Mycotoxins

Mycotoxins are secondary metabolites of fungi that are recognized as toxic to other life forms.

1.Fungal growth

a.Field fungi : grow under conditions occurring *prior to harvest.* (*Fusarium*)

b.Storage fungi : do not invade intact grain *prior to harvest. (Aspergillus & Penicillium)*

2.Characteristics of mycotoxin-induced disease

a. not transmitted among animals

b. Pharmaceutical treatment does not alter the course of disease

c. Mycotoxicosis most often presents as a uncertain, subacute or chronic condition

3.Treatment of mycotoxin-induced disease

a. For most mycotoxins, there is no specific treatment or antidote

b. Supplement with vitamins & selenium may be helpful, and provision of adequate high-quality protein

4.Prevention of mycotoxin-induced disease

a. Avoiding

b. Diluting

c. Cleaning

d. Testing

e. Drying

f. Adding (organic acids will prevent mold growth)

Aflatoxin

1.Sources : *Aspergillus flavus & A.paraciticus*

: corn, peanuts

2.Factor favoring production of aflatoxins

a. Temperature : 25-30 ◦c

b. Dryness stress

c. Grain moisture

3.Chemical characteristics

exhibit intense blue or green fluorescence under UV.

: aflatoxins B1, B2, G1 and G2

: aflatoxin M1 is a metabolites of AFB1 found in animal urine, milk or tissues.

4.Toxicokinetics

One major metabolite of AFB1 is a highly reactive electrophilic epoxide that forms covalent adducts with DNA, RNA and protein.

5.Mechanism of toxicologic damage

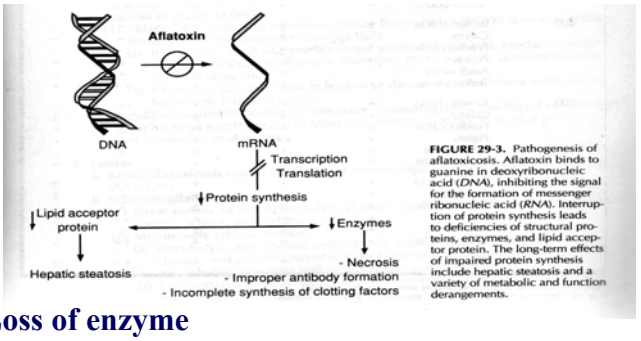

FIGURE 29-3. Pathogenesis of aflatoxicosis. Aflatoxin binds to guanine in deoxyribonucleic acid (*DNA*), inhibiting the signal for the formation of messenger ribonucleic acid (*RNA*). Interruption of protein synthesis leads to deficiencies of structural proteins, enzymes, and lipid acceptor protein. The long-term effects of impaired protein synthesis include hepatic steatosis and a variety of metabolic and function derangements.

a. Loss of enzyme

b. Lack of formation of lipid acceptor protein in liver

c. Decreased cellulose digestion, volatile fatty acid formation & proteolysis

d. Necrosis

6.Toxicity

a. Young animals are more susceptible than adult

b. Nutrition deficiency increase susceptible

7.Diagnosis

Clinical sign : decreased growth rate, reduced feed efficiency, steatorrhea, icterus, mild anemia, ascites and increased susceptibility to infectious disease.

8.Treatment & Prevention

a. Detoxification : Hydrated sodium calcium aluminosilicate (HSCAS) can absorb aflatoxins

b. Supportive : Vit.E & selenium

c. Prevention

- Mold inhibitor
- Treatment of grain with anhydrous ammonia for 10-14 days.

Zearalenone

1.Sources : *Fusarium roseum (F.graminearum)*

: corn, wheat, barley, oats

2.Factor favoring production

a. High moisture 22% - 25%

b. Alternating high and low temp. (7-21 °c)

3.Mechanism of toxicologic damage

a. binds to cytosolic receptors for estradiol-17 beta

----> initiating specific RNA synthesis

b. Function as a weak estrogen.

4.Toxicity

a. Swine are most susceptible

b. low for all effects except reproductive function.

5.Clinical sign (Swine)

a. Prepubertal female (gilts) : syndrome of hyper-estrogenism (behavioral estrus, swelling & edema of vulva, mammary gland enlargement and rectal & vaginal prolapse)

b. Mature sows : Nymphomania, Anestrus & pseudopregnancy

c. Castrated males : prepuce & nipple enlargement

d. Immature boars : reduced libido & retard testicular development ,whereas mature boars are not affected

6.Treatment

a. Detoxification : activated charcoal, Alfafa

b. Supportive

-Vaginal & rectal prolapse must be treated

- 10 mg of prostaglandin F_{2alpha}

- Bentonite added to contaminated diets.

Ergot

1.Source : *Claviceps purpurea*

: rye, barley, wheat & oats

2.Factor favoring : Warm & humid

3.Mechanism of toxic

a. potent initiators of contraction in smooth muscle

b. mimic the action of dopamine in CNS.

4.Clinical sign

a. dry gangrene, lameness, swelling of the feet & feltlocks and sharply demarcated necrosis of the feet, ears and tail

b. increased temp., pulse & respiration rate and anorexia

c. lactation does not occur

d. hyper-excitability & tremors

e. heat intolerance in cattle

E.Treatment

a. animals should be provided with a warm, clean, stress-free environment

b. Control secondary bacterial infection

c. Colostrum & milk supplement

Ochratoxin & Citrinin

1.Sources : *Aspergillus orchraceus &*
Penicillium viridicatum

2. Mechanism of toxic : target the renal proximal tubule

-Bind strongly to protein (albumin)

-Interfere with synthesis of tRNA & mRNA

-Interfere with protein synthesis

-Disrupt carbohydrate metabolism

-Increase the generation of free radical

4.Clinical sign

a. Acute : anorexia, vomiting, diarrhea, dehydration & depression

b. Subacute to chronic : weight loss, ↓ feed efficiency, polyuria, polydipsia & dehydration. Immunosupression, teratogenicity, carcinogenesis & hemorrhage

5.Treatment & prevention

a. Detoxification : Activated charcoal

b. Supportive : chronic renal insufficiency

c.Prevention :

- proper harvest and drying for storage
- Questionable grain supplies should be tested

THE END