Nutritional Disorders in Poultry

Nutritional disorders in poultry may be evaluated in 3 groups

1- Resulted from feedstuffs

*Physical form of feeds
*Antinutrional factors in feeds
*Microbial contamination of feeds

2- Resulted from composition of the diet
* Energy or nutrient surplus or deficiencies

3- Resulted from more than one nutrient surplus or deficiencies or another factors

1- Disorders resulted from Feeds

- A- Disorders resulted from physical form of the feeds:
- Cereal proteins, known as gluten, disperse starch molecules. If cereals, (especially wheat that is rich in gluten) are grinded too much (fine feed) and glutens combine with water it will make a mass, like concurements, around the beak and under the lingue of the bird. This mass make difficult to intake feed or water for the bird.
- Fine wheat meal should not be used in poultry diets.
- Too much grit, and fine or fibrous feed consumption results in ingestion in stomach, softness or hardness in gizzard. Fibrous-bony feeds leads to perforation in the crop

B-Disorders related to antinutritional factors in feedstuffs

- **Rye:** Ergotamine increases blood pressure by occuring arterial contraction
- **Milo**: tannin decrease feed consumption, depress in growth and constipation
- **Barley:** Barley contain 4-8 % β-glucan. The main problem of these β-glucans is the bird's inability to digest the structure, resulting in the formation of a more viscous digesta. This increased viscosity slows the rate of mixing with digestive enzymes and also adversely affects the transport of digested nutrients to the absorbtive mucosal surface. Adding synthetic **β-glucanase enzymes** to diets containing more than 15-20% barley seems to resolve many of these problems.

- **Vicia:** Vicine or vicianine, glucoside with cyanic acid
- **Soybean Meal:** Soybeans contain a number of toxins for poultry, the most problematic being trypsin inhibitor. The trypsin inhibitors will disrupt protein digestion and their presence is characyerized by compensatory hypertrophy of the pancreas. Apart from reduced growth rate and egg production, presence of inhibitors diagnosed by a 50-100% increase in size of pancreas. Heat treatment at 110-120C is adequate to destroy trypsin inhibitor and other toxins such as hemaglutinins, urease, guatrogenic, anticoagulan, allergic and ostrogenic substances. Heat sensitivity characteristics of urease similar to those of trypsin inhibitors and urease levels are much easier to measure. Residual urease in soybean meal has therefore become standard in quality control programs.
- Urease is assesses in terms of change in pH during assay, where acceptance values range between 0.05- and 0.15.

- Cottonseed meal: Cottonseed contains gossypol being a yellow polyphenolic pigment. In most meals, the gossypol content will be around 1%, although 0.1% will be free gossypol. The remaining bound gossypol is fairly inert, although binding can have occured with lysine during processing, making both the gossypol and lysine unavailable to the bird. Characteristically the gossypol causes a green-brown-black discolorisation in the yolk depending upon gossypol level and duration of egg storage. Gossypol will also depress growth by impeding vitamin A, Ca, Fe and amino acid utilisation.

- Gossypol does complex with iron, and this activity can be used to effectively detoxfy the meal. Adding iron at a 1:1 ration in relation to free gossypol greatly increases the dietary inclusion rate possible in broiler diet.

- Canola meal: While canola was derived from varieties of rapeseed, its composition has been altered through genetic selection. The level of goitrogens and eurisic acid, two of the more detrimental constituents of the original rapeseed cultivars, have been markedly reduced. Euricic acid levels are now negligible. Canola still has enough goitrogen activity to result in measurable increase in thyroid weight, although this does not appear to be a problem affecting performance of poultry. The tannin and sinapine levels in canola can also be relatively high.
- There are several reports which suggest that increased leg problems resulting from feeding canola may be due to its having a different minearl balance than soybean meal. Canola is also high in phytic acid and so the high level of this compound may be sequestering zinc and this affects bone development. There have been reports suggesting that high levels of sulfur in canola meal may be responsible for some of the leg problems and reduced feed intake

- Flaxseed (Linseed) meal:Flaxseed contains a number of antinutrients including mucilage, trypsin inhibitor, cyanogenic glycosides and phytic acid. Mucilage contributes to more viscous excreta.

C- Disorders resulted from the feeds contaminated by microorganism

- Feeds may be contaminated by microorganism
- in the field,
- harvesting
- storage
- İn the plant

Mould : < 1000 /g of feed

Bacteria: < 10.000/g of feed

Microorganism in feeds or their toxins may cause diseases

There are two important diseases resulted from microorganism in feeds: Candidiasis and Mycotoxicosis

1- Candidiasis

Candida albicans, found normally in intestinal flora, will cause this disease

- Candida albicans don't transfer by contact from animal to animal. Generally it is spread by drinking water and feed.
- No treatment has been shown to be universally effective in controlling this disease.
- Gentian violet, administared in the feed at a concentration of 8 ppm suppress the growth of C. albicans
- Preventative measures include cleanliness and disinfection of all environments. Well ventilating housing to avoid moist litter assists in avoiding candidiasis.
- Antibiotic terapy should be discontinued if candidiasis is observed. Long term antibiotic therapy leads to colonization

2-Mycotoxicosis

- Mycotoxicoses are defined as those intoxicants that result in animals from the consumption of feedstuffs contaminated by one or more poisons of fungal origin.
- These fungal poisons are collectively referred to as mycotoxins.
- The most common toxins produced by mold are aflatoxin (Aspergillus flavus, Aspergillus parasiticus) , ocratoxin (Aspergillus ochraceus), T-2 toxin - tricotescene - (Fsarium tricinctum), F-2 toxin - zearalenon - (Fusarium roseum) and sitrinin (Penicillum citrinin).

Aflatoxin

- Produced by *Aspergillus flavus* mold, aflatoxin is one of the most potent carsinogens known.
- Present in cereals in ppb levels, acute toxicity will occur at 1.2 ppm.
- Aflatoxin B1 is the most common form of the toxin – the toxin produce a blue color when exposed to ultraviolet light.
- According to feed law the highest level of aflatoxin in compound feed must be 50 ppb (0.05mg/kg) and 20 ppb (0.02mg/kg) in poultry feed.
- Aflatoxin is found in most cereals-corn and milo, some meal such as groundnut meal, cottonseed meal, soybeanmeal are the most common hosts.

- Aspergilus growth, as with any mold, is greatly reduced when corn or milo moisture levels are less than 15%.
- Symptoms change according to dose and duration of aflatoxin consumed.
- Death is inevitable in acute conditions. In chronic conditions mortality rate is not high. There seems to be retardation of growth, reduction in feed efficiency and egg production, thickness in egg shell, decrease in egg pigmentation, accumulation of fat in liver and cancer.

- Aflatoxin is a potent hepatotoxin, and so varying degrees of liver breakdown occur.
- As toxicity develops, normal liver function declines and reduced growth rate is quickly followed by death.
- There also seems to be a nutrient interaction, because toxicity is more severe when diets are low in either CP or methionine, riboflavin, folic acid or Vitamin D3.

- There is no treatment for acute aflatoxicosis
- There are a number of effective preventative measures
- Firstly, feeds contaminated with aflatoxin must be changed. New feeds should have higher energy and protein level as well as fat solubles vitamins.
- Adding toxin binding agents to the feeds seems to reduce the adverse effect of aflatoxins.
- There are some toxin binders:
- *Aluminosilicates (10-15kg/tonne of hydrated Na-Ca aluminosilicate)
- *Bentonite clays
- *Yeast cell walls

2. Disorders resulted from energy and nutrient surplus or deficiencies

- **-Energy deficiency: As the energy decrease in the diet, birds consume more feed to meet their enegy requirements.**
- **As long as energy levels meet the maintanence of bird:**
- **It is seen only depression in growth, reduction fat reserve in the body. But,**
- **İf the energy levels too low to meet maintanence of bird then it is observed;**
- **-Weight loss will occure.**
- **_Bird will use glucogen in its body after then use its fat reserves and protein to meet energy requirement. As a result of this it is inavitable that bird will Die**

- **Energy Surplus:** As the energy levels increases the bird reduces its feed intake
- As long as the balance between energy and protein is stay stable and the diet contain enough vitamin and mineral_____no problem on the health or performance of the bird.
- Only energy levels increase but not other nutrients:
- Decreased feed intake consequently less protein consumption____Decreased growth and production

  Increased fat reserves

  Specific disorders related to vitamin or mineral deficieny.

- **Nutrient Deficiency**
- **Water: Water consumption twofold or threefold of feed intake**
- **Water Deficiency: Reduced digestibility of the feed.**
- **During the long term deficiency:**

  Nephroz
  Polisitemi
  Dried skin and other dehydration symptoms
  Reduced egg size and shell weight
  Depressed growth rate and feed efficiency

  Water Surplus: is not common in practice.

  Pendulous crop: it is oAflatoxin is found in most cereals-corn and milo are the most common hosts.

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ccure because of too much water intake during long term hot weather. The crop is enlarged and pendulous and the contents are not impacted, digestion of the feed reduces, because of inflamation in the lungs mortality reaches 50%.

It has been suggested that there is a genetic predisposition to the condition in some birds.

- **Carbohydrate:** No specific disorder
- **Lipids:**
- Fats and Oils: Energy sources in the body. Sufficient energy level in diet encouregе egg production, hatchability and feed efficiency
- **Insufficiency of Esential fatty acids:**

  Linoleic, linolenic and arachidonic acids are essential for poultry. Minimum linoleic acid level is 1% for chicken and laying hen and 2% for breeder diets.
- Deppressed in growth in chicks. Vegetable oils are a good sources of linoleic acid.
- In chickens fed diet insufficient in essential fatty acid

  mortality rate will high within 10-12 weeks,

  In laying hens, egg production, egg size and hatchability decrease, embrionic mortality and susceptibility to the respiratory diseases will increase. Liver will also grow and fat accumulation increase.

- **Rancidity in Lipids:** Hydrolytic or oxidative rancidity will occure in fats or oils. Rancidity also leads to destruction in fat soluble vitamins.
- Symptoms:
- Feed refusal
- Growth depression in the chicks
- Weakness
- Anemia
- Difficulty in walking-moving (peguin walking)
- Mortality reaches 50 %
- Determine: Peroxide count: 3-5mEq/kg in poultry feed. 6-10mEq/kg acceptable

  Higher than 10mEq/kg not acceptable

  20mEq/kg in feedgrade
- Rancidity in lipids can be prevent by adding antioxidan to the feeds.

- Wet Litter:
- It is occure especially during winter and fall.
- **Causes and Symptoms are very complex**
- **There appears asites, diharhoe decrease in productivity, fatty liver as well as wet litter.**
- **Causes: Rancid fat or oils**
- **Any factor that increases water consumption will increase the likelihood of wet litter**
- **Drinking water quality**
- **Mineral level of feed (Na, K, Mg)**
- **Protein in excess,leads to increased water intake to allow the excreation of higher uric acid levels.**
- **Symptoms**
- **Wet litter, diarrhoea, asites, degeneration in the liver**

- Protein:
- All essential amino acids must be included and balanced in the diet. Protein quality is important as well as protein quantity in poultry diet.
- Protein quality is related to digestibility and amino acid content of protein.
- **Protein Deficiency:** Both quantity and quality of protein is important. In protein defficincy:
- Growth depression, weight loss, small egg size, decrease in egg production and hatchability, increased fat reserves in the body from excessive energy and amino acids (Because they can not be used for production).
- **Protein Surplus:** slightly decrease in fat reserves and growth rate, increase in blood uric acid. Excessive water intake to excreate uric acid from body

- **Minerals:**
- **Rickets and Osteomalasia**
- **Causes: inadequate dietary Ca, P or Vit D3; or Ca:P imbalance**
- **Rachitic deformities develop especially in the legs, producing painful, hard joint swelling and lameness, abnormality being most clearly seen in the structure of the proximal tibiotarsus.**
- **The bones, beak and claws bocome soft and pliable. Growth is retarded, feather development is poor.**
- **In the laying bird, egg production decreases, with thin or soft shelled eggs and reduced hatchability.**

CANNIBALISM

- **Cannibalism is a problem that is associated with large poultry flocks kept in restricted cage. This can result in significant mortality within the flock when a wound is generated and it will also cause a decrease in egg production**

What Are The Causes of Cannibalism In Poultry Flocks?

The causes of this vice are not well understood, but the onset of cannibalism has been attributed to a number of things which are outlined below:

***The problem may simply arise because of the normal pecking behavior of this type of animal when searching for food or exploring an environment.**

The birds are kept in barren, crowded conditions and may have little else to peck besides their pen mates. Once one hen has picked up this technique other hens, observing the behavior, will learn from the initial pecker and a serious episode will develop.

*A lack or a deficiency of nutrients in a poultry ration may lead to cannibalism in the flock. Imbalance of the diet, usually protein or sodium level of the ration is involved in the outbreak

*Deficiencies can also be caused by insufficient feeding and water space.

*An abrupt change in the palatability or form of a flock's ration may also be a contributing factor in the onset of cannibalism as the birds might impulsively seek alternative sources of food.

*Poor ventilation, high temperature, low humidity, excessive population density, and excess illumination are all factors in the flock's environment that may precipitate an outbreak of this vice, especially with the lighter breeds.

During egg laying the cloaca may become damaged and distended especially with the passage of large eggs and this protrusion of the vent may be an attractant to other birds due to its stark color difference against the white body.

How Can An Outbreak Of Cannibalism Be Prevented In a Flock?

- The onset of this vice can be prevented by paying particular attention to the

- **dietary factors** (protein, sodium, and palatability),
- **environmental factors** (ventilation, temperature, humidity, population density, and lighting)
- **feeding and water space** should be available for each bird.
- **light intensity** should be reduced, perhaps by changing to a light which is of a red hue.
- **beak trimming** **t**he most common and cost effective mode of prevention is the use of beak trimming. Beak trimming is usually done at 4-6 weeks of age

Several studies report that the risk of cannibalism is lower when hens are fed a **mash diet** which takes longer to eat, than when fed a diet consisting of pellets, which are quickly consumed.

An increase in the time spent foraging reduces the incidence of cannibalism. This has practical value, as poultry farmers could potentially reduce or prevent cannibalistic behavior among their flocks simply by increasing the time birds spend feeding.

- It should be remembered when approaching a problem of cannibalism in the poultry industry that the best method of control is to prevent it from starting at all, since **once it has begun it will be very difficult to stop.**

- **Outbreaks of feather pecking and cannibalism in laying hens remain a serious problem in the egg industry, in terms of both welfare and economics. In developing strategies to reduce cannibalism among laying hens, it is important to understand the causation of cannibalistic behavior.**

GOUT

Uric acid is one of the end breakdown products of dietary protein in birds and other animals. The uric acid is removed from the blood by the kidneys and excreted in the urine.

Gout can occur if the level of uric acid in the blood exceeds the ability of the kidneys to remove it. In articular or synovial gout, the uric acid crystallizes in the joints, ligaments, and tendon sheaths.

In visceral gout, uric acid deposits are found in the liver, spleen, pericardial sac (the covering of the heart), kidneys, and air sacs. When the uric acid crystallizes in tissues it forms small, white nodules called "tophi."

There are two types of gout.

In primary gout, the high uric acid level is a result of an abnormal breakdown of protein. Primary gout is thought to be hereditary in humans.

In secondary gout, the high level is due to the inability of the kidneys to adequately excrete the uric acid. This can be caused by medications, *chronic* diseases, kidney disease, overeating, improper diet (high protein, and possibly high Vitamin D or low Vitamin A), poor blood circulation, inactivity, decreased water intake or chronic dehydration, some infections, and other environmental factors

How is gout treated?

- Birds with gout will be placed **on a low protein diet.**
- **Vitamin A** may be given to birds
- **Proper hydration** is necessary and fluids may need to be administered.
- **Medications** may be used, but the exact dosage and safety of drugs in birds have not been determined.

  Most birds will need to be treated for life or the condition will quickly reappear if therapy is discontinued.

  If arthritis from gout is severe, it is possible to surgically remove the uric acid crystals from the joint. Often the damage to the joints or organs is irreversible.
- **Pain medications** such as aspirin may be given.
- **The prognosis for a bird with gout is generally poor.**

Caged Layer Fatigue

- **Cage layer fatigue is a condition that is unique to hens that are in a high state of egg production, primarily caged layer hens.**

Causes of CLF

The cause of the condition is not known exactly. It is thought to be associated in an imbalance of minerals/electrolytes in the body.

***High energy density and high enviromental tempruture, which reduce feed intake of bird**

***Infestation with parasites and management mistakes may play a role for arising the condition**

*Genetic sensitivity

*Failure to retention of phosphourus from kidney which is damaged because of viral diseases

*It is not found there is a relationship between CLF and inorganic P level of ration or high egg production.

*In layers under thirty weeks of age, the cause is usually a temporary calcium deficiency when egg production reaches 80% or higher. If intake of calcium does not satisfy the need for egg production, the hen will remove calcium stored in the bones. Ultimately, osteoporosis develops, bones become soft and hens are subject to bone fractures. Crippled and unable to stand, the hen suffers from the caged fatigue symptoms.

- **Many hens show spontaneous recovery if removed from the cages and allowed to walk normally on the floor.**

  This indicates that a lack of exercise may be a partial cause. Cage layer fatigue is more prevalent in single-hen cages than in multiple-hen cages. When two or more hens are caged together, they get more exercise because of competition for feed and water.

- Supplementation of the diet with phosphate, calcium and vitamin D3 is usually helpful.
- Adding calcium to young birds will often help the condition.
- In older hens, calcium deficiency is less likely than phosphorus or vitamin D3 deficiencies. Adding a vitamin/electrolyte supplement to drinking water is recommended in any age bird suffering from this condition.

Fatty Liver Syndrome

This problem is commonly referred to as "**Fatty Liver Hemorrhagic Syndrome**." It results when large amounts of fat is deposited in the hen's liver and abdomen.

- Fatty liver syndrome is a condition that affects either hens at the begining of laying period or broilers at 2-4 weeks of age.

Causes of Fatty Liver

- **Hereditary:** Hereditary tendencies vary among various strains of egg production stock, but heredity is not the entire cause for this malady. It is thougt to be Leghorn breeds more susceptible.

- **Diet**
- **The basic cause is thought to be excessive dietary energy intake.**

  When laying hens are fed diets containing high levels of dietary energy the hens tend to deposit excess energy as fat deposits in their bodies, especially the liver.
- **The problem is more common when feeds containing high levels of corn or other high energy ingredients is fed.**
- **Biotin deficiency may be effective on the condition**

- **Climate:** It is occure more often during spring or summer.
- **Raise:**Laying hens housed in cages are most often affected since they are less able to get sufficient exercise and dispose of the extra dietary energy.
- **Toxication:** Elementary P, carbontetrachlorure toxication and micotoxines may cause fat degeneration in liver.

- **Reduced egg production and size are the most common symptoms of fatty liver. Egg production is reduced from 75-85% to 45-55% within a week.**

- **The condition is most often seen in birds that appear to be healthy and in a state of high egg production. Non-laying hens will not eat as much of the high-energy feed and therefore are not affected as much as high producing hens.**

- **Mortality varies considerably among flocks but can become excessive in some cases. Lesions include accumulation of large amount of abdominal fat; enlarged, easily damaged liver and presence of blood clots that indicate that hemorrhages have occurred prior to death.**

- **The primary treatment for this condition requires an alteration of the diet or amount of dietary energy consumed. Replacement of some of the corn in the diet with lower energy feedstuffs like wheat bran can provide a lower energy diet. If a complete layer ration is being fed, addition of vitamins can be of benefit. If grains are the primary feedstuff, it is suggested that the birds be switched to a complete layer diet. Control of body fat is the only successful remedy for this condition and is best accomplished by regulation and reduction of total energy intake.**

- To prevent excessive fat accumulation
- Choline+methionine+Vit B12
- Balanced diet in Se and Vit E may prevent cell degeneration and bleeding
- Biotin and cholin addition to the drinking water

Acidosis-Alkalosis

- Biologic reactions will occure at an optimum pH. Some mechanisms regulate this optimum pH.
- 1-Diluation
- 2-Buffer systems
- 3-Respiration, CO_2 loss
- 4-Renal mechanism

- Acidosis-alkalosis arise when bicarbonate levels increase or decrease in blood and it is compansated by respiration.
- Acidosis and alkalosis may be metabolic or respiratoric.
- In primer bicarbonate (HCO_3) insufficiency
- ⟶ Metabolic acidosis
- In primer carbonic acid (H_2CO_3) surplus
- ⟶ Respiratoric acidosis
- Metabolic and respiratoric alkalosis will occure in the reverse conditions.

- As birds pant, they tend to lose more CO2 and so changes in acid-base balance can quickly develop. With mild to severe alkalosis, blood pH may change from 7.2 through 7.5 to 7.7 in extreme conditions. This change in blood pH, together with loss of bicarbonate ions can influence eggshell quality and general bird health and metabolism.

- Once an egg come into uterus acidity will start to increase and reach maximum level at 22 hours. During this time bicarbonate levels decrease by 30%.
- Respiratory center is stimulated and excess CO_2 is removed by respiration. This situation will be partly compansated with reduction of CO_2 pressure at the rate of 15%.

- Shell formation normally induces a renal acidosis related to the respiration of filtered bicarbonate. At the same time, shell secretion induces a metabolic acidosis because the formation of insoluble $CaCO_3$ from HCO_3 and Ca^{2+} involves the liberation of H^- release would induce very acidic and physiologically destructive conditions,and be necessarily balanced by the bicarbonate buffer system in the fluid of uterus.

- Severe electrolyte imbalance can be prevented by considering the ratio of cation:anion in diet formulations.
- Electrolyte balance is usually a consideration of Na+K-Cl in the diet.
- Electrolyte balance is usually expressed in terms of mEq of the various electrolytes, and for an individual electrolyte this is calculated as Mwt/1000

- For example:
- A diet containing 0.17%Na, 0.80%K and 0.22%Cl
- Electrolyte balance of the diet:
- Mwt mEq
- Na: 23 23 mg/kg 1700/23=73.9 mEq
- K: 39.1 39.1 mg/kg 8000/39.1=204.6 mEq
- Cl: 35.5 35.5 mg/kg 2200/35.5=62mEq

- Overal diet balance:
- Na+K-Cl= 73.9+204.6-62=216.5mEq
- A balance of around 250 mEq/kg is usual

- While a mild metabolic acidosis is normal during eggshell formation a more severe situation leads to reduced shell production because of intense competition for HCO3 as a buffer rather than shell formation.

Gizzard Erosion

- Gizzard erosion is a condition, usually of broiler chickens, in which the lining of the gizzard is erroded and darkened by crater-like lesions. Affected birds have signs ranging from small localized cracks in the gizzard lining, through to severe erosion and hemorrhage.
- Causes of GE:
- Bacteria (erisipelas)
- Viruses (AI, Gumboro)
- Mycotoxins
- Non-infectious reasons (hemorragic syndrome, giserosine and histamine in fish meal, physical form of feed)
- Some vitamin-mineral (Vitamin E and B6, Zn, Cu, Se, Pb, Ar, Hg) defficiencies
- Yeast (candida albicans)
- Parasites (Nematode, gizzard worm)

- Gizzard erosion was initially thought to be associated with histamine levels in fish meal. Fish meals contain histamine, and following microbial degredation during pre-cooking storage, bacteria possessing histidine decarboxylase will convert variable quantities from histidine to histamine. Histamine has the effect of stimulating excessive acid production by the proventriculus, and it is this acid environment that initiates breakdown of gizzard lining. A product known as gizzerosine has been isolated from fish meal, and this has histamine-type properties in terms of stimulating acid secrection.

- Gizzerosine is formed by heating histidine and a protein during manufacture of fish meal. Gizzerosine is almost 10x as potent as is histamine in stimulating proventricular acid production and some 300x more potent in causing gizzard erosion.
- Because the mode of action of gizzerosine is via acid production and a change in gizzard pH, there have been attempts at adding buffers to prevent the problem. For example adding sodium bicarbonate has been reported to lessen the severty of gizzard erosion. (10kg/ton change only 0.3)

Chondrodystrophy, Slipped Tendon or Perosis

- A syndrome characterized with
- Short legs
- Lameness
- Distortion of hock
- Slipping of Achilles tendon (or perosis)
- Malposition of leg distal to hock

- The fact that leg problems are more prevalent in broilers (and turkey) than egg-type birds, has led to the speculation of growth rate and/or body weight as causative factors.

- General nutritional factors can influence leg problems. For example:
- Energy restriction in the first few weeks,
- Deficiency of manganese, choline, zinc, either singly or in combination (although deficiencies of pyridoxine, biotin, folic acid, niacin may also be involved)
  Diets high in protein can interfere with folic acid metabolism and in so doing, increase the incidence of leg problem
- Mycotoxins
- High Chloride levels
- Canola meal (having a different mineral balance and high phytic acid)