MYCOTOXICOSIS

- Disease related to one or more mycotoxins
- **Mycotoxin** - "mycos" - mold, toxic metabolite
- Produced by filamentous fungi while growing on substrate such as feedstuffs and litter.
- Deleterious results are from ingestion of mycotoxin, not active infection in affected animal.
- Over 300 mycotoxins known.
- Many antibiotics are mycotoxins.
DISCOVERY AND ISOLATION

• In poultry, 1969 in England, X disease in turkey poults, killed 100,000.

• Turned out to be aflatoxin in peanut meal.

• Previous concern - food deterioration.

• Now feel the effect on production efficiency may be most costly.
MAIN MYCOTOXINS PRESENTLY CONSIDERED PATHOGENIC

1. Aflatoxin - most common.
2. Fusarium toxins (trichothecenes)
   T-2 toxin DAS, HT2, deoxynivalenol
   (Don) "Vomitoxin", Zearalenone (F-2)
   "Estrogenic toxins", Fumonisins (B₁).
3. Ochratoxin
4. Oosporein
5. Cyclopiazonic acid (CPA).
SEVERITY OF MYCOTOXICOSIS MAY VARY WIDELY—INFLUENCED BY THE FOLLOWING PARAMETERS

- Level in substrate (feed or litter)
- Age
- Sex
- Genetic complement
- Environment, temperature, etc.
- State or plane of nutrition
- State of production (laying birds)
- Presence of other disease
AFLATOXICOSIS

- Received most attention in last few years.

- Mainly due to feed (corn, milo, canola) contamination in poultry.

- Responsible for poor feed conversion and loss of egg production.
AFLATOXICOSIS

- Acute (less common) and chronic toxicity
- In chickens, ducks, and turkeys.
- Same etiology as "moldy" corn in swine.
ETIOLOGY

AFLATOXINS

- Type B₁ (most important in poultry)
- B₂, G₁, G₂, M₁*, M₂*, and others
  - *Not an avian problem

Known toxin producers:
- *Aspergillus flavus, A. parasiticus, and some species of Penicillium.*
- Substrate - corn, barley, millet, peanuts, peas, rice, sorghum, wheat, coastal hay(s), oats.
CHARACTERISTICS OF AFLATOXIN

- Ubiquitous in nature.
- Carcinogenic, teratogenic.
- Aflatoxin is the most potent mycotoxin, and is responsible for severe economic loss.
DOSE EFFECT

- 400 PPB - weight depression in 10 days to 2 weeks
- 500 PPB - kill pouls in 2 weeks
- 1 PPM = 1000 PPB
CLINICAL SIGNS

- Depressed growth and feed conversion.
- Pale and off feed with increase in mortality - common.
- Impaired immune system with increased susceptibility to other conditions.
- Reduced fertility and hatchability.
- Egg production loss depending on dose.
POSTMORTEM LESIONS

- Pale yellow fatty livers & distended gall bladder
- Ecchymotic hemorrhage in muscles & petechial hemorrhages in viscera
- Regressed bursa of Fabricius
- Congested kidneys (usually enlarged)
POSTMORTEM LESIONS (CONT.)

• Enlarged spleen & pancreas
• Hydropericardium and ascites
• Pale bone marrow
• Bile is dilute and lime to fluorescent green in color
Fatty Liver
Normal / Affected
Bruising
Hydropericardium
Ascites
IMPLICATIONS

- Some mycotoxins are carcinogenic.
- Documented synergism with coccidiosis, Salmonella, crop mycosis, nutritional deficiency, and infectious bursal disease.
- Impairment of immune ability of affected animal may occur.
- Specifically impairs protein synthesis (building block protein as well as globulins).
- Increased capillary fragility causing bruising and condemnations.
COMMENT

The target organ is the liver and the most sensitive metabolic systems are protein synthesis, lipid synthesis, and lipid transport.
DIAGNOSIS

- Extraction & quantification of toxins from feed with chromatography, monoclonal antibody technology.
- Gross pathology - swollen fatty livers - non-specific.
- Histopathology
HISTOPATHOLOGY

- Fatty changes of the liver with focal necrosis.

- Progressive bile duct proliferation with portal cirrhosis.

- Acinar degeneration of the pancreas.
Histology
Bile Duct Proliferation
• Detection of metabolites in tissue.
  – This is rarely used.
  – The half-life is extremely short, i.e. 1-2 days.
BIOLOGICAL TEST

- Feed suspect feed to 1 day old ducklings
- Age susceptibility - young more susceptible

  Susceptibility to toxin

  Ducks > Turkey > Chicken
COMMENT

- Allowed level for feedstuffs in interstate commerce is no greater than 20 PPB (Delaney Clause, FDA).
- This number was arbitrarily chosen.
  This was the level they were able to detect with technology present at that time.
PREVENTION

1. Establish an ingredient assay program. Kojic acid causes this mold to glow under a black light. But the presence of mold does not mean aflatoxin is present.

2. Reject contaminated feed or ingredients. Aflatoxin is a warm weather problem.

3. Reduce feed storage time. (Keep under 9 days) This is dependent on price and storage capacity.
4. Keep feed bins and handling equipment clean. Use chlorox, rotate bins.

5. Use low moisture ingredients and don't allow it to become damp.
TREATMENT

• Discontinue use of contaminated feed.

• Increase protein, fat, vitamin, and electrolyte level of diet. This is to oversupply the systems affected by aflatoxin.

• Fat soluble vitamins have a sparing effect.
50 related compounds from genus *Fusarium*.

E.G.: Type A - T-2, HT-2, DAS  
(Diacetoxyscirpenol)  
Type B - nivalenol, deoxynivalenol  
(Don)

*Fusaria* are cold weather molds. Contamination of overwintered wheat and stored corn, and soybeans is common.
EFFECTS

- Immunosuppression - decreased spleen size, decreased cell function, and thymus atrophy.
- Depressed WBC and RBC numbers.
- Hemorrhages in musculature and viscera.
- Oral, proventricular, ventricular lesions/ulcers.
- Decreased feed intake.
- Depressed weight gains and conversions.
- Depressed egg production.
- Poor egg shell quality.
- More egg breakage.
- Depressed hatch.
- Enteritis with necrosis.
EFFECTS (CONT.)

- Lymphoid depletion in "GALT", "BALT".
- Bone lesions like "TD", rachitic lesions.
- Neural abnormalities.
- Footpad lesions.
- Stunting.
- Depressed hemoglobin.
- Lowered grain quality due to mold contamination.
- Feed refusal factors.
- Depressed fertility.
- Anti-thiamine activity (*F. moniliforme*).
Oral lesions
Oral Lesions
Oral Lesions
DIAGNOSIS

• Detection of toxin
  – Thin layer chromatography
  – HPLC, gas chromatography
TREATMENT

• Remove offending feed.

• Supportive - Increase vitamins
  Increase protein
  Increase copper
OCHRATOXICOSIS

- A disease caused by a very potent Mycotoxin produced by *Aspergillus spp.* and *Penicillium spp.*
- Nephrotoxic in all classes of poultry.
- Most nephrotoxic mycotoxin.
ETIOLOGY

• Ochratoxin A & B

• Produced by *Aspergillus ochraceus*; *Aspergillus melleus* and *Penicillium viridicatum*.

Poor grades of corn important

This is a warm weather mold.
MORBIDITY

• Variable; depends on the age of the bird and level of ochratoxin consumed.
MORTALITY

• Variable; depends on amount of ochratoxin consumed, and the age of the bird.
CLINICAL SIGNS

- Diarrhea, urate content very high (Polyuria)
- Listlessness
- Decreased feed consumption
- Rickets
- Decreased egg production
- Decreased weights
- Increase in egg stains in caged birds
POSTMORTEM LESIONS

- Kidneys enlarged and pale - renal disease is permanent
- Reduction in bursal size due to depletion of lymphoid elements
- Suppression of hematopoietic activity in bone marrow
- Decreased bone strength in the legs
- Cachexia in advanced cases
Stained Egg
Kidney lesions
Kidney lesions
Gout
DIAGNOSIS

• Enlarged kidneys suggestive, but IBV can also cause kidney lesions.

• Feed assays conclusive.

• Histopathologic changes on H&E.
  • Histo: coagulation necrosis of proximal tubules of kidney.
TREATMENT

1. Remove suspect feed
2. Treat secondary problems
PREVENTION

1. Purchase good quality corn

2. Establish a quality control program
OOSPOREIN

• Toxic metabolite of *Chaetomium trilaterale* and other mold species found in animal feeds and cereal grains.
• Will kill chickens at 200-400 ppm of feed in 4 days.
• Uric acid secretion is impaired.
• Birds become lethargic and show lesions of visceral and articular gout.
• Broiler chickens are 4 times more susceptible than poults.
• Chickens may have pseudomembranes on the mucosa of the proventriculus lining and Gizzard erosions.
• Fairly uncommon.
The toxin cyclopiazonic acid is produced by several Aspergillus and Penicillium species when they grow on corn, mixed poultry feeds, peanuts, and other substrates.

100 ppm of CPA reduced growth by one-half and produced high mortality (60%) when fed from 1 day old to 7 wks. of age in chickens.

Levels above 50 ppm cause mucosal erosion, hyperemia and flaccid dilatation of the proventriculus on gross examination, ulcerative proventriculitis, mucosal necrosis of the gizzard, hepatic and splenic necrosis, and inflammation on histopathological examination.

Fairly uncommon.

Have increased ingesta in carcasses at processing.