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HIDDEN KILLERS: MOLDS AND MYCOTOXINS

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Mold Diseases

Many diseases in horses have been associated with the presence of molds. These diseases involve guttural pouches, lungs, eyes, skin, the reproductive system, and the body as a whole.

GUTTURAL POUCHES

Molds have been isolated from infected guttural pouches in horses worldwide. Such associations have been made in India (Pal, 1996), Korea (Ha Tae Yong et al., 1995), France (Guillot et al., 1996), Japan (Takatori et al., 1984; Yoshihara et al., 1994), Italy (Gresti et al., 1993), and the United States.

The most common molds isolated from equine guttural pouches are *Aspergillus*, *Penicillium*, and *Candida* (Grabner, 1987). Perhaps the most important report for this group is a case where a liquid pellet feed binder was found to be the source of infection for a horse with guttural pouch disease. *Aspergillus* sp. was cultured from the guttural pouch, the mixed feed, and the liquid pellet binder (McLaughlin and O'Brien, 1986).

Early reports did not identify the particular species of *Aspergillus*. Several secondary problems were identified early on, including erosion of the internal carotid artery, cranial nerve damage (Hilbert et al., 1981), and blindness (Hatzios et al., 1975). Later studies reported the species of *Aspergillus* responsible for disease.

Aspergillus nidulans is the *Aspergillus* species most frequently isolated from equine guttural pouches. The association between *Aspergillus nidulans* and guttural pouch mycosis was first recognized in the early 1970s (Johnson et al., 1973; Johnson and Attleberger, 1973). Soon thereafter an association between *Aspergillus nidulans* guttural pouch mycosis and nosebleeds was made (Lingard et al., 1974). Coughing, nasal discharge, and loss of 100 kg in 16 days occurred in a horse with *Aspergillus nidulans* guttural pouch mycosis (Krogh and Lundegaard, 1986). *Aspergillus nidulans* guttural pouch mycosis has been recognized in India (Pal,

1996), Korea (Ha TaeYong et al., 1995), France (Guillot et al., 1996), and Japan (Takatori et al., 1984; Kanemitsu et al., 1995). *Aspergillus nidulans* has recently been renamed *Emericella nidulans*. Horses have bled to death after erosion of the carotid artery due to *Emericella nidulans* infection of the guttural pouch (Guillot et al., 1997; Matsuda et al., 1999). *Emericella nidulans* and *Aspergillus fumigatus* were isolated from the guttural pouches of four Thoroughbreds using endoscopy. Three of the horses were killed because of their poor prognosis (Anzai et al., 2000).

Two other species of *Aspergillus* are commonly isolated from equine guttural pouches. *Aspergillus fumigatus* from a guttural pouch infection has caused an atlanto-occipital joint infection (Dixon and Rowlands, 1981) and nasal discharge (Greet, 1981). Guttural pouch mycosis has also been caused by *Aspergillus ochraceus* (Gresti et al., 1993).

A *Penicillium* sp. mold was isolated from the guttural pouch of a horse with a fistula that developed from a guttural pouch mycosis (Jacobs and Fretz, 1982).

LUNGS

Aspergillus has also been associated with lung lesions in horses. Both acute and chronic forms of the disease have been identified (Sudaric et al., 1979). An association between GI disease and pulmonary aspergillosis has been suspected. Invasive pulmonary aspergillosis was identified in 19 horses; 16 of them also had enterocolitis (Slocombe and Slauson, 1988). Endocarditis and pulmonary aspergillosis developed in an 8-year-old Quarter Horse after surgery (Pace et al., 1994). Three Thoroughbreds died after a five-day illness of apathy, fever, lacrimation, and dyspnea after being transferred to a new stable. They died with thrombosis, hemorrhage, and tissue necrosis. A diagnosis of pneumonia caused by *Aspergillus niger* was made (*Rhizopus stonifer* was also isolated) (Carrasco et al., 1997). The sudden death of two horses was attributed to the rapid and acute development of pulmonary aspergillosis. One horse developed it after surgery, the other while being treated for equine protozoal myelitis (Johnson et al., 1999).

EYES

A variety of molds has been isolated from the eyes of horses with keratitis (Hamilton et al., 1994). *Alternaria*, *Aspergillus*, *Actinomyces*, *Candida*, *Fusarium*, *Penicillium*, and *Mucor* have been isolated from 11 cases of keratomycoses in Pennsylvania (Beech et al., 1983). Additionally, *Rhizopus* (Scherzer et al., 1998), *Cephalosporium*, and *Phycomyces* have been isolated. *Aspergillus* is the most prevalent (Moore et al., 1983). Of 31 keratomycosis cases in Texas, 11 were *Aspergillus* and four were *Penicillium* (Coad et al., 1985).

Aspergillus and *Fusarium* are most commonly isolated. Of 39 horses treated for ulcerative keratomycosis, *Aspergillus* was isolated from 13 and *Fusarium* from 10 (Andrew et al., 1998). *Aspergillus* and *Fusarium* sp. were also reported in a second study (Kern et al., 1983). *Aspergillus flavus* (Grahn et al., 1993; Collins et al., 1994), *Aspergillus fumigatus* (Aho et al., 1991), and *Aspergillus oryzae* (Marolt et al., 1984) are the most commonly reported *Aspergillus* species. *Fusarium* has been isolated in a number of cases also (Mitchell and Attleburger, 1973; Hodgson and Jacobs, 1982). Of six cases with keratomycosis, *Aspergillus* was isolated from three, *Fusarium* from two, and *Cladosporium* from one (Peiffer, 1979).

REPRODUCTIVE SYSTEM

Molds have also been reported to cause abortions as well as uterine and placental infections. Fungi were cultured from nine of 100 aborted horses in India. *Candida tropicalis* was in three, *Aspergillus fumigatus* in three, *Candida albicans* in two, and *Cryptococcus laurentii* in one (Monga et al., 1983). Of 2000 pregnancies followed in India, 175 abortions occurred. Six were caused by fungi. The fungi involved were *Mucor* (three), *Aspergillus* (two), and *Microsporum* (one) (Garg and Manchanda, 1986). *Aspergillus fumigatus* has been diagnosed as the cause of abortion in two Thoroughbred mares (Plagemann et al., 1992).

Aspergillus fumigatus and *Candida albicans* were isolated from mares with uterine infections (Blue, 1983). Fungi isolated from the uteri of mares with endometritis are *Actinomyces*, *Aspergillus*, *Candida*, *Coccidioides*, *Hansenula*, *Monosporium*, *Mucor*, *Nocardia*, *Paecilomyces*, and *Trichosporon* (Pugh et al., 1986). Of 27 mares with chronic infertility problems, *Alternaria* sp., *Aspergillus flavus*, *A. fumigatus*, *A. niger*, *Mortierella wolffi*, and *Mucor* sp. were isolated from cervical, vaginal, or clitoral fossa swabs (Verma and Gupta, 1983). Of 200 cases of infective placentitis, 37 were caused by *Aspergillus fumigatus* and 14 by *Absidia* sp. (Whitwell and Powell, 1988).

SKIN

Of 1090 horses examined, most had *Trichophyton equinum* skin disease, but *Aspergillus* infection was common (Takatori et al., 1981).

SEPTICEMIA

An 18-year-old Morgan had a 10-day history of watery diarrhea, depression, and dysphagia. It died four days after being referred to a veterinary teaching hospital. *Aspergillus niger* was identified as the cause of vasculitis and brain infarction (Tunev et al., 1999). *Mucor* and *Rhizopus* were associated with a horse that developed myocarditis and nephritis after surgery (Peet et al., 1981).

IMMUNOSUPPRESSION

Horses have developed systemic mold infections after corticosteroid treatment or natural immunosuppression. Fatal pulmonary infections with *Aspergillus flavus* and *A. niger* developed after corticosteroid immunosuppression (Weiler et al., 1994) or colic treatment (Smith et al., 1981). A horse with myelomonocytic leukemia developed pulmonary aspergillosis after phenylbutazone and corticosteroid therapy (Blue et al., 1987) or without such therapy (Buechner-Maxwell et al., 1994). A chronic bronchopulmonary *Aspergillus* infection was diagnosed in a 30-year-old Saddlebred with Cushing's syndrome (Carrasco et al., 1996).

Mold Allergies

Chronic obstructive pulmonary disease (COPD) is also referred to as "heaves," "broken wind," or "pulmonary emphysema." The syndrome was first reported in the early 1970s. Eight horses were reported as having an allergic pneumonitis that was clinically and pathologically similar to farmer's lung in humans (Paul et al., 1972). The pathology has been described as peribronchitis, perivascularitis, and interstitial pneumonitis with foci and nodules of macrophages containing refractile particles of inorganic dust in alveoli (Chen et al., 1989). Considerable work has gone into determining the factors that predispose horses to the disease.

Molds were one of the predisposing factors initially considered. Using the method available at the time, exposure of horses to *Penicillium*, *Alternaria*, *Epicoccum*, and *Cladosporium* molds was found to have little relationship to the existence of the disease (Halliwell et al., 1979). Poor ventilation of the stable did seem to increase the chance of a horse becoming affected with COPD. However, gender, body weight, and season of onset of coughing did not seem to influence occurrence of the disease (McPherson et al., 1979b). After ruling out several factors, an immune component was investigated.

The possibility of COPD as a hypersensitivity disease has been considered (McPherson et al., 1979a; McGorum et al., 1993) and gained acceptance. Currently, evidence indicates that COPD is a delayed hypersensitivity response to inhaled antigens, particularly molds. It involves increased histamine, thromboxane, and 15-hydroxyeicosatetraenoic acid in bronchoalveolar lavage fluid (BALF), and decreased prostaglandin E₂ in airway mucosa (Robinson et al., 1996).

Despite these advances, the diagnosis of COPD was elusive, largely because investigators focused on skin reactions and serum antibody titers. Although horses with COPD had strong skin reactions after intradermal injections of mold extracts, there was no correlation between fungal contamination and the incidence of COPD. *Aspergillus*, *Alternaria*, and *Hormodendron* extracts were tested (Eyre et al., 1972). Of 237 horses tested, 100 of which had COPD, no relationship between COPD and type I skin reactions was seen. Although many horses gave a type III reaction

to *Micropolyspora faeni* (Eriksen and Olson, 1990), more recent studies of dermal and pulmonary reactivities to *M. faeni*, *A. fumigatus*, and *T. vulgaris* indicate that intradermal testing is of limited value in investigating COPD (McGorum, 1993b).

Studies focusing on serum antibody titers have been equally disappointing. Circulating precipitins to *Micropolyspora faeni* and *Aspergillus fumigatus* were not restricted to horses with COPD, but did occur more frequently in horses with COPD (Lawson et al., 1979). In 119 serum samples, antibodies against *M. faeni* were demonstrated in 11, and among these, COPD was only confirmed in four (Eriksen et al., 1986). Serological tests are of little value in the diagnosis of COPD (Madelin et al., 1991). In eight horses tested with 67 extracts from different allergens, significant difference was evident between horses with COPD and healthy horses in only 3% of the possible extracts (Evans et al., 1992). Higher titers of anti-*Micropolyspora faeni*, anti-*Aspergillus fumigatus*, and anti-hay mold precipitins were observed in the serum samples of horses positive for equine influenza antibodies (Chabchoub et al., 1994). Fortunately, BALF has been investigated.

Recently, use of BALF has shed light on the diagnosis of COPD. *Micropolyspora faeni* and *Aspergillus fumigatus* were identified as common causes of respiratory hypersensitivity in horses affected with COPD (McPherson et al., 1979a). An ELISA was used to measure specific antibodies to *Micropolyspora faeni* and to *Aspergillus fumigatus* in the serum and BALF of normal horses, horses with COPD, and horses with other respiratory diseases. Elevated antibody results were not detected in the sera of any horses, but IgE and IgA antibodies to both allergens were significantly elevated in BALF of COPD horses (Halliwell et al., 1993). Horses with COPD have significantly higher levels of BALF IgE and IgG to *A. fumigatus* antigens but no significant differences in serum (Schmallenbach et al., 1998).

Treatment has been developed. Sodium cromoglycate (80 mg) prevented exacerbation of the respiratory disease for four to five days after exposure to *Micropolyspora faeni* (Murphy et al., 1979).

Mycotoxins (Forage)

FESCUE

Fescue toxicosis in horses has been recognized for decades. Nevertheless, the mechanism of action and successful management practices are only now being reported. The prevalence of exposure, clinical signs, management, and treatment reports are briefly summarized.

Despite decades of knowledge of the potential for toxicosis from endophyte-infested fescue, many horses remain exposed. Of 207 equine owners and veterinarians responding to a recent survey, fescue was the predominant forage on 50% of pastures and was present on 70%. Almost 50% of the broodmares in the

survey were exposed to endophyte-infected fescue, and 43% had signs of toxicosis requiring treatment or management to reduce the problem (Anas et al., 1998).

The clinical signs of fescue toxicosis are well known. Pregnant mares develop agalactia, stillbirth, and thickened and retained placentas (Bennett-Wimbush and Loch, 1998). In addition, mares have dystocias (McCann et al., 1992), increased gestation length, increased foal and mare mortality, weak and dysmature foals, and increased sweating during warm weather (Cross et al., 1995). The prevalence of clinical signs is not the same in each case. For example, in one study approximately 26% of 1010 mares on fescue pasture had fescue toxicosis, 53% had agalactia, 38% had prolonged pregnancy, 18% had abortions, and 9% had thickened placentas (Garrett et al., 1980).

Mares are not the only horses affected. Average daily gain for yearlings is lower on fescue with high infection rates versus low infection rates (Aiken et al., 1993). Fiber digestibility of endophyte-positive hay is lower than that for endophyte-negative hay (McCann et al., 1992).

The mechanism for these signs long eluded researchers but may now have been discovered. Decreased perfusion of peripheral tissues (Adney et al., 1993) and impaired endometrial cup function mechanisms were investigated (Brendemuehl et al., 1996). Reduced serum prolactin and progesterone and increased serum estradiol 17 beta levels have been observed (Cross et al., 1995). At this point, it appears that ergovaline in tall fescue infested with *Neotyphodium coenophialum* explains the clinical signs and laboratory results previously reported (McClusky et al., 1999).

Fescue toxicosis may be dealt with by management or treatment. A rotational grazing technique allows use of fescue for growing horses. Even though endophyte-infected tall fescue hay may be less digestible in horses than uninfected hay (Redmond et al., 1991), young growing horses being exercised can efficiently use the endophyte-infected fescue on a short-term basis (Pendergraft et al., 1993). Similar techniques can be used in mares.

If mares are removed from fescue late in gestation, most signs of toxicosis can be eliminated or reduced. Withdrawal from infected fescue before parturition results in a rise in serum prolactin levels, allowing milk production (Redmond et al., 1991). Mares moved to endophyte-free pasture at 305 to 310 days of gestation delivered live foals and lactated normally. Supplementation of energy requirements to these mares while grazing endophyte-infected fescue was of little or no benefit (Earle et al., 1990).

Effective treatments after signs develop are also being developed. Selenium treatment is not effective (Monroe et al., 1988). Fluphanazine has been considered (Bennett-Wimbush and Loch, 1997), and domperidone looks promising. Daily oral doses of 1.1 mg/kg body weight domperidone prevented symptoms of fescue toxicosis in late gestation mares on endophyte-infested fescue forage (Cross et al., 1999). A single injection of a long-acting dopamine receptor antagonist may be

beneficial in reducing the effects of fescue toxicosis in pregnant mares grazing endophyte-infected tall fescue pastures (Bennett-Wimbush and Loch, 1998).

RYEGRASS

Ataxia, tremors, and paralysis were observed in a group of horses and then several weeks later in a second group ingesting the same hay. The horses were ingesting ryegrass hay containing 5 to 6 mg lolitrem B/kg (Van Oldruitenborgh-Oosterbaan et al., 1999). A stallion ingesting 1.5 and 2.5 mg lolitrem B/kg also experienced ryegrass staggers. Trembling, hyperexcitability, and abdominal muscular spasms developed suddenly in ponies fed exclusively ryegrass seed cleanings shown to contain 5.3 mg lolitrem B/kg (Munday et al., 1985; Hintz, 1990).

SWEET CLOVER

Spontaneous nosebleeds developed in a 6-year-old Percheron mare fed weathered sweet clover (McDonald, 1980).

RED CLOVER

Excessive salivation and increased water consumption were observed in horses eating red clover or lucerne infested with *Rhizoctonia leguminicola*. Slaframine was associated with the parasympathetic signs (Socket et al., 1982). The slaframine breaks down with time. It fell from 100 mg/kg to 7 mg/kg after ten months of storage (Hagler and Behlow, 1981).

ALSIKE CLOVER

Photosensitization and biliary fibrosis may occur in horses ingesting alsike clover (*Trifolium hybridum*) (Nation, 1989). Chronic or nervous clinical signs and liver disease, including biliary fibrosis and epithelial proliferation, may occur (Nation, 1991). Icterus and photosensitization are followed by nervous signs in almost 80% of cases (Zientara, 1993). Icterus of the sclera, oral and vulvar membranes and dermatitis of the muzzle and vulva, as well as increased serum liver enzymes have recently been reported in horses ingesting alsike clover (Colon et al., 1996).

Mycotoxins (Grains)

FUMONISIN

Fumonisin are new mycotoxins that are of great significance to horse owners. Like aflatoxin, they are suspected of being carcinogenic, so feed entering interstate

commerce will be subject to regulation based on its fumonisin content. FDA's guidance document issued June 6, 2000 indicates that corn or corn by-products intended for horses may contain five ppm fumonisin B₁, plus B₂, plus B₃, but comprise no more than 20% of the diet.

Fumonisin is produced by *Fusarium moniliforme*, which causes "stalk rot" in corn. Fumonisin toxicosis in horses has primarily been caused by corn screenings or corn-containing feed. Fumonisin causes equine leukoencephalomalacia (ELEM), liver necrosis, and occasionally death in horses. Quite a number of field and experimental reports have substantiated this.

Although "moldy corn poisoning" of horses has been recognized for decades, fumonisin was not identified as the causative agent until 1988. Investigators in South Africa were the first to make the connection (Marasas et al., 1988; Kellerman et al., 1990; Thiel et al., 1991; Sydenham et al., 1992; Sydenham et al., 1993). Since then, fumonisin toxicosis in horses has been recognized in Italy (Carmelli et al., 1993), Australia (Shanks et al., 1995), Hungary (Fazekas and Bajmicy, 1996; Fazekas et al., 1997), Mexico (Rosiles et al., 1996), France (Guerre et al., 1997), Turkey (Akar and Sari, 1998), and the United States. The reports from North America are summarized.

In the United States, several cases of fumonisin toxicosis were diagnosed in 1989 and 1990. No clinical cases occurred in horses ingesting feed with less than eight ppm fumonisins (Wilson et al., 1990; Ross et al., 1991a,b). In 1991 and 1992, fumonisin B₁ and B₂ were detected at concentrations higher than 10 ppm in 16% of 291 Indiana corn samples tested (Binkerd et al., 1993), so studies of the dose to produce toxicity were initiated.

A pony fed a diet of 22 ppm fumonisin for 55 days suddenly died (Wilson et al., 1991; Wilson et al., 1992; Ross et al., 1993). *Fusarium moniliforme* was isolated from each feed sample of 125 horses affected with ELEM (Wilson et al., 1990a,b,c). One hundred donkeys died in Mexico with ELEM after ingesting feed containing 0.67 to 13.3 mg fumonisin B₁ (Rosiles et al., 1998), and four Thoroughbreds developed ELEM after ingesting corn containing 46 to 53 µg/g fumonisin B₁ (Mallmann et al., 1999).

Fusarium moniliforme produces fumonisin B₁, B₂, and B₃. Early studies focused on fumonisin B₁, but it now appears that fumonisin B₂ can also contribute to toxicosis (Ross et al., 1994). Fumonisin B₂ at 75 ppm (0.75 mg/kg body weight daily) caused hepatotoxicity and ELEM in ponies. Fumonisin B₂ is more effective than fumonisin B₃ in causing toxicity (Riley et al., 1997). The fumonisins appear to cause ELEM by interfering with myelin synthesis.

Both fumonisin B₁ and B₂ disrupt sphingolipid metabolism (Riley et al., 1997). So, the ratio of sphinganine to sphingosine may be elevated in horses exposed to fumonisin B₁ or B₂ (Goel et al., 1996). This testing of serum or liver is being used to diagnose exposure to fumonisin. Aflatoxin is the other mycotoxin that is a suspected carcinogen.

AFLATOXIN

Aflatoxin is primarily produced by *Aspergillus* molds and occasionally by *Penicillium* molds. Grain in interstate commerce is currently regulated based on its aflatoxin concentration because it is a suspected carcinogen. The major concerns to horse owners though are liver disease, death, and abortion.

Signs of aflatoxicosis in nonpregnant horses include mild fever, anorexia, depression, incoordination, and marked swelling of the supraorbital fossae (Poomvises et al., 1982; Asquith et al., 1983). Signs of inappetence, depression, tremors, and prostration have also been reported (Cysewski et al., 1982). Liver disease develops in horses receiving more than 0.075 mg/kg aflatoxin B₁ (Cysewski et al., 1982), but liver enzymes return to normal within 10 days of removing the source (Aller et al., 1981).

Equine deaths have been reported in field and experimental cases. Three horses had severe hepatic necrosis and died after ingesting corn containing aflatoxin B₁, B₂ and M₁ at 114, 10 and 6 ppb, respectively (Vesonder et al., 1991).

All ponies given 4 mg/kg aflatoxin B₁ died and half given 2 mg/kg died (Bortell et al., 1983). Death occurred at 12 to 16 days in horses dosed with 0.3 mg/kg aflatoxin B₁, at 25 to 32 days if dosed with 0.15 mg/kg, and at 36 to 39 days if dosed with 0.075 mg/kg (Cysewski et al., 1982). Mortality rates of 25% are reported in field cases (Poomvises et al., 1982).

Abortion is not reported in most species ingesting aflatoxin unless the dam is quite ill. However, a report of 17 of 63 mares aborting 6- to 9-month-old feti after ingesting feed containing 250 ppb aflatoxin B₁ exists (Xie et al., 1991).

ERGOT

Symptoms of ergot toxicosis developed in several horses fed Bermuda grass hay (Lindley, 1978). *Achnatherum inebrians* (drunken horse grass) in China contained ergonovine and lysergic acid amide at 2500 and 400 mg/kg, respectively (Miles et al., 1996).

DEOXYNIVALENOL

Reduced general condition was noted in horses fed for six to eight weeks on oat samples containing 20 ppm deoxynivalenol and 2 ppm zearalenone (Bauer and Gedek, 1980).

Table 1. Molds, mycoses and allergies in horses.

<i>Mold</i>	<i>System</i>	<i>Mold</i>	<i>System</i>
<i>Absidia</i>	Reproductive	<i>Candida tropicalis</i>	Reproductive
<i>Actinomyces</i>	Eyes	<i>Cephalosporium</i>	Eyes
<i>Actinomyces</i>	Reproductive	<i>Cladosporium</i>	Eyes
<i>Alternaria</i>	Eyes	<i>Coccidiodes</i>	Reproductive
<i>Alternaria</i>	Reproductive	<i>Cryptococcus laurentii</i>	Reproductive
<i>Aspergillus</i>	Eyes	<i>Emmericella nidulans</i>	Guttural pouch disease
<i>Aspergillus</i>	Guttural pouch disease	<i>Fusarium</i>	Eyes
<i>Aspergillus</i>	Lungs	<i>Hansenula</i>	Reproductive
<i>Aspergillus</i>	Reproductive	<i>Micropolyspora faeni</i>	COPD
<i>Aspergillus</i>	Skin	<i>Microsporium</i>	Reproductive
<i>Aspergillus flavus</i>	Eyes	<i>Monosporium</i>	Reproductive
<i>Aspergillus flavus</i>	Reproductive	<i>Mortierella wolffi</i>	Reproductive
<i>Aspergillus flavus</i>	Septicemia	<i>Mucor</i>	Eyes
<i>Aspergillus fumigatus</i>	COPD	<i>Mucor</i>	Reproductive
<i>Aspergillus fumigatus</i>	Eyes	<i>Mucor</i>	Septicemia
<i>Aspergillus fumigatus</i>	Guttural pouch disease	<i>Nocardia</i>	Reproductive
<i>Aspergillus fumigatus</i>	Reproductive	<i>Paecilomyces</i>	Reproductive
<i>Aspergillus nidulans</i>	Guttural pouch disease	<i>Penicillium</i>	Eyes
<i>Aspergillus niger</i>	Lungs	<i>Penicillium</i>	Guttural pouch disease
<i>Aspergillus niger</i>	Reproductive	<i>Phycomyces</i>	Eyes
<i>Aspergillus niger</i>	Septicemia	<i>Rhizopus</i>	Septicemia
<i>Aspergillus ochraceus</i>	Guttural pouch disease	<i>Rhizopus stonifer</i>	Eyes
<i>Aspergillus oryzae</i>	Eyes	<i>Rhizopus stonifer</i>	Lungs
<i>Candida</i>	Eyes	<i>Trichophyton equinum</i>	Skin
<i>Candida</i>	Guttural pouch disease	<i>Trichosporon</i>	Reproductive
<i>Candida</i>	Reproductive		
<i>Candida albicans</i>	Reproductive		

Table 2. Molds and mycotoxins in horse feeds.

<i>Mold</i>	<i>Source</i>	<i>Mycotoxin</i>
<i>Acremonium coenophialum</i>	Fescue	Ergovaline
<i>Aspergillus</i>	Grain	Aflatoxin
<i>Claviceps purpurea</i>	Small grains	Ergot
<i>Fusarium</i>	Grain	Oxynivalenol
<i>Fusarium moniliforme</i>	Grain	Fumonisin
<i>Neotyphodium coenophialum</i>	Fescue	Ergovaline
<i>Penicillium</i>	Grain	Aflatoxin
<i>Rhizoctonia leguminicola</i>	Legumes	Slaframine

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