Mycotic Diseases and Mycotoxicosis

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Mycotic Diseases and Mycotoxicosis

- It is unicellular or multicellular organisms, possess cell wall, reproduce asexually, sexually or both and live on organic matter.
- Mycoses are relatively uncommon diseases.
- * The most important mycotic diseases in poultry are:
 - Aspergillosis
 - Dactylariosis
 - Candidiasis
 - Favus
 - Mycotoxicosis

Fungi and yeasts produce disease in two ways:

■Firstly

They invade and destroys body tissues where

- Birds may be exposed to pathogenic strains.
- ▶ Decreased resistance of the birds, may allow invasion by normally non pathogenic strains.

■Secondly

► Fungi can infect growing grains or finished feed and produced mycotoxins which cause disease when consumed by birds.

Aspergillosis Brooder pneumonia Neurnomycosis Mycotic pneumonia

Definition

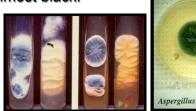
Aspergillosis is a mycotic disease characterized mainly by respiratory signs, affect most domestic and wild birds, it also affect mammals and human particularly pigeon feeders.

Etiology

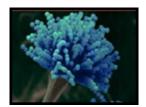
- There are about 600 species of this genus as Aspergillus furnigatus, A. Flavus, A. Niger, A. Nidulans.
- The two major agents causing Aspergillosis in poultry are A. fumigatus and A. flavus.
- These organisms are:
 - Common soil saprophytes world wide.
 - Also may present normally in air and litter in poultry houses.
 - Grow on organic matter in warm (>25°C) and humid environments including damaged eggs in hatcheries, poultry litter and feed.

- Spores are widely distributed in nature and birds frequently contact them through contaminated feed or litter.
- Spores of A. fumigatus is smaller than that of A. flavus, so can reach the lower respiatory tract easily and this explain why A. furnigatus is more common and more pathogenic.
- A. fumigatus is quite thermostable and some of them resist chemicals as sulphuric acid, copper sulphate, formalin.
- These fungi grow readily on ordinary culture media as sabouraud dextrose agar, Czapek's agar, may be used with antibiotics at room temperature (37° C and higher); optimum temperature is (40° C).

Colonies are green to bluish green at first and darken with age to appear almost black.



Colonies vary from velvety to floccose, vesicles are apical and flask shaped in case of A. fumigatus.





Toxins

- A. fumigatus has a toxin which is hemotoxic, neurotoxic and histotoxic.
- Pigeons which are susceptible to spontaneous infection are very resist to this toxin.

Predisposing factors

- ▶ Bad ventilation.
- ► High ammonia.
- ▶ Dusty environment.
- ▶ Overcrowding.
- ► Cold stress.
- ► Concurrent debilitating factors and immunosuppresion.

All these factors increase the incidence and severity of infection.

Susceptibility

- Nearly all species of birds may be affected.
- Turkey poults and duckling are more susceptible to infection than other birds
- Newly hatched birds are highly susceptible (1-3 weeks of age) and the embryos may die before hatching due to contamination of egg shells with Aspergillus.

Transmission

- **4** Egg borne disease:
 - ► The fungi penetrate the egg shell due to contamination of egg shell during incubation,
 - ► Lead to embryonic mortality or hatched infected chicks
- Hatchery borne disease:
 - ▶ Infection to recently healthy hatched chicks occur by inhalation of spores during hatching.
- Inhalation of spores from contaminated feed or poultry house litter.
- Some reported that ingestion of contaminated feed may cause infection, <u>both routes occur in brooding poultry</u> <u>house</u>.
- Infection may occur through eyes at injuries.

Clinical signs

There are 2 forms of Aspergillosis

- Acute Aspergellosis characterized by severe outbreaks in young birds and high morbidity and high mortality.
- Chronic Aspergellosis occur in adult birds and its incidence is of little importance.

Clinical signs include the following pictures:

General signs

- Depression, increase thirst, pyrexia, dysphagia was noted in cases in which esophageal mucosa was involved.
- Dropping of wings, retardation of growth, emaciation, diarrhea in the later stage.
- In acute respiratory disease in young birds may cause high morbidity and mortality.

Respiratory signs (pulmonary Aspergellosis).

- Dyspnea, gasping, accelerated breathing and serous excretions from nose and eyes.
- Birds may suffer coughing, sneezing, rattling noises only if complicated with IB or ILT.
- Asphyxia due to blockage of the larynx or trachea with exudates.







Eye infection

- Usually unilateral in turkey poults and chicks characterized by yellow cheesy (caseous) exudates beneath the nictating membrane (third eye lid) causing bulging of the eyelids.
- Central ulceration of the cornea in older chicks which lead to blindness.

Nervous signs (encephalitis)

- Occur in turkey poults, chicks, duckling and goosling if fungi and its toxin reach brain.
- Encephalitis including torticollis, incoordination of movement and convulsions.
- These signs attributed to toxin production (toxigenic origin)







Central ulceration of the cornea

Yellow cheesy (caseous) exudates beneath the nictating membrane (third eye lid) causing bulging of the eyelids

Osteomycosis:

A. fumigatus caused deformed vertebrae result in partial paralysis in young chickens.

Systemic Aspergillosis:

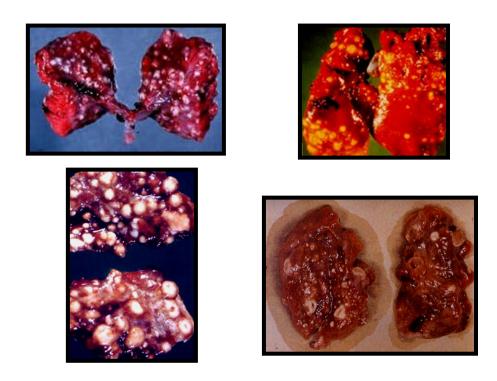
In acute form in young birds if expose to severe infection or may be due to caponization or sternal bone infection hematogenous infection by spores may lead to infection of brain, eyes . . . etc.

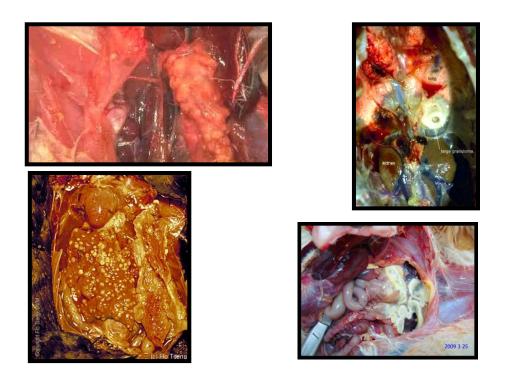
Cutaneous Aspergillosis (dermatitis):

- Outaneous lesions in pigeons and chickens caused by A. glaucus and A. niger, but this infection is rare.
- Necrotic or granulomatous dermatitis in any part of the body and the feathers are dry and easily broken.

Gross lesions

- Congestion of parenchymatous organs and tracheal mucosa with mucopurulent to gelatinous exudates, later on caseous exudates may be present in larynx and trachea and buccal cavity.
- This disease characterized by nodular formation in the lungs, trachea, bronchi, air sacs leading to thickening of the wall.
- Nodules may be present in the thoracic cavity on the pleural surface or in the abdominal cavity on intestinal serosa, liver, spleen, kidney, peritoneum.
- Nodules may be in the cerebrum or the cerebellum or both.
- In the early infection the nodules is white then become greenish.
- The nodules ranged from pin head to millet size and up to a pea.
- Caseous material under the eyelids.
- Ascites.

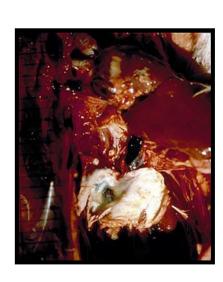












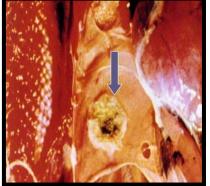






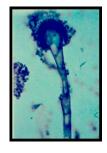






Diagnosis

- Case history, Clinical signs, PM lesions.
- Smear or imprints of nodules.
- Special fungal stains periodic acid-schiff. Spores may be present in nodules of good aerated parts as bronchi and air sacs.
- Isolation and identification of the fungus by staining the colonies with ink dye or lactophenol methylene blue.
- Serology: Serological tests as IFT, AGPT and ELISA are of limited value due to the non specific nature of the antigens.





Differential diagnosis

- From other diseases causing respiratory symptoms.
- From other diseases causing nervous symptoms.
- ■Nodules from that of salmonella gallinarum pullorurn.
- ■Eye lesion from E.coli, coryza, vit. A deficiency.

Prevention

- # Avoidance of moldy feed or litter.
- ➡ Premises or materials used for feed, water or litter should be thoroughly cleaned and disinfected.
- # Frequent moving of feeding troughs and watering places is advisable to avoid wet places.
- Feed store, bins, water tanks must be thoroughly cleaned and disinfected regularly every 2-3 month by fumigation using paraformaldehyde or formalin with potassium permangnate.
- ★ Litter treated by spraying of nystatin or copper sulphate or thiabendazole solution.
- # Incubators, hatcheries, air ducts, and brooding house sanitation.
- ¥ You must add mold inhibitor to the feed, some act on fungi only, others act on toxin, and some of them act on both.
- # Reducing dust and good ventilation in the poultry house.

Treatment

- Mycostatin.
- Amphotericin B.
- Thiabendazole.
- Crystal violet.
- Brilliant green.
- Kanamycin.
- Garlic juice.

Dactylariosis

Definition

②Dactylariosis is a fungal disease characterized mainly by nervous signs in turkey poults and chicks causing severe losses.

Etiology

♣Dactylaria gallopava, it is thermophilic fungus. It grows well at 25 - 37° C but optimally at 45° C, hyphae is brown.

Susceptibility

- ■Species affected are turkey poults, young chickens and quail.
- ■Age: 1-5 weeks old is the high susceptible age.

Transmission

■Infection occurs via inhalation of spores from moldy litter or moldy egg incubators.

Clinical Signs

- >Nervous signs: Torticollis, incoordination, tremors, paralysis, and death.
- >Eye lesion: In few cases ocular lesion develops and produce blindness.
- **Dyspnea**: In rare cases pulmonary granulomas develop and cause dyspnea as in Aspergillosis, mortality 3-20 %.

Lesion

- The lesions differ from the mycotic encephalitis of Aspergillosis characterized by more malacia and hemorrhages and a large numbers of giant cells.
- Severe large, hardened, circumscribed cerebellar and cerebral lesion (necrosis that is gray or yellow).
- Pulmonary granulomas are minimum, indistinguishable from those of Aspergillosis.
- Ocular lesions appear similar to Aspergillosis grossly.



Diagnosis

- Signs, lesions, demonstration of pigmented hyphae with characteristic oval two-celled brownish conidia and large number of giant cells from brain lesions are diagnostic.
- Culture from brain lesions or litter on Sabouraud dextrose agar or others supplemented with antibiotics at 45°C; colonies produce brown pigment which diffuses into the surrounding media.

Differential diagnosis

- Viral (ND, AE).
- Bacterial meningitis.
- Mycotic (Aspergillosis).
- Nutritional (crazy chick disease).

Control

- There is no effective treatment for dactylariosis so prevention of exposure to moldy litter.
- Decontamination of incubators by fumigation is the only means of prevention.

Candidiasis Thrush Moniliasis Sour Crop Crop Mycosis Mycosis of the digestive tract Stomatitis Oidica Oidiomycosis

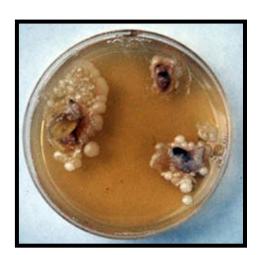
Definition

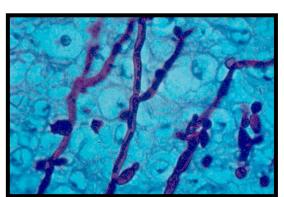
■ Candidiasis is a mycotic disease affecting digestive system especially crop occurs mainly in chicken and turkey.

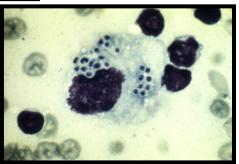
Etioloy

- Candida albicans, yeast like fungi appear as round to oval budding yeasts.
- It is ubiquitous in the environment and is often present in the upper gastrointestinal tract of normal birds.
- Grows on Sabouraud dextrose agar at room temperature or at 37° C and after 1-2 days produce whitish creamy high convex colons.
- Young cultures consist of oval budding yeast cells but old culture show septated hyphae or pseudohyphae and occasionally spherical swollen cells with thickened membrane, the so-called chlamydiospores.
- Soluble endotoxin which is toxic for mice has been isolated from C. albicans.









Transmission

- Ingestion of contaminated water and food by yeast.
- With inhibition of competing microflora or immunosuppression, candidas proliferate and hyphae or pseudohyphae invade superficial epithelial layers.
- These invasions stimulate epithelial hyperplasia and pseudo membrane or diphtheritic membrane formation.

Clinical signs

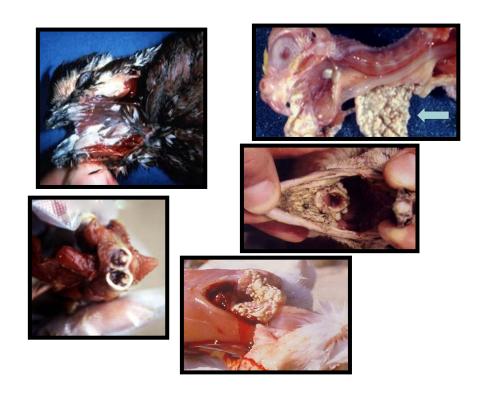
- ✗ Infections is common but clinical signs is present in only severely affected birds.
- ★ Signs appears as depression, retardation of growth, stunted appearance, reduced food intake ruffling feathers, listlessness, diarrhea, may drooling mucus exudates of offensive odor from mouth.
- ★ Pendulous crop in some of the affected birds may occur, when candidiasis occurs as a secondary infection.
- The signs of the predisposing disease may predominate the clinical signs.
- X Drop of egg production in layers.
- Mortality due to candidiasis is low to non existent.





Lesions

- Lesions most frequently in the crop and to less extent in oesophagus and pharynx, may extend to the buccal cavity and proventriculus.
- → The lesion appear grossly as thickening of the mucosa with raised multifocal (circular) to confluent whitish cheesy material.
- ◆ Often there are pseudo or diphtheritic membranous necrotic batches that are peeled easily from the eroded mucosal surface.
- Sloughed ulcer formation may occur.
- When the proventriculus is affected, its mucosa is hemorrhagic with whitish membrane and appears swollen.

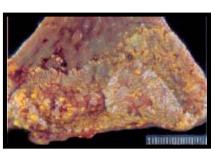




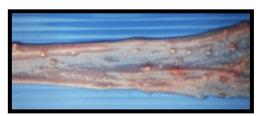
















Diagnosis

- Clinical signs including poor hygiene or history of long term antimicrobial therapy are supportive and candidiasis is suspected.
- Q Lesions are pathognomonic.
- @ Crop swap or tissue scarping
- @ Histological section reveal yeasts and hyphae or pseudo-hyphae in large numbers after staining with gram's stain (gram positive) or lactophenol methylene blue.
- © Cultural characteristics where an original heavy growth is considered essential for diagnosis.

Differential diagnosis

- Diphtheritic form of pox.
- Paratyphoid in pigeons.
- Trichothecene mycotoxines.
- Trichomoniases in pigeon.
- Vitamin A deficiency.

Prevention

- Avoid predisposing factors especially long administration of drugs.
- Through cleaning and disinfections of watering and feeding troughs.
- Addition of mould inhibitor to the feed.
- Feed birds with balanced feeds have adequate amounts of vitamin A.

Treatment

- Copper sulphate 1: 2000 to flush GIT during the course of outbreaks.
- lodine solution used as the sole source of drinking water during the course of outbreaks.
- Nystatin.
- →Amphotericin B
- Gentian violet 1 lb/ton.
- Disinfection of premises to kill yeast which descend in faeces by:
 - ⇒2 % formaldehyde + 1 % sodium hydroxide VV for / 1 hours.
 - ◆1 % solution of iodine monochloride in HCL for 3 hours.
- Oral lesions can be treated locally with suitable antiseptic.

Favus Dermatomycosis White comb Ring worm

Definition

♣ Favus is contagious chronic mycotic disease of the skin affecting chickens, occasionally turkey and some other birds.

Etiology

- Microsporum gallinae.
- These fungi cultivated, on Sabouraud's dextrose agar.
- Reddish pigment varying from rose or strawberry red to a deep raspberry diffuse through the medium.

Susceptibility

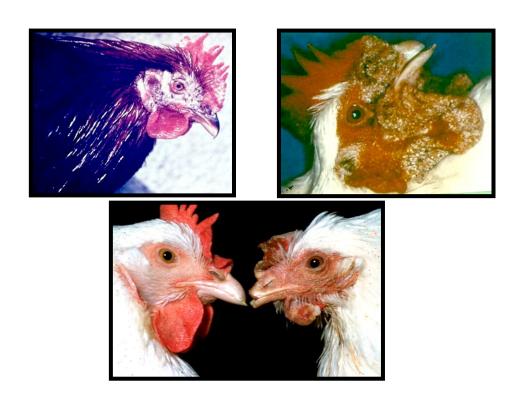
- Favus affect mainly chickens and to less extent turkey and other birds (duck quail, canary), may affect mammals and human.
- Favus affect only adult birds because it is a chronic disease which develop slowly.

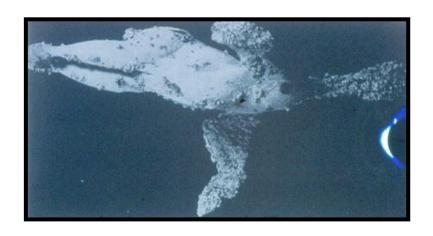
Transmission

*By direct and indirect contact through detached scales contaminating the premises.

Signs and lesions

- Lesions develop firstly in unfeathered skin as comb, wattles and shanks.
- It's covered with dry white spots and appear as if sprinkled with flour.
- ■By progress of the disease, the scaly spots become thicker and form crust which lead to thickening of the skin in the affected areas due to epidermal hyperplasia and hyperkeratosis.
- In severe cases the disease spread to the feathered portions of the body and hence loss of feathers in batches occur in some birds.
- ♣There were necrotic foci, nodules and yellowish caseous materials on mucosa of upper respiratory tract, occasionally bronchi and lungs and may be in crop and small intestine.





Diagnosis

- Lesions.
- Demonstrate fungi microscopically.
- Isolation of the fungus on Sabouraud's dextrose agar.

Prevention

- → Avoid predisposing factors.
- Care should be noticed in adding birds with lesions of favus to the flock.

Control

- Sanitation and sound management:
- → Isolation of diseased birds from apparently healthy ones.
- Hygienic disposal of dead and badly affected birds.
- → Thorough cleaning and disinfection of the premises with 2 -3 % formalin or 0.5 -1% copper sulphate or 1 % NaOH.

Treatment

- Treat mildly affected birds locally after removal of scales by warm water and soap using:
 - Tincture iodine and glycerin 1: 6.
 - Soap and 5 % phenol solution.
 - Sulphur ointment.
 - Use antifungal ointment.
- Systemic antifungal therapy may attempted in valuable birds.
- Spontaneous recovery in mildly infected birds occurs in many infections by time.

Mycotoxicosis

- @ Growing fungi produce many byproducts. Some are toxic (mycotoxins), some to bacteria (antibiotics) and some have both effects.
- Mycotoxicosis is type of food poisoning or disease produced by toxic metabolites of certain fungi (toxins), these toxins affect poultry as well as human and animals.
- Mycotoxins in poultry caused by many fungi which attack grains before to harvest, during storage or after inclusion in finished feeds or contaminated litter.
- Many mycotoxins are stable during milling and feed storage, so toxins can be present in grains after the fungi that produced them are dead as aflatoxin.
- The most harmful toxins in poultry are aflatoxins, ochratoxins and fusariotoxins.

Factors essential for growth of fungi

- Warm temperature about 30-35°C.
- High humidity content of grains: the most problems of grains comes from corn where it contains more than 13 % moisture content.
- Other grains are also affected such as maize, wheat, millet, barley, sorghum. Also cottonseed cake, peanut cake, soybean meal, and fish meal.

Factors stimulate and increase production of toxins

- Spoilage of grains by insects (insect damage).
- Delaying harvesting grains.
- Drought.
- Poor nutrition.
- Improper storage of grains
- Unsanitary instruments used in manufacturing and distribution of foods.
- Consider that importation of grains or feeds with permissible level of mycotoxins may cause problems due to growth of fungi and more toxin production.
- Transportation of contaminated grains with mycotoxins.

Economic losses

- Increased mortalities.
- Immunosuppressive effect, vaccination failure and increase susceptibility to other disease.
- → Decrease growth rate in broilers, may lead to paleness of the color of the skin and these fattening birds are unfit for marketing.
- Decrease egg production, hatchability and fertility in adults.
- → Aflatoxicosis can influence drug effectiveness due to decrease drug binding to plasma protein.
- Hazard on human Health.

Aflatoxicosis

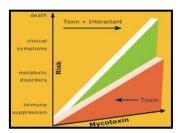
Aflatoxicosis is the most prevalent, highly toxic, carcinogenic and economically significant mycotoxins consumed by poultry.

Etiology

A. flavus, A. parasiticus, Penicillium species

- → A. flavus produce the majority of the toxin and gives it its name.
- Aflatoxins are relatively stable once formed in grains and is not degraded during normal milling and storage but sensitive to oxidizing agents such as hypochlorite.
- Aflatoxins give blue or green color to fluorescent light.
- Aflatoxins is produced mainly in feeds and in litter.

- ◆There are 4 types of aflatoxins B1, B2, G1 and G2
- B1 is the most toxic.
- →Aflatoxins are carcinogenic, so B1 has public health significance because it is considered a model hepatocarcinogen.
- ➡These toxins are rapidly excreted in the bile, urine and intestinal content, does not accumulate or persist in the body tissues and this proved by rapid improvement of the affected flock just stop the contaminated food.
- Other toxins produced with aflatoxins under field conditions may play a synergistic role.

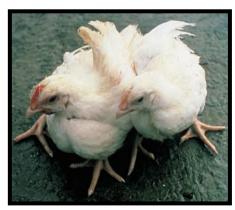


Susceptibility

- Species: ducks, turkeys, geese, pheasant, chicken and other birds in order.
- Young birds are more susceptible than adults.

Clinical signs

- ◆The most economically significant effects in growing birds are decreased growth rate and poor feed conversion because.
- Immunosuppressive effect leading to vaccination failure and decrease resistance to infectious disease.
- Passage of undigested food in the dropping.
- ◆Anemia of the infected birds due to decrease food intake and hence occur failure of normal pigmentation and decrease PCV.
- **→**Decrease egg production.
- Decrease of hatchability and fertility.
- ⇒Sometimes, lameness, ataxia, convulsions and opisthotonos.
- Mortality depends on amount and type of toxin consumed, species of birds and age.



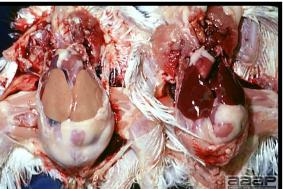


Post mortem findings

- Aflatoxins have hepatotoxic effect so the liver is swollen, pale or yellow due to deposition of fat.
- Hyperplasia of bile ducts epithelium.
- Gallbladder is enlarged.
- In the late stage fibrosis of the liver may occur.
- ➡ Enlargement of the kidneys, spleen and pancreas.
- Atrophy of bursa, thymus gland, spleen and testes.
- Hemorrhages present under the skin, on muscles and on the internal organs.
- ➡ In chronic stage ascites and hydropericardium developed.









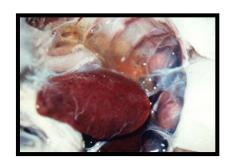












Diagnosis

- History and clinical signs.
- **■** Examination of grains or feeds grossly.
- Failure of isolation the infectious agents other than fungi.
- Persistence of deaths inspite of trials with continuous treatment.
- Gross lesions.
- Definite diagnosis depend on identification of toxin.

N.B. you must analyze more than one feed batches due to the unequal distribution of mycotoxin in the batch, and mycotoxin can be detected by:

- ►ELISA test (Monoclonal antibody detection kits).
- ▶Thin layer chromatography.
- **▶**Gas chromatography.
- ▶ High performance liquid chromatography (HPLC).

Differential diagnosis

- Aflatoxicosis must be differentiated from:
- ▶Disease causing decrease growth rate.
- ▶Disease causing decrease egg production.
- ▶ Disease causing Immunosuppression.
- ▶Disease causing Haemorrhagic syndrome.
- ▶Diseases causing enlargement of organs especially liver.

Prevention

- ■Shipments falling outside acceptable levels should be rejected.
- ■Antifungal agents as organic acids, copper sulphate, gentian violet, thiabendazde, ammonium hydroxide, must be added routinely to the feeds to prevent fungal growth in storage pins or finished feeds.
- **■**Ozone treatment of grain.

- ■Feed must be stored in well ventilated place, far away from floor and walls to avoid excess moisture (must be less than 12 %) and prefer to prepared fresh (store time must not exceed than 14 days), also well ventilation of poultry house to prevent fungal growth.
- ■Litter must stored in well ventilated places and in poultry house must preserved dry.
- Also wet places around watering troughs must be replaced by dry one to prevent fungal growth.

- Poultry farms should have two bins per house to allow rotation and cleaning.
- Thorough cleaning and disinfections of grain mills, feed store, bins, feeding troughs and pen after each batch with antifungal substances as formalin 2-3 % or copper sulphate.
- Analyze prepared feed to sure its freedom from mycotoxin.
- Use pelleted feed because during their manufacturing process cause destruction of fungal spores.
- Irradiation and fumigation of feeds may prevent mold growth.
- Regular inspection of feed bins.

Control

- **Solution** Contaminated feed must be stopped and replaced by another feed free from aflatoxin, birds recover from most mycotoxins soon after replacing contaminated food by uncontaminated one.
- Treat the birds with some antitoxic substances that having the ability to bind with aflatoxin and prevent its absorption, some of these substances can be used in the feed and the other in water as sodium calcium aluminosilicates or bentonites or zeolites.

- Because some vitamins as vitamin E and C for example counteract the toxicity of T2 toxin or ochratoxin A; Vitamins, Trace elements especially selenium, proteins especially methionine and cysteine which counteract aflatoxins and lipids must be compensated by feed formulation.
- Birds vaccinated during the clinical signs of aflatoxicosis must be revaccinated.
- Treat concurrent bacterial or parasitic infections.

Ochratoxicosis

Occurs less frequently in poultry than aflatoxicosis but is more lethal based on its acute toxicity, it is nephrotoxic, and its name derived from *Aspergillus ochraceous*.

Etiology

- → Aspergillus ochraceous and five other species of Aspergillus.
- → Penicillium species, both species produce ochratoxin which is relatively stable.
- → Ochratoxin A is the most common, and most toxic, other ochratoxins are B, C, and D.
- → Ochratoxins are rapidly excreted in bile and urine, the biological half- life in chickens was about 4 hrs. and residues persist for only 4 days or less.

Susceptibility

- > Ducks, quail, turkey, and chickens respectively.
- > young poultry are more sensitive to ochratoxin.

Mode of infection

Ingestion of contaminated feed with ochartoxin.

Clinical signs

- Acutely intoxicated birds are depressed, dehydrated and often polyuric and die due to acute renal failure.
- Survivors will be stunted due to decrease growth rate, poor food conversion, and anemia.
- There may be loss of pigmentation due to impairs utilization of dietary carotenoids.
- Poorly feathered, increase clotting time and immunodepression due to lymphocytes depletion (both cell mediated and humoral immunity decreased).
- Laying hens may delayed sexual maturity, decreased egg production, egg weight and egg shell quality.
- Wet dropping causing increased numbers of stained eggs.
- Decreased hatchability and fertility.

P.M. Lesions

- Affected **kidneys** are pale, swollen, hard and may have white urate crystals.
- White urates in the ureter cause renal failure.
- There is dehydration, hyperuricaemia and visceral urates deposition on pericardial, perihepatic and peritoneal surfaces, white urates are deposited also on the articular surfaces.
- Liver in addition to the renal lesions there is mild to moderate, pale or yellow **enlarged liver**.
- There is also mild decreased in bursa and thymus size causing immunosuppression.

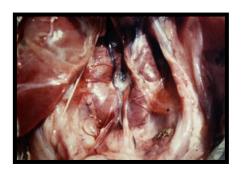
Differential diagnosis

→ From visceral and articular gout.

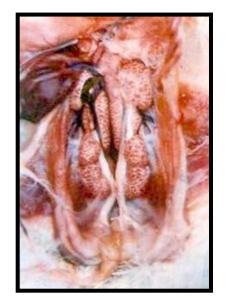


















Fusariotoxicosis

The fusarium toxins are covered by chemical groupings sush as **trichothecenes**.

Trichothecene mycotoxicosis

- Trichothecene mycotoxicosis occurs fairly in commercial poultry but does not usually cause mortality.
- Environmental conditions suitable for growth of fungus need very low temperature to grow (less than 20 C), its occurrence in our country is rare except in winter months or when feeds stored in cold places.
- There are several chemically related trichothecenes, the most known are:
 - 12,13 epoxy trichothecenes (T-2) are the most effective.
 - **Di-acetoxy-scirpenol** (DAS), **Zearalenone** (F-2)

Etiology

Trichothecene mycotoxines produced by many species of fusarium.

Signs

- ■Decrease food intake due to ulcerative stomatitis.
- ■Decrease growth rate, decrease feed efficiency, poor feathering, and anemia.
- ■Immunosuppresion due to bursal and thymus atrophy.
- ■Affected adult birds will develop oral ulcers.

P.M. lesions

- Necrosis or ulcers in the mucous membrane of the mouth, on the dorsal surface of the tongue, also on the hard palate adjacent to the beak and oropharynx .
- → Lesions is rarely present in esophagus <u>due to presence of</u> moisture and mucus.
- → Ulcer appear 3-4 days after initial toxin intake.
- → Atrophy of bursa, thymus and spleen.
- → Reddening of the gastrointestinal mucosa with visceral hemorrhages.
- → Mottling of the liver.
- → Gall bladder distention.
- → In severe cases ulcer in the intestine may present.
- → Anemia and pale bone marrow.











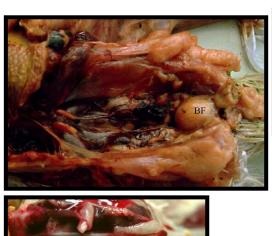
















Oral lesions of trichothecenes must be differentiated from:

- ▶Lesion of candidiasis.
- ► Lesion of vitamin A deficiency.
- Lesion of copper sulphate.
- ▶ Lesion of Quaternary ammonium disinfectants.
- Lesion caused by high concentration of fines in the feed.