Table 7 - Toxic Plants Which Affect the Liver and Cause Cirrhosis

Scientific Name	Common Name
*Amsinckia intermedia	Fiddleneck
*Crotalaris spp	
*Echium plantagineum	Viper's bugloss
*Heliotropium europeum	
Phyllanthus abnormis	Abnormal leafflower
Sartwellia flaveriae	
*Senecio spp	Groundsel

* Contain Pyrrolizidine Alkaloids

Table 8 - Toxic Plants Associated with Photosensitization

Scientific Name	Common Name
Ammi majus	Bishop's weed
Agave lecheguilla	Lechugilla
Avena sativa	Oats
*Cymopterus watsonii	Desert Parsley
Cynodon dactylon	Bermuda Grass
*Hypericum performatum	St. Johnswort
Lantana spp	
Medicago spp	Alfalfa
Nolina spp	Sacahuista
Panicum coloratum	Kleingrass
*Polygonum fagopyrum	Buckwheat
Tetradymia spp	Horsebrush
Tribulus terrestris	Goat Head, Puncture Vine

* Primary Photosensitizer

Table 9 - Toxic Plants Which Accumulate Selenium

Scientific Name	Common Name
Astragalus spp	Locoweeds
Atriplex nuttallii	Saltbrush
Oonopsis spp	Goldenweed
Stanleya pinnata	Prince's Plum
Xylorrhiza spp	Woody Aster

Mycotoxicoses of the Bovine with Reference to Fungi and Toxins Associated with Disease

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Introduction

The mycotoxicoses are a group of diseases caused by toxins produced by a number of different fungi. In many instances, the toxins represent secondary metabolites produced by fungi following a growth phase of the fungus. There are approximately 50 different genera of fungi in four major toxonomic groups capable of producing over 100 different toxins. We are concerned, however, only with those that may become involved with animal health because they (1) produce toxins orally active or potentially poisonous by other exposure routes for animals and man, (2) can be isolated from food and feedstuffs or (3) (toxins) are found naturally occurring in food and feedstuffs in amounts sufficient to be responsible for disease.

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The Fungi

The major genera of fungi concerned with bovine mycotoxicoses discussed in this paper are *Aspergillus, Penicillium, Fusarium, Rhizoctonia,* and *Claviceps. Pithomyces, Stachybotrys, Phompsis, Phoma Diplodia,* and *Myrothecium* are genera of fungi implicated in disease in other countries. Although it is helpful to have identification of fungal contaminants of foods and feedstuffs, definitive indentification of the toxin is necessary to prove the diagnosis. There are several reasons for this. First presence of the fungus is no assurrance it was producing its toxin. Second, a given toxin may persist in a food or feedstuffs when the fungus producing it is no longer present. Third, a given fungus may be capable of producing more than one toxin, i.e., *Aspergillus flavus* may produce both aflatoxin and a tremorgenic mycotoxin, and *Aspergillus parasiticus* may produce both aflatoxin and rubratoxin. Fourth, a given toxin may be produced by

different genera of fungi, i.e., ochratoxin A, a nephrotoxic mycotoxin, is produced by *Aspergillus ochraceus,* as well as by *Penicillium viridicatrum.*

In spite of these variables, it is helpful to be aware of these fungi (the environmental conditions necessary for their development and foods and feedstuffs where they occur), the toxins they produce and the experimental or naturally occurring disease syndromes associated with them. For example, the temperature extremes under which certain fungi grow and produce ther toxin vary from *Aspergillus flavus* producing alfatoxin at 32-38°C (89.6-100.6°F) to *Fusarium tricinctum* producing trichothecene (T-2) toxins at 8° (46°F) or lower with *Penicillium sp* in a wide range of 4°C (39.2°F) to 35°C (95°F).[1] Toxigenic fungi have been divided into field and storage fungi, i.e., those which invade developing plants and seeds prior to harvest and those which invade stored grains and forages.[2] The field fungi invade developing plants and seed prior to harvest under moderately warm, moist weather conditions (usual moisture content of 90-100%) and include fungi such as *Fusarium* and *Rhizoctonia sp.* discussed in this paper. The storage fungi invade stored grains and forages and consist mainly of *Penicillium* and *Aspergillus* growing at ranges from 25-35°C in relative humidity of 70% to 90%. However, with the discovery that aflatoxin may be present in field corn, the division of field and storage fungi is not as clear-cut as once presumed.

The Toxins

The toxins themselves are an interesting group of chemical compounds. Many are pigments, others posess antibiotic or toxic properties and these are the ones in which we, as veterinarians, have the most interest.[1] Patterson has emphasized the fine line existing between antibiotic and toxic properties of mycotoxins since antibiotics are simply mycotoxins with selective toxicities for certain pathogens.[3] It is not clearly understood why fungi produce these chemical toxins. It has been postulated to be a means of disposal of intermediates that accumulate in fungal cultures once they stop dividing.[4] At any rate, the fact that fungi do produce these compounds have had far reaching implications for animal and human health beyond those of fungus contamination **per se.**[1]

The Mycotoxicoses

General--As a group of diseases, the mycotoxicoses represent a real challenge to the practicing veterinarian and to those of us in veterinary medical diagnostic laboratories. Many of the syndromes are subtle and are difficult for the practitioner to diagnose and the laboratory to confirm. The greatest overall loss may well be in decreased productivity and performance through the insidious and debilitating chronic effects of mycotoxins, i.e., reduced growth rate, reduced feed conversion and permanent stunting. These effects can be attributed to a decreased palatibility of foods, the fact that major nutrients used in the growth process of molds are unavailable to animals, and accumulation of the toxic metabolites of molds in foods and feedstuffs. As the latter occurs, a number of more acute disease problems may be identified.

Virtually every system of the animal's body may be affected by one or a combination of mycotoxic substances, i.e., (1) **Vascular system** (increased vascular fragility, hemorrhage into body tissues (aflatoxin, dicoumarin), (2) digestive system (diarrhea, intestinal hemorrhage and hepatotoxic effects manifested as liver necrosis, bile duct proliferation and fibrosis (aflatoxin)) (caustic effects on mucous membranes (T-2 toxin, Stachybotrys)), (bile duct occlusion (sporidesemin)) (salivation, (slaframine)), (3) **respiratory system** (adenomatosis (4-ipomeanol)), (4) **nervous system** (tremors, incoordination, mania, ataxia, coma (tremorgens, ergotamine and related alkaloids)), (5) **cutaneous system** (photesensitization (sporidesmin) and necrosis and sloughing of the extremities (fescue foot, ergot)), (6) **urinary system** (nephrosis, uremia (ochratoxin, citrinin)), and (7) **reproductive system** (infertility, prolonged estrous (zearlenone or F2 toxin)).

Specific Clinical Syndromes

Aflatoxicosis - The chemical toxin(2) responsible for this disease syndrome is aflatoxin, named from the fungus that produces it, *Aspergillus flavus.* Although a number of metabolites, chemically known as bisfuranocoumarin compounds, have been identified, the discussions in this paper will apply to aflatoxin B^1 and M^1. Aflatoxin B^1 has been the subject of worldwide study and concern since its association with "Turkey X" disease in England,[5] and, most significantly, its establishment as a potent carcinogen.[6]

Although aflatoxin may be produced on many foods and feedstuffs, most emphasis has been placed on corn, likely because of the large acreages and its extensive use as livestock feed. Further emphasis was placed on the problem of aflatoxin in corn with the serious problems in both field and storage corn throughout the southeast in the late summer and fall of 1977. In the southeastern United States, it has been estimated that approximately 50% of the corn harvested in the fall of 1977 contained more than 400 parts per billion (ppb) of aflatoxin B_1.[7]

As with many other species, an important feature of aflatoxin poisoning in cattle is reduced growth rate and, in the case of dairy cattle, decreased milk production.[8,9] Also as with other species, the young are the most susceptible. This is especially true in the calf where dietary levels of aflatoxin as low as 200 ppb in 4-day-old calves over a 3-month period decreased both weight gain and feed efficiency.[10] Thirty-day-old calves fed 0.2 mg/kg/day aflatoxin B^1 for 14 days developed mild clinical signs of depression and inappetance but weight gain ceased after day 14 and 0.5 mg/kg/day proved fatal on day 14.[11]

In 5-to 8-week old calves on a 2,000 ppb aflatoxin B_1 diet, loss of weight was apparent at 1 month of age and terminal signs developed at 4-6 months with evidence of abdominal pain and blood-stained diarrhea.[12] In 6- to 8-month-old steers, aflatoxin dietary levels of 700-1,000 ppb resulted in significant decreases of weight gain, food consumption and feed efficiency over a 4-6 month period.[8]

Dairy cattle on dietary levels of 2,300-4,200 ppb of aflatoxin for a period of 3 weeks to 1 month had marked deterioration in health, and marked reduction of feed consumption and milk production.[8] Concern has been expressed that the carcinogenic metabolite M_1 is excreted in milk of cattle and that a relationship does exist between dietary intake of aflatoxin B_1 and the concentration of M_1 in the milk.[13] At least 3 practical safeguards have been emphasized concerning M_1 contamination of milk, i.e., (1) milk is usually bulked thus a dilution factor exists, (2) there is evidence that M_1 is more weakly carcinogenic than B^1, and (3) pasteurization may reduce M_1 contamination.[14] Nonetheless, a lower tolerance level for aflatoxin has been set for aflatoxin in dairy cattle feedstuffs (20 ppb)[14] than is required for beef cattle (100 ppb).[15]

Necropsy findings may include generalized icterus, ascites, visceral edema, congestion of intestines and kidneys, hemorrhages in the mesentery, and an enlarged fibrous liver. Microscopically, there may be proliferation of bile duct epithelium, chronic obliterating endophlebitis of centrolobular and hepatic veins, a variation in size and shape of parenchymal cells, and a diffuse fibrosis of the liver.

Ochratoxin and Citrinin Toxicosis--These compounds are primarily nephrotoxic and are produced by a number of fungal species of *Aspergillus* and *Penicillium.* Polyuria, low urine specific gravity, pale kidneys at necropsy with microscopic evidence of tubular degeneration and interstitial fibrosis are all changes reported to occur in experimental exposure of young calves to ochratoxin.[11] It has been pointed out that because of the relatively low levels of ochratoxin occurring in nature and the higher levels required to kill adult cattle that natural outbreaks in older cattle would be more chronic in nature; this has been supported experimentally when adult cattle fed more than 20 times the amount of ochratoxin expected to occur in nature exhibited clinical signs only of transitory anorexia, diarrhea and a abrupt cessation of milk production.[16] Nonetheless, it should be remembered that acute toxicosis could be expected in calves not yet having a functional rumen, and it has been shown experimentally that naturally occurring levels of ochratoxin sufficient to produce toxicosis could easily be consumed by young calves.[11]

A very recent report describes strong circumstantial evidence for a case of citrinin toxicosis in a herd of Iowa feedlot cattle.[17] Moldy corn and silage *(Penicillium citrinin* and *Aspergillus niger)* fed to the cattle was the source of the citrinin (2-3 ppm). Nephritis and nephroses were salient histopathologic lesions at necropsy. No new cases occurred after

discontinuance of feeding moldy feedstuffs.[17]

Trichothecene Toxicosis (T-2 Toxin)

More than 30 chemical compounds produced by fungi of *Fusarium* and *Myrothecium sp.* are mycotoxins known as the trichothecene toxins. Some are highly irritating to skin and mucous membranes and experimental animals develop diarrhea, rectal hemorrhaging and necrotic lesions of the oral cavity following their administration. Degenerative changes in bone marrow and massive hemorrhages are reported to develop in the lumen of the small intestine of large animals.[18]

T-2 toxin in moldy corn and in commercially prepared cattle rations has been associated with hemorrhaging and poor weight gain in cattle[19] and experimental studies with calves (at dosage levels of 0.16 mg/kg to 0.64 mg/kg) have verified responses of inappetance, dehydration, abomasal ulcers and acute enteric responses with bloody feces following T-2 administration orally.[11] Other workers have reported hemorrhages confined to the epicardium of calves and ecchymotic hemorrhages in the rumen, abomasum, small intestine, lungs and cervical lymph nodes.[20] The Ld50 in young calves with nonfunctional rumens has been established at 0.6 mg/kg body weight.[20] Interestingly, recently published work failed to confirm hemorrhaging in swine or in one cow following administration of purified T-2 toxin, however, the workers did verify toxic and caustic properties of the toxin.[21] Nonetheless, hemorrhaging in cattle associated with presence of T-2 toxicoses.[19,22,23] One case involved moldy corn,[19] one involved a commercial feed[22] and one, in our laboratory, involved hay in the ration of a group of yearling steers.[23] In all cases, T-2 toxin was demonstrated, and there was history of moldy feeds.

Zearalenone Toxicosis (F-2 Toxin)

Zearalenone, or F-2 toxin, is a natural metabolite produced by at least four species of the genus *Fusarium.* Its primary effects are recognized in swine with typical clinical signs of hyperestrogenism. It also has been reported to cause clinical signs in dairy cattle,[24] although apparently little or no experimental work has been done. There is enough solid circumstantial evidence, however, to alert us to infertility problems associated with zearalenone toxicity in the bovine. In one dairy herd with an infertility problem, 25 ppm of F-2 toxin was found in the meal feed of the animals, *Fusarium graminearum* and *Fusarium culmorum* (both F-2 producers) were isolated from the feed and clinical signs of turbid mucous discharge from the vagina and obvious estrus behaviour were in evidence.[25]

Another report described a bad lot of hay which had resulted in decreased fertility following feeding to dairy cattle. Fourteen ppm of zearalenone was found in the hay samples.[26]

Stachybotryotoxicosis

Most reports of this condition are from eastern Europe and Russia, but it seems worthy of mention here because the saprophytic fungus *Stachybotrys atra,* producing an as yet uncharacterized mycotoxin, does occur in this country. It is a cellulose decomposing fungus and, therefore, grows best in cellulose rich feedstuffs such as straw and hay.

The toxin incites a necrotic dermatitis in rabbit skin tests. It is, therefore, highly irritating to skin and mucous membranes, resembling very closely the effects of T-2 toxin. Both acute and chronic effects of this toxin have recently been reviewed in cattle, varying all the way from debility, refusal of food and sudden death to fever, depression, conjunctivitis, necrotic foci of oral membranes, gums, teeth, and tongue.[27]

Fescue Foot

There may still be reason to question exactly where the "fescue foot" syndrome fits into the overall mycotoxin picture. A number of research scientists have found numerous genera of fungi on toxic fescue, but most attention has focused on *Fusarium tricinctum.* This fungus has been found to increase with the favorable environment in yearly accumulated growth of tall fescue. This led one group of scientists to initiate studies on *F. tricinitum* that led to isolation of a toxic butenolide compound providing some, but not all, of the lesions associated with fescue foot.[28] Although the cause of the total pathologic entity associated with fescue foot has not been established definitively, its association with fungi capable of producing toxins at least partially responsible experimentally for the entity, has given fescue foot a prominent place in the disease syndromes associated with mycotoxins.

Fescue foot is a disease solely of cattle with onset of clinical signs occurring from one to several weeks, depending on the level of toxicity in the pasture. Salient clinical features in older cattle include arching of the back, roughened haircoat, soreness in one or both rear limbs, formation of a necrotic line at the coronary band, loss of hooves, and sloughing of the distal end of the ears and tip of tail. A recent extensive review of this disease emphasized its widespread distribution in the United States and pointed to some important potentiating factors, including excessive nitrogen fertilizer with minimum rainfall, grazing on feeding of late fall cut forage in the coldest part of the year and feeding of fescue with minimum supplementation.[29] Therefore, limiting intake of toxic grass and maintaining the rumen flora by supplemental feeding are practical approaches to control.

Ergotism in Cattle

In retrospect, ergotism is undoubtedly the oldest mycotoxic condition known, with descriptions of the condition in man and animals dating to biblical times. It takes two quite distinct forms in cattle, i.e., a gangrenous form with necrosis and sloughing of the extremities and a CNS form characterized by staggers, ataxia, tremors, and convulsions. The former syndrome is attributed to the fungus *Claviceps purpurea* producing the toxins ergotamine and related alkaloids and the latter is attributed to the fungus *Claviceps paspali,* also producing ergotamine and lysergic acid derivatives. Both fungi grow under warm, moist conditions with the former invading rye, oats, wheat, and Kentucky bluegrass, and the latter parasitizing *Paspalum* or Dallis grass. The fungus replaces the grass ovary with a dark brown to purple body known as a sclerotium.[30]

The gangrene of the extremities associated with *Claviceps purpurea* may be very similar to those described for fescue foot but some very important differences should be kept in mind, i.e., (1) fescue foot affects only cattle, (2) fescue foot occurs in the winter when *Claviceps* would be least active, (3) the fescue foot syndrome has been reproduced with extracts of tall fescue devoid of ergot alkaloids, (4) abortion is seldom associated with fescue foot, and (5) forelimb involvement occurs only infrequently in fescue foot but is more common in ergotism. A detailed discussion of these important differences by a number of investigators has recently been published[29] and leads one to feel with certainty that ergot is not involved in the fescue foot syndrome, Prevention of losses due to ergotism depends principally on restricting access to ergotized pastures, grasses or silages. As in fescue foot, the lameness is usually reversible when toxic feeds are removed unless the lesions have progressed to a gangrenous stage.

Paspalum staggers, the central nervous system form of ergotism, has long been recognized both experimentally and from field observations. Affected animals may become extremely hypersensitive, with a wide range of clinical signs associated with nervous incoordination. Very recent information may shed additional light on the exact nature of the toxic alkaloid responsible for this syndrome with the experimental demonstration that a group of tremorgenic mycotoxins caused paspalum staggers.[31]

The Tremorgenic Mycotoxic Syndrome

Since the first report was made of a tremorgenic mycotoxin from *Penicillium cyclopium,*[32] at least 10 such mycotoxins produced by *Penicillium* and *Aspergillus sp.* have been described.[33] One of these toxins, penitrem A, has produced neuropathic effects experimentally in calves consisting initially of fine tremors, progressing to marked tremors, ataxia, paddling in lateral recumbency and opisthotonus.[34] Calves recovered after administration of the toxin ceased.

An isolate of a *Penicillium sp.* producing the mycotoxin penitrem A, was made from a moldy commercial feed suspected to be involved in the deaths of dairy cattle.[35]

These experimental and field reports again serve to remind us that several naturally occurring mycotoxins may cause marked neurologic clinical signs in cattle. It is of further interest to note that one review of the subject in 1973 included a discussion of paspalum staggers and tremorgen intoxication,[36] and that recent findings have shown a mycotoxic tremorgen to be

Rhizoctonia Toxicity or "Slobber Syndrome"

The fungus responsible for this disease, *Rhizoctonia Leguminicola,* also is responsible for a condition known as "black patch" disease of red clover. It produces a mycotoxin, slaframine, capable of initiating an intense salivation response.

There are no definitive pathologic changes associated with the profuse salivation and lacrymation.[37] Over a short period, this entity may primarily cause inconvenience in handling of animals, but prolonged exposure may result in feed refusal, diarrhea, loss of condition, and drop in milk production. It, therefore, should be recognized early for what it is, and animals should be removed from the source of the toxic clover.

Facial Eczema

This mycotoxocosis of cattle is named after one of its salient clinical features rather than for the basic pathologic mechanism responsible for the entity. The condition has been of major economic importance in cattle and sheep in Australia and New Zealand for a number of years,[38] where the fungus responsible has been identified as *Pithomyces chartarum* and the toxin produced is sporidesmin. Clinical signs include irritation, reddening and edema of nonpigmented areas of skin followed by serum exudation of swollen areas that may proceed to deep necrosis and sloughing of tissue.

The basic pathologic mechanism responsible has been shown to be a fibrous occlusion of the bile duct due to the effects of sporidesmin. This allows phylloerythrin, a normal breakdown product of chlorophyll usually excreted in the bile, to build up in the blood. Since it is a photodynamic chemical increased sensitivity to sunlight occurs.[39]

It is puzzling that isolates of *P. chartarum* in this country produce little or no sporidesmin, yet photosensitization diseases similiar to facial eczema do occur here.[1,40] It may well be that a combination of agronomic practices and environmental conditions explain why mycotoxic fungi known to cause major disease problems in one country may be present in another country with neglible, if any, disease impact. Nonetheless, with changing management practices, we must remain alert for the emergence of these disease entities.

Cases of hepatogenous photosensitization have been reported in our southern states from cattle grazing Bermuda grass,[41] and severe outbreaks of photosensitization occured in Oklahoma in cattle fed mold damaged alfalfa.[42] An extensive review of bovine photosensitizations on a global scale has recently been completed.[43]

Regardless of the primary cause, principal control procedures wherever photosensitivity occurs is recognizance of clinical signs, removal from direct sunlight, avoidance of dangerous forages or pastures as they can be determined and management of animals to restrict ingestion of toxin.

Sweet Clover Toxicity

This puzzling disease entity confounded research workers for more than 2 decades before the mycotoxin, 3',3' methylenebis (4-hydroxycoumarin) or dicoumarin produced by the growth of *Aspergillus* and *Penicillium sp.* on improperly cured sweet clover hay, was identified as the causative agent.[44] This bleeding disease of cattle fed on this type of hay has been reported in Canada and the United States since the early 1920's.

Early workers described this disease as one of extensive and uncontrollable hemorrhage with no apparent damage to the vascular bed where it occurred.[45] Fatal hemorrhage characteristically occurs subcutaneously in areas where normal activity would result only in mild trauma, i.e., joints, flanks and areas where the animals might lie down. The underlying cause of the uncontrolled hemorrhaging was described many years ago as a failure of blood coagulation due to loss of prothrombin.[46]

This mycotoxic disease is still with us and must be considered whenever uncontrollable hemorrhages and history of access to moldy and an improperly cured sweet clover hay is in evidence.

Adenomatosis in Cattle

This condition, also known as atypical interstitial pneumonia, is characterized by acute pulmonary edema and emphysema. One of its etiologies in recent years has been shown to be due to a toxin, 4-ipomeanol, found in mold damaged sweet potatoes from which *Fusarium solani* was isolated.[47] It was later postulated that a precursor to this lung-toxic substance was produced by sweet potatoes in response to various injuries and that the fungus *Fusarium solani* was necessary to convert the precursor into a series of lung-toxic substances.[48] A very recent report[49] describes the experimental reproduction of atypical interstitial pneumonia with the toxin 4-ipomeanol that was originally obtained from sweet potatoes infected with *Fusarium solani,* thus demonstrating how fungi may indirectly result in the formation of substances in feedstuffs that are toxic to animals.

Mycotoxicoses in Foreign Countries

The following mycotoxicoses are mentioned to indicate conditions known to affect cattle in other countries, but which, to the best of this author's knowledge, have not been reported as clearly defined mycotoxicoses in this country. The toxigenic fungi responsible for these entities, however, are found here, and it is for this reason they seem worthy to mention to bovine practitioners of the United States.

In *Lupinosis,* the fungus is *Phomopsis leptostromoformis,* which grows on lupin species. The toxin may be Phomopsin A* (*Recent report by Culvenor et al (1977) Abstr. U.S.-Australia Symposium on Poisonous Plants, Logan, UT, June 19-24 1977. Auspices U.S.-Australian Agreement for Scientific and Technological Cooperation.)

The clinical signs consist of icterus and hemoglobinuria. Pathologically, marked fatty infiltration of the liver is observed with bile ductule proliferation in more chronic cases.

In *Phoma* toxicity, the causal, fungus is *Phoma herbarum* (the fungus associated with "black stem" disease of alfalfa), and the toxins producing clinical signs of inappetance, weakness, unsteady gait and a fetid diarrhea in cattle are *brefeldin A* and cytochalasin B.

In *Diplodiosis* of cattle, the fungus incriminated is *Duplodia maydis* (responsible for ear rot of maize). A toxin, diplodiatoxin, is known to be produced by this fungus but its vital role in the causation of clinical signs of lacrymation, salivation, tremors, ataxia, incoordination, and paralysis has not been clearly established.

In *Myrotheciotoxicoses,* the fungi of the genus *Myrothecium* (found on rye grasses and white clover), are involved producing the highly toxic trichothecenes verrucarin A and roridin B. Experimentally, an acute high mortality has been reported with severe hemorrhagic enteritis and a more chronic ill-thrift syndrome.

In-depth descriptions of the fungi,[50-53] the chemistry of the toxins,[54-55] and the clinical signs in cattle associated with ingestion of these toxins[56-59] have recently been published by research workers observing these entities in various countries.

Recognition and Control of Mycotoxicoses

This paper has described a wide range of mycotoxicoses of the bovine. The disease syndromes discussed may effect virtually every system of the body resulting in a number of clinical signs that may be difficult to differentiate from various infectious diseases.

Recognition or early diagnosis of the problem is the obvious first step toward control. Some well known problems interfering with definitive diagnosis are (1) even though a known toxigenic mold is present it may not be producing its toxin, (2) isolation of identification of the specific chemical toxin is necessary for confirmatory evidence of a specific mycotoxicoses, (3) proper sampling (contamination may vary widely within the same storage area so feed samples from a number of locations in the suspect feed storage unit should be taken), and (4) the feed in question may be gone by the time the disease problem is presented.

With recognition of the above limitations, there are a number of factors that still may aid the practitioner in making a presumptive diagnosis. These include (1) recognizing clinical signs consistent with a given mycotoxicosis, (2) failure to isolate infectious agents or observation of failure to respond to antibiotic therapy, (3) probability of exposure to fungal toxins, i.e., careful examination of feed, sources, bunkers, troughs, etc., and consideration of environmental factors favorable to fungus development and toxin production, (4) evidence of feed refusal or other indications of lack of palatibility, and (5) test feeding of small groups of animals with suspect feed, keeping in mind that the young, as a rule, are the most susceptible.

associated with *Claviceps paspali* described earlier in this discussion of paspalum staggers.[31]

Attention to the above factors, along with the knowledge that a number of veterinary diagnostic laboratories may now be able to analyze (or refer to other laboratories) a number of the toxins described, there is reason to be optimistic that more of our mycotoxic diseases can be diagnosed.

The two most important control measures are (1) removal of toxic feed sources and (2) maintenance of feed storage and management practices that hold mold contamination to a minimum.

In regard to the first control measure, it should be remembered that the effects of many of the mycotoxic substances may be only temporary and that recovery may follow removal of the toxic feed. Even in those instances where this is not the case, mold contamination may be "spotty" and removal of the toxic feed source is the best preventive measure for future occurrence of toxicosis. Where such a move is not economically feasible, "blending" of feeds may be possible for salvage of livestock feed on the premise in question, i.e., diluting moldy feed with good feed, although the risk of obtaining a safe "blend" should be kept in mind and such practices would not be recommended for lactating animals or for feeds entering trade channels.[60]

In regard to the second control measure, it is reemphasized that two factors are critical in determination of fungal growth, i.e., temperature and moisture. It has been pointed out that *Aspergillus flavus* grows well at temperatures of 32-38°C (89.6-100.6°F) and at moisture levels of 18% or above equilibrating with a relative humidity of approximately 85%. The combination of these factors are critical in the estimation of the length of time that shelled corn can be held at various moistures and air temperatures.[61] The advantages and disadvantages of high moisture grain storage, of chemical preservation, of high temperature versus low temperature grain drying, of chemical preservation of grains to limit fungus growth, and of chemical compounds now under study for detoxification of contaminated grains represent other dimensions of feed storage and management practices that are too extensive for incorporation into this paper. These have recently been thoroughly reviewed.[62]

General feed management practices, however, i.e., systematic clean-up of feedlines, augers, and feeder grain storage at proper moisture content and inspection of grain regularly (for temperature, insects, moistness and wet spots) are all practical, realistic and relatively inexpensive procedures that may be followed to limit the possibility of fungal development in feeds and feedstuffs and resultant exposure to mycotoxins.

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Management and Treatment of Toxicosis in Cattle

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Intoxications in domestic livestock continue to confront practitioners with therapeutic and prophylactic problems. The current widespread and necessary use of pesticides in American agriculture continues to cause accidental intoxications in animals. Because of the continued likelihood of intoxications in animals, it is imperative that veterinarians continue their attempt to educate their clients to use proper handling and storage techniques. Only when the actual users and applicators of toxic chemicals control these chemicals properly will the incidence of intoxications be lowered.

The increased incidence of intoxications in domestic animals makes necessary the continued emphasis on treating disease conditions caused by toxicants. Consequently, the purpose of this communication is to briefly identify some of the more common toxicants which cause intoxications in animals, and describe the therapeutic and management procedures which should be instituted to treat the resulting disease states.

Basic Concepts of Intoxication Therapy

The primary goals of therapy in cases of intoxication are:

1. Emergency intervention and prevention of further exposure.