DERMATOMYCOSIS – CONDITIONS THAT CONTRIBUTE TO THE DISEASE DEVELOPMENT

ABSTRACT: Skin lesions caused by dermatophytes are classified depending on the infected skin structure: surface layer of cutis, fur layer, clutches or nails. Surface mycoses are caused by dermatophytes: Epidermophyton, Microsporum and Trichophyton species (also important are Malassezia spp., Candida spp., and Trichosporon). Skin is the target tissue for fungal infections if the epithelial layer is damaged and immune system cannot cope with the infection, or if the conditions are favorable for dermatophytes, which spread in the cutis due to the enzyme activities. Dermatophytes can be found on skin surface if they contaminate or colonize epidermis or hair follicles. However, clinical symptoms of lesion on the skin are sometimes absent. According to the literature data 6-9% of skin lesions are caused by dermatophyte in human medicine. Similar situation is in veterinary medicine. Fungus that cause dermatomycosis are widespread in the nature and could be divided into: zoophilic, geophilic and anthropophilic.

The goal of this paper is to present the latest knowledge in pathogenesis on dermatomycosis, predisposing factors important for the outcome of the disease, and immunological reaction of organism to the fungal infection. Our intention is to summarize the subject and present the facts related to specific problems in dermatomycosis.

KEY WORDS: dermatomycosis, dermatophyte, pathogenesis, immunity

INTRODUCTION

Among many microorganisms that are present in nature there are over 300 fungi that are actually pathogenic for animals and people (Outerbridge and Catherine, 2006). Mycoses are manifested differently and appear if the immune system of the host is weak, or under various conditions that support the growth of fungi. It is important to determine the factors that contribute to the mycoses development, such as: 1. Fungi are widespread in nature so eradication is difficult, 2. Clinical manifestation is variable (inflammation, allergic reaction), 3. Diagnosing is not easy since clinical appearance is different and depends on the host, 4. Therapy is difficult since number of available drugs is

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Igor M. Stojanov, Jasna Z. Prodanov Radulović, Ivan M. Pušić, Miloš Kapetanov, Radomir D. Ratajac, Sandra Jakšić

Scientific Veterinary Institute “Novi Sad”, Rumenački put 20, Novi Sad, Serbia
igor@niv.ns.ac.rs
restricted, 5. Prevention is available for some fungi and only for some animal species (B lanco and Garcia, 2008).

Dermatophytes present a part of the above mentioned group of fungi, among which zoonoses are some of the most important, since they are common for both people and animals. These fungi are widespread in nature and its classification depends on the habitat and their presence in various ecology niches. They are classified into zoophilic dermatophytes (which also include silvatic ones, those found in woods), geophilic and anthropilic (Chabasea and Piheta, 2008).

Most of dermatophytes are located superficially and are localized on the surfaces of cutis, hair and nails. However, the mechanism between the host and fungus that actually contributes to the disease is not well understood. Lesions on skin induced by fungus depend on the location and structure of the skin, as well as on the skin product (superficial layer of the cutis, hair or nails). Dermatophytes cause superficial mycoses (most often Microsporum, Trichophyton and also Malassezia spp., Candida spp. and Trichosporon). If the protective barrier is damaged the skin presents main “door” for fungal infection. The skin infection may occur when fungus contaminates or colonizes epidermis or hair follicles, although it has been reported that clinical changes are not always present. According to the literature data, dermatomycosis in human medicine encompasses 6 to 9% of cases of all pathological changes in skin. This is similar in veterinary medicine (Stojanov et al., 2009).

The most significant aspects of dermatomycosis are related to the broadening of knowledge on all the factors that participate in pathogenesis, such as: proteases, secretory enzymes, adhesion possibilities and ability to modulate defense mechanisms of the host (Sandey et al., 2008). These data lead to the research of two problems: investigation of the pathogenicity mechanisms that transform ubiquitous fungi to pathogenic, and research on resistance mechanisms of the host related to the infection and disease.

The main goal of this paper is to present the latest knowledge on pathogenesis of dermatomycosis, predisposing factor important for the outcome of the disease, and immunological reaction of the organism to the fungal infection. Our intention is to summarize the subject and present the facts related to specific problems in dermatomycosis.

DERMATOPHYTES

Dermatophytes (gr. derma = skin + phyton = plant) (Klan and Šipka, 2006) includes three genera: Epidermophyton, Microsporum and Trichophyton. Fungi that belong to these genera can grow on keratinized tissue of animals and people (skin, hair, fur, nails, clutches) and induce dermatophytosis.

Infection appears on cutis and is restricted to dead cornified layers, since dermatophytes cannot penetrate deeply into the skin, and immune system of the host prevents the spread of this agent (Sandey et al., 2008). These fungi are not part of the normal microflora of the skin in people and their presence on
the skin is a consequence of their ability to utilize keratin as a food source, that is opposite from other fungi (Weitzman and Summerbell, 1995).

Dermatophytes are classified in three anamorphic orders (asexual and imperfect): *Epidermophyton* spp., *Microsporidium* and *Trichophyton* from the class of anamorphic Hyphomycetes which belong to Fungi imperfecti. These genera are described in literature, depending on morphology and production of conidia.

*Epidermophyton* spp. Macroconidia of this type of dermatophytes have enlarged wedge with typical smooth, thin and slightly bold wall. Macroconidia have one to nine barriers that are 20 to 60 μm wide and 4–13 μm long. They appear in large number, single, or in a group. This genus has two species among which the pathogenic one is *E. floccosum*.

*Microsporum* spp. Macroconidia have walls that are rough, uneven, wart and serrated. Macroconidia of first isolated dermatophyte were described as fusiform, but later on new species were described with ovoid macroconidia (*Microsporum nanum*) (Fuentes, 1956), cylindrical form (*Microsporum vanbreuseghemii*) (George et al., 1962). Macroconidia have thin, slightly large or large walls with 1–15 barriers, and are 6–160 μm x 6–25 μm in size. Macroconidia can be stocky, with stalk or sphenoid appearance, usually individually situated along hyphae.

*Trichophyton* spp. Macroconidia are smooth, with thin wall having 1 or 12 barriers. They appear alone or in the group and could have long appearance, as a pencil; they may become cylindrical, or resemble a long wedge. Macroconidia are 8–86 μm x 4–14 μm in size. In comparison to Macroconidia, Microconidia are present in larger number, have a shape of a ball or pear. They can be sphenoid, stocky or stalk “like”, and can appear either individually, at one side of hyphae, or in a cluster.

**DERMATOMYCOSIS**

*Dermatomycosis of dogs and cats*

Most often, dermatomycosis is induced by *Microsporum canis* in both cats and dogs, while in dogs, the disease is most often caused by *Trichopyton mentagrophytes* and *T. mentagrophytes* var. *erinacci*. From the clinical point of view, characteristic lesions are: round hairless spots, with broken hair and inflamed skin, and milliary dermatitis present. Lesions are rarely generalized and appear if immunosuppression occurs, as well as in the case of hyperadrenocorticism. Folliculitis and onychomycosis (mycosis of clutches) can occur in dogs. Dogs’ lesions can appear on muzzle and this is related to their activities (Quinn et al., 2002), such as digging and machination in ground with muzzle, hunting of rodents and attacking hedgehogs. As a consequence of such activities, specific type of dermatophytes that reside in the ground, rodents and wild animals, could be found on dogs. *M. gypseum* is present in ground, while *T. mentagrophytes* var. *erinacci* can be found in hedgehogs. It was confirmed
that arthropore of dermatophytes could be found on fur of dogs and cats, even if the clinical manifestation of the disease is absent.

**Dermatomycosis of cattle**

The main causative agent of dermatomycosis in cattle is *Trichophyton verrucosum*. Affected animals have lesions around their eyes and on head. In heifers and cows, lesions could appear on legs and neck. The characteristic lesions are: alopecia and spots with gray and white deposits similar to scabs (Quinn et al., 2002; Gudding and Lund, 1995). Infection is most common in winter. In spring, when animals are outside on grasslands, the disease vanishes. If immune system is not able to cope with the disease, therapy or vaccine prevention is obligatory. Dermatomycosis are rare in goats and sheep, but if the infection does occur, it is caused by *M. canis*, *T. mentagrophytes* and *T. verrucosum*.

**Dermatomycosis in horses**

*Trichophyton equinum* is the main agent inducing dermatomycosis in horses. Still, there are two more types of dermatophytes that infect animals in various geographic regions, and those are *M. equinum* and *T. equinum var autotrophicum*. The agent is transferred by direct contact with infected animal or through contaminated equipment used for horse care. Changes are often present on parts of the skin that are in contact with belt and saddle, but could appear on all parts of the body if the brush, contaminated with this agent, is used for horse grooming. Infection with dermatophytes from the ground is possible with *M. gyseum*, if horses are rolling on the ground, with *M. canis* and *T. mentagrophytes* if they are in contact with dogs and cats, or with *T. verrucosum* if they are in contact with cows. Young animals are more susceptible to infection than the older ones.

**Dermatomycosis in pigs**

Dermatomycosis is significant in pig production. It could appear in all ages and is usually related to poor management. The main cause of this disease comes from the *M. nanum* that resides in the ground.

**Dermatomycosis in poultry**

Dermatomycosis in poultry is rare. It is caused by *M. gallinae* as a consequence of poor management. In hens and turkeys, it appears in the form of
white deposits on scabs and wattle, in severe cases it could attack the feather follicles and cause systemic disease.

**Dermatomycosis in people**

Infections caused by dermatophyte (ringworm) are named depending on the location of lesions. Word naming the lesion location is added after the Latin word *tinae*. *Tinae barbae* – stands for the infection of the chin which could be superficial or deep, with severe inflammatory pustular folliculitis. They are caused by zoophilic dermatophytes (Kwon–Chung and Bennett, 1992). *Tinae capitis* – represents head covered with hair. Changes could be subclinical with erythema or severe folliculitis, alopecia, sometimes with lymphadenopathy as well. It is caused by *Microsporum* and *Trichophyton* (Rippon, 1985). *Tinae corporis* – dermatomycosis that appears on body, shoulders and legs, and may also appear on face. Clinical signs could be severe with clearly limited erythematous vesicular spots. *Tinea cruris* – infection of crotch, perianal and perineal region. It appears mostly in older mail persons. Causative agents are *T. rubrum* and *E. floccosum*. The symptoms are flushing with dry dandruff. *Tinea favus* – causes lesion on head that appear as prominent yellow scabs and dry dandruff. It has been recorded mostly in Euro Asia and Africa. *Tinea imbricate* is a chronic infection that appears in places where skin folds, and causative agent is *T. concentricum*. It can be found in Asia, North and South America and Oceania. It is strictly anthropilic dermatophyte (Rippon, 1988). *Tinea manuum* – its causative agent is *T. rubrum*. Lesions are found on the palms and interdigital areas of hands. Hyperkeratosis and cracking of the skin are present. *Tinea pedis* – is present on soles of feet and toes. It is also called athletes foot. It could be chronic with squamose epithelia, hyperkeratosis, redness and inflammation. Causative agents could be *Epidermaphyton floccosum* and a member of genera *Trychophyton*. *Tinea unguim* – attacks nails and appears under nails or superficially. The most frequent agents are *T. rubrum* and *T. mentagrophytes*.

Tab. 1 – Main type of fungi that could cause mycoses in people (Deacon J., 2005)

<table>
<thead>
<tr>
<th>Primary site of pathogen entrance</th>
<th>Fungi</th>
<th>Pole stage</th>
<th>Disease</th>
<th>Type and the place of invasion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td><em>Trichophyton</em> (22 species)</td>
<td></td>
<td>Dermatomycoses: ringworm, tinea, athletes foot</td>
<td>Keratinized tissue people, wild and domestic animals</td>
</tr>
<tr>
<td></td>
<td><em>Microsporum</em> (19 spp.)</td>
<td><em>Arthroderma</em> (Ascomycota)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>but only 9 are involved in infections</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td><em>Epidermophyton</em> (2 spp)</td>
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</tbody>
</table>
Table 2. Some of the major dermatophytes that could infect people (Deacon J., 2005)

<table>
<thead>
<tr>
<th>Anthrophilic</th>
<th>Zoophilic</th>
<th>Geophilic</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Epidermophyton floccosum</em></td>
<td><em>Microsporum canis</em> (dogs, cats)</td>
<td><em>Microsporum gypseum</em> (the most common infection in people)</td>
</tr>
<tr>
<td><em>Microsporum audouinii</em></td>
<td><em>Microsporum equinum</em> (horses)</td>
<td><em>Trichophyton terrestre</em></td>
</tr>
<tr>
<td><em>Microsporum ferrugineum</em></td>
<td><em>Microsporum nanum</em> (ground/pigs)</td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton mentagrophytes var interdigitale</em></td>
<td><em>Microsporum persicolor</em> (rodents)</td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton rubrum</em></td>
<td><em>Trichophyton equinum</em> (horses)</td>
<td></td>
</tr>
<tr>
<td><em>Trichophyton tonsurans</em></td>
<td><em>Trichophyton mentagrophytes var mentagrophytes</em> (mice, rodents)</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Trichophyton verrucosum</em> (cattle)</td>
<td></td>
</tr>
</tbody>
</table>

PREDISPOSING FACTORS FOR SKIN FUNGAL DISEASE

It is very important to have the knowledge on predisposing factors that contribute to the development of fungal skin diseases. High humidity and hot climate, such as in tropical countries, contribute to the development of dermatomycosis (Blank et al., 1969). If the skin of laboratory animals or people is covered at the place of fungus inoculation it becomes softened and paired (Greenberg et al., 1976). The covered place enhances the humidity of the skin and keeps CO₂ produced by skin. This helps the growth of dermatophytes.

Many medical reasons contribute to predisposing factors for dermatomycosis. Dermatomycosis is usually found in chronically ill patients and animals suffering from vascular disease, corticosteroid therapy, Cushing disease, hematological malignancy, chronic candidosis, diabetes mellitus or atopic dermatitis (allergy to many allergens present in the house and nature) (Hay, 1982). The age of a patient is also important for the development of dermatomycosis, and such infections usually occur without symptoms (Gilchrist, 1979). Vascular disorders in peripheral blood stream that have not been diagnosed, and keratinization problems are related to chronic dermatomycosis. Nowadays, the predisposing factors for this disease are number of different allergies that are widespread in the world (Wagner and Sohnle, 1995). Research has shown that sensitivity to dermatophytes could be connected to hereditary factors and that some recessive autosomal genes could transfer higher susceptibility to dermatomycosis (Serjeantson and Lawrence, 1977).

Effects of dermatophytes on host immune system

Results of the research indicate that dermatophytes are capable of "avoiding" the immune system and causing lesions in the host. Fungi may express several effects, including inhibition of lymphocyte by mannans – plant polysaccharides, impaired function of macrophages, disturbed activation of
keratinocytes and secretion of different protease (Gi d d e y et al., 2007). The level of immune response and inflammation depend on how deep fungi have penetrated in the skin. Less invasive dermatophytes are sheltered from soluble components of the immune system (D a h l and G r a n d o, 1994). Also, secretion of subtilisin (Sub3) and metalloprotease (Mep3) produced by M. canis (B r o u t a et al., 2003) participate in the immunomodulation in host. Subtilisin and dipeptidyl protease V, secreted by T. rubrum and T. tonsurans, may induce immunity causing acute dermatomycosis and delayed type of hypersensitivity reaction (DTH) (W o o d f o l k and P l a t t s – M i l l s, 2001) with high IgE and IgG4 level. Molecules of Trichophyton rubrum cell wall mannan (TRM) act immunosuppressively. They can inhibit the proliferation of mononuclear leukocytes against several antigens, including antigens of dermatophytes under laboratory conditions (M a c C a r t h y et al., 1994). Keratinocytes and monocytes/macrophages play an important role in the modulation of immune response. However, level of inter leukins (IL-1) secreted by these cells was lower when they were in contact with T. Rubrum than with T. mentagrophytes (O g a w a et al., 1998). The enzymes of dermatophytes, like dipeptidyl protease IV, may influence the immune response by dissolution of soluble immune components (L a n d i s et al., 2008).

PATHOGENESIS OF DERMATOMYCOSES

Adherence and invasion of skin surface

Dynamics of dermatophyte adherence to skin and keratinized skin tissues was studied in experimental models by using microscope techniques. It was determined that depending on time, the number of spores attached to skin and, consequently the number of germinated spores, increased. Also, the penetration through stratum corneum and spreading in different directions was observed (S a n d y et al., 2008). Zurita and Hay determined that maximal adherence of arthroconidia Trichophyton spp. to keratocytes occurred in the first 3 to 4 hours. For some species of Trichophyton spp. (T. mentagrophytes) the adherence takes place during the first 6 hours and the germination starts after only 4 hours (R a s h i d et al., 1995). In laboratory conditions, when skin sample of live tissue (explant) was used, maximal adherence was reached in the first 12 hours, and the spore germination started after one day (D u e k et al., 2004).

It is well known that there are factors that mediate the adhesion of dermatophytes. For example, at the surface of T. rubrum macroconidia, specific carbohydrate adhesives enable the adhesion of dermatophytes to the epithelial cells (E s q u e n a z i et al., 2004). Interestingly, one research showed that, on skin surface, long and free fibrils connect arthroconidia of fungi and keratocytes, while in deeper layers newly formed arthroconidia spread through the tissue creating a contact surface between skin and fungi (K a u f m a n et al., 2007).

Similar to the findings that confirmed aspartic protease in Candida albicans, which is necessary for the adherence to the host (D e B e r n a r d i s et
al., 2007), it was observed that protease enzymes, secreted by dermatophytes, facilitate the adherence, or are required for this process. The secretion of proteolytic enzyme subtilisin, metalloproteases and dipeptidyl peptidases by \textit{M. canis} is regarded as important for the adhesion, or for the early phase of invasion of this microorganism (K u m a g a i et al., 2005).

\textbf{Growth on hard keratinized skin products}

Dermatophytes have several proteases essential for the transformation of keratine into useful oligopeptides or amino acids. Fungi secrete different forms of serine and metalloendoproteases (J o u s s o n et al., 2004) which are called keratinases. The level of importance of hydrolases, such as lipases or ceramidases, has not been precisely determined yet, but Viani et al. Have found that potent keratolytic hydrolase of \textit{M. canis} is responsible for clinical infections. However, it remains unresolved whether the symptoms are caused by the activity of dermatophyte's keratinases, or the lesions develop because of inflammation and immune reaction. In any case, keratolytic effect of these enzymes is possible only after the reduction of disulphite bonds which maintain the protein structure of keratin tissue (K u n e r t, 1992). The excretion of sulphite depends on sulphite efflux pump that enables sulphitolysis of proteins and makes them available for proteases.

The secretion of proteases by fungi occurs under the circumstances of complex protein compounds being the only source of carbon and nitrogen, but not glucose and easily digestible peptides (J o u s s o n et al., 2004). This means that the keratolytic activity of dermatophytes is expressed under restricted nutrient conditions. Successful survival and growth of dermatophytes in some species actually depend on secretion of numerous proteases. The protein and protease structure differ depending on the species, in spite of extremely high similarity of orthologous genes. Specific features of dermatophytes that cause severe inflammation in host are probably related to different regulation of protein and protease secretion.

It should be mentioned that skin damage may occur without any activities of lytic enzymes of dermatophytes, but as a consequence of other biotic factors, such as bacteria or parasites. Moreover, host proteases may be activated and may contribute to the development of altered skin structures because of allergic reaction.

\textbf{Skin immune response and inflammation}

Superficial infections with dermatophytes cause different inflammatory reactions in organism depending on the pathogenicity of agent and chronicity of the process. Anthrophilic dermatophytes, such as \textit{T. rubrum} and \textit{E. Floccosum}, generally cause mild inflammation and small lesions on skin, but usually long term or persistent infections. On the other hand, geophilic and zoophilic
dermatophytes cause strong inflammatory reaction restricted to smaller surfaces because of higher immune response. The above mentioned points to the significant role of localized inflammatory process and the immune response of the host against dermatophytes (Wagner and Sohnle, 1995).

Chemotactic mechanism

Superficial fungal infections are limited to the surface layers of skin, but sometimes the infection can be spread more deeply and cause strong inflammation. These deep changes include the occurrence of desquamation, vesicle and pustule formation, as well as considerable skin damage. Under microscope, aggregation of large number of neutrophils and formation of microabscesses can be observed in the acute phase, while in chronic cases monocytes dominate and hyper or para keratosis develop. Chemotactic mechanisms, complement activation, that participate in mobilization of neutrophils, are very important for inflammation (Swann et al., 1983). The reason why some fungal infections like *T. rubrum* cause only mild inflammation is the fact that this fungus secretes substances which disable chemotactic mechanisms and hinder the activity of neutrophils (Davis and Zaini, 1984). Chemotactic mechanisms that activate keratinocytes by secretion of cytokines, contribute to the inflammation and defense of the host are still unclear.

Role of phagocytic cells

The role of neutrophils in inflammatory reaction after the infection with dermatophytes is different. Their microbiocidic activity depends on oxidative activity of superoxide and hydrogen peroxide, hipochlorine acid and monochlor amines (Test et al., 1984). Nonoxidative substances, such as cathepsine, proteins that increase bactericidal effect or permeability, lactopherin, lysozime, elastase, azuricide and others, may act bactericidally (Gaby et al., 1986). Antimicrobial features of macrophages/monocytes are expressed through the production of nitrogen oxide which inhibits fungal pathogens.

Superficial skin infections with severe clinical forms occur more often in immune compromised individuals, which leads to the conclusion that preserved functions of immune system are crucial for the protection against dermatomycoses. Proper functioning of defense system is necessary even in case of superficial infections reaching the stratum corneum (Findling et al., 1981). Numerous researches indicate that epidermis is not just passive barrier against infectious agents, but also acts as immune surveillance which, by means of cell cooperation, successfully protects the organism from a wide palette of different noxae (Wagner and Sohnle, 1995).

Cell wall of dermatophytes is primarily comprised of chitin and glucan that make glycopeptides, main antigens of these microorganisms. Like other fungi, dermatophytes possess complex antigens, such as glycopeptides, pep-
tides or carbohydrates (Moser and Pollack, 1978). Antigen features of these molecules are good and they sensibilize immune system even in case of superficial infection. Antibodies against *T. rubrum* were determined in people not infected with this fungus, although cross reaction with antigen of some other microorganism remained possible (Sohnle et al., 1983). By using different serologic methods (ELISA, complement fixation, immunodiffusion and agglutination), investigations of humoral immune response in humans showed presence of antibodies against dermatophytes (Papini and Simonetti, 1985). Special immunologic problem is the occurrence of hypersensibilization mostly manifested as late allergic reaction of type DTH. It is not completely clear whether chronic dermatomycosis influences the development of IgE or the organism is predisposed due to atopa presence (Sergeant and Lawrence, 1977). This allergic reaction is also related to dermatophyte species. In humans, DTH more frequently occurs in acute form of infection with *T. Mentagrophytes*, in comparison to the chronic form of infection with *T. rubrum* (Jones et al., 1973).

In conclusion, the presence of dermatophytes in humans and animals is often without clinical signs or with nonspecific skin changes, which delays prompt diagnostics. Dermatophytes are widely distributed and are well adjusted to specific ecologic niches. Therefore, immune system is not always prepared to respond in a completely satisfying manner. It can be stated that there are some differences between dermatomycoses in humans and animals, mainly because of the living conditions, that is the environment. The relationship between animals and humans, as well as the contemporary lifestyle, their living together or in close proximity, claims for better understanding of all the factors that can influence the infection, its spread and the reaction to dermatophytes in both humans and animals.

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ДЕРМАТОМИКОЗЕ – УСЛОВИ КОЈИ ДОПРИНОСЕ НАСТАНКУ БОЛЕСТИ

Игор М. Стојанов, Јасна З. Проданов Радуловић, Иван М. Пушић, Милош Капетанов, Радомир Д. Ратајац, Сандра Јакшић

Научни институт за ветеринарство „Нови Сад“, Руменачки пут 20, Нови Сад, Србија

Резиме

Промене на кожи изазване дерматомицитима могу бити систематизоване у зависности од структуре или продукта коже који је захваћен: површински слој кутиса, крзнио-длачни покривач или канце – нокти. Површинске мицозе изазивају дерматомицете *Epidermophyton, Microsporum* и *Trichophiton* врсте (поред наведених врста значајне су још *Malassezia spp.* и *Candida spp.* и *Trichosporon*). Кожа представља узазна врата за гљивичне инфекције када је заштитна епителна ба- ријера оштећена и имунолошки систем није у стању да се избори са инфекцијом или када су створени услови да дерматомфите својом ензимском активносту насе- ље кожу и прошире се ткивом кутиса. На површини коже се могу наћи дерматофите које контаминарију и/или колонизују површину епидерма или длачног фоликула, али се клинички знаци, промене на кожи, није увек јавити. Дерматомфите у хуманој медицини, према доступним подацима из литератури, представљају узрочнике 6% – 9% свих промена везаних за кожу и продукте коже. Сличан на- лаз је и код клиничких и лабораторијских испитивања узорака у ветеринарској практици. Гљивице које узрокују дерматомикозу су група микроорганизама веома расширених у природи и њихова заступљеност, у односу на природно станиште и присуство појединих врста у појединим деловима животног станишта, делу их у зоофилне, геофилне и антропофилне.

Задатак нашег рада је да презентује досадашња истраживања везана за па- тогенезу дерматомикоза, предвидирајуће факторе који имају важну улогу у на- станку болести као и да прикаже имунолошку реакцију организма на гљивичну инфекцију. Намера нам је да се сумарним приказивањем наведене тематике на јед- ном месту изнесу чињенице везане за специфичну проблематику дерматомикоза.