergovernmental Panel on Climate Change (IPCC 2007), climate change can be defined as a variation in climate over a period of time, whether in average weather or extreme events. Increasing temperatures, decreasing rainfall reliability, and increasing frequency and severity of extreme climatic events are the main outcome of variation in climate change. Climate change after the post-industrialization period has altered the molecular biology of pathogens, vectors (if any), farming practices and land use, and environmental and zoological factors. It has established new microenvironments and microclimates and influenced the occurrence, distribution, and prevalence of livestock diseases under the changing ecological conditions. The implication is the drier subtropical regions warming more than the moister tropics (Alcamo et al. 2007; Speranzaa 2010). Climate variability, by affecting the environmental conditions, has the consequence of impacting pasture growth and quality, availability of water resources, and, thus, the distribution of livestock diseases (Gale et al. 2008). Plant, animal, and human epidemics are influenced climatically (Bosch et al. 2007; Thomson et al. 2006; Wint et al. 2002).

Fungi are an organism that have the capacity to spread in many different habitats around the world and can affect living and non-living creatures. The fungi, which are thought to number around 140,000 on Earth, play an important role in the global ecosystem. Fungi help the ecosystem by promoting nutrient conversion, the formation of soil, and decomposition of organic matter (Bal et al. 2017). They live as symbiotic, parasitic (mostly plant pathogens), and saprotrophic (Kauserud et al. 2005; Durugbo et al. 2013; Sevindik et al. 2017), in which

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parasitic fungi are the second largest group, of whose members do a lot of serious damage. Their (fungi) spores can spread through meteorological activities and can cause harm to living and non-living organisms in the areas in which they spread (Garcia et al. 2012).

Veterinary mycology refers to the branch dealing with the detection and identification of veterinary fungi, pathophysiology and diagnosis of fungal infections, the pharmacodynamics of antifungals in animals, epidemiology of animal mycoses, eradication, and promotion of vaccine development against invasive veterinary fungal infections. It is different from human mycology in mainly two different aspects. First, the phenomenon of pathological or iatrogenic immunosuppression has affected an ever-increasing number of people after 1980, which has led to the emergence of fungal infections mainly caused by opportunistic fungi previously not or only rarely involved in human mycoses (Enoch et al. 2017). These new phenomena promoted the discovery of multiple ranges of antimycotic drugs, many of which are prohibitively expensive for veterinary use. This phenomenon has not been seen in animals since the gamut of fungi associated with veterinary mycoses has remained largely the same, with differences primarily from factors such as improved diagnostic methods and raised awareness (Elad 2011). The second aspect that separates veterinary from human mycology is the cost-effectiveness. The price and cost associated with diagnosis, identification, etiology, and cure are major hindrances in the eradication of fungal infections in animals, especially cattle.

As bovines are warm-blooded animals, "A major factor protecting warm-blooded animals from fungal disease is the difference in temperature between the body temperature and the outside environment. As fungi adapt to living at higher temperatures after post-industrial temperature rise, animals may begin to lose some of that natural protection" (Casadevall 2010). For a heat-tolerant fungus, the animal body might become pastureland. Casadevall and colleagues have demonstrated that some fungi have already started to adapt to warmer temperatures. When they analyzed temperature susceptibility data for several strains of yeast (a type of fungus) gathered since 1985, they found an increase in the temperature tolerance in the past 20 years in some fungal strains. The findings, say the researchers, are a "clear warning that global warming may cause fungi to become significantly more thermally resistant," though it is not yet possible to predict which species may become new threats.

Dufour et al. (2008) conducted an experiment to study the impact of climatic changes on the vector, host reservoir, characteristics, and epidemiology of the parasite. This study was an attempt to analyze susceptibility to the emergence and development of contagious disease in France as a result of global warming and found that climate change affects the incidence of livestock diseases transmitted by direct contact due to changes in the frequency and duration of animal contacts. Changes in the degree of mixing of cattle will affect the prevalence of some diseases (Fig. 6.1).

Fig. 6.1 The proportion of plant and animal disease alerts recorded by the Program for Monitoring Emerging Diseases attributable to fungi has risen in recent years. (Diagram courtesy of Nature Publishing Group)

6.2 Mycotic Diseases in Cattle

6.2.1 Mycotic Abortion

Bovine mycotic abortions (BMAs) take place mostly in the third trimester of pregnancy and are mostly sporadic. They are the major cause of mycotic abortions in some countries (Hugh-Jones and Austwick 1967). Bovines are most sensitive for mycotic abortion in comparison with ovines, camels, and caprines (Dehkordi et al. 2012).

6.2.1.1 Pathogen

One of the most common causes of mycotic reproductive failure, abortions due to *Aspergillus fumigatus*, has a tendency to occur in the second and third trimesters (McCausland et al. 1987). It is a storage fungus and grows well in conditions of relatively high moisture and temperature. Temperature rise, as well as extreme rain in some places, may promote the fungal growth, as this fungus proliferates in decomposing hay, badly preserved silage and soil, and produces a non-airborne, pathogenic spore (Cordes et al. 1964). The spores confine to the uterine caruncle and induce inflammation-induced abortion after 2–5 weeks of proliferation. The placenta often has swollen, necrotic cotyledons and the intercotyledonary membrane may be diffusely thickened, wrinkled, and leather-like. Occasionally, aborted fetuses have characteristic fungal plaques, 1–10 cm in diameter involving the skin around the eyelids, neck, dorsum, and thorax (Cordes and Shortridge 1968). This is due to fungal proliferation in the amnion, penetration of the epidermis, and the fetal inflammatory reaction, as well as hyperkeratosis. Mycotic abortion can be diagnosed by culture, and histologically from placental changes, with visible fungal hyphae confirming infection (Hill et al. 1971).

Mortierella wolfii is sporadically reported in Australian cattle and often reported in the North Island of New Zealand (Gabor 2003). The appearance of *M. wolfii* infection in pregnant cows is the same as *A. fumigatus*, except that abortion is followed by fatal pneumonia 4–5 days later in about 20% of cases (Cordes et al. 1972). Inoculation of spores occurs through inhalation from contaminated silage, and pass into arterial circulation via the pulmonary vascular bed (Curtis et al. 2017). Pathogenesis is also the same as *A. fumigatus* and, after hematogenous spread, fungal growth at the uterine caruncle causes widespread tissue necrosis and inflammation, leading to placentitis and abortion. In a nutshell, the placenta appears thickened, edematous, and necrotic, and other lesions appear consistent with mycotic abortion described above.

6.2.1.2 Symptoms

Symptoms comprise modifications in the placenta, which becomes thick and tough, may have puffy cotyledons and necrotic spots, with raised, hyperkeratotic plaques (Glover et al. 2011) on the fetus' skin, although the frequency of this symptom is doubtful. A tentative clinical diagnosis may be made on the appearance of the placenta and particularly the cotyledons, and also on the presence of skin lesions on the fetus.

6.2.1.3 Pathogenesis

The pathogenesis of mycotic abortions has significant implication for the analysis of the diagnostic results. Examining the fetus alone does not reveal the abortive agent in cattle, as the contact between the fetal and the maternal part of the cattle placenta is not contiguous but take places in contact organs called placentomes, composed of the fetal part (the cotyledon) and the maternal part (the caruncle), and, also, there is no direct contact between the fetal and maternal blood vessels. Thus, infectious agents that spread hematogenously do not do so by the maternal to fetal blood but through the placentome and cause infection. If the pathological process is too severe to lead to an early abortion, as happens in about 70% of cases, the agent will not diagnose in the fetal organs, but if the abortion is delayed, the microorganism will spread through the amniotic fluid first to the fetus' stomach (abomasum), then to the lungs, and, finally, to the other organs (Sheridan et al. 1985).

Laboratory diagnosis is based on the identification of the fungus by culturing the sample from the placenta and/or the fetus. Since the culture of *Aspergillus* spp. may be the result of contamination and can contradict the result, it is vital to assess tissue invasion by histopathology (Jensen et al. 1991). Contaminant overgrowth is another hindrance in the mycological identification of the fungus causing the abortion in a comparison between three diagnostic methods. Better conformity was found between immunofluorescence and histopathological or mycological identifi-

cation (kappa D 0.4 and 0.48, respectively). Jensen et al. (1993) also assessed the presence of galactomannan in the sera and urine of calves infected artificially and intravenously with *A. fumigatus* in cows with mycotic placentitis and abortion (confirmed by histopathology and culture), cows that aborted for other reasons, cows that did not abort but had other infections, and healthy cows at the slaughterhouse by inhibition enzyme-linked immunosorbent assay (ELISA). The results showed that the test was not specific enough to be considered a dependable means to diagnose mycotic abortions in cattle. Additionally, the clinical and microbiological diagnosis and possibilities of serological diagnosis of mycotic abortions in cattle was examined, but there is no considerable result in the diagnostic approaches (Corbel et al. 1973; Wiseman et al. 1984; Jensen et al. 1991, 1993)

6.3 Cryptococcosis

6.3.1 Disease Name

Cryptococcosis.

6.3.2 Syn. (synonym)

Torulosis, European blastomycosis, Busse–Buschke disease.

6.3.3 Pathogen

Cryptococcus neoformans.

6.3.4 Symptoms

Cryptococcosis is a chronic and often fatal infection in most of the cases, with no clear clinical pattern. In small animals, the central nervous system is often infected. In cattle, there may be mastitis, pneumonia, encephalitis, and cryptococcal meningitis. On the other hand, cattle are likely to develop cryptococcal mastitis as a result of ascending infection of the mammary gland via the teat canal. Affected cows have anorexia, reduced milk flow, swelling and hardness of affected quarters, and enlarged supramammary lymph nodes. The milk becomes viscid, mucoid, and gray-white, or it may be watery with flakes. The clinical signs included multifocal

neurological deficits manifested by hypermetria, ataxia, depression, circling, impaired vision, head pressing, low head carriage, wide-based stance, and falling to the side or backwards. Diagnosis is made by demonstrating capsulated yeast cells in tissues, spinal fluid, pus, or other exudate and confirmed by culture of the pathogen. No reliable treatment is known.

6.3.5 Cryptococcosis in Bovines

Cryptococcus infections were mostly reported in association with mastitis. Klein (1901) was the first to isolate a yeast from a case of mastitis and reported it to be identical to strains of *C. neoformans* of human and plant origin. Almost 50 years later, *C. neoformans* was diagnosed as the causative agent of severe outbreaks of mastitis in cattle (Carter and Young 1950; Stuart 1951; Emmons 1952; Hulse 1952). Pounden et al. (1952) studied the clinical aspects of an outbreak, in which they found 106 cows affected in a herd of 235 cows. They observed that *Cryptococcus* has been isolated from samples without any visible change either in the gland or milk, and the cases with visible signs varied from mild and brief swelling of one or more quarters of the udder to severe swelling of the affected glands. Cryptococcal mastitis was also detected in sporadic cases by El-Ghany et al. (1978), Rahman et al. (1983), and Moawad (1991). On the other hand, Rippon (1982) emphasized the worldwide presence of cryptococcal mastitis in dairy cows. Among the cryptococci, *C. neoformans* is a common pathogen, but other cryptococcal species express putative virulence factors, such as polysaccharide capsule and melanization (Petter et al. 2001). As the temperature is increasing day by day, acquisition of thermotolerance by other species in the post-industrial temperature rise era may increase their potential to become pathogenic. One such case can be attributed to *C. laurentii*, which normally does not grow at 37 °C, but thermotolerant strains have increasingly been associated with disease in extremely immunosuppressed hosts (Khawcharoenporn et al. 2007) and it may also affect the prevalence of cryptococcosis in bovines.

6.3.6 Pathogen

In cows, *C. neoformans* was the most commonly recorded species (El-Far et al. 1987; Saleh 2005; Abou-Elmagd et al. 2011). *Cryptococcus neoformans* as a causative agent for mastitis in buffaloes was found by Jand and Dhillon (1975) and Pal and Mehrotra (1983). Other species, such as *C. laurentii*, *C. flavus*, *C. lactativorus*, *C. albidus*, *C. luteolus*, *C. uniguttulatus*, and *C. terreus*, were also reported in several studies (El-Far et al. 1987; Costa et al. 1993; Klimaite et al. 2003; Türkyılmaz and Kaynarca 2010; Wawron et al. 2010; Fadda et al. 2013; Zhou et al. 2013; Akodouch et al. 2014).

Table 6.1 Cryptococcosis in bovines (Refai et al. 2017)

Species	References
Cryptococcal mastitis	
C. neoformans	Carter and Young (1950); Stuart (1951); Emmons (1952); Hulse (1952); Pounden et al. (1952); Innes et al. (1952); Simon et al. (1953); Redaelli (1957); Menhnert et al. (1964); Galli (1965); Monga and Kalra (1971); Jand and Dhillon (1975); Sipka and Petrović (1975); El-Ghany et al. (1978); Rippon (1982); Rahman et al. (1983); Pal and Mehrotra (1983); El-Far et al. (1987); Kotb (1990); Moawad (1991); Pal (1991); Pengov (2002); Moshref (2004); Saleh (2005); Asfour et al. (2009); Türkyılmaz and Kaynarca (2010); Abou-Elmagd et al. (2011); Saleh et al. (2011); Sharma et al. (2012); Hassan et al. (2013); Al-Ameed (2013)
C. albidus	El-Far et al. (1987); Costa et al. (1993)
C. laurentii	Wawron et al. (2010); Costa et al. (1993); Klimaite et al. (2003); Türkyılmaz and Kaynarca (2010); Zhou et al. (2013)
C. flavus	Costa et al. (1993)
C. luteolus	Costa et al. (1993)
C. uniguttulatus	Zhou et al. (2013)
C. lactativorus	Al-Khalidi et al. (2012)
C. terreus	Akodouch et al. (2014)
Cryptococcus species	Jand and Dhillon (1975); Kirk and Bartlett (1986); Costa et al. (1993)
Cryptococcal pneumonia	
C. neoformans	Hassan et al. (2013); Akodouch et al. (2014)
C. laurentii	Pal (2007)
Systemic cryptococcosis	
C. neoformans	Riet-Correa et al. (2011)
Cryptococcus species	Akange et al. (2013)

Cryptococcal pneumonia was occasionally reported in bovines (Pal 2007; Hassan et al. 2013). Bovine systemic cryptococcosis was also rarely diagnosed. Only two reports indicate its presence (Riet-Correa et al. 2011; Akange et al. 2013). Similar findings were observed in the case of abortion due to *Cryptococcus* species (Riet-Correa et al. 2011; Akange et al. 2013) (Table 6.1).

6.3.7 Cattle

Cryptococcus neoformans was first isolated from peach juice in 1894 in San Felice in Italy. Madsen (1942) isolated a *Cryptococcus* sp. from a cow in New York. The most impressive outbreak of bovine cryptococcosis during recent years, however,

was of cryptococcal mastitis described in detail by Pounden et al. (1952), Innes et al. (1952), and Emmons (1952), in which, over the course of 12 months, 106 of 235 cows of a Holstein Friesian herd in Maryland became infected. Fifty-four of the infected animals developed a visible abnormality of the udder or milk and, in severe cases, the udder was greatly enlarged. A similar outbreak involving 50 of a herd of 280 cows was recorded by Simon et al. (1953). Hammer and Engler (1956) recorded *C. neoformans* as the cause of ulcerative endocarditis in a bull.

6.4 Coccidioidomycosis

6.4.1 Syn

Coccidioidal granuloma, oidiomycosis.

6.4.2 Pathogen

Coccidioides immitis. (A partial list of synonyms is given by Conant et al. 1954).

It has been more than a century since coccidioidomycosis was first recognized as a serious disease, and its etiology and epidemiology have been well documented. However, the disease remains an enigma to many, and it often goes undiagnosed, even in endemic areas. As management of this chronic disease remains problematic, new preventive or therapeutic options are needed.

6.4.3 Etiology and Epidemiology

Coccidioidomycosis is a fungal disease found only in the Western Hemisphere. It is caused by two nearly identical species, *C. immitis* and *C. posadasii*, generically referred to as the "Californian" and "non-Californian" species**,** respectively (Fisher et al. 2002). The fungus grows in a mycelial phase in the soil within a geographically delineated area of the United States known as the Lower Sonoran Life Zone(Maddy 1957). This semi-arid zone encompasses the southern parts of Texas, Arizona, New Mexico, and much of central and southern California. *Coccidioides immitis* is sensitive to climate variability and responds to changes in temperature and precipitation. Previous studies have found a relationship between climatic conditions like temperature, precipitation, humidity, wind, and occurrence of dust storms with growth of *C. immitis* or the distribution of the arthrospores.

Mycelial phase—the growth form in the soil, composed of filamentous hyphae and reproductive spores called arthroconidia.
Arthroconidia—reproductive spores, highly resistant to desication, which are the infectious particles inhaled by man and animals.
Spherules—the parasitic phase of this dimorphic fungus; spherules are round cells of 30–100 μM or more than reproducing the progeny endospores.
Endospores—the progeny units of the parasitic phase, derived from spherules.

6.4.4 Cattle

Infection in cattle was first recorded by Giltner (1918) in a cow from the San Joaquin Valley, California and, since then, more than 3000 cases have been reported in cattle from the same state (Maddy 1954). In cattle, the infection is usually benign. Typically, there is a granulomatous involvement of the bronchial and mediastinal lymph nodes. More rarely, the lungs are attacked (Davis et al. 1937).

6.5 Rhinosporidiosis

6.5.1 Pathogen

The disease is caused by *Rhinosporidium seeberi*, an organism that was earlier known as a fungus but has been regrouped into the class Mesomycetozoa (family Rhinosporideacae). This class consists of several parasitic and saprophytic organisms, most of which infect fish and amphibians; only *R. seeberi* infects mammals (Adl et al. 2005).

Rhinosporidiosis is a non-infectious, sporadic, benign, generally non-fatal, and chronic granulomatous disease of humans and animals (Pal 2007). It is characterized by the development of nasal polyps. Diagnosis is confirmed by finding many large sporangia in the diseased tissues. The standard treatment is surgical excision of the growths.

6.5.2 Geographical Distribution

It is endemic in India (Rao and Narayan 1938) and sporadic in South Africa (Zschokke 1913; Quinlan and De Kock 1926), Uruguay (Saunders 1948), Argentina (Prieto and Pires 1944), the USA (Robinson 1951), and Australia (Albiston and Gorrie 1935).

6.5.3 Symptomology

In animals, the site attacked is the mucous membrane of the nose and the infection is unilateral. According to Rao and Narayan (1938), in cattle and horses, the tumors are rarely larger than 2–3 cm in diameter and may be either pedunculated or sessile. They are lobulated, soft, and pink in color, bleed readily, and are dotted with small white specks (the sporangia). In man, the nasal polyps are similar but often larger, and other sites, including the pharynx, larynx, eye, ear, skin, vagina, and rectum, may be infected.

6.6 Mucormycosis

Species of *Mucor*, *Absidia*, and *Rhizopus*.

6.6.1 Disease Name

Mucormycosis, including mycotic ulceration.

6.6.2 Pathogen

Absidia corymbifera (Cohn) Sacco (Vink 1941). Syn. *A. lichtheimi* (Christiansen 1922). *Mucor javanicus* (Porges et al. 1935). *Mucor pusillus* (Tscherniak 1928). *Mucor racemosus* (Frank 1890). *Mucor rhizopodiformis* (Christiansen 1922).

Host: Cattle (Gleiser 1953).

6.6.3 Geographical Distribution: Europe and North America

Although the pathogenicity of mucoraceous fungi for experimental animals has been known since the work of Lichtheim (1884), spontaneous mucormycosis in animals has rarely been reported, with there being only some 40 contributions appearing in the literature. The early accounts are concerned with the disease in man (Paltauf 1885) and are of limited value owing to their lack of adequate descriptions, so it is impossible to be certain whether the fungi concerned were, in fact, mucoraceous at all. To some extent, the characteristics of the lesions described are a guide, but isolation of fungi was not often accomplished. When the species was identified, it invariably proved to be non-pathogenic, e.g., *Mucor racemosus* (Frank 1890). For

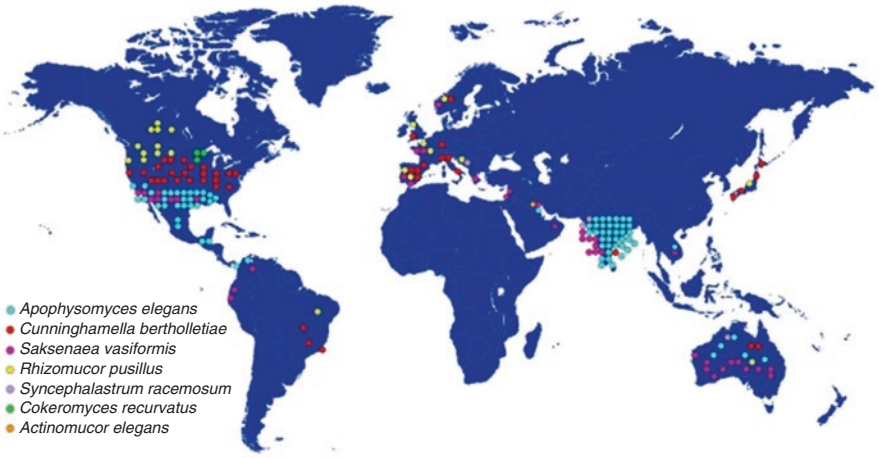

Fig. 6.2 Geographical distribution of reported cases of unusual mucormycosis (Gomes et al. 2011)

this reason, no reliable account of mucormycosis in animals has been located before that of Christiansen (1922), who described two cases in pigs. Subsequently, the disease has been defined in several domestic animals and differentiated from other granulomatous and ulcerative diseases. The diseases are now recognized as having regular occurrence in the Northern Hemisphere, whereas *R. pusillus* (formerly known as *Mucor pusillus*) is thermophilic and, thus, grows in high-temperature environments. To understand the impact of climate change on mucormycosis, comprehensive epidemiological surveillance, better methods of detection, and multidisciplinary collaboration will be required (Fig. 6.2).

6.6.4 Symptomatology

Mucormycosis is chiefly known as a granulomatous disease generally involving the lymph nodes. As it rarely produces any signs until either the granulomata become visible as protuberances or the animal wastes away, most of the reported cases in pigs and cattle have been discovered during a routine inspection in abattoirs.

6.6.5 Cattle

Gleiser (1953) first mentioned the occurrence of caseocalcareous lesions in the bronchial and mediastinal lymph nodes of a yearling heifer. Davis et al. (1955) also found similar lesions in 11 cattle during meat inspection. The most frequent lesions

were in the mesenteric lymph nodes and lungs and consisted of a greenish-yellow caseocalcareous mass with a variable amount of scar formation. None of the animals had shown any apparent symptoms of the disease. Abomasal ulceration has been the more frequently reported form of bovine mucormycosis and most likely accounted for a proportion of the early literature on peptic ulcers in animals. Sanchez Botija (1951) first found mucoraceous hyphae in a single ulcer 5–6 cm in diameter in an ox, whilst Gentles and O'sullivan (1957) summarized seven acute cases in 47 young calves, stressing that these represented every case of abomasal ulceration seen in 208 consecutive post-mortem examinations of calves. All except one of the calves died following the onset of severe scouring and one to several ulcers up to 4 cm in diameter were found on each occasion. The early lesions were small, raised, inflamed foci which developed into ulcers with raised hemorrhagic margins and gray, depressed, necrotic central areas.

6.7 The Ringworm Fungi

Dermatophytes infecting cattle.

6.7.1 Pathogen

Trichophyton asteroides: cattle (Davidson et al. 1934; Muende and Webb 1937; Gentles and O'sullivan 1957). *Microsporum* and *Epidermophyton* (Gudding and Lund 1995). *Trichophyton verrucosum*: cattle (Lebasque 1933; Blank 1955, El-Diasty et al. 2013).

6.7.2 Pathogenicity

Ringworm is a highly infectious skin disease of cattle all over the world. It affects the keratinized structures of the skin and hair of animals superficially. The causative agent of this disease is a faction of keratinophilic filamentous fungi called dermatophytes in the genera *Trichophyton*, *Microsporum*, and *Epidermophyton* (Gudding and Lund 1995). *Trichophyton verrucosum* is the most observed etiologic agent of cattle (Akbarmehr 2011; El-Diasty et al. 2013). Infection is caused mainly by contact with arthrospores (asexual spores formed in the parasitic stage) or conidia (sexual or asexual spores formed in the normal environmental stage). Transmission between hosts usually occurs by direct contact with a host (Murray et al. 2005). It was observed that housing animals in close proximity to the presence of infected debris was responsible for the high occurrence of the disease in winter (Al-Ani et al. 2002). However, it appears that the disease is more common in tropical than

temperate climates, and particularly in areas having tropical or subtropical climatic conditions (Radostits and Done 2007). Dermatophytosis is mainly observed in young calves, whose high skin pH and ill-developed immune system are more sensitive to the infection (Radostits et al. 1997).

6.7.3 Symptoms

The characteristic lesion symptoms include patches of hair loss (10–50 mm), desquamation, and crust formation usually confined to the head, dewlap, and sometimes other parts of the body surface. In a study by El-Diasty et al. (2013), they collected skin scrapings and hair samples from beef calves and observed clinical signs of dermatophytosis. The collected samples were examined for fungal elements by microscopy and fungal culture preparation. Fungal culture revealed *T. verrucosum*. A second study was conducted by El-Ashmawy et al. (2015) during September 2013 till April 2014 (autumn and winter months) in a farm containing 250 beef calves (local mixed breeds) aged 6–9 months. Clinical signs of skin lesions appeared in 30 animals after 2 weeks in the form of circumscribed areas of hair loss filled with puffy white scales all over the body. All examined samples were found to be positive for fungal elements (ectothrix spores and endothrix hyphae) by microscopic examination. The fungus grew slowly on SDA, producing white, cottony, and heaped colonies with some submerged growth and yellow pigmentation. Microscopic examination after staining with lactophenol cotton blue revealed septate hyphae with numerous clavate microconidia borne across the hyphae and many chlamydospores arranged in chains (chains of pearls) characteristic of *T. verrucosum*.

The disease causes great economic loss to animal products (wool, meat, etc.) due to skin injuries and many deformities, as reported by Weber (2000). Dermatophytosis has also been considered the most common zoonosis worldwide (Achterman and White 2012; Adeleke et al. 2008).

6.8 Antifungal Immunotherapy: Vaccines

Given their increasing frequency and unacceptably high morbidity and mortality rates, the prevention of invasive fungal infections has become of vital importance (Spellberg 2011; Medici and Del Poeta 2015). Vaccination of high-risk groups is a particularly promising strategy to check invasive fungal infections because easily identifiable risk factors are clearly defined from any such infections (Perlroth et al. 2007; Spellberg 2011). Advances in our understanding of the host defense and pathogenic mechanisms underlying fungal infections have supported the development of effective vaccines to combat these diseases. Invasive fungal infections can be some of the trickiest medical problems to treat. A dearth of antifungal medications is one reason for this, says Edwards: “There just aren’t a lot of drug options

for preventing or treating fungal disease. We have vaccines for parasites, viruses, bacteria but no fungal vaccines." Recently, the feasibility of vaccination of mice with crude antigen preparations from an *Aspergillus* strain, *A. fumigatus*, has been demonstrated in animals that were, afterwards, immunocompromised (Cenci et al. 2000; Ito and Lyons 2002). In these studies, vaccination was found to be effective for survival against both inhaled as well as intravenously administered fungi. Researchers have dedicated studies towards developing robust, durable, and safe fungal vaccines. For most active vaccines studied against invasive fungal infections, the key to protection has been the induction of cell-mediated, proinflammatory, Th1 or Th17 responses, which improve phagocytic killing of the fungus. It is also clear that antigens targeted for vaccination need not be restricted to virulence factors but also focus on niche vaccination of patients in restricted geographical areas. The lack of complete understanding of the market potential for such vaccines and the requisite capital to develop these vaccines are also a matter of concern in this area (Spellberg 2011).

6.9 Future Prospects

Economically sustainable therapeutic techniques and improved diagnostic methods can significantly improve the prognosis of cattle mycotic diseases. Reports of adapting modifications of human kits such as that aimed at galactomannan detection to animals, especially pets, are scarce and should be the matter of more detailed assessment for cattle aspergillosis for clear use recommendations. The role of imaging techniques has also been found to be often unreliable and should be investigated further. While great advances in antifungal therapy have been made in the last several years, cost-effectiveness is still a major hindrance, as the price of the newer drugs is unaffordable for animal use, especially considering the necessity of extended, possibly life-long, treatments. This has restricted their use to a very low number of cases, mostly for scientific researches. As the research in this field is ongoing, the price of these drugs will decrease in the future, but the development of specific antifungal drugs for veterinary use should be promoted in order to achieve the required level in treatment. The discovery of drug-resistant aspergillosis in patients who have never previously taken antifungal medications suggests that drug resistance in some cases could be coming from the environment and not from the previous use of antifungals. Resistance acquired through the environment needs special attention from researchers, dairy owners, and policymakers, as it can pose a new challenge to antifungal therapy and treatment in bovines.

The conventional identification of pathogenic fungi in the clinical microbiology laboratory is not appropriate, as it is based on morphological and physiological tests, which often require three or more days and may be inaccurate. The early, quick, and accurate detection of the pathogenic fungus is critical for timely, suitable dairy management. In recent years, a multiplex polymerase chain reaction (PCR) method was developed to simultaneously identify multiple fungal pathogens in a

single reaction (Luo and Mitchell 2002, which can prove to be a very useful tool for timely detection and, so, curing the animal. It is also the need of the hour to relate weather-based plant disease forecasts to recent climate change models, and, hence, predict the effects of climate change on where, which, and by how much mycotoxins will be changed.

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