NUTRITIONAL PROBLEMS

- Human error
- Improper formulation
- Ingredient variation
- Malabsorption
- Overproduction -- under consumption
- Feed separation - especially with calcium in chain feeders
- Ingredient antagonism
- Oxidation of ingredients
Most companies run their own analytical chemical lab tests to test the feed ingredients.

Changing genetics of the birds requires constant nutritional changes.
MOST COMMON PROBLEMS

1. Ca/P. Imbalance
   (A) Rickets
   (B) Osteomalacia

2. Sel./Vit. E deficiencies - this is still seen especially with corn from the Midwest. The soils there are selenium deficient. Therefore supplements are used.
   - Add 0.1 ppm to chicken feed
   - Add 0.2 ppm to turkey feed

This is not high enough to meet requirements which are 0.2-0.3 ppm but these levels are legal.
RICKETS AND OSTEOPOROSIS

- Soft bones in bird due to a deficiency or improper balance of Ca, P & Vitamin D$_3$.
- Chickens need Vit. D$_3$ to absorb calcium.
- Approximate proper levels:
  - Broilers and pullets: 0.8-1% Ca-0.6% P. The ratio of Ca and P is just as critical as the total amount. An excess of one results in an effective deficiency in the other.
  - Pre-lay diet: 2% Ca++
  - Layers: 3% Ca - 0.6% P
RICKETS AND OSTEOPEROSIS

• Problem usually caused by human error or miscalculation.

• Frequently seen as a sequela to the malabsorption syndrome.

• The fastest producing broilers are on the edge of rickets all the time due to increased growth with low calcium levels.
CLINICAL SIGNS

- Osteoporosis (a decrease in mineralization of structural bone)

- Usually seen in young high producing cage layers. Called “cage fatigue” - produces pathologic fractures.

- Layers go down in their cages and stop laying eggs.

- Many times birds appear to have spontaneous fractures of wings and legs, ribs, vertebrae, and other bones.

- Weight bearing bones are brittle and easily broken.

- Poor producing individuals not affected.

- This has almost been eliminated with the use of pre-lay diets.
Downer bird
Broken Wing
CLINICAL SIGNS

Rickets

- Poultry 3 to 6 weeks of age often affected.
- Crippled birds that squat.
- Swollen extremities of long bones - lesions in growth plate of broilers.
Soft bones
Soft, bent bones
POSTMORTEM LESIONS

- If you kill the bird by pulling its head you may also break its legs.

- Bones break quite easily.

- Ova may be beginning to show regression.

- Rib joints may fold - rachetic rosary.

- Malformation of the sternum.
Soft bones
Soft beak
Radiograph

Ricketts Normal
Bent Keel
Enlarged costochondral junctions
Normal
Calcium
Phosphorus
Tibial dyschondroplasia
TREATMENT AND PREVENTION

• Maintain proper amounts and proper ratio of Ca, P, and Vitamin D₃.

• Top dress feed with dicalcium phosphate.
Caged layers may be on NRC recommended Ca, Phos. & Vit. D$_3$ and still get “cage fatigue.”

Usually this is a low Phos. problem caused by chickens being denied access to their own droppings.

Increasing the Phos. to 0.8% will alleviate this problem.
VITAMIN E DEFICIENCY

Encephalomalacia
(Crazy chick syndrome)

• Fat soluble vitamin, easily destroyed by oxidation.
• Required for proper maintenance of brain tissue, particularly in growing birds.
• Required for proper muscle development in gizzard of turkeys; selenium is involved in Vit. E metabolism.
ETIOLOGY

• Oxidative destruction of fat (rancid).

• Vegetable oils are abundant in alpha tocopheral.

• Vitamin E required for proper embryo development and proper brain development in young chickens.
CLINICAL SIGNS

• Usually occurs between 15th and 30th day of age. Has been seen up to 56th day. Usually occurs at 3 weeks of age.
• Chicks appear depressed.
• Assume fixed posture. Motor paralysis due to decreased cerebellar function.
• Intense excitement when stimulated. Birds flap wings and sit up on their tails and fall backward.
Paralysis & typical posture
Paralysis
POSTMORTEM LESIONS

Gross

- Chicks: hemorrhage and edema in cerebellum & soft area in any part of the brain
- Poult's: require both Vit. E & selenium deficiency to have encephalomalacia.
- Myopathy in chickens rare because cystine and selenium deficiency along with Vitamin E deficiency required.
Cerebellum
Cerebellum
Normal vs. affected
POSTMORTEM LESIONS

**HISTOPATH.** - Encephalomalacia. Must differentiate from A.E. especially in chicks.

**DIAGNOSIS** - Histopathology - typical microscopic lesions of encephalomalacia: necrosis, hemorrhage.
TREATMENT

• Addition of 50 mg/lb or 10,000 units of Vit. E/ton of feed.

• For best results, make sure selenium is added to the feed at 0.1 ppm for chickens and 0.2 ppm for turkeys.
PREVENTION

• Do not use rancid oils or feed products in poultry feed.

• Do not store feed over 3 to 4 weeks. Make sure breeders are getting proper Vit. E in diet.

• Use anti-oxidants such as Santoquin or BHT in all poultry feeds.
COMMENT

- Vitamin E is not required for egg production or fertility in male birds.

- But Vitamin E for the 1st 7 days of life comes from the yolk.
SELENIUM

• Minute amounts of this trace mineral are needed for proper metabolism. Closely involved with Vit. E metabolism.

• FDA passed regulations to allow for addition of the pure form of this mineral to feeds.
ETIOLOGY

• Feeds (grain) from soils deficient in selenium.

• Diets low in animal protein - especially fish-meal.

• Certain sulfur containing amino acids may be tied to selenium deficiency. The amino acids are probably abundant in fish-meal.

• Deficiency interferes with peroxidase production.
CLINICAL SIGNS

• First notice pale staggering birds with deformed wing feather, stargazers.
• Usually seen in 3 to 8 wk. old birds.
• Fluid noted over keel bone and under wing, and intermandibular edema.
• Chicks with encephalomalacia may also have selenium deficiency.
• Poults will be unthrifty with above normal mortality.
Submandibular edema
Weeping skin
Weeping skin
POSTMORTEM LESIONS

- Subcutaneous edema - exudative diathesis.
- Areas of weeping dermatitis with scabbing - usually caused by secondary bacterial invasion.
- White muscle streaks in gizzards of poults (rare in chickens - require deficiency of selenium, Vit. E, & cystine).
Subcutaneous exudate
SQ exudate
SQ exudate
SQ exudate
Pale gizzard
Pale gizzard
COMMENT

- This condition was misdiagnosed when it was first observed. It was thought to be a subcutaneous clostridium infection.

- Clostridium can be cultured frequently, but probably is a secondary invader.
DIAGNOSIS

- Depends on response to addition of selenium along with symptoms and lesions. Affected flocks usually respond to treatment in about 3 days.

- Selenium toxicity in breeders will cause blind chicks with crooked beaks.
PREVENTION AND TREATMENT

- Selenium at 0.1 ppm for chickens up to 16 wks. of age and 0.2 ppm in turkeys.

- Cannot be used in chickens that lay eggs for human consumption.
RIBOFLAVIN - VIT. B$_2$

- **Chicks & poultts** - early mortality, poor growth & curled toe paralysis.

- **Breeders** - decreased egg production, high embryonic mortality, decreased hatchability.

- **Post mortem lesion** - pronounced swelling of sciatic nerves. These chicks will be too young to have Marek’s disease.
Curled tow paralysis
Curled toe paralysis
Curled toe paralysis
Enlarged sciatic nerve
BIOTIN

- Usually seen as dermatitis of the bottom of the feet in turkeys.

- A deficiency prevents the healing of sores. Supplementation costs about $0.35/ton.
Sore footed turkeys
Foot pad dermatitis
Foot pad dermatitis
Foot pad dermatitis
FOLIC ACID

- Occasionally seen in turkeys. They are affected by poor growth, poor feathering, anemia & perosis (slipped tendon).

- Early signs may be seen in the form of neck extension, as birds are too weak to lift their heads.
Rough feathers
Perosis
Neck extension
VITAMIN A

• May appear as pustule-like lesions in the pharynx and esophagus.
Lesions in pharynx & esophagus
Esophageal lesions
Esophageal lesions