Avian Nutrition in Tropics

Basic & Applied Nutritional Aspects



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FOREWARD

PREFACE

This book is intended to serve as a reference and as a textbook for advanced courses in avian nutrition. It gives information on the chemical profile, digestion & metabolism of nutrients. A major emphasis is placed on antinutritional factors, feed additives, feed processing and ration formulation. Chapters are included on feeding management of different species of poultry. The text is well illustrated with tables and photographs. In addition it includes nutrient requirements for different classes and species of poultry.

I strongly believe that this book will bring up comfort and practical help for those individuals engaged in the poultry industry. I still look forward to suggestions, comments and healthy criticism for the forth-coming edition.

Javed I. Sultan

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PART ONE

Chapter 1

Introduction

In a successful commercial poultry operation, balanced and cost effective feed formulation is the key to success as feed price accounts up to 77% of total production cost. A very precise feed formulation is required for the provision of long list of nutrients. There are numbers of considerations (Figure 1), which a nutritionist must emphasize while formulating a ration for a flock.

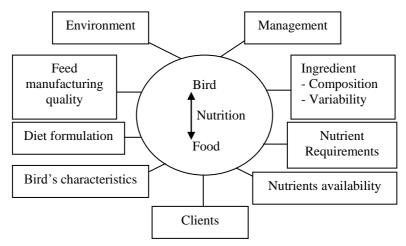


Figure 1. Factors affecting avian nutrition

The diet of avian species contains hundreds if not thousands of different molecules, but the bulk of the ingested nutrients are in the form of huge macro-molecules that cannot be absorbed into blood without first being reduced to much simpler and smaller forms; even sucrose cannot be absorbed without first being enzymatically ripped apart into glucose and fructose. Chickens use feed for two main purposes, firstly, as an energy source to maintain body temperature and to carry out activities such as breathing, walking, eating and digesting the feed, and secondly as building material for the development of bone, flesh, feathers and eggs.

Any chemical substance that provides nourishment to the body is known as nutrient. There are six basic classes of nutrients, each of which serves a specific need. These include

- > Water
- > Carbohydrates
- ➢ Proteins,
- \succ Lipids,
- \blacktriangleright Minerals and
- ➢ Vitamins.

They will be discussed in detail in forthcoming chapters.

Water

Water accounts for 55% to 80% of the body weight of a chicken, about 79% of the body of chick and 65% of the egg (Swick, 1998). About 70% is inside the cells and 30% is in fluid surrounding the cells and in blood. As the bird gets older, the percentage changes, but not the need for water. A chick needs a constant supply of fresh and clean water to stay healthy. It can't drink a lot of water at one time; therefore, it has to drink often. However it is not considered in ration formulation because it does not have any calorific value but it is most essential nutrient as bird can live for weeks or months without other nutrients, but will die within days without water. The body has access to water from 3 sources.

- 1. Drinking water
- 2. Water in feeds
- 3. Metabolic water

1. Drinking water:

The need for water as a nutrient should not be underestimated. It is not necessary only to make water available but quality should also be maintained. Quality, as dictated by number of factors (pollutants, minerals, pH etc.), is as important as availability. A particular problem of drinking water is salinity. Dissolved salts can affect the intake of water. The levels of different salts can also effect production. There are several factors affecting water intake, includes, climatic conditions, species and physiological status, the amount of water required by the various classes of different avian species (/100 birds) are given in table 1.

2. Water in feeds:

It is the water available from feeds. Usually the water content of finished feeds varies from 8-15%. In Pakistan it is usually avoided to give feeds to chicken soaked in water. But the wet feeds are still practiced in many parts of the world. These types of feeds are usually provided to psittacines and pet birds.

3. Metabolic water:

It is the chemically bound water, which is released during metabolic processes. This amount varies with type of nutrient e.g. 0.6g water is released per gram of carbohydrates consumed as compared to 0.4 and 1g water per gram of protein and fat consumed, respectively. However there are some losses of water in the oxidation of both proteins and fats.

Water is used for the excretion of nitrogen produced in the deamination process of proteins thus reducing the net availability of water. The oxidation of fats requires increased respiration. Water is lost from the lungs during this increased respiration and thus net yield of water produced from fat is less than that from the oxidation of carbohydrates.

| Species | Condition | Age | Water quantity |
|----------|-----------|------------|----------------|
| | | (weeks) | (L/day) |
| Chickens | | 1-3 | 2-10 |
| | | 3-6 | 6-14 |
| | | 6-10 | 12-18 |
| | | 9-13 | 16-22 |
| | Mature | Non-laying | 20+ |
| | Mature | Laying | 20-32 |
| Turkeys | | 1-3 | 4.5-12 |
| | | 4-7 | 16-34 |
| | | 9-13 | 35-57 |
| | | 15-19 | 64-66 |
| | | 21-26 | 52-60 |

 Table 1. Water requirement for various classes/species of poultry (per 100 birds)

(Ensminger et al., 1990)

Functions of water:

Water is a solvent for number of chemicals, which can subsequently be detected by taste buds. In body water acts

- ➢ As a lubricant
- > As a constituent of synovial fluid, it lubricates joints
- As a component of cerebrospinal fluid, provides cushion for nervous system; and, involved with sight and provide lubrication for eyes
- Water is an important transport medium for various substances, which serve to nourish the cells and remove waste products out of body
- > It also assists the thermoregulation in body and helps to control temperature in thermo neutral zone.

The total body water involved in all of these functions is contained in two major compartments in the body. The extracellular water, which is present outside the cell, comprises 20% of the body weight and the intracellular water, which is present inside the cell, constitutes 45% of the total body mass.

Water consumption:

Water intake increases with age. Water consumption can be affected by feed type, stage of production, growth, health status and environmental temperature. Water requirements of poultry are often crudely estimated by multiplying the amount of feed eaten by 2 (e.g. 1kg feed: 2L of water). Under hot conditions however, they will drink substantially more water (up to twice as much). Water consumption increases by approximately 7% for each 1°C above 21°C (Swick, 1998). This will be greater if the water is cooler than the air and less if the water is warmer than the air.

Water quality:

Water quality is determined by analysis of water. A bacterial analysis indicates if water contains microorganisms, such as bacteria, which may be harmful. A chemical analysis determines the levels of various minerals present in water. Water quality is an important consideration when planning to raise birds. Measurements of water quality include the items shown below. In some cases concentrations, which may be of concern, are mentioned.

1. Filterable residue (mg/L):

Filterable residue or total dissolved solids (TDS) is the main indicator of water quality. The TDS includes all of the dissolved minerals in the water. Its effect on birds will depend upon specifically, which mineral is present. Water with a TDS of less than 1,000mg/L is acceptable for all classes of livestock. Between 1,000 and 7,000mg/L the effects of TDS are less clear-cut and may range from no noticeable effect to temporary diarrhea and decreased productivity. If the TDS is between 7,000 and 10,000mg/L, serious health problems can develop and water refusal by livestock can occur. Water with a TDS over 10,000mg/L should not be used for animal consumption (Table 2).

Any mineral can elevate TDS, e.g., Ca and Mg contribute to TDS, but have very different physiological effects compared to sulfate, another contributor to TDS. The concentrations of mineral salts, which produce significant reductions in growth or significant increases in mortality, are shown in table 3.

Table 2. Guidelines for the suitability of water with different concentrations of total dissolved solids (mg/L)

| Total dissolved | Water quality |
|------------------|--|
| solids | |
| Less than 1000 | Considered low; Excellent for all classes of poultry |
| 1000-2999 | Very satisfactory for all classes of livestock and |
| | poultry. Watery droppings in poultry may be noticed in birds not accustomed to this level of salinity |
| 3000-4999 | It is poor water for poultry. Watery droppings, increased mortality and increased morbidity associated with poor growth may occur. Turkeys are particularly susceptible |
| 5000-6999 | It is not suitable for poultry |
| 7000-10,000 | Not fit for poultry |
| More than 10,000 | Not fit for any class of livestock |

(Runyan, 1996)

Table 3. Concentrations of mineral salts in drinking water (g/L) associated with reduced growth and increased mortality in newly hatched chicks

| Species | Mineral salt | Reduced growth | Increased mortality | References |
|----------|-------------------|-------------------|------------------------|-----------------------------|
| Chickens | NaCl | 7 | 7 | Krista et al. (1961) |
| | NaCl | 5.6 | 7 | Connor <i>et al.</i> (1969) |
| | CaCl ₂ | 3.9 | 7.8 | Connor <i>et al.</i> (1969) |
| | MgCl ₂ | 2.6 | 5.6 | Connor <i>et al.</i> (1969) |
| | Na_2SO_4 | 5.7 | 8 | Connor <i>et al.</i> (1969) |
| | MgSO ₄ | 8.7 | >10.6 | Connor et al. (1969) |
| | $MgSO_4$ | 0.7 | >10.0 | |

(Balnave, 1988)

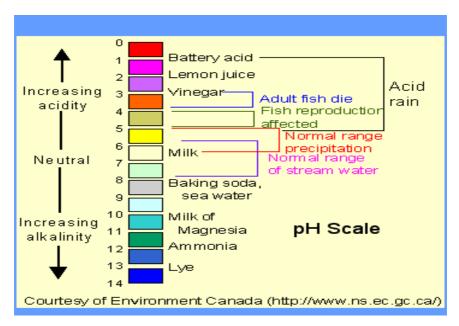
It has been suggested that greater than 1500 mg/L is unacceptable for poults less than 3-weeks of age; greater than 3000 mg/L is not good for chicks and ducklings; greater than 4000 mg/L can cause an increase in wet droppings in hens and turkeys; and greater than 7000 mg/L is not acceptable for any avian species.

2. Water pH:

The hydrogen ion concentration is expressed as pH. A pH value of 7.0 indicates "neutral" water. Values less than 7.0 are

increasingly acidic and values greater than 7.0 are increasingly alkaline. Most water falls within an acceptable range of 6.5 to 8.5.

Water pH is an important factor in determining the effectiveness of various water treatments. Chlorination efficiency is reduced at a high pH. A low pH may cause precipitation of some antibacterial agents delivered through the water system. For example, sulphonamides are a particular concern as precipitated medication may leak back into the water after treatment has ended, contributing to potential sulphur residues in carcasses.



3. Salinity:

Salinity is a measurement of the different salts in the water, and the term is often used interchangeably with TDS. Because there can be various mineral salts present, the effects on birds will vary. Saline water has been found to have a negative effect on poultry performance. Egg shell quality in breeders and layers is rapidly reduced being noticeable within 4-6 weeks in young pullets and within a few days in older hens (Balnave,

1996). Poor egg production, high embryonic death, increased cracking and poor hatchability have been observed. The adverse effects in hens do not appear to be reversible. Egg defects from hens exposed to saline water do not improve after being returned to normal water. Table 4 describes the effect of saline water on egg shell quality.

| | Chloride | | |
|-------------------------------|----------|---------|--|
| Performance | <14mg/L | 420mg/L | |
| Egg weight, g | 60.9 | 60 | |
| Egg shell weight, % | 9.57 | 9.03** | |
| Egg shell break strength, g | 2852 | 2030* | |
| Egg shell thickness, μm | 392 | 355** | |
| *. (P<0.01), | | · | |

Table 4. Effect of saline water on egg-shell quality

**. (P<0.001)

4. Sulfates, nitrates and nitrites:

Sulfates have a laxative effect on birds and can cause wet litter if concentrations are greater than 500 mg/L. Birds may build up a resistance to this effect. It has also been suggested that concentrations greater than 500 mg/L are not suitable for poults and greater than 1500 mg/L are not good for chicks.

The presence of nitrates and nitrites may indicate bacterial contamination from human or livestock waste and/or decomposing animals. In large quantities they will affect the capability of blood to carry oxygen and can be toxic. The recommendations for maximum nitrate concentrations vary. Some indicate that concentrations of less than 300 mg/L nitrate nitrogen should be tolerable for poultry, while others suggest a maximum of 50 mg/L.

5. Iron and other elements:

Iron (Fe) in water may stain equipment and laundry, and affect the taste, but is not generally considered a health risk for

⁽Balnave, 1996)

poultry. Iron may block pipes and drainage, but are not directly a threat to bird's health. Excessively high or low concentrations of other chemicals can produce recognizable symptoms. Excessive amounts of manganese (Mn) can produce a flavor problem. Too much copper (Cu) can give the water a bitter taste and may cause liver damage. High phosphate levels may indicate contamination from sewage. Calcium does not seem to have any negative effect at levels as high as 400 mg/L, and it appears that a level of 350 mg/L or more may be desirable.

| Contaminant | Average | Maximum | Comments |
|-----------------------|------------|------------------|---|
| | level | acceptable level | |
| Bacteria, count/m | l | | |
| Total bacteria | 0 | 100 | 0/ml is desirable |
| Coliforms | 0 | 50 | 0/ml is desirable |
| Nitrogen compour | ıds, mg/ml | | |
| Nitrate | 10 | 30 | Performance affected above 50mg/ml |
| Acidity and hardn | ess | | |
| pH | 6.8-7.5 | - | High or low may degrade medicaments or cause precipitation of minerals in water lines |
| Total hardness, | 60-189 | | <60 water is soft; >180 water |
| mg/L | | | is hard |
| Minerals, mg/L | | | |
| Calcium | 60 | 100 | Binding with tetracyclines; precipitation in water systems; bacterial buildup; high levels may alter nutrition |
| Chloride | 14 | 250 | If Na is high, low Cl may be detrimental; high NaCl may reduce performance and reduce egg shell quality |
| Sodium | 32 | 50 | If sulfate or Cl is high performance will be reduced |
| Sulfate, <i>mg/ml</i> | 125 | 250 | Laxative effect with high Mg, fast bleeding and edema |
| Copper | 0.002 | 0.6 | Higher levels produce a bitter flavor |
| Iron, <i>mg/ml</i> | 0.2 | 0.3 | Precipitation clogs water systems; higher levels produce a bad odor and taste |

Table 5. Maximum acceptable levels of contaminants in water for poultry

(Swick, 1998)

Carbohydrates

Carbohydrates are the organic compounds composed of carbon, hydrogen and oxygen and include sugars, starch, cellulose and gums etc. The diversity of dietary carbohydrates necessitates discussion of several classes of these molecules. ranging from simple sugars to huge, branched polymers. Monosaccharides or simple sugars are either hexoses (6-carbon) like glucose, galactose, fructose and mannose, or pentoses (5carbon) like arabinose, ribose and xylose. These are the breakdown products of more complex carbohydrates and can be efficiently absorbed across the wall of the digestive tract and transported into blood. The other monosaccharides are trioses (dihydroxy acetone phosphate), tetroses (erythrose) and heptoses (sedoheptulose). The trioses and tetroses occur as intermediates in the metabolism of other carbohydrates. Disaccharides are simply two monosaccharides linked together by a glycosidic linkage. The most important disaccharides in nutrition are lactose or "milk sugar" (glucose + galactose), sucrose or "table sugar" (glucose + fructose) and maltose (glucose + glucose).

Oligosaccharides are relatively short chains of monosaccharides, which typically are intermediates in the breakdown of polysaccharides to monosaccharides. Polysaccharides are the most abundant dietary carbohydrates for all except very young animals. There are two important storage polysaccharides, each of which is a large polymer of glucose:

1. Starch:

It is a major plant storage form of glucose. It occurs in 2 forms: α -amylose, in which the glucoses are linked together in

straight chains, and amylopectin, in which the glucose chains are highly branched. Except for the branch points of amylopectin, the glucose monomers in starch are linked via $\alpha(1-4)$ glycosidic linkage, which are hydrolyzed by amylases.

2. Glycogen:

It is the large polymer of glucose and is the major animal storage carbohydrate. Like starch, the glucose molecules in glycogen are linked together by $\alpha(1-4)$ glycosidic linkage.

NON-STARCH POLYSACCHARIDES:

The fiber component of the grain consists primarily of non-starch polysaccharides (NSP), which is, in cereals form part of the cell wall structure. The role of fiber in monogastrics diets has attracted much attention in recent years, due to the facts that, soluble NSP elicit anti-nutritive effects, and utilization of NSP as a feed material in monogastrics is very poor. These two factors are of significant concern because of the increasing population of world as compared to the static food production. More efficient utilization of potentially utilizable nutrients for food production is therefore of paramount importance to the sustainability of agriculture in the future.

Monosaccharides commonly present in cereal cell walls are Dglucose, D-galactose and D-mannose (hexoses); L-arabinose and D-xylose (pentoses); and, D-galacturonic acid, D-glucuronic acid and its 4-O-methyl ether (acidic sugars).

The term NSP covers a large variety of polysaccharide molecules excluding α -glucans (starch). The classification of NSP was based originally on the methodology used for extraction and isolation of polysaccharides. The residue remaining after a series of alkaline extractions of cell wall materials was called cellulose, and the fraction of this residue solubilized by alkali was called hemicellulose. The word hemicellulose was adopted because early researchers mistakenly regarded these polysaccharides as the precursors of cellulose.

This is now known to be incorrect but the term is still used. Some workers used the terms hemicelluloses and pentosans interchangeably because the pentose-containing polysaccharides make up the bulk of hemicelluloses (Neukom *et al.*, 1967; Neukom, 1976).

Chemical structure of non-starch polysaccharides (NSP):

Cellulose:

Cellulose is the most abundant organic compound in nature, comprising over 50% of all the carbon in vegetation. It is the major constituent of plant cell walls and of high molecular weight. Cellulose is composed of 7,000-10,000 glucose units (Goring and Timell, 1962). Individual cellulose chains lie side by side in bundles, held together by hydrogen bonds between numerous neighboring-OH groups, to form a "ribbon-like" twofold helix (Gardner and Blackwell, 1974; Figure 1) as compared to straight chain starch molecules. Cellulose is believed to be identical in chemical composition regardless of the source, and it is insoluble in water and aqueous solutions of alkalis. Cellulose in cereal grain cell walls can be recovered from the insoluble residue left after vigorous extraction of cell wall material matrix components with alkalis (Mares and Stone 1973a).

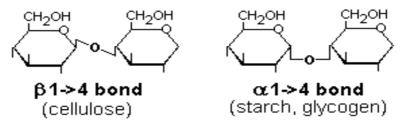


Figure 1. Chemical structure of cellulose, starch and glycogen

Pentosans (arabinoxylans, xylans):

The structures of cereal pentosans (arabinoxylans) are composed predominantly of two pentoses, arabinose and xylose, and their molecular structure consist of a linear (1-4)- β -xylan backbone to which substituents are attached through O₂ and O₃ atoms of the xylosyl residues (Perlin, 1951). The substituents are single arabinose residues, hexoses and hexuronic acids (Fincher, 1975), phenolics and proteins (Geissmann and Neukom, 1973a; Neukom, 1976). Most of the arabinoxylans in cereal grains are insoluble in water because they are anchored in the cell walls by alkali-labile ester-like cross-linkages rather than by a simple physical entrapment (Mares and Stone, 1973b). But the arabinoxylans not bound to the cell walls can form highly viscous solutions and they can absorb about 10 times water of their weight.

Arabinoxylans can rapidly develop a gel network in the presence of oxidative agents like hydrogen-peroxide/peroxidase (Geissmann and Neukom, 1973a, b). This gel formation coincides with disappearance of feruloyl groups. Fully developed cross-linked arabinoxylans can hold up to 100g of water/g polymer (Izydorczyk *et al.*, 1990). Besides establishing covalent cross-links arabinoxylans may also form "junction zones" by inter-molecular hydrogen bonding between un-substituted regions of the xylans backbone (Fincher and Stone, 1986). This non-covalent interaction of arabinoxylans may be of great importance in determining their conformational changes and solubility properties, and hence their anti-nutritional activities.

Pectic polysaccharides:

The term pectic polysaccharides refer to galacturonans or more commonly rhamno- galacturonans in which (1-4)-a-D galacturonan chains are interrupted at intervals by insertion of (1-2)-a-L-rhamnose residues. Other constituent sugars attached as side chains include D-galactose, L-arabinose, D-xylose, and less frequently L-fucose and D-glucuronic acid. Most of these sugars occur in short side chains, although D-galactose and L-arabinose are often found in multiple units (Choct, 1997). Extremely complicated side chains containing neutral pectic-polymers such as galactans and arabinans, xyloglucans and galactomannans have been reported. Pectic polysaccharides are found in cell walls of stems and leaves of cereals.

Mannans:

In some plant cell walls glucomannans and galactomannans may be present as the major non-cellulosic hexosans. The glucomannans are comprised of (1-4)-B-linked glucose and mannose units, whilst the galactomannans consist of a (1-4)- β -mannan backbone substituted with single units of (1-6)a-galactose (Choct, 1997). Glucomannans have been found as a minor component in cereal grains. No evidence has been reported for occurrence of galactomannans in cereal grains (Meier and Reid, 1977). Glucomannans have been found as a minor component in cereal grains (Mares and Stone, 1973a; Fincher, 1975). Table 1 shows the NSP contents and estimated β -mannan content of different feedstuffs.

Xyloglucans:

Another unusual group of NSP, xyloglucans, has been found in rice (Shibuya and Misaki, 1978). The structure of xyloglucans is a (1-4)- β -linked glucan backbone with single units of a-xylose attached to the O₆ atoms of the main chain. The physicochemical properties and nutritional activities of these NSP are yet to be established.

Table 1. Non-starch polysaccharide and β -mannan contents (%) of different feeds (on DM basis)

| | Non-starch | | |
|------------------|-----------------|---------------------|--|
| | Polysaccharides | Estimated-β-mannans | |
| Barley | 12.6 | 0.5 | |
| Corn | 11.7 | 0.1 | |
| Lupine seed meal | 34.8 | 0.4 | |

| Potato | 5.1 | 0.06 |
|--------------|------|------|
| Soybean meal | 22.7 | 1.2 |
| Wheat | 11.9 | 0.1 |
| Wheat bran | 33.7 | 0.07 |
| Whole peas | 13.8 | - |

(Chesson, 1987)

Quality & quantity of NSP:

The NSP content varies not only between different ingredients, but also within the same ingredient due to variety and geographical location where it is grown. However, the main structural feature of the NSP in a particular ingredient is not affected by environmental or varietal factors.

Other unusual ingredients:

The byproducts of roots and tubers, vegetable oil sources such as coconut (copra meal) are used as possible feed resources. Copra meal is the residue of coconut oil production. Its use in monogastric diets is limited due to a very high level of NSP (Purwadaria *et al.*, 1995). It contains about 45-60% of NSP, which consist predominantly of mannans (galactomannans and mannans), just over 10% cellulose and trace amounts of other polymers (arabinoxylogalactans, arabinomannogalactan and galactoglucomannans; Saittagaroon *et al.*, 1983; Zamora *et al.*, 1989).

Anti-nutritional effect of soluble NSP:

The NSP include a range of compounds possessing different physicochemical properties. Their nutritional effects in monogastrics are diverse and, in some cases, extreme. It is, however, generally conceded that the major detrimental effects of NSP are associated with the viscous nature of these polysaccharides, their physiological and morphological effects on the digestive tract and the interaction with the microflora of the gut. The mechanisms include altered intestinal transit time, modification of the intestinal mucosa and changes in hormonal regulation due to a varied rate of nutrient absorption (Vahouny 1982).

1. Viscosity:

The viscosity of NSP depends on their solubility and molecular weights. Generally, high gut viscosity decreases the rate of diffusion of substrates and digestive enzymes and hinders their effective interaction at the mucosal surface (Edwards *et al.*, 1988; Ikegami *et al.*, 1990). Soluble NSP interact with the glycocalyx of the intestinal brush border and thicken the rate-limiting unstirred water layer of the mucosa, which reduces the efficiency of nutrient absorption through the intestinal wall (Johnson and Gee, 1981). The fact that the viscous property of NSP is a major factor in the anti-nutritive effect of NSP in the diet of monogastrics is supported by the widespread use of enzymes. The enzymes cleave the large molecules of NSP into smaller polymers, thereby reducing the thickness of the gut content and increasing the nutritive value of the feed (Bedford *et al.*, 1991; Choct and Annison, 1992).

2. Modification of gut physiology:

The soluble NSP cannot only act as a physical barrier to nutrient digestion and absorption by increasing gut viscosity, but also change gut functions by modifying endogenous secretion of water, proteins, electrolytes and lipids (Johnson and Gee, 1981; Angkanaporn et al., 1994). The changes in the gut are characterized by enlargement of the digestive organs and increased secretion of digestive juices, accompanied by a decrease of nutrient digestion. The ability of certain NSP to bind bile salts, lipids and cholesterol is also well documented (Vahouny et al., 1980; Vahouny et al., 1981). This property of NSP may influence lipid metabolism in the intestine. Furthermore, viscous NSP can enhance bile acid secretion and subsequently result in significant loss of these acids in the feces (Ide et al., 1989; Ikegami et al., 1990). This, in turn, can result in increased hepatic synthesis of bile acids from cholesterol to reestablish the composite pool of these metabolites in the enterohepatic circulation. The continued "drain" of bile acids and lipids by sequestration, and increased elimination as fecal acidic and neutral sterols, may ultimately influence the absorption of lipids and cholesterol in the intestine. These effects could lead to major changes in the digestive and absorptive dynamics of the gut, with consequent poor overall efficiency in nutrient assimilation by the bird.

Future options for NSP in monogastric diets:

The anti-nutritive activity of soluble NSP with welldefined chemical structures, e.g., arabinoxylans and β -glucans in cereal grains, is eliminated effectively by supplementation of the feed with xylanases and β -glucanases which cause a partial depolymerisation of the NSP to smaller polymers so that their ability to form highly viscous digesta is greatly reduced. Enzymes capable of effectively cleaving various pectic polysaccharides are, however, yet to be produced.

Protein and Amino Acids

Dietary protein is important for maintenance, growth and development. Proteins are polymers of amino acids linked together by peptide bonds. The chain length of protein varies tremendously. Very short proteins, typically 3-10 amino acids in length, are called peptides. Although very small peptides can be absorbed to a limited degree, for all intents and purposes, proteins must be reduced to single amino acid before they can be absorbed. Protein chain may contain minerals (hemoglobin high in iron, casein high in phosphorus) or lipids (lipoproteins, e.g. cholesterol) or carbohydrates {glycoproteins, e.g. antibodies, antigenic determinants (portion of an antigen, which antibodies recognize and bind to)}.

Enzymes that cause the break down of peptide bonds and reduce proteins or peptides to amino acids are called proteases or peptidases (Figure 1). All living organisms, from viruses to man, require protein for structural units and for metabolically active compounds called enzymes. These are made up of amino acids that are linked together in specific arrangements; each particular sequence creating unique 2- and 3-dimensional structures, as required for the proper function of the protein.

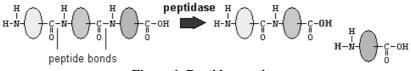


Figure 1. Peptidase action

Functions:

The amazing array of functions performed by proteins includes

- Muscle contraction (actin and myosin filaments),
- Nucleic acid synthesis {deoxyribose nucleic acid (DNA) and ribonucleic acid (RNA)}
- Digestion and metabolism of food with the help of enzymes
- Stimulation and regulation of growth with hormones
- Protein in the form of immunoglobulins helps to deactivate antigens
- Play important role in collection and processing of light during vision process
- Involved in the formation of various functional structures required for life processes like heart, lungs liver etc.
- ➤ As a component of hair/skin it protects the body.

Amino acids (AA):

Amino acids are the building blocks of proteins. These are simple organic compounds that contain a central carbon to which is attached a carboxyl group (COOH), an amino group (NH₂), and a side chain of varying complexity. The side chains Physical AA's determine the identity. and chemical characteristics are derived from sequence and linkages of AA chain, which is controlled by DNA. Amino acids link together by way of peptide bonds, which result when the carboxyl group of one amino acid binds to the nitrogen group of another. In the chicken's body, proteins contain 22 different AAs, all of which are needed for health. Plants and many bacteria are able to synthesize their own amino acids. There is no de-novo synthesis of AA in birds and higher mammals so these are dietary essential.

Indispensable & dispensable amino acids:

Indispensable amino acids (IAA) are those, which are not synthesized in the body of chicken so they must be exogenously added in the diet. Dispensable amino acids (DAAs) can be made from IAA within the body. However these too are often supplied in the ration so that the bird does not have to use up energy in synthesizing them.

There are several hundred amino acids existing in nature, but only 20-25 are found in plant and animal proteins. Most of the simple stomach animals require 10 IAA in the diet because they are unable to synthesize them, including phenylalanine, valine, tryptophan, threonine, isoleucine, methionine, histidine, arginine, lysine and leucine. Cystein and tyrosine are semiessential in the sense that, they can be synthesized from methionine and phenylalanine, respectively. The other AAs can be synthesized from other compounds and IAA therefore referred as DAAs.

Indispensable Amino Acids:

Names Valine Leucine

Isoleucine

 $\begin{array}{c} CH_{3} \\ CH_{3} \\ CH_{3} \\ CH_{2} \\ CH_{3} \\ CH_{2} \\ CH_{2} \\ CH_{2} \\ CH_{3} \\ CH_{2} \\ CH_{3} \\ CH_{2} \\ CH_{3} \\ CH_{2} \\ CH_{3} \\ CH_{3$

NH₂

Structural Formulas

| Lysine | H2N-CH2-CH2-CH2-CH2-CH-COOH |
|--------------------------|---|
| Methionine | CH3—S—CH2—CH2—CH—COOH NH2 |
| Phenylalanine | CH2—CH—COOH |
| Threonine | CH₃—CH—CH—COOH │ │ │ OH NH₂ |
| Tryptophan | CH2-CH-COOH NH2 H |
| Arginine | NH C-NH-CH2-CH2-CH2-CH2-CH-COOH H2N NH2 |
| Histidine | |
| Dispensable Amino Acids: | |
| Alanine | CH₃—CH—COOH I NH₂ |
| Asparagine | H2NOC-CH2-CH-COOH |

| Aspartate | HOOC-CH2-CH-COOH |
|-----------|----------------------------|
| Cysteine | HS-CH2-CH-COOH I NH2 |
| Glutamate | HOOC-CH2-CH2-CH-COOH |
| Glutamine | H2NOC-CH2-CH2-CH-COOH |
| Glycine | NH2-CH2-COOH |
| Proline | |
| Serine | CH2-CH-COOH |
| Tyrosine | |

Role of amino acids in the chicken:

Amino acids make up the body proteins which are used to form muscle, skin, feathers, bone matrix, ligaments and tendons. The requirement of the bird for protein varies at different stages of life with a higher level required in the rearing period when birds are growing. During lay provision of the right level of amino acids in the diet is one of the main factors influencing the laying performance particularly in relation to egg number and size, and is also important to maintain healthy plumage. Proteins are also vital for immune system function. Any protein deficiency will render the bird much more vulnerable to infectious and disease. It is not important to have all EAA in the ration but must be at balanced level and that there is no excess or deficiency of a particular amino acid, which might have an adverse effect.

Physiological basis of amino acid requirements:

Amino acid requirements have been determined using a variety of experimental techniques, which usually employ purified diets with graded levels of the particular free AA in question. Free AAs are 100% digestible and available. Many factors may influence the requirement, the foremost being the genotype and age of the bird. Other factors which might affect growth and egg production include the level of other nutrients, disease states and environmental conditions such as temperature, humidity, water quality and air quality. Amino acid requirements have been established for poultry and data is available from a number of sources (ARC, NRC).

Amino acid requirement can be partitioned into a component required for maintenance and a component required for protein accretion. The AAs requirement for maintenance consists of AAs destroyed in the body, excreted in urine, used for synthesis of essential body metabolites and lost from skin and gastrointestinal tract (GIT).

In a growing chicken, the need for AAs for protein accretion comprises the largest portion of the AAs requirement. However, the contribution of the maintenance component to AAs requirement becomes increasingly important when the animal matures or increases in age and when the animal increases in body weight. Amino acids having a high requirement for threonine. methionine/cystine maintenance include and tryptophan. Factors affecting AAs requirements are age, body weight, sex, genotype, environment (climatic, microbial, social) and dietary factors. With these multiple factors, it is rather difficult and impractical to conduct experiments to determine the AAs requirements for each and individual situation. However,

this dilemma may be overcome by applying the concept of ideal protein in diet formulation.

Meeting the amino acid requirements of poultry:

The challenge to the nutritionist is to formulate diets, which provide as closely as possible the AA requirements of the bird and at the same time, the economical. This can present some difficulties because the AA profiles of raw feed materials do not match the requirements of the birds. Amino acid imbalances may occur when using too much protein. Antagonism can exist when the excess of an AA is associated with a deficiency of another whose requirements will therefore be increased. For example, if the lysine to arginine ratio exceeds 1:2, reduced growth rates of young birds may occur. Also, excess leucine, which may occur when using high levels of gluten meal or blood meal, causes reduced growth, feed intake is depressed through a metabolic effect, catabolism of valine and isoleucine is also stimulated.

Protein quality:

In the past, animal diets have been formulated based on crude protein (CP) level, which is measured on the basis of nitrogen (N) content of a feed ingredient. Protein requirements are greater during the young and growing stages, due to increase needs as the animal assimilates more tissue mass. It is important that the proper amount of protein should be included in diet, but of greater importance is the balance of various amino acids that make up the dietary protein. Amino acid balance is known as protein quality.

Quality of protein, judged by its biological value that how digestible are the proteins in a protein source; and how well the amino acid supplied match the animal's requirements. Protein quality is important because poultry require amino acids in a balanced ratio in order that all appropriate proteins can be synthesized. Additionally, during metabolic processes, concentrations of substrates and intermediates, including amino acid, must be appropriate to drive the reactions in the proper direction.

Excess protein:

Protein in excess of its requirement can be a problem. The avian body doesn't store excess amino acids; only proteins in the body are in the form of organized tissue, i.e. skeletal muscle (compared to carbohydrates stored as glycogen, triglycerides stored as adipose tissue, some fat-soluble vitamins are stored in liver). If protein is present in excess amount in feed, amino acids are not required for protein synthesis, then the excess protein will be utilized for energy.

In avian species, deamination (removal of amino acids), either transferred to another carbon skeleton to form a nonessential amino acid, or degraded to urea and flushed through urine. Remaining amino acids will be degraded for energy. Avian species do not have the ability to utilize ammonia (NH₃). For them NH₃ is toxic substance which must be removed from system. In monogastric species, generally accepted that excess protein will not cause major problems as long as animal has sufficient water to drink to flush NH₃/urea from body.

Protein excess may cause a decreased intake, which may then cause deficiencies of other nutrients. It may also exacerbate hypersensitivity to feed antigens, thus causing malabsorption and diarrhea. Excessive protein in the diet may increase systemic NH₃ levels, causing an increased burden on kidneys and can lead to kidney and urinary stones.

Protein deficiency:

In practical poultry nutrition, the amino acids most likely to be deficient are lysine, methionine and tryptophan. Cereal grains, which are the primary energy sources used, are quite low in these amino acids. The major symptoms of protein deficiency are poor growth, poor feed conversion ratio and decreased egg production (Kino and Okumura, 1986). Inadequate lysine is known to cause depigmentation of the wing feathers in turkey poults. A variety of abnormalities in feather development occur with deficiencies of valine, leucine, isoleucine, tryptophan, phenylalanine and tyrosine in growing chicks. Footpad dermatitis has been observed in poults with methionine deficiency.

Practical ration formulation:

A fundamental assumption in poultry feed formulation is that the nutrient supply of individual feedstuffs can be added together to meet the nutrient specifications of the diet. Additivity is assumed to be applicable under practical situations.

The energy requirements of birds are met in most diets by the use of cereals and fat. Cereals have low levels of proteins with a poor amino acid balance. This requires the use of additional protein supplements. Plant derived protein sources have low levels of some of the IAA and thus the balance of the amino acids differs from bird's requirement. This can be a particular problem with the supply of methionine.

Apart from the difficulties in exactly matching plant and animal protein sources to the amino acid requirements, these feed ingredients have other problems associated with them such as anti-nutritional factors and variability in composition and quality. Soybean meal is a relatively high quality ingredient. Its protein is highly digestible and the amino acid balance closely matches that of the bird except in the case of the sulfur containing amino acids, where it is deficient. Depending on the source, the protein content can vary considerably (44-50%) and if not properly treated it may contain high levels of some factors, which can inhibit protein digestion. Conversely, sunflower meal is high in sulfur containing amino acids but relatively low in lysine and may contain high fiber and tannins. One of the problems of this ingredient is its low overall protein level (approx 34%).

Fish meal is high in all amino acids and is a particularly good source of lysine and methionine compared to the plant protein sources. Fish meal protein is also highly digestible. Fish meal, however, is highly variable in quality. Although most researchers list fish meal as containing 60-70% protein.

The feeding of poultry was based on CP contents of diet previously. That did not account for any of the amino acid needs of the birds. With more research the formulations shifted to an amino acid requirements basis. Thereafter poultry feed industry worldwide moved to routine laboratory analysis for amino acids and formulas to predict amino acid contents based on the CP content of the feedstuffs. More recently the use of amino acid digestibility values to formulate diets with the same amino acid contents using different feedstuffs is popular. The relatively newer concept is ideal proteins and the use of tissue accretion methods to predict retention efficiency and define the exact requirements for amino acids (Figure 2).

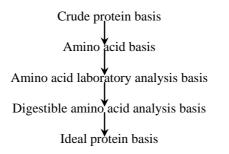


Figure 2. Development of nutritional knowledge based on formulation methodology and inputs for amino acid nutrition (Firman and Boling, 1998)

Digestible amino acids (DAA):

It has been known for many years that not all of the amino acids in a feedingstuff are digested by the bird and then available for protein synthesis. It is also known that there is considerable variation between, and often within, protein sources in the digestibility of AAs. Digestible amino acids result in the requirement being expressed on a more accurate basis. The most common approach to the measurement of available amino acids has been digestibility studies. This assumes that availability is a direct function of digestibility. For poultry, this is largely correct. Because of the mixing of faeces and urine in birds, digestibility is normally measured as the difference between amino acids consumed in the feed and in the corresponding excreta (faeces plus urine).

The methods used to measure DAA vary considerably and these have been reviewed on numerous occasions (Sibbald, 1987; Johnson, 1992; McNab, 1995). The *in vivo* methods revolve around some form of feeding the bird and collection of excreta. The cockerel is removed from feed to clear the gut; force-fed a known quantity of feed and its excreta collected for a period of time. Another bird is either left off feed or fed a nonnitrogenous feedstuff and excreta collected for adjustment to account for endogenous loss or loss that occurs regardless of the feed being fed. Other methods involve feeding with an indigestible marker that allows quantitation of feed in the gut and collection of feces from the small intestine after killing the bird. This tends to be more expensive and time consuming. Site of sampling is important and the indigestible markers used to calculate illeal digestibility may also vary.

The use of cecectomized cockerels for estimating DAA is not without problems. The age, strain or sex of birds may influence the ability to digest and use certain nutrients. In addition to it surgery is required to cecectomize the birds. Cecectomized birds must be constantly maintained and held for this purpose, whereas young chicks or poults can generally be readily obtained at most research institutions. A potential method of determining amino acids availability that may overcome many of these problems is that of ileal digestibility in intact birds. Payne *et al.*, (1971) proposed the use of ileal digestion as a means of estimating DAA for broilers and were able to find significant differences in amino acids availability between proteins of widely different quality. This technique eliminates the need to perform surgery on birds and can be used in virtually any housing situation, even on litter.

A novel technique based on the guanidination of dietary proteins to distinguish between endogenous secretions and exogenous or dietary sources of amino acids in intestinal digesta proposed by Hagemeister and Erbersdobler (1985). was Guanidination is the chemical process wherein the lysine moieties in dietary proteins are transformed to homoarginine (2amino-6-guanidino-hexanoic acid) by reaction with methylisourea under alkaline conditions. This approach has been applied to the measurement of ileal endogenous amino acids loss in poultry (Angkanaporn et al., 1996; Siriwan et al., 1994). Ileal endogenous amino acids loss determined by the use of guanidinated casein were about twice greater than those estimated either by feeding a nitrogen free diet or by extrapolation to zero nitrogen intake. The values generated by the homoarginine technique were of similar magnitude to those determined using ¹⁵N-dilution technique (Roos *et al.*, 1994).

The recent development in methodology for assessing the DAA in feedstuffs through the use of cecectomized cockerels has resulted in the generation of data regarding the DAA of common feed ingredients. Synthetic amino acids are generally considered as almost 100% digestible, use of digestible values increases the relative value of pure amino acids.

Factors affecting feedstuff digestibility:

A number of factors can affect the digestibility of feedstuffs both from the bird standpoint, diet standpoint and from the feedstuff standpoint. In case of birds the variation may occur due to ages, gender, species or strain, environmental temperature, gut length, gut conditions and level of feed intake. It appears that very young birds does not digest feed as well as after several weeks of life and probably the older bird may depress digestibility somewhat as well. From the standpoint of dietary factors it has been shown that high levels of dietary fat can improve digestibility of feeds with lower digestibility coefficients. High levels of dietary fiber can reduce digestibility of feeds as well.

Feedstuff digestibility may also occur with fewer differentials in the grains and well-controlled processed feeds such as soybean meal. More problems occur with byproduct meals where there may be differential inputs of fat, ash (from bone), whole carcass etc. as well as different cooking conditions due to these changes. This can result in over-cooking in some cases and leads to changes in digestibility values. Similar effects can be had in processed grain products where different levels of hulls may be left in product causing changes in digestibilities of the product. Table 1 depicts the percent DAA in common feed ingredients calculated with adult roosters.

| Feedstuff | Arginine | Serine | Histidine | Isoleucine | Leucine | Lysine | Methionine | Phenylalanine e | Tyrosine | Valine | Asparganine | Glutamine | Proline | Alanine | Average |
|-------------------------|----------|--------|-----------|------------|---------|--------|------------|--------------------|----------|--------|-------------|-----------|---------|---------|---------|
| Barley, steam rolled | 87.9 | 83.4 | 79 | 76.9 | 77.5 | 67.5 | 76.2 | 85.7 | 83.2 | 78.3 | 81.8 | 84.2 | 79.3 | 73.1 | 79.7 |
| Blood meal | 92.5 | 94.1 | 88.4 | 86.2 | 91.3 | 92.5 | 93.3 | 92.2 | 94.7 | 89.6 | 90.4 | 89.1 | 90.5 | 92.7 | 91.2 |
| Corn gluten meal | 99.9 | 100 | 98.4 | 99.5 | 99.5 | 99.5 | 99.5 | 99.2 | 99.2 | 99.6 | 99.7 | 99.8 | 99.5 | 99.7 | 99.5 |
| Corn gluten feed w/bran | 81.5 | 66.8 | 64.8 | 73.1 | 81.5 | 57.4 | 76.1 | 75 | 81.5 | 59 | 68 | 75.7 | 69.5 | 76.1 | 70.5 |
| Corn grain | 99.3 | 10 | 95.9 | 100 | 99.2 | 86.2 | 98.3 | 98.2 | 99.1 | 99.8 | 96.3 | 98.2 | 100 | 97.9 | 97.2 |
| Oats | 100 | 82.8 | 89.5 | 91.2 | 93.9 | 89.4 | 91.9 | 95.2 | 89.6 | 85.8 | 90.8 | 92.8 | 86 | 79.8 | 88.3 |
| Poultry byproduct meal | 93.2 | 85.7 | 80.8 | 90.6 | 91.1 | 90.9 | 92.1 | 90.4 | 93.9 | 88.1 | 73.3 | 87.6 | 80.9 | 86.5 | 87.3 |
| Feather meal | 84.2 | 76.4 | 84.2 | 82.3 | 76.8 | 73.3 | 77.5 | 79.6 | 78.9 | 77.5 | 58 | 71.8 | 63.1 | 72.3 | 73.6 |
| Wheat bran | 87.9 | 85.8 | 86.1 | 74.3 | 80.2 | 74.8 | 75.6 | 78 | 79.5 | 70.4 | 76.4 | 86.3 | 89 | 56.8 | 78.6 |

 Table 1. Digestibility (%) of common feedstuffs in roosters

(Firman, 2001)

Ideal amino acids:

The ideal amino acid is the exact ratio of AA needed to provide optimal performance without excess and is based on lysine at 100% with all other amino acids in a ratio to lysine. Theoretically as the requirement for lysine changes (e.g. from strain x to strain y), all other amino acids will change related to that. For chickens lysine is the reference amino acid in ideal amino acid ratio (Baker and Han, 1994). The logic involved in expressing amino acid requirements as ideal ratios to lysine is that a multitude of dietary factors (e.g., protein level, energy level and feed intake), environmental factors (e.g., disease, crowding, feeder space and heat stress) and genetic factors (e.g., sex and capacity for lean vs. growth) may affect amino acid requirements, but the ideal ratio of indispensable amino acids to lysine should remain largely unaffected by these variables (Baker and Han, 1994). Lysine is selected as the reference amino acid for 3 primary reasons, firstly, its analysis in feedstuffs unlike tryptophan and sulfur containing amino acids is relatively simple and straightforward; secondly, a considerable body of data exists for digestible lysine needs of poultry; and lastly, unlike several other amino acids (e.g., methionine, cystine and tryptophan), absorbed lysine is used only for protein accretion.

In order to apply ideal amino acid concept correctly in diet formulation, knowledge of lysine requirement for a given stage of growth of chicken is required. Therefore the correct level of dietary lysine must be determined before ideal amino acid ratios can be evaluated in practical diets. It is critical to feed diets at, or possibly slightly below the bird's required level of lysine, because lysine is used in the development of ideal amino acid ratios (Knowles and Southern, 1998). Determination of ratios of individual amino acid to lysine is not important when birds are fed diets containing excess lysine (i.e. ratios of individual amino acid to lysine would appear lower than they actually are). Thus the ratios of individual amino acid to lysine are meaningful when chickens are fed a level of lysine that is not in excess of their requirements. The use of ideal amino acid concept in practical diet formulation is advantageous because it is simple and flexible; can maintain dietary amino acid balance when using or changing combination of alternative feed ingredients; and, provide estimates when information on requirements for IAA is not available. This can be applicable equally well to diets formulated for animals of either sex, raised on high or low energy and protein diets, animals with slow or fast growing rates, different lean gain potentials and raised in thermo neutral or heat stressed environments.

The predicted requirements for lysine, sulfur containing amino acid and threonine at 8 different growth periods of broilers (Table 2) on the basis of the Illinois ideal protein ratio (Table 3) are given.

| (SAA) and three at 8 growth periods | | | | | | | |
|-------------------------------------|-------------------|----------------|----------------------|--|--|--|--|
| Period | Digestible lysine | Digestible SAA | Digestible threonine | | | | |
| (days) | | (% of diet) | | | | | |
| 0-7 | 1.2 | 0.8 | 0.8 | | | | |
| 7-14 | 1.1 | 0.8 | 0.7 | | | | |
| 14-21 | 1.4 | 0.7 | 0.7 | | | | |
| 21-28 | 1.0 | 0.7 | 0.7 | | | | |
| 28-35 | 0.9 | 0.7 | 0.6 | | | | |
| 35-42 | 0.9 | 0.6 | 0.6 | | | | |
| 42-49 | 0.8 | 0.6 | 0.5 | | | | |
| 49-56 | 0.8 | 0.5 | 0.5 | | | | |
| - | | | | | | | |

 Table 2. Predicted requirements for lysine, sulfur containing amino acids (SAA) and threonine at 8 growth periods

(Firman, 2001)

Table 3. Illinois ideal chick protein

| Amino acids | Percent |
|------------------|---------|
| Lysine | 100 |
| Arginine | 105 |
| Histidine | 37 |
| Methionine | 36 |
| Cystine | 36 |
| Phenylalanine | 55 |
| Tyrosine | 50 |
| Tryptophan | 16 |
| Glycine + Serine | 65 |
| Proline | 44 |

(Firman, 2001)

The data on protein and amino acid requirements of turkey has been reviewed (Firman 1994a). It appeared that lysine and sulfur containing amino acids are most limiting in commercial diets. A number of studies (Jackson et al., 1983; Jackson and Potter, 1984) have been performed using deletion methods to determine that threonine, valine and isoleucine were next limiting in corn-soybean meal diets. Firman (1994a) confirmed the results by using low protein diet in an addition method. It has been suggested that the amino acid profiles of broilers would be close to those for turkeys and that additional threonine, sulfur containing amino acid and tryptophan would aid growing turkeys 16 to 20 week old (Baker and Chung, 1992). The NRC (1994) recommends 1.60% total dietary lysine for turkeys 0-4 weeks of age. However Boling and Firman (1998) suggest that the digestible lysine requirements for turkey poults during the starter period is 1.32% for optimal body weight gain and 1.34% for optimal feed conversion at relatively higher energy levels used in this study. The dietary treatments exceeded NRC recommendations for energy, but this excess was not believed to affect the outcome of the experiments. Ideal ratios for the turkeys are given in table 4.

| Amino acid | Percent | | | | | |
|-------------------------------|---------|--|--|--|--|--|
| Lysine | 100 | | | | | |
| Sulfur containing amino acids | 59 | | | | | |
| Threonine | 55 | | | | | |
| Valine | 76 | | | | | |
| Arginine | 105 | | | | | |
| Histidine | 36 | | | | | |
| Isoleucine | 69 | | | | | |
| Leucine | 124 | | | | | |
| Phenylalanine + Tyrosine | 105 | | | | | |
| Tryptophan | 16 | | | | | |
| | | | | | | |

 Table 4. Estimated ideal protein ratio for turkeys (starting hens)

(Firman and Boling, 1998)

Lipids and Fats

Lipid is a term referring to the compounds that are soluble in chloroform, benzene, petroleum or ether. It includes fats, oil, waxes, sterols, and complex compounds such as phospholipids and sphingolipids. Not all the lipids are the fat but all the fats are lipids. Nutritionally, fats are the source of energy, essential fatty acids (EFA) and fat-soluble vitamins (A, D, E, and K). Fatty acids are present in only small amounts in animal and plant tissues, but are the building blocks of many important complex lipids.

True fatty acids possess a long hydrocarbon chain terminating in a carboxyl group. Nearly all fatty acids have an even number of carbons and have chains between 2 and 24 carbons in length. If hydrogen molecules occupy all the carbons, it is saturated; with one double bond, it is unsaturated and with 2 or more double bonds, polyunsaturated. The degree of saturation is going to affect the melting point of the oil and also how quickly it's going to oxidize. The more saturated a fat is, the more solid it is at room temperature, whereas more unsaturated a fat is, the more liquid it is and going to oxidize faster. The principle differences among the key fatty acids are the length of the chain (usually 16 or 18 carbons) and the positions of unsaturated or double bonds.

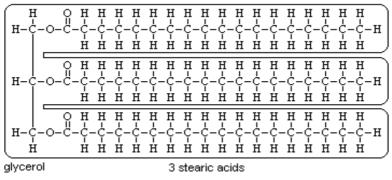


Figure 1. A molecule of tristearin

The most abundant storage form of fat is neutral fat or triglyceride. A molecule of triglyceride is composed of a molecule of glycerol in which each of the 3 carbons is linked through an ester bond to a fatty acid. Triglycerides cannot be efficiently absorbed, and are enzymatically digested by pancreatic lipase into a 2-monoglyceride and 2 free fatty acids, all of which can be absorbed. Other lipases hydrolyze a triglyceride into glycerol and 3 fatty acids (Figure 1).

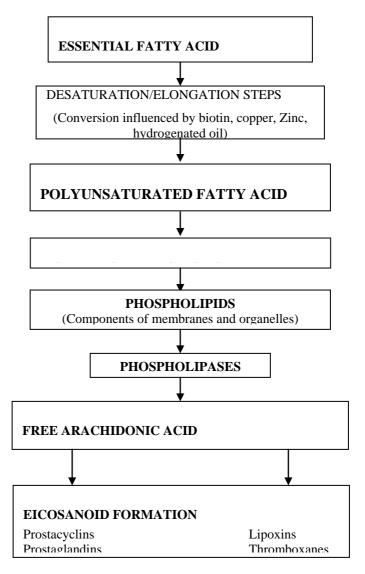
Poultry, fed primarily unsaturated fats will have higher degree of unsaturated fatty acids in body fat. This affects the characteristics because unsaturated are softer, have lower melting point, which affects processing and carcass quality. Poultry fed primarily saturated fats are going to have harder fat.

Polyunsaturated and essential fatty acids:

The investigations for the determination of EFA requirements of poultry began in the 1950's and established linoleic acid requirements in 1960's. Presently, a dietary level of 1% linoleic acid is recommended for adequate growth of chickens, turkeys and quails. Essential fatty acids are necessary for proper cell structure, synthesis of certain metabolites and production of prostaglandins.

The EFA follow a number of metabolic pathways, which include oxidation in mitochondria to generate adenosine tri-

phosphate (ATP), desaturation and chain elongation leading to the long-chain polyunsaturated fatty acids (PUFA), and incorporation into glycerolipids. Several hormones and dietary factors influence desaturation of PUFA in mammals and probably also influence desaturation/elongation of EFA in poultry. Unfortunately the important biochemical steps in EFA conversion to PUFA have not been studied in poultry. Figure 2 illustrates the desaturation and chain elongation steps for linoleic acid in the liver and the formation of eicosanoids. The desaturases facilitate the transport of dietary and *de novo* synthesized fatty acids and generate PUFA, which serve as components of phospholipids and as precursors of eicosanoids. Figure 2. Conversion of linoleic acid to n-6 polyunsaturated fatty acids; incorporation into phospholipids and subsequent biosynthesis of eicosanoids



Polyunsaturated fatty acids that are derived from EFA possess biological activity. Furthermore, the eicosanoids, which collectively include prostaglandin, leukotrienes and lipoxins, are biosynthesized from specific PUFA and act as cell-to-cell signaling agents. The biological effects of eicosanoids can be described as either autocrine (acting on the same cell of origin) or paracrine (acting on a different cell). The eicosanoids participate in biochemical reactions and physiological processes to affect tissue growth and development, and metabolism. Knowledge of these biologically active lipids in poultry has expanded to include their role in embryonic development, reproduction and bone growth.

Deficiency symptoms of essential fatty acids:

Linoleic acid (18 carbon) is one of the most important EFA. Linoleic acid in sufficient amounts can produce other necessary fatty acids like arachidonic acid (20 carbon). Although linolenic acid is required for nervous tissue and the retina, the EFA for poultry is linoleic acid. Linoleic $(18:2^{n6})$ and linolenic $(18:3^{n3})$ acids contain 2 and 3 double bonds, respectively, and are both 18 carbons in length. Linoleic acid belongs to the omega-6 (n-6) series of PUFA since the terminal double bond is located at 6th carbon counting from the methyl end of the molecule. Likewise, linolenic acid is a member of the omega-3 (n-3) series of PUFA since the terminal double bond is at the third carbon from the methyl end.

Embryonic viability and hatchability are compromised during EFA deficiency. Other EFA deficiency symptoms in chicks include retarded growth, increased water consumption, reduced resistance to disease, and enlarged liver with increased lipid content and an alternation of tissue fatty acid composition. In males, deficiency symptoms also include reduced testes size and delayed development of secondary sexual characteristics. In the laying hen, decreased egg size, lowered egg weight, and changes in egg yolk fatty acid composition result from linoleic acid deficiency. However, reproductive failure and increased susceptibility to disease might be related more to defective eicosanoid biosynthesis than to strictly EFA deficiency.

Sources of essential fatty acids:

Poultry diets should provide an adequate level of linoleic acid since plant oils are generally rich sources of this EFA. However, under some conditions poultry diets may not contain an adequate amount of EFA. Both linoleic and linolenic acids are readily absorbed through the intestinal wall where resynthesis of triacylglycerols and the packaging of lipids into portomicrons occur for transport to the liver. Long-chain n-3 PUFA present in marine oils and fish meals seem to be absorbed and metabolized to the same extent as the n-6 PUFA are absorbed and metabolized.

Since varying the dietary levels of EFA and PUFA will modify the composition of long-chain PUFA (n-6 and n-3) present in poultry tissues, enriching poultry meat and eggs with specified PUFA can be done to meet consumer demands, Furthermore, the changes in the types of PUFA in tissues of poultry may offer potential benefits to the bird by modulating eicosanoid production to enhance or depress immune responses.

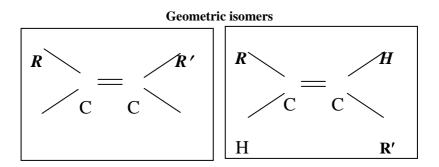


Figure 3. Double bond conformation for *cis* and *trans* geometric isomers of mono unsaturated fatty acids

Trans fatty acids and essential fatty acid requirements:

Fats should be stabilized by an antioxidant; otherwise they are likely to become rancid, especially in hot weather. Poultry nutritionists routinely request that antioxidants must be added during processing. Antioxidants help to prevent oxidative damage to unsaturated fatty acids and fat-soluble vitamins and to insure that ingredient quality is maintained until the animal consumes the final feed. The addition of feed grade fat to poultry feed provides numerous benefits including improved growth rate and feed conversion.

Dietary factors affecting fatty acid composition:

Dietary unsaturated fats can dramatically modify the fatty acid composition of lipids in the hen egg yolk and in tissues of rapidly growing poultry. The long-chain n-3 PUFA (especially eicosapentaenoic and docosahexaenoic acids) present in fish oils are extremely effective in lowering total n-6 PUFA in chick liver, egg yolk, edible muscle, bursa and thymus tissues.

Eicosanoid biosynthesis:

The majority of eicosanoids (prostaglandins, leukotrienes and lipoxins) are biosynthesized from arachidonic acid maintained in membrane phospholipids. Arachidonic acid is the precursor of 2-series prostaglandins, but $20:3^{n6}$ and $20:5^{n3}$ are substrates for the 1- and 3-series prostaglandins, respectively. Prior to eicosanoid formation, phospholipase-A₂ cleaves arachidonic acid from the sn-2 position on the glycerol backbone of phospholipids. Once arachidonic acid is liberated, it can undergo controlled oxidative metabolism to form a variety of eicosanoids with differing physiological effects. The enzymes inherent to the cell dictate the types and amounts of eicosanoids produced.

Minerals

The mineral portion of the feed is inorganic matter. They are required in varying amounts for proper nutrition. Minerals represent about 3.5% of the total body composition, of which 46% is Ca, 29% is Phosphorus (P) and 24% included K, S, Na, Cl and Mg. Minerals, especially Ca and P, help to build bones and make them strong and rigid. Laying hens also require minerals for egg shell formation. Other minerals are needed in trace amounts for all metabolic functions.

Grains are low in minerals, so it is necessary to provide supplements. Calcium, P and NaCl are needed in the greatest amounts. Bone meal, defluorinated rock phosphate and dicalcium phosphates (DCP) supply additional Ca and P. Ground limestone and oyster shells are good Ca sources. Trace levels of iodine (I), Fe, Mn and Zn are also included in mineral supplements.

Any mineral fed at levels either below or in excess of requirements is going to have some sort of affect on production, but most minerals in excess are not toxic they are just going to probably affect absorption of some other mineral (thereby causing a deficiency of that mineral) or increase feed cost. There are dozens of interactions between different compounds that can affect absorption e.g., too much P will bind Ca, making it unavailable for absorption and causing Ca deficiency. Also, excessive Ca will affect absorption of Mg (and vice versa), excessive Cu will affect Fe and P etc.

Macro-Elements:

Dietary minerals can be divided into 2 broad groups; Macro-minerals and micro- or trace-minerals. Macro-elements include Ca, P, Mg, Na, K, Cl and S. These will be discussed in the following paragraphs.

Calcium and phosphorus:

These two elements are needed in large quantities for proper bone formation. Production of eggs requires increased intake of both Ca and P, so the requirement for these macroelements can change tremendously within a single season. The 99% of Ca is stored in bones and teeth, whereas 80% of P. The real concern with Ca and P is that these two elements share a somewhat antagonistic relationship. Excess P, e.g. will form insoluble complexes with Ca, thus increasing the dietary need for Ca. Proper dietary Ca: P ratios range from about 1.3:1 to about 2.3:1, depending on the species and phase of growth.

Functions:

Calcium plays an important role in a wide variety of essential functions in metabolism.

- Calcium is an essential component of bones and helps in egg shell formation. It is involved in the normal blood clotting process
- Involved in the contraction of skeletal, smooth and cardiac muscles
- Engages in the regulation of normal heartbeat and helps in transmission of nerve impulses
- It is also involved in secretion of a number of hormones and activates or stabilizes enzymes.

Phosphorus is involved in virtually every metabolic reaction in the body and is considered to be the most versatile of all the mineral elements.

- Phosphorus is essential for the building of muscle tissues, egg and bone formation.
- It also aids in maintaining cellular osmotic pressure and acidbase balance.
- It has very important role in amino acid metabolism and protein formation. Phosphorus is an activator of many enzyme systems e.g. enzymes of energy metabolism.
- As a component of phospholipid, it acts as a major carrier to transport fatty acids across the cell membrane.
- As a component of nucleic acids, it has an important role in genetic transmission and control of cellular metabolism.
- Energy transfer in most metabolic systems involves phosphate compounds such as ATP and creatine phosphate.

In the body, at a normal blood pH of 7.4, the two forms of P, dibasic and monobasic, normally exist in a ratio of 80:20, respectively. During acidosis (excess H^+ which lowers the pH), the dibasic form is important because of its ability to accept a proton and thus be converted into the monobasic form, removing the proton from body fluids and helping to bring the blood pH back up to normal. Likewise, the monobasic form is beneficial during alkalosis (low H^+ which increases the pH) in the animal body. In this case, the monobasic form donates a proton to body fluids, which helps to lower the pH back to normal. Phosphate, in the monobasic and dibasic forms, thus provides one of the important buffer systems in the body, which assists in maintaining acid-base balance.

Metabolism:

There are many factors influencing the utilization and metabolism of Ca and P in the body. Some of the most important includes the ratio of the two elements in the diet, the amount of vitamin D present, the biological availability of the supplements used to provide the elements, and the age and physiological state of the birds. Young birds with a rapidly developing skeletal system tend to use the minerals more efficiently than do older birds; hens in active egg production utilize minerals more effectively than non-layers.

Phosphorus is absorbed chiefly in the duodenal area of the small intestine. As in the case of most nutrients, the greater the need, the more efficient is the absorption. Phosphorus absorbed from the intestine is circulated throughout the body and is readily withdrawn from the blood for bone development. It may be withdrawn from bones to maintain normal blood plasma levels. Plasma Ca and P levels are regulated by the parathyroid hormone.

Sources:

The plant ingredients such as grains, soybean meal and cottonseed meal all have low levels of Ca, whereas young mammals receive their Ca from milk, this is obviously not the case for the growing chick. When the hen enters egg production, her need for Ca more than triples in order to support shell formation. Clearly, a source of supplemental Ca is needed. Limestone is the most common source of Ca for poultry feeds, containing about 37% Ca. The use of dolomite limestone should be avoided because of high levels of Mg, which can be harmful to poultry. A relatively fine product should be used in chick diets. Oyster shell is also a preferred source of Ca, where available.

Phytase is a bacterial enzyme that can degrade phytic acid in the plant material, freeing additional P for absorption by the animal. It is coming under increasing use in poultry diets to decrease the amount of inorganic P that needs to be added to the diet. This strategy also decreases the amount of P excreted in animal waste, thus reducing its environmental impact.

| Sources | Calcium | Phosphorus | Magnesium |
|--------------------------------|---------|------------|-----------|
| Calcium carbonate (limestone) | 36 | - | - |
| Dolomitic limestone (dolomite) | 22 | - | 10 |
| Oyster shell | 35 | - | 0.3 |
| Calcium sulfate (gypsum) | 29 | - | - |
| Bone meal (steamed) | 29 | 14 | 0.6 |
| Phosphoric acid | - | 31.6 | - |
| Sodium phosphate (Monobasic) | - | 22.4 | - |
| Sodium tripolyphosphate | - | 30.8 | - |
| Sodium tripolyphosphate | - | | - |

Table 1. Composition (%) of calcium and phosphorus sources used in feeds and mineral mixtures

(Cheeke, 1991)

The most common forms of phosphate supplemented to poultry diets are defluorinated rock phosphate, steamed bone meal, guano deposits such as Curacao phosphate, colloidal phosphates and either monocalcium or DCP. Phosphorus in meat and bone meal is almost completely absorbed by the bird. Bone meal is typically of high biological quality, the P content and bioavailability of the other products is generally lower and more variable than processed phosphates. The majority of the feed phosphates used in poultry feeding are chemically processed materials. All these sources of P also contain appreciable amounts of Ca, making it much easier to meet the bird's requirement for both minerals. It is most often supplied in commercial diets from inorganic sources such as DCP. The table 1 gives common sources of Ca and P and their mineral contents.

Bioavailability of calcium and phosphorus:

Biological availability is a measure of the degree to which Ca and P (or any other mineral) source can support the physiological processes of an animal. The P from any source is never completely available or utilized. Some of it is always lost in normal digestive and metabolic processes. Further, many factors influence P absorption. The "true" or "absolute" availability of the P from any source is a goal that is often sought but is unlikely to be obtained, due to the myriad of factors that are involved. It has been stated that monocalcium phosphates have the highest bioavailability, with DCP about 5% less and defluorinated phosphates about 10% less in comparative value. In some countries, availability of feed-grade phosphates is limited and expensive and questions arise about the utilization of raw rock phosphates or fertilizer grade phosphates as sources of P in poultry diets. It is apparent that some sources of rock phosphate, either raw unprocessed supplies or partially processed fertilizer grade products, can be used to supply part or all of the P in poultry diets provided that adjustments are made for their bioavailability and concern given to contents of fluorine and vanadium. The level of fluorine in the final diet should not exceed 500ppm.

The most often over-looked aspect of mineral nutrition is adjustment of dietary Ca-level in respect to phytate-P levels. This is especially important when feed ingredients high in phytate-P are included in the diet, such as rice bran, wheat bran, canola meal, or sunflower meal. Failure to adjust the minimum Ca content of the diet in such situations may lead to a Ca deficiency. Nelson (1984) suggested the following formula to adjust dietary Ca levels in the presence of phytate-P:

Dietary Ca (%) = $0.6 + (\text{phytate P} \times 1.1)$

For example, a corn-soy diet formulated to provide a minimum of 0.45% available-P may have a total P contents of about 0.70%, thus providing about 0.25% phytate-P using the equation above, a minimum Ca level of $0.6 + (0.25 \times 1.1) = 0.88\%$ would be required. However using a corn-rice bran-wheat bran diet with some sunflower or canola meal formulated to provide the same minimum of 0.45% available P may result in a total P content of 1.05% phytate-P, so the minimum dietary Ca needs would be about $0.6 + (1.05 \times 1.1) = 1.75\%$

Although considerable information is available regarding total Ca content of different plant feedstuffs and different Ca and P sources, there is little direct information regarding the bioavailability of such products. In contrast to P, Ca is typically inexpensive to provide to poultry diets and little economic emphasis has been placed on determining biological values for Ca. The response to Ca source or particle size is sensitive to dietary Ca levels, being of greater concern when dietary Ca levels are minimal or in situations where egg shell quality is stressed.

Sodium and chloride:

Even though the body only contains about 0.2% Na, it is essential for life and is highly regulated. Sodium makes up about 93% of the basic mineral elements in the blood serum. About half of the Na in the body is in the soft tissues, and half in bones. Chloride is the blood's primary anion and makes up $\frac{2}{3}$ of the acidic ions.

Functions:

- Sodium is almost absent from the blood cells, but it constitutes approximately 93% of the total cations concentration of the blood plasma and hence the chief cation regulating blood pH
- Muscle contraction is also dependent on proper Na concentrations
- Sodium plays an essential role in nerve impulse transmission as energy for impulse transmission in nerves is obtained from the potential energy produced from the separation of K and Na by the cell membranes
- Efficient absorption of amino acids and monosaccharides from the small intestine also requires adequate amount of Na
- Sodium is needed to maintain electrolyte balance
- It is present in very small amounts in cell nuclei and mitochondrial enzymes. However, a small increase in Na concentration inhibits mitochondrial enzymes. Actually K and Mg ions are involved in activation of mitochondrial enzymes and Na ions have adverse effects on their activity.

Chloride is needed in maintaining the acid-base balance.

- Chlorine is found distributed in high concentrations in interand extracellular spaces.
- It in the form of NaCl constitutes over 60% of the blood anions. It does not have the affinity for combination with protein molecules and therefore can remain in salt and ion forms. So it contributes massively in maintaining the ionic strength of the extracellular fluids and as the most matching anion for combining with Na, which predominates in the fluids.
 - ➤ The gastric secretions contain Cl in the form of hydrochloric acid and as Cl salts.

Sources:

The source of NaCl for poultry rations is common table salt. There are other salts but NaCl is the most commonly used. Salt is added to the diet to satisfy the bird's requirement for Na and Cl and to improve the bird's appetite. Poultry diets have recommendations for levels of Na and Cl. Though salt is the major source of Na and Cl, grains, meals and supplements also contain these molecular nutrients. Meat contains plenty of Na, whereas, plant and cereal-based feeds are generally deficient in Na. The salt problem usually occurs due to unbalanced ration formulation i.e. the use of wrong values for ingredient nutrients, abnormal Na value of ingredients, computer error in nutrient specification, and. malfunction of mill equipments.

Potassium:

The K and Na contents of the body are almost similar, but there exist a difference in their distribution in the body. Potassium is mainly a cellular constituent. Its concentration in the body fluids is very low. Like Na it is also rapidly absorbed.

Functions:

- Potassium performs the same functions inside the cell that Na performs in the plasma and interstitial fluid
- It helps to maintain the acid-base balance and proper osmotic balance in the cells
- > It is also the activator of many intracellular enzymes
- It is needed for normal heartbeat and exerts its effect favoring relaxation, opposing the Ca, which favors contraction
- It is also involved in the increasing cell membrane permeability and increases the rate of entry of free and neutral amino acids and leaves the cell when the amino acid is entering
- > It is also involved in the lysine metabolism.

Magnesium:

There is a close association between Mg, Ca and P in their distribution as well as metabolism in animal body. About 70% of the body Mg is present in the skeletal system. Most of the Mg found in the egg is present in the shell portion. Like K it is generally found in sufficient amounts in most feed sources and hence occurrence of its deficiency is quite rare. The common Mg salts are highly soluble and therefore absorbed easily from the small intestine.

Functions:

- Magnesium is an important constituent of bones
- It also serves an important role in many enzyme systems, including DNA replication, transcription and translation.
- Specifically it activates all the enzymes concerned with the transfer of phosphate from ATP to ADP and, therefore, is essential in the energy exchange reactions.

Sulfur:

Sulfur is widely distributed in the body as a component of sulfur containing amino acids (methionine, cysteine, cystine, taurine), vitamins (thiamin and biotin), mucopolysaccharides in connective tissues and mucus secretions. In birds almost 50% of the S is found in the muscle tissues. Inorganic sulfate, which is a component of mucopolysaccharides, can be derived from the metabolism of sulfur containing amino acids. Thus S is not the dietary requirement of non-ruminants, although inorganic sulfates in the diet have a sparing effect on the sulfur containing amino acid requirements.

Functions:

- It is the structural component of the various body tissues (bones, feathers and cartilage).
- In the bones and cartilages S is present as chondroitin sulfate; in the feathers of bird as cystine; while in sperm and in the cuticle of gizzard, present as unidentified protein; and, in egg white and yolk it is present as sulfate.
- It is also the component of various hormones. The disulfide bridges (-S-S), which interlink the amino acid chains and stabilize the protein structure are contained in the molecules of several hormones (Insulin, prolactin etc.). When the bridges are split, the hormonal activity is lost.
- Other compounds having S as its integral components include the coenzyme A, lipoic acid, acetyl-CoA, sulfur acetyl lipoate, heparin, glutathione, thiamin, biotin, ergothionine, insulin etc.

TRACE-MINERALS:

Trace- or micro-minerals include elements such as Fe, Cu, selenium (Se), Mn, cobalt (Co), I and Zn. As the name implies, they are required in lesser amounts than are macroelements. Iron, I, Cu and Se deficiencies are the most common disorders noted in captive or domestic species. Trace-mineral sub clinical deficiencies occur more frequently and a bigger problem than clinical mineral deficiencies, because of the lack of farmer awareness about specific subclinical trace-mineral deficiency symptoms. The immune system is depressed, the animal begins to grow more slowly and fertility is impaired. The end result is inefficient production and lower profitability. Therefore, a profitable and efficient farm operation must provide the supplemental trace-mineral elements.

Iron:

Iron has been recognized as an essential nutrient for over 100 years. Approximately $\frac{2}{3}$ of body Fe is present in hemoglobin in red blood cells and myoglobin in muscle, 20% is in labile forms in liver, spleen and other tissues with the remainder in unavailable forms in tissues such as myosin and actomyosin and in metalloenzymes. In hemoglobin, which contains 0.34% Fe, an atom of ferrous (Fe⁺⁺) in the center of a porphyrin ring connects heme (prosthetic group) with globin (a protein). Excess Fe forms insoluble iron phosphate, which results in a decreased P absorption leading to rickets. Other trace-minerals and vitamins may get adsorbed on the colloidal suspension formed by the insoluble iron phosphate and remain unabsorbed.

Functions:

- It is an essential constituent of several metalloproteins, including hemoglobin, cytochrome-C and myoglobins
- The Fe in hemoglobin is essential for the proper function of every organ and tissue of the body
- It also plays a role in enzymes involved in oxygen transport and the oxidative process, including catalases and peroxidases
- Iron in blood plasma is bound in the ferric state (Fe⁺⁺⁺) to a specific protein called transferrin. Transferrin is the carrier of

Fe in the blood and is saturated normally only to 30-60% of it Fe-binding capacity.

Copper:

A small quantity of Cu is essential along with Fe, for synthesis of hemoglobin. Copper is another trace-mineral in which deficiencies can occur. A minimum requirement for Cu cannot be given with great accuracy, since Cu absorption and utilization in the animal can be markedly affected by several mineral elements and other dietary factors. Zinc, Fe, molybdenum (Mo), inorganic sulfate and other nutrients can reduce Cu absorption.

Functions:

- It is required for the activity of enzymes associated with Fe metabolism, elastin and collagen formation, melanin production and the integrity of the central nervous system
- It is required for normal red blood cells formation by allowing Fe absorption from the small intestine and release of Fe in the tissue into the blood plasma. Ceruloplasmin is the Cu-containing transport protein
- Copper is required for bone formation by promoting structural integrity of bone collagen and for normal elastin formation in the cardiovascular system
- Copper is required for normal myelination of brain cells and spinal cord as a component of the enzyme cytochrome oxidase, which is essential for myelin formation
- Maximum immune response is also dependent on Cu as indicated by depressed titers in deficient animals.

Iodine:

Iodine deficiencies are expressed mostly as thyroid disorders. Many feedstuffs are deficient in I, thus enlargement of the thyroid is common in captive birds. Iodine can also be derived from the soil but its content can vary considerably across geographic regions. As a result, I concentration in plants and other feedstuffs may also vary, depending upon their region of origin. The thyroid gland contains the highest concentration (0.2-5% on a dry weight basis) of I in the body, 70-80% of the total body stores. Approximately 90% of the I, which passes through the thyroid gland, is captured by it. Iodine is then combined with tyrosine in the thyroid to form diiodotyrosine. Two molecules of this compound are then combined to form thyroxin. Approximately 80% of the thyroxin entering the circulation is broken down through de-iodinization by the liver, kidney and other tissues.

Functions:

- The only known metabolic role of I is as a component of thyroid hormones (thyroxin and triiodothyronine). Thyroid hormones play an integral role in the regulation of the rate of cellular metabolism.
- When thyroid activity is inadequate, metabolic and growth rate and egg production and egg size are reduced. Iodine deficiency in breeders results in low I content of the egg and, consequently, decreased hatchability and thyroid enlargement in the embryos.

Selenium:

The role of Se in preventing exudative diathesis in chicks was established in 1957. Selenium is present in all cells of the body, but the concentration is normally less than 1ppm. Toxic concentrations in liver and kidney are normally between 5-10ppm. Like Fe and I, Se also exists in the soil and its concentration can vary tremendously within a small geographic region.

Functions:

Selenium is an important part of the enzyme glutathione peroxidase. This enzyme destroys peroxides before they can damage body tissues. Vitamin E is also effective as an antioxidant. Therefore, Se works in concert with vitamin E to prevent free radicals that can lead to oxidation of lipids and destruction of cells. This aids the body's defense mechanisms against stress. Most feeds contain compounds that can form peroxides. Unsaturated fatty acids are a good example. Rancidity in feeds causes formation of peroxides that destroy nutrients. Vitamin E for example, is easily destroyed by rancidity. Selenium spares vitamin E by its antioxidant effect as a constituent of glutathione peroxidase.

Selenium and vitamin E are interrelated. Birds need both and both have metabolic roles in the body in addition to an antioxidant effect. In some instances, vitamin E will substitute in varying degrees for Se, or vice versa. However, there are deficiency symptoms that respond only to Se or vitamin E. Although Se cannot replace vitamin E in nutrition, it reduces the amount of vitamin E required and delays the onset of E deficiency symptoms. Selenium also plays a critical role in increasing the immune response.

Sources:

Selenium can be added to diets of chicken either as sodium selenite or sodium selenate up to 0.1ppm in complete feed. Turkeys may be fed up to 0.2ppm. Game birds can also be fed up to 0.1ppm Se in the total diet.

Interaction of other dietary supplements with selenium:

The interaction of Se with other dietary supplements used in poultry feeds and with disease conditions undoubtedly influenced the quantitative requirement of the element for optimum production. Furthermore, the change from complex diets to more simple ones for both broilers and turkeys played a role. This removed the protection of having feed ingredients grown on a variety of soils more apt to supply the needs for Se.

A greater possibility was the presence of higher concentrations of Cu and Zn that would interfere with normal metabolism. Both Cu and Zn are used as dietary supplements in feed manufacturing. Broiler rations commonly contain Cu supplements providing 120-240ppm in addition to the 5-8ppm included in the trace-mineral mix. These levels have been used for years, because they improve performance and are believed to protect against fungal and perhaps other diseases. Adding 1,000ppm Cu to diets containing toxic levels of Se modified the toxicity. A 70% of chicks fed a diet with 80ppm Se died by 2weeks but only 3% in those supplemented with 1,000ppm Cu. A more than 13 times increase in the Se content of the liver in the Cu-fed chicks indicated that a non-deleterious form of the element was stored in this organ.

Manganese:

Manganese was first recognized as a necessary nutrient for animals in the early 1930s. Because Mn is found in many different feeds, a deficiency is less likely than with most of the other trace-minerals. Bone, kidney, liver, pancreas and pituitary gland are the sites of highest Mn concentration. The absorption of Mn in poultry is poor. High dietary intake of Ca, P and Fe reduce Mn absorption, whereas, body has only a limited storage of mobilizable Mn reserves. The absorption as well as excretion of dietary Mn is affected by the extent of the formation of natural chelates in the gut primarily with bile salts.

Functions:

Manganese is essential for chondroitin sulfate synthesis, which is critical to the organic matrix of bone. Many enzymes required for the synthesis of mucopolysaccharides and glycoproteins require Mn

- Manganese is a key component of the metalloenzymes, pyruvate carboxylase and a critical enzyme in carbohydrate metabolism
- Lipid metabolism is also dependent on Mn to allow the liver to convert mevalonic acid to squalene, in cholesterol synthesis.

Zinc:

Zinc has many biochemical functions. Supplemental Zn is usually added to bird diets in the form of zinc oxide or zinc sulfate. Recent comparisons of bioavailability in chicks suggest that feed grade zinc oxide has only 44-78% the availability of zinc sulfate when added to purified or practical diets. Zinc is absorbed equally well when provided as the oxide, carbonate, and sulfate or as metallic Zn. Absorption of Zn occurs throughout the small intestine. Transfer of Zn out of the intestinal mucosal cells to the plasma is regulated by metallothionein. Zinc absorption is reduced whenever diets are high in Ca or phytate.

Functions:

- > Zinc is present in many enzyme systems, which are concerned with the metabolism of feed constituents. For example, it is a constituent of carbonic anhydrase, carboxypeptidase A and B, several dehydrogenases, alkaline phosphatase, ribonuclease and DNA polymerase. Carbonic anhydrase is involved in maintaining the acid-base balance of the blood and other body fluids; it plays an important role in the release of carbondioxide (CO₂) in the lungs, in bone calcification and in the egg shell formation
- Zinc is required for normal protein synthesis and metabolism and it is also a component of insulin so that it functions in carbohydrate metabolism.

Cobalt:

The only known requirement for Co is as a constituent of vitamin B_{12} , which has 4% Co in its chemical structure. The vitamin has very complex chemical structure and contains Co in chelated form. This means that a Co deficiency is really a vitamin B_{12} deficiency. In ruminants microorganisms in the rumen are able to synthesize vitamin B_{12} needs of ruminants, if the diet is adequate in cobalt. Cobalt is necessary for the synthesis of hemoglobin and myoglobin and a component of various enzymes.

Cobalt deficiency has not been demonstrated in poultry. In a few instances where Co has created some response, it is assumed that the diet lacked vitamin B_{12} . If the diet is adequate in vitamin B_{12} , there is no need for Co.

Vitamins

Vitamins are organic substances, which are necessary for the proper operation of vital functions in humans and animals. These substances occur in feeds in small amounts, but are absolutely necessary for most physiological processes like normal growth, feathering, leg development, reproduction and maintenance of health. Deficiencies of various vitamins may cause problems such as, skin lesions, nervous disorders, muscle problems, reduced egg production in layers, reduced growth in meat birds and improper chick development in eggs from breeding birds. The severity of any of these problems will depend on which vitamin is inadequate, and how deficient the diet is.

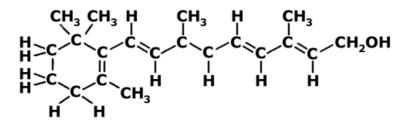
The major factor, which limits the nutritional value of naturally occurring vitamins, is bioavailability. Although chemical analysis may indicate a high level of a vitamin, its bioavailability may be less than 100%. For example biotin is 100% bioavailable in corn but less than 10% available in wheat and barley. The unstable nature of naturally occurring vitamins and the great variation in their levels means that formulating diets to meet the requirements of birds in the intensive poultry keeping based only on natural vitamin levels is almost impossible. It is for this reason that commercially available vitamins are used extensively in the feed industry.

Vitamins are categorized by their solubility in either water or fat. This has relevance to how and where they can be absorbed by the gut. Vitamin concentrations in a diet are generally discussed in terms of activity (International Unit; IU) since, for most vitamins, several forms of differing activity can occur. As with minerals, vitamins are often incorporated into commercial feeds by way of premixes, which are intended to be used at specific concentrations in order to provide the appropriate amounts of individual vitamins.

FAT SOLUBLE VITAMINS:

Vitamin A:

McCollum and Davis first noted Vitamin A requirement in early decades of 20^{th} century. A major storage organ for vitamin A is the liver, with much lesser amounts being found in other organs and muscle. Cod liver oil and other fish liver oils were found to be the best sources of vitamin A. It is not present in plants, although a precursor (β -carotene) is available from plants, especially in leafy green vegetation. Destruction of vitamin A occurs in pelleting and during feed storage. Loss of vitamin A during storage varies from 6-30%, depending upon whether the free alcohol or esterified form is used, presence of an antioxidant, temperature reached during pelleting, etc.



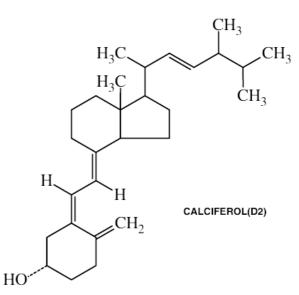
Functions:

- Vitamin A is necessary for the visual processes, health and proper functioning of the skin, mucus membranes and lining of the digestive, reproductive and respiratory tracts and immune system
- In the vision process, as a component of visual pigments it traps light in the eye and trigger nerve impulses via the optic nerve to the brain

- It has an essential role in the synthesis of glycoproteins and mucopolysaccharides, because it is necessary for the addition of glycosyl (carbohydrate like) groups to protein to form glycoproteins that are constituents of cell membranes, connective tissue and mucus secretions
- It is needed for maintaining the integrity of epithelial linings of all the openings of the body to air like alimentary canal, respiratory tract, corneal epithelium, genitourinary tract and the soft tissues around the eyes
- Vitamin A also has role in the cellular proliferation and differentiation.

Vitamin D:

It is aptly known as "sunshine vitamin". It is formed by the irradiation of sterols in plants and in the skin of animals. Vitamin D found in two major forms D₂ (Ergocalciferol) and D₃ (Cholecalciferol). Ergocalciferol



is activated plant sterol whereas cholecalciferol is an animal sterol. They differ slightly in chemical structure. Sunlight plays an important role with regard to vitamin D needs. Ultra violet light contacting the skin converts 7-dehydrocholesterol to vitamin D_3 precursor. It is interesting that vitamin D_2 production

does not require sunlight. In all animals vitamin D_2 is converted to D_3 , the metabolically active form.

Functions:

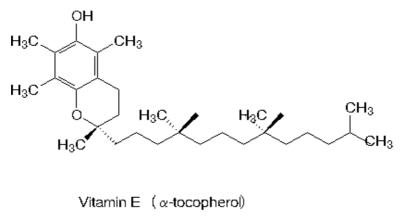
- Vitamin D plays an important role in Ca metabolism, being involved in the Ca absorption and bone mineralization
- The metabolically active form of vitamin D in the body is 1,25-dihydroxy-cholecalciferol (1,25-OHD₃), which is synthesized in the kidneys
- It acts like hormone in controlling Ca absorption and bone mineralization, through inducing the synthesis of Ca-binding protein synthesis.

Sources:

Fish and fish byproducts generally contain sufficient levels of vitamin D_3 , although improper or lengthy storage can greatly diminish these levels. Commercial forms of vitamin D are available but large excesses can lead to toxicities. Bone abnormalities can result with excess as well as a deficiency of this vitamin.

Vitamin E:

The missing factor responsible for the unsuccessful reproduction was first recognized in 1936 as α -tocopherol.



The word *tocopherol* is taken from the Greek, *tokos* meaning "offspring" *pherein* "to bear" and *ol* denoted to "alcohol group attached to benzene ring". Vitamin E exists in several chemical forms known as Tocopherols, with α -tocopherol having the highest potency.

Functions:

- Vitamin E plays a significant role in the reduction of free radicals, which can damage cell membranes and other lipidcontaining structures
- Vitamin E and Se compliment each other in the role of free radical reduction; however vitamin E deficiency cannot be alleviated by increasing Se or vice versa.
- It may also play a role in alleviating heat stress in layers. In some experiments conducted by Rhône-Poulenc, layers were maintained in controlled environment housing at 22°C, then held for one month at 32°C, and finally returned to 22°C. Dietary vitamin E levels of 315IU/kg resulted in higher rates of lay and better-feed conversion efficiency during the hot period and in the following months.
- Other studies have confirmed that elevated dietary vitamin E can enhance disease resistance in pullets raised under heat stress conditions of 34°C for 14 hour/day and 24°C for 10 hour/day (Ward, 1995).

Vitamin E and antioxidant functions:

Vitamin E is known to be a lipid component of biological membranes and is considered a major chain-breaking antioxidant during heat stress. Vitamin E is found mainly in the hydrocarbon part of membrane lipid bilayer towards the membrane interface and in close proximity to oxidase enzymes, which initiate the production of free radicals. Vitamin E is capable of interacting with free radicals in both the aqueous phase and the membrane. It protects cells and tissues from oxidative damage induced by free radicals. It does this primarily by scavenging free radicals, which can directly induce initiation (O_2 , O^- , HO^- , etc) or propagation (lipid peroxyl radicals) of lipid peroxidation. Vitamin E suppresses the radical in reaction by donating its phenolic hydrogen to the oxygen radicals. For example, lipid hydroperoxide is formed together with a vitamin E radical, when vitamin E reacts with the lipid peroxyl radical.

In the peroxidation of lipids, the lipid peroxyl radical acts as a chain carrier (1), where LH, LOO \cdot , LOOH and L \cdot are lipid, lipid peroxyl radical, lipid hydroperoxide and lipid radical, respectively. Vitamin E must scavenge this lipid peroxyl radical before it attacks the lipid molecule (2), where EH and E is tocopherol and -tocopheroxyl radical, respectively.

> $LOO \cdot + LH LOOH + L \cdot (1)$ $LOO \cdot + EH LOOH + E \cdot (2)$

In organic homogeneous solutions, tocopherol scavenges the peroxyl radical about 10 times faster than lipid reacts with the radical. Approximately 90% of the peroxyl radicals are scavenged by tocopherol before they attack the lipid molecules. The tocopheroxyl radical can react with another lipid peroxyl radical to give adduct, which may be reduced to tocopherylquinone.

Vitamin E & meat quality:

Poultry meat is susceptible to off flavors and odors because of its high concentration of poly unsaturated fatty acids (PUFAs). These may give poultry a competitive advantage over red meat among consumers worried about saturated fat intake, but PUFAs are also more prone to oxidation. In addition, restructuring and pre-cooking of meat products significantly increase the susceptibility of muscle tissue to oxidative deterioration.

This wide range in supplemental levels to preserve meat quality is necessary because the optimum level will depend on many factors. Feeding oxidized fats or oils limits oxidative stability within the bird's tissues, as does increased feeding of PUFAs to enhance the meat's fatty acid profile. Environmental stress also affects vitamin E requirements and, thus, the total supplementation needed to elevate tissue levels and avoid oxidation.

Apart from its effectiveness in limiting oxidation, vitamin E supplementation offers the poultry industry two other benefits in improving meat quality. First, vitamin E is a natural dietary antioxidant, thus minimizing any consumer concerns about food additives. Second, supplementation at these levels significantly improves poultry's nutritional profile.

Vitamin E & bird health:

The importance of vitamin E adequacy to a strong immune response has been well documented. Researchers have demonstrated improved response to a wide variety of organisms and diseases, including *E. coli*, Newcastle disease (ND) and infectious bursal disease (IBD), with increased vitamin E supplementation.

Sources:

Several forms of this vitamin exist as compounds known as tocopherols. Vitamin E is only synthesized in plant and bacterial cells. Levels of vitamin E are generally high in fish byproducts, however these levels quickly decrease during storage, especially if the fish contains excessive fats and oils. Cereals are also a good source of vitamin E.

| | No of Sub-straight | | | % Deviation from commercial target | | |
|----------------|---|---|----------------------|------------------------------------|--------------------|--------------------|
| Flock category | No. of flocksSub clinical IBD1 status | Sub clinical IBD ¹ status | Dietary vitamin E | Feed efficiency | Weight per bird | Flock mortality |
| А | 19 | Negative | High | -2.58 | +0.33 | -0.53 |
| В | 17 | Negative | Medium | -2.13 | -0.57 | -1.11 |
| С | 20 | Positive | High | -0.13 | -3.70 | -0.71 |
| D | 23 | Positive | Medium | +1.32 | -5.90 | -0.39 |

 Table 1. Vitamin E supplementation and broiler performance

<u>1</u>. Infectious bursal disease

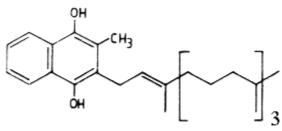
(Mcllroy et al., 1993)

Vitamin K:

The name vitamin K was proposed for it from Danish word "Koagulation" which stands for coagulation i.e. why it is also

as

anti-



trans-vitamin K -hydroquinone

coagulation vitamin. There are two natural forms of vitamin K exist; phylloquinone (K_1), which is synthesized by green plants and menaquinone (K_2), synthesized by gut bacteria. Menadione is a commercial water-soluble form of the vitamin. Vitamin K is generally not toxic except when included at very high concentrations, thus the vitamin can be routinely supplemented in most diets without much concern.

Functions:

known

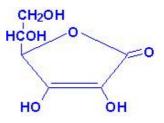
- Vitamin K functions in the blood clotting processes
- It is essential for the activation of prothrombin (a plasma protein) by catalyzing the addition of carbondioxide (CO₂) to glutamic acid residues in prothrombin, creating Ca binding sites
- During the clotting process, prothrombin is activated to thrombin by addition of Ca; thrombin converts the soluble protein fibrinogen to insoluble fibrin, forming the clot.

WATER-SOLUBLE VITAMINS:

The B-vitamins function as cofactors of enzymes involved in energy metabolism. They include thiamine (B_1) , riboflavin (B_2) , pyridoxin (B_6) , pantothenic acid, niacin, choline, folic acid, biotin, ascorbic acid (C), cyanocobalamine (B_{12}) etc.

Vitamin C:

Vitamin C (ascorbic acid) can be synthesized in the kidneys of birds. Because the ability to synthesize this vitamin is variable among species, and excess can be given with little concern for toxicity, this vitamin should be routinely supplemented for all captive birds.



Vitamin C & stress:

Vitamin C has been shown to have some beneficial effects in counteracting the effects of heat stress with egg production, fertility and hatchability all responding positively to Vitamin C supplementation to 100mg/kg. Although it has not been determined exactly how vitamin C supplementation helps negate the effects of stress.

The level of dietary supplemental vitamin C needed to help combat the harmful effects of stress depends on a number of factors, including the type and severity of stress and the age and reproductive status of the bird.

Vitamin C & bone development:

The increase in bone strength is especially important because stress on leg bones increased as body weights rose while frame size remained essentially unchanged. It has been estimated that weak bones cause losses up to 15% of broilers and turkeys. McDowell (2000) has suggested that many cases of "field rickets" in poults may ultimately be due to stress-induced vitamin C deficiency.

In addition, as improvements in egg shell quality suggest, vitamin C adequacy is also required for proper Ca utilization. Specifically, ascorbic acid is a cofactor in the conversion of vitamin D to the active hormonal metabolite calcitriol $[1,25-(OH)_2D_3]$. It is this metabolite, not vitamin D itself, which stimulates intestinal absorption of Ca by elevating activities of duodenal Ca-binding protein and thus elevates plasma Ca to a level that supports normal mineralization of bones. Reductions in collagen synthesis and (or) calcitriol conversion would be especially likely in young birds because the ability to biosynthesize vitamin C develops gradually.

Role of vitamin C in various classes of poultry:

Growing birds:

Vitamin C supplementation to stressed growing birds shows benefits from day first to end. Whether the stress comes from necessary management practices like beak trimming and vaccinations or from disease, heat stress or pre-slaughter cooping, studies have shown that the performance of growing birds receiving dietary vitamin C exceeds that of unsupplemented birds.

Without supplemental vitamin C, the young chick may be particularly handicapped in responding to stress, because the ability of poultry to synthesize vitamin C appears to be age related. It has been estimated that the day-old chick's ability to synthesize vitamin C is only $1/6^{th}$ to $1/3^{rd}$ that of a 3-4 week old bird. With the growth birds face new environmental and immunological stress, either alone or in combination, the benefits of supplemental vitamin C continue.

Breeders and layers:

The supplemental vitamin C may favorably affect reproductive performance in poultry during stress, due to its involvement in the synthesis of steroid hormones, including the sex hormones. Breeding and laying hens also exhibit heat stress in a number of ways. Mortality was noticed to be more than 1% less during a 3-month period, when older heat-stressed hens received 100 or 200ppm of dietary ascorbic acid. Both levels of ascorbic acid also favorably affected internal egg quality and egg shell quality.

Egg shell weight and egg specific gravity also increased with 250 or 500ppm of dietary supplemental vitamin C in the study conducted by Zapata and Gernat (1995). Egg production increased by about 5% with either level of vitamin C fortification in this study. Hens that received neither vitamin D nor vitamin C averaged as many as 16.48% cracked or soft-shelled eggs by 79weeks of age (Figure 1).

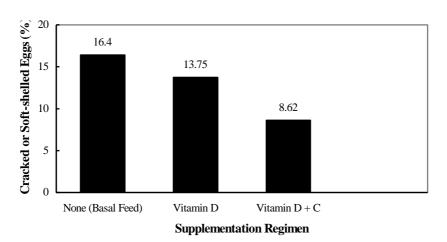
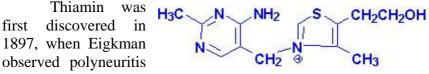


Figure 1. Vitamin supplementation and cracked or softshelled eggs in older hens

<u>Note</u>. Vitamin D = $2\mu g \ 1,25$ -(OH)₂D₃/kg of feed; vitamin C = 100ppm (Weiser, *et al.*, 1990)

With vitamin D supplementation of $2\mu g$ of calcitriol, cracked and soft-shelled eggs in comparable hens were reduced to 13.75%. More impressive still, however, including 100ppm of vitamin C as well as D reduced cracked and soft-shelled eggs to 8.62%. This represented a 37% reduction in cracked or soft-shelled eggs over the hens that received only vitamin D supplementation.

Thiamin (B₁):



in chickens fed only polished rice. This water-soluble antineuretic factor was named as vitamin B_1 . Upon the establishment of its chemical nature, it was named as thiamin. It is one of the most important B-complex vitamins. It is involved as cofactor in several important biochemical reactions in the body.

Absorption:

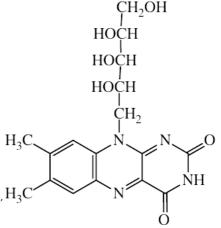
Absorption of vitamin occurs in small intestine, from where it is transported to the liver for phosphorylation under the action of ATP to form cocarboxylase. A very little vitamin is stored in the body and excreted readily from the body through urine. Thus body requires a constant supply and the unneeded intakes go to waste.

Functions:

- The major function of thiamin is to regulate carbohydrate metabolism. It is important for normal function of nerve tissue and heart muscle
- Thiamin is the component of enzymes involved in decarboxyaltion (CO₂ removal) and transketolation- type reactions such as the conversion of pyruvate to acetate in carbohydrate HOCH HOCH

Riboflavin (B₂):

After the complex nature of this water-soluble



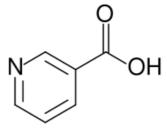
vitamin was recognized, it was assumed that the effects, which could not be ascribed to a lack of anti-neuretic factor, were due to lack of another single factor, termed vitamin B2. The term B2 is now reserved for vitamin Riboflavin. Riboflavin acts as a cofactor for many enzymes involved in oxidation-reduction. It is commercially available as crystalline vitamin. It is stable to heat but sensitive to degradation by light. Cereals and their byproducts are relatively low in riboflavin in contrast to their abundant thiamin contents.

Functions:

- It constitutes the prosthetic group of number of enzymes in the body, which are involved in protein and fat metabolism, including, cytochrome reductase, lipoamide dehydrogenase, xanthine oxidase, L- and D- amino acid oxidases and histaminase. All of these enzymes are concerned with the oxidation-reduction reaction, involved in tissue respiration
- The major function of flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) is to transfer hydrogen between the nicotinic acid containing coenzymes, nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP) and the Iron porphyrin-cytochromes.

Niacin:

Niacin is available in commercially two forms. nicotinamide and nicotinic acid. It is stable in the presence of heat, oxygen, moisture and light. Niacin in cereal grains (especially corn) is bound to amino acids and has low



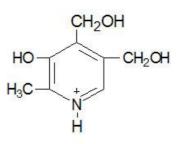
bioavailability. Tryptophan may be converted into niacin; however the efficiency is poor and not recommended as a substitute for diet supplementation. Nicotinic acid is widely distributed among common poultry feeds, but the availability from cereal grains is very low. Animal and fish byproducts are good sources of this vitamin.

Functions:

- Niacin is a constituent of two important coenzymes; NAD and NADP, which serve in number of reactions involved in the carbohydrate, protein and fat metabolism
- In biological oxidation-reduction systems they serve as electron transfer agents
- The NAD is specific for hydrogenases concerned in passing electrons to oxygen via the electron transport system in the Krebs's cycle, serving as electron acceptor in 3 of the 4 dehydrogenation reactions. Whereas NADP is specific for dehydrogenases concerned with the biosynthetic reductions.

Pyridoxine (**B**₆):

Gyorgy (1934) who separated vitamin B_6 from the B-complex defined it as the factor "responsible for the cure of a specific dermatitis developed by young rats on the vitamin free diets supplemented with B_1 and riboflavin". Pyridoxine, pyridoxal and pyridoxamine are the three active forms of vitamin B_6 .



Functions:

It has several roles in protein metabolism, being involved in amino acid inter-conversions (transamination) and decarboxylation. Decarboxylations lead to at least 4-amines that affect nervous system functioning. Transaminations of certain glycolysis and Krebs's cycle intermediates form most of the nonessential amino acids, whereas the reverse is the basis of gluconeogenesis from protein.

Folic Acid:



acid (pteroyl- γ -monoglutamic acid) and the array of extended glutamic acid conjugates. Folic acid is available commercially in crystalline form. It is sensitive to heat and light but is not affected by oxidation.

Functions:

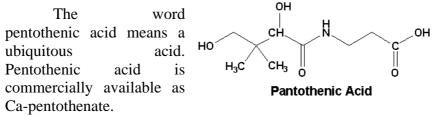
- Folic Acid, a B-vitamin, acts as a carrier of single carbon fragments and is involved in various methylation reactions and enzyme systems
- Because of its role in DNA formation, it is essential for normal cell development and growth. At the cellular level, folacin is essential to the transfer of single carbon units in various reactions, much as pantothenic acid is essential to the transfer of 2-carbon units

Requirements:

The intensive nature of modern poultry production has led researchers to reconsider the folic acid requirements of poultry for optimum nutrition. Although there is still uncertainty over these requirements, recent research suggests a need for dietary supplemental levels greater than NRC requirements. Like other species, poultry rely on both feed sources and microbial synthesis in the intestine to meet their folacin (folic acid) feedstuffs. folacin mainly requirements. In occurs in polyglutamate forms, which must be converted via hydrolysis into monoglutamate derivatives before they are absorbed,

primarily in the duodenum and jejunum. These derivatives are then transported in plasma to the cells, where they are built up again in step like fashion as pteroyl-polyglutamates.

Pentothenic acid:

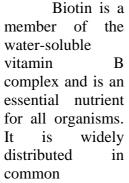


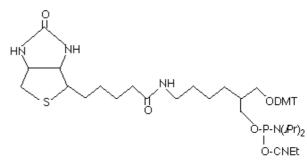
Bioavailability of pentothenic acid is high from barley, wheat and soybean meal but low in corn and sorghum.

Functions:

Pentothenic acid is best known for its role as prosthetic group in coenzyme A and thereby is essential in energy metabolism. Acetate and other fatty acids occur esterified to coenzyme A (acetyl-CoA, fatty acyl-CoA) in metabolism.

Biotin:





feedstuffs but its bioavailability is low in some feeds (wheat, barley, sorghum and oats). Synthetic Biotin is quite expensive

and should be carefully considered before supplementation. It can be readily destroyed in the feed by rancidity so protected by adding antioxidant to the feed.

Functions:

- Biotin is important for poultry in several different ways. As a co-enzyme, the function of biotin is to fix and hold CO₂ in such a way as to permit the enzyme to transfer the CO₂ to the appropriate substrate
- Carboxylation (CO₂ transfer) plays an essential role during the process of gluconeogenesis and fatty acid synthesis
- All living creatures must maintain a steady supply of glucose in the blood to provide active cells with the energy they continuously need. Here, biotin is indispensable through its role in gluconeogenesis. Pyruvate carboxylase is a biotindependent enzyme in the gluconeogenesis pathway where it permits the maintenance of normal blood sugar concentration by controlling the conversion of stored energy into glucose
- In fatty acid synthesis, it controls the carboxylation of acetylcoenzyme A to malonyl-coenzyme A (acetyl-CoA carboxylase is biotin dependent). Biotin also affects protein synthesis through its influence on the nature and rate of formation of ribonucleic acid.

Sources:

Biotin is present in many feeds, but is only partially available to poultry. In particular the biotin in many cereals, such as wheat, barley and oats is very poorly available compared to biotin in maize or soybean meal (Table 2).

 Table 2. Average available biotin contents in raw materials used in poultry diets

| Raw material | Raw material Average biotin contents | | Available biotin contents | |
|--------------|--------------------------------------|-----|------------------------------|--|
| | (µg/kg DM) | (%) | $(\mu g/kg DM)$ | |
| Wheat | 101 | 5 | 5 | |

| Barley | 140 | 20 | 28 |
|-----------------|-----|-----|-----|
| Oats | 246 | 40 | 98 |
| Maize | 52 | 100 | 52 |
| Sorghums | 287 | 25 | 72 |
| Cottonseed meal | 230 | 90 | 207 |
| Fish meal | 135 | 100 | 135 |

(Roche Vitamins, 2000) Table 3 Recommended highlight levels (mg/kg) for various avian species

| Table 5. Recommended bloth levels (ing/kg) for various avian species | | | |
|--|---------|-----------------------------|--------|
| | | Supplementary | Age |
| | | biotin in feed ¹ | (days) |
| Chick | Starter | 0.1-0.2 | 1-21 |

| | | biotin in feed ¹ | (days) |
|---------|-----------------|-----------------------------|--------|
| Chick | Starter | 0.1-0.2 | 1-21 |
| | Grower | 0.1-0.15 | 22+ |
| Broiler | Starter | 0.1-0.2 | 1-21 |
| | Grower/finisher | 0.15-0.25 | 22+ |
| | Breeder | 0.2-0.4 | |

1. Wheat based diets for poultry should use the higher recommendation. Note: Where birds are known to be stressed by extremes of the environment or by disease the biotin supply should be increased by a factor of 2 or 3

(Roche Vitamins, 2000)

Requirements:

The recommended supplements of biotin listed in table 3 are the optimum additions for most feed mixtures for the specific species and age. They include the amounts necessary for normal nutrition, a safety margin and where appropriate an additional amount for optimum performance.

Cyanocobalamine (B₁₂):

Vitamin B_{12} is isolated from liver in 1948 and is also called as anti-pernicious anemia factor. It is dietary essential for poultry because it does not occur in plants. The concentration of vitamin B_{12} is high in spleen, bone marrow, liver, kidney and skin. It is available in crystalline form as cyanocobalamine. It has good stability in feeds. The complex structure of cyanocobalamine is shown in the figure 2.

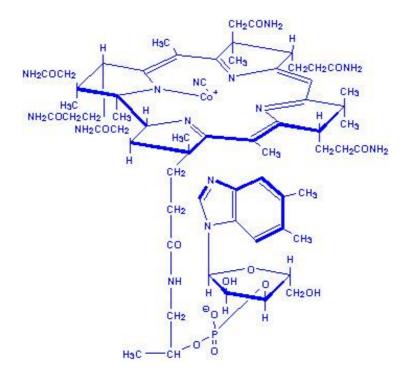


Figure 2. A molecule of vitamin B₁₂

Functions:

- Vitamin B₁₂ is a cofactor for enzymes transferring 1-carbon units and catalyzing rearrangements in the carbon skeleton of several metabolic intermediates
- > In fowl, vitamin B_{12} mediated 1-carbon transfers involve methionine, serine, choline and thymidine, whereas the interconversion of methylmalonyl coenzyme A to succinyl coenzyme A is one of the rearrangement reactions requiring vitamin B_{12}
- It along with folic acid has a dual role in the formation of erythrocytes.

Choline:

Choline is often considered as vitamin, although it is not. However it is dietary essential. It can be synthesized in the body using methyl groups from methionine but it is less expensive to supplement choline.

Functions:

- Choline is a constituent of phospholipids, lecithin and sphingiomyelin
- It is also essential for nerve function and as a component of acetylcholine it released at the termination of the parasympathetic nerves
- Choline after its conversion to betaine provides labile methyl groups for the formation of methionine from homocystein (Figure 3) and of creatine from guanidoacetic acid.
- Therefore, choline spares dietary methionine for other metabolic functions.

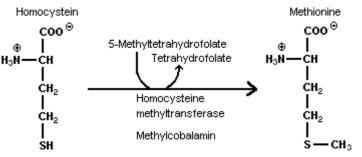


Figure 3. Transfer of homocystein to methionine

Provitamins:

Provitamins are a class of substances closely related to the vitamins. Most of them resemble carotene. The term "carotenoid" is derived from β -carotene, the pigment of carrots. Carotenoids are widely distributed in nature and are found in

grasses, carrots, paprika and citrus fruits. Chemically they are very similar to vitamin A.

Vitamin withdrawal and performance:

In a broiler study, the effects of withdrawing supplemental vitamins and trace-minerals from the feed in days 28 through 49 were reported (Table 4). When both were withdrawn, growth rate and feed efficiency declined even when the birds weren't heat stressed. Trace-mineral withdrawal from the diet did not affect live production performance, carcass characteristics or immunocompetence. When the trace-minerals were left in the diet and vitamins withdrawn, gain, feed efficiency, survivability and carcass traits all suffered. It appears from these studies that vitamin withdrawal, or inadequate levels of vitamins, can be damaging to growth rate and immune response.

Table 4. Effects of vitamin and trace-mineral withdrawal on broiler performance

| | Basal diet | Withdrawa l of vitamins | Withdrawa l of trace- minerals | Withdrawa l of vitamin and trace- minerals | Poole d SEM ¹ |
|--------------------------------|-------------------|-------------------------------|--------------------------------------|---|--------------------------------|
| Weight gain, g | 1279 ^a | 1103 ^c | 1254 ^{ab} | 1242 ^b | 13 |
| Feed efficiency | 0.37 ^a | 0.32 ^c | 0.36 ^{ab} | 0.35 ^b | 0.01 |
| Survivability | 90 ^a | 83 ^b | 88 ^a | 87 ^{ab} | 2 |
| Antibody titre ² | 8.8 | 8.6 | 9.09 | 9.02 | 0.22 |
| Specific gravity | 1.051 a | 1.047 ^b | 1.05 ^a | 1.05 ^a | 0.00 |
| Fat pad, % CW^3 | 1.6 | 1.7 | 1.7 | 1.9 | 0.14 |

1. Standard error of mean

(Deyhim and Teeter, 1993)

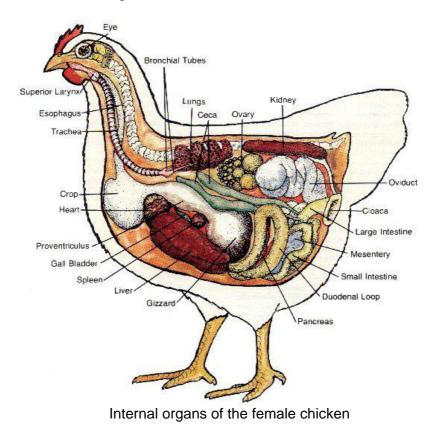
<u>**2**</u>. Antibody titer to sheep red blood cells

3. Carcass Weight $\frac{abc}{abc}$ Means within a row without common superscripts differ significantly (P<0.05)

Chapter 8

Gastrointestinal Tract

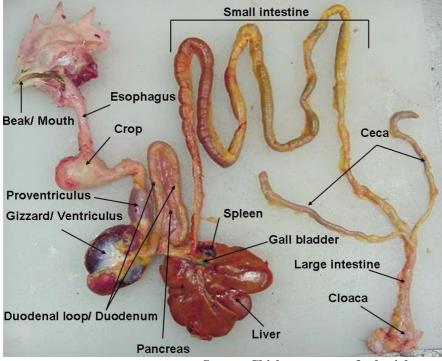
Avian species vary in their gastrointestinal organization and nutritional needs. Most of the knowledge of avian nutrition comes from research on production birds, including chickens and turkeys. However, other species like raptors, sea birds and browsers (grouse) still require much research just to define their basic nutrient requirements.



AVIAN DIGESTIVE TRACT:

The digestive tract is a tube running from mouth to cloaca. This tube is like an assembly line, or more properly, a disassembly line. Its chief goal is to break down huge macromolecules (proteins, fats and starch), which cannot be absorbed intact, into smaller molecules (amino acids, fatty acids and glucose) that can be absorbed across the wall of the tube, and into the circulatory system for dissemination around body. Digestion in birds involves a lot of organs, each performing a specific function. It begins with entry of food via beak and ends with exiting of refuse at vent. Digestive system of poultry is depicted in figure 1.

Proper function of the digestive system requires robust control systems. These systems must facilitate communication among different sections of the digestive tract and between the digestive tract and the brain. Control of digestive function is achieved through a combination of electrical and hormonal messages, which originate either within the digestive systems, own nervous and endocrine systems, as well as from the central nervous system (CNS) and from endocrine organs such as the adrenal gland.



Source: Chicken anatomy & physiology

Figure 1. Digestive system of poultry

The mouth:

The avian beak (rostrum) is composed of bone and an outer horny keratin sheath. Keratin replacement continues throughout the life of the bird. In large parrots, the keration is worn down by digging, eating and chewing on hard objects and is completely replaced in 6 months on the upper bill (maxillary rostrum) and in 2-3 months on the lower bill (mandibular rostrum). The cutting edges of the beak are tomia, which take the place of teeth. In some parrots, the under-side of the upper beak has ridges for holding food and maintaining the edge of the lower beak. The tip of the upper and lower beak is richly supplied with nerve endings. The tongue, just as the beak, is adapted to the type of food the bird consumes. Parrots, birds of prey and finches have short, thick, fleshy tongues, which allow them to manipulate their food. Fowl and pelicans have non-protrudible tongues with caudally directed papillae, which allow the food to be easily shoved to the back of the mouth for swallowing. Birds do not have an epiglottis to prevent food from entering their larynx. Instead, the opening of the glottis closes during swallowing.

Numerous small salivary glands are found in the oral cavity. These are best developed in birds that eat a dry diet such as seed and insect eaters and least developed in birds that eat a moist diet such as fish. Mucus is the major substance secreted by the avian salivary glands. It acts as a lubricant in swallowing.

Esophagus and crop:

The esophagus transports food from the mouth to the stomach. Many species of birds (chicken and parrots) have an enlarged area of the esophagus known as a crop anterior to the stomach. The size and shape of crops vary according to the eating habits of the species, and may be unilobular, bilobed or spindle shaped. The crops of certain granivorus birds are bilobed and large. The crop can serve several purposes, depending on the species. Additionally, in some birds, the crop serves as a transport device for food destined for nestlings that are not yet able to forage on their own.

The stomach:

Birds have a two-part stomach, a glandular portion known as the proventriculus and a muscular portion known as the gizzard (Figure 2). Specialized cells in the proventriculus where chemical digestion begins secrete hydrochloric acid (HCl), mucus and digestive enzyme pepsin. The gizzard has a thick muscular wall and plays an important part in the mechanical digestion by crushing and grinding food.

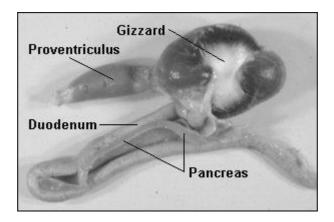


Figure 2. Gizzard, proventriculus, duodenum and pancreas

Fish and meat eaters have a thin-walled sack like stomach that is adapted for storage rather than mechanical digestion. The distinction between the proventriculus and the gizzard is difficult to determine. Seedeaters (such as parrots), insectivores and herbivores have a very well developed gizzard adapted for mechanical breakdown of indigestible food. The proventriculus is easy to distinguish from the gizzard in these birds. The stomachs of carnivores also secrete greater amounts of HCl that destroy any harmful bacteria, which may be present in the meat they consume.

The small intestine:

A bulk of the small intestine is suspended from the body wall by an extension of the peritoneum called the mesentery. The small intestine is the primary site of chemical digestion and nutrient absorption. It is the longest section of the digestive tube and consists of 3 segments forming a passage from the pylorus to the large intestine and arranged in U-shaped loops in the abdominal cavity. 1) Duodenum, a short section that receives secretions from pancreas and liver via pancreatic and common bile ducts, 2) Jejunum considered to be roughly closer to 90% in animals, and 3) Ileum is the portion that empties into the large intestine. It is the short terminal section of small intestine.

The liver lies in the abdominal cavity, in contact with diaphragm. The large liver is divided into a right and left lobe. In most species the right lobe is larger than the left. In some species, such as songbirds, the right lobe is subdivided. The liver has numerous functions among which, is the production of bile. Bile is important in the breakdown of fat and is stored in the gallbladder.

The digestive tract is sterile at hatching. Altricial chicks are the chicks born blind and helpless and are inoculated with microflora when parents feed them. Pre-cocial chicks are the chicks that are able to feed on their own shortly after hatching, are inoculated by bacteria from their environment during feeding.

The microorganisms in the intestine produce the enzyme cellulase, which assists in the breakdown of cellulose; a fibrous component of plant cell walls. Enzymes in the plant food itself will also aid in the breakdown of cellulose in the intestinal tract. The parrot utilizes some of the carbohydrates, vitamins, minerals and protein from the cellulose material.

The large intestine:

The large intestine is the part of digestive tube between terminal ileum and anus. Ingesta from the small intestine enter large intestine through ileo-cecal valve. Within large intestine, major segments are recognized as: 1) Cecum, which is a blindended pouch and unlike mammals, these are usually paired structures (caeca) in poultry; 2) Colon, that constitutes the majority of the length of large intestine and is sub-classified into ascending, transverse and descending segments; and 3) Cloaca is short, terminal segment of digestive tube, continuous with the anal canal. The large intestine is the last attraction in digestive tube and the location of terminal phases of digestion. It functions in 3 processes:

1. Recovery of water and electrolytes from ingesta:

By the time ingesta reaches the terminal ileum, roughly 90% of its water has been absorbed, but considerable water and electrolytes like sodium (Na) and chloride (Cl) remain and must be recovered by absorption in the large gut.

2. Formation and storage of faeces:

As ingesta moves through the large intestine, it is dehydrated, mixed with bacteria and mucus, and formed into feces.

3. Microbial Fermentation:

The large intestine of all species contains microbial life. These microbes produce enzymes capable of digesting many of molecules that are indigestible, cellulose being a premier example. The extent and benefit of fermentation also varies greatly among species.

The large intestine is composed of paired caeca and rectum. The shape and size of the caeca vary in different species of birds. The caeca of the chickens are well developed while those of the songbirds are very small. Caeca are very rudimentary or absent in some species such as parrots and pigeons. Cellulose breakdown by bacteria occurs in the caeca. The caeca in birds appear to perform much the same function as that of mammals, allowing further degradation of substrates that are more difficult to digest. The large intestine, as in mammals is mostly where fermentation takes place along with absorption of water and water-soluble vitamins, minerals, etc.

GASTROINTESTINAL TRACT SECRETIONS:

Salivary secretions:

Saliva production is exocrine in nature (secreted into ducts which deliver the product to the oral cavity). Salivary secretions serve several important functions. It acts to dissolve food and disperse it in mouth for taste perception and prepare it for further enzymatic action. Saliva also provides some digestive action e.g., amylase, which is a common constituent of saliva, can break down polysaccharides (starch), into simple sugars (maltose and glucose This buffering action is primarily a function of bicarbonate (HCO₃⁻) and phosphate (PO₄) ions, along with electrolytes including Na, Cl and potassium.

Gastric secretions:

Control of gastric activities is accomplished through a complicated array of hormonal and nervous inputs. To complicate matters further, several of the peptide hormones that affect the gastrointestinal tract (GIT) are found both in nerve tissues (including the brain) and in endocrine cells in the gut. A few of gastric secretions are discussed as under.

Gastrin:

Gastrin is released from gastrin cells (G-cells) in the pyloric glandular region of the proventriculus. It stimulates acid (HCl) production by the proventriculus. Gastrin is released in response to both luminal and nervous stimuli. Amino acids and protein fragments in the proventriculus are the main luminal stimuli. Vagus nerves then release acetylcholine (ACH), which stimulates the G- cells to produce gastrin. The GRP is released in response to anticipation of feeding, distention of the proventriculus and other signals.

Pepsin:

Peptic cells (or chief cells), found in the oxyntic glands of the proventriculus, produce pepsinogen. Pepsinogen is a zymogen (pro-enzyme), which is quickly converted to the active proteolytic enzyme, pepsin, in an acid environment. Pepsin can digest most protein, including collagen. This is important since pancreatic proteases and other enzymes released downstream of the stomach have little ability to digest collagen.

Acid:

Hydrochloric acid is secreted from parietal cells into the lumen where it establishes an extremely acidic environment. This acid is important for activation of pepsinogen and inactivation of ingested microorganisms such as bacteria.

Mucus:

The most abundant epithelial cells are mucous cells, which cover the entire lumenal surface and extend down into the glands. These cells secrete a bicarbonate-rich mucus that coats and lubricates the gastric surface, and serves an important role in protecting the epithelium from acid and other chemical insults.

Intestinal secretions:

By the time ingesta reach the small intestine, feedstuffs have been mechanically broken down and reduced to a liquid by mastication and grinding in the stomach. Once within the small intestine, these macro-molecular aggregates are exposed to pancreatic enzymes and bile, which enables digestion to molecules capable or almost capable of being absorbed.

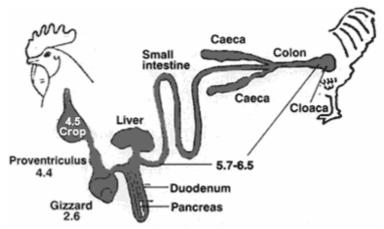


Figure 3. pH in different segments of gastrointestinal tract of poultry

Stomach contents are passed into the small intestine by the pyloric valve. This material is still very acidic at this point, however bile and pancreatic ducts open into the duodenum a short distance downstream of the pylorus and both provide secretions with a high alkaline content that neutralizes the gastric chyme. Figure 3 illustrates the pH in the different important segments of the digestive tract of the poultry.

Food substrates (proteins, fats and carbohydrates) stimulate the production of hormones in the small intestine, as in the proventriculus. While the GIT might not be thought of as an endocrine organ, it has been estimated that the total mass of endocrine cells in the small intestine and stomach exceeds that of all other endocrine glands. Small intestinal hormones include secretin, cholecystokinin (CCK), gastric inhibitory polypeptide (GIP) and motilin, which are produced by specialized glands in the duodenum and upper jejunum.

Large quantities of water are also secreted into the lumen of the small intestine during the digestive process. Almost all of this water is also reabsorbed in the small intestine. Regardless of whether it is being secreted or absorbed, water flows across the mucosa in response to osmotic gradients.

Pancreatic secretions:

As chyme floods into the small intestine from the stomach, 2 things happen, firstly, acid must be quickly and efficiently neutralized to prevent damage to the duodenal mucosa, and secondly macro-molecular nutrients (proteins, fats and starch) must be broken down much further before their constituents can be absorbed through the mucosa into blood. The pancreas produces trypsin, chymotrypsin, carboxy- and amino-peptidases, lipase and ribonucleases.

Trypsin is an endopeptidase, which digests proteins at the carboxyl end of arginine and lysine, two dibasic amino acids. It also plays a major role in activating chymotrypsin from chymotrypsinogen, its respective zymogen. Chymotrypsin is another endopeptidase. Like pepsin, this enzyme cleaves proteins on the carboxyl side of the 3 aliphatic amino acids, tyrosine, phenylalanine and tryptophan. The breakdown of starch by pancreatic amylase; carbohydrates by sucrase, maltase, lactase and other saccharidases; nucleic acids by poly-nucleotidases; purine-containing nucleosides by nucleosidases; and, lecithins by lecithinases, are other salient functions of pancreatic secretions.

In addition to its role as an exocrine organ, the pancreas is also an endocrine organ and secreting insulin and glucagon, which play a vital role in carbohydrate and lipid metabolism. They are absolutely necessary for maintaining normal blood concentrations of glucose.

Bile secretions:

The liver and gall bladder play a major role in the digestion of nutrients, by producing and secreting bile. Bile is a complex fluid containing water, electrolytes and a battery of organic molecules including bile acids, cholesterol, phospholipids and bilirubin that flow through the biliary tract into the small intestine. The primary acids normally enter the bile as taurine and glycine conjugates (taurocholic, glychocholic acids in the case of cholic acid). All are derived from cholesterol through a series of direct or indirect pathways. Once in the intestine, primary acids may be further converted to the secondary acids. More than 90% of bile acids are recycled, via reabsorption by the intestine. Because they are derived from cholesterol, and since cells cannot break down the steroid nucleus of cholesterol, excretion of unabsorbed bile acids is the primary way in which cholesterol is lost from the body. Many waste products are eliminated from the body by secretion into bile and elimination in feces.

The liver is well known to metabolize and excrete into bile many compounds and toxins, thus eliminating them from the body. Examples can be found among both endogenous molecules (steroid hormones, calcium) and exogenous compounds (many antibiotics and metabolites of drugs). The most important and clinically relevant examples of waste elimination via bile is that of bilirubin. Additionally, the mechanisms involved in elimination of bilirubin are similar to those used for elimination of many drugs and toxins.

HORMONES OF THE GASTROINTESTINAL TRACT:

Their are 3 important hormones of the GIT namely CCK, secretin and gastrin.

1. Cholecystokinin:

This hormone is synthesized and secreted by enteric endocrine cells located in the duodenum. The name of this hormone describes its effect on the biliary system. *Cholecysto* means gallbladder and *kinin* is movement once released, it stimulates contractions of the gallbladder and common bile duct, resulting in delivery of bile into the gut. As chyme floods into the small intestine, CCK is released into blood and binds to receptors on pancreatic acinar cells, ordering them to secrete large quantities of digestive enzymes.

2. Secretin:

This hormone is also a product of endocrinocytes located in the epithelium of the proximal small intestine. Secretin is secreted in response to acid in the duodenum, which of course occurs when acid-laden chyme from the stomach flows through the pylorus. The bicarbonate and water expands the volume of bile and increases its flow out into the intestine.

3. Gastrin:

This hormone is secreted in large amounts by the G-cells, scattered among the secretory epithelial cells in the stomach, in response to gastric distention and irritation. Being a hormone, gastrin is secreted into blood, not into the lumen of the stomach.

The enteric nervous system:

The nervous system exerts a profound influence on all digestive processes, namely motility, ion transport associated with secretion and absorption, and gastrointestinal blood flow. Some of this control emanates from connections between the digestive system and CNS, but the digestive system is endowed with its own, local nervous system referred to as the enteric or intrinsic nervous system.

Enteric neurons secrete an intimidating array of neurotransmitters. The major neurotransmitter produced by enteric neurons is acetylcholine. In general, neurons that secrete acetylcholine are excitatory, stimulating smooth muscle contraction, increases in intestinal secretions, release of enteric hormones and dilation of blood vessels.

The enteric endocrine system:

The second of the two important systems that control digestive function is the endocrine system, which regulates function by secreting hormones. Hormones are chemical messengers secreted into blood that modify the physiology of target cells.

Hormones produced in many endocrine glands affect digestive function, but hormones produced within the GIT exert the most profound control. The GIT is the largest endocrine organ in the body and the endocrine cells within it are referred to collectively as the "enteric endocrine system". Three of the enteric hormones are, Gastrin, CCK and secretin. Gastrin is secreted from the stomach and plays an important role in control of gastric acid secretion.

To illustrate how control is implemented through the enteric endocrine system, consider the important example of preventing stomach acid from burning the epithelium of the small intestine. Acid-laden ingesta flow out of the stomach, into the small intestine, where acid in the small intestine stimulates the secretion of the hormone secretin from endocrine cells in the intestinal epithelium. Secretin stimulates the pancreas to dump a bicarbonate-rich fluid into the lumen of the intestine.

Gastrointestinal motility:

The digestive tube conducts fundamental patterns of motility, which is "propulsion". Feedstuffs must be propelled along the length of the digestive tube in order to be subjected to the sequential series of processing involved in disassembly and absorption. The principal type of propulsive motility, seen in the esophagus and small intestine, is "peristalsis". A ring of muscle contraction appears on the oral side of a bolus of ingesta and moves towards the anus, propelling the contents of the lumen in that direction; as the ring moves, the muscle on the other side of the distended area relaxes, facilitating smooth passage of the bolus. Chapter 9

Absorption and Metabolism

Absorption:

The small intestine is the portal for absorption of virtually all nutrients into blood. Accomplishing this transport entails breaking down large supramolecular aggregates into small molecules that can be transported across the epithelium. The net effect of passage through the small intestine is absorption of most of the water and electrolytes {sodium (Na), chloride (Cl), potassium (K)} and essentially all dietary organic molecules (including glucose, amino acids and fatty acid). Absorption processes fall into 3 broad categories.

Simple diffusion:

It can be driven by concentration gradients, if the membrane is permeable to the substrate. Volatile fatty acids (VFA) and simple lipids are absorbed through this process. Once the compounds cross into a cell membrane they can be altered or moved by other means so that the gradient can persist for further absorption of like molecules.

Facilitated diffusion:

It is again driven by concentration gradients. However, in this case the membrane is not permeable to the substance and carrier molecules are required to traverse the membrane. As such, these systems are saturable, since only a limited number of carrier molecules may be available. This type of mechanism absorbs some sugars.

Active Transport:

It can occur in the absence of a concentration gradient or even against a gradient. This mode of absorption requires energy, usually in the form of adenosine triphosphate (ATP). This process absorbs most sugars, amino acids and short peptides.

All of this absorption and much of the enzymatic digestion takes place on the surface of small intestine epithelial cells and to accommodate these processes, a huge mucosal surface area is required.

Virtually all nutrients, including amino acids and sugars, enter the body across the epithelium covering small intestine villi. Each villus contains a capillary bed and a blunt-ended lymphatic vessel referred to as the "central lacteal". After crossing the epithelium, most of these molecules diffuse into a capillary network inside the villus, and hence into systemic blood. Some molecules, fats in particular, are transported not into capillaries, but rather into the lymphatic vessel, which drains from the intestine and rapidly flows into blood via the thoracic duct.

It's probably fair to say that the single most important process that takes place in the small intestine to make such absorption possible is establishment of an electrochemical gradient of Na across the epithelial cell boundary of the lumen. To remain viable, all cells are required to maintain a low intracellular concentration of Na. Low intracellular Na is maintained by a large number of Na⁺/K⁺ ATPases, so called "Na-K pumps" embedded in the basolateral membrane.

Sodium is absorbed into the cell by several mechanisms, but chief among them is by co-transport with glucose and amino acids. It reflects that efficient Na absorption is dependent on absorption of these organic solutes. When a lot of Na is entering the cell, a lot of Na is also pumped out of the cell, which establishes a high osmolarity in the small intercellular spaces between adjacent enterocytes.

Water absorption:

Water is absorbed into the intercellular space by diffusion down an osmotic gradient. Transport of water from lumen to blood is often against an osmotic gradient. This is important because it means that the intestine can absorb water into blood even when the osmolarity in the lumen is higher than osmolarity of blood. This ability is best explained by the "3-compartment model" for absorption of water and, like many aspects of gut permeability (Figure 1).

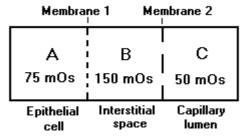


Figure 1. 3-compartment model for water absorption

Carbohydrate absorption:

Polysaccharides and disaccharides must be digested to monosaccharides prior to absorption and the key players in these processes are the brush border hydrolases, which include maltase, lactase and sucrase. Dietary lactose and sucrose and maltose derived from digestion of starch, diffuse in the small intestine lumen and come in contact with the surface of absorptive epithelial cells covering the villi where they engage with brush border hydrolases. Maltase, lactase and sucrase cleave maltose, lactose and sucrose, respectively, into simpler molecules of glucose, galactose and fructose.

Absorption of glucose entails transport from the intestinal lumen, across the epithelium and into blood. The transporter that carries glucose and galactose into the enterocyte is the "Nadependent-hexose-transporter", known more formally as SGLUT-1. This molecule transports both glucose and Na into the cell and in fact, will not transport either alone. Once inside the enterocyte, glucose and Na must be exported from the cell into blood. Glucose is transported out of the enterocyte through a different transporter (called GLUT-2) in the basolateral membrane. Glucose then diffuses "down" its concentration gradient into capillary blood within the villus.

Fermenting microflora in the caeca produces VFA. Substrates for fermentation may be fiber and complex oligosaccharides. However, starch, which is found in large concentrations in the grain-based diets, is more likely the substrate responsible for VFA production in monogastrics. Regardless of the impact of VFA on the total energy balance of monogastrics, it should be remembered that VFA are probably the primary source of energy for the gut mass; with some reports indicating these organic acids contribute up to 70% of intestinal energy needs.

Amino acid and peptide absorption:

The brush border of the small intestine is equipped with a family of peptidases. Like lactase and maltase, these peptidases are integral membrane proteins rather than soluble enzymes. They function to further hydrolysis of lumenal peptides, converting them to free amino acids and very small peptides. These end products of digestion, formed on the surface of the enterocyte, are ready for absorption.

The lumenal plasma membrane of the absorptive cell bears at least 4 Na-dependent amino acid transporters; one each for acidic, basic, neutral and aliphatic amino acids. These transporters bind amino acids only after binding Na. Thus, absorption of amino acid is also absolutely dependent on the electrochemical gradient of Na across the epithelium.

Neutral amino acids such as leucine, glycine and valine are all transported by a common system. Basic amino acids such as arginine and lysine also share a common transport system, as do acidic amino acids, including aspartate and glutamate. Proline and hydroxyproline, lending to their unusual structures, require yet another separate transport system. Recent evidence also suggests active transport of short peptides across the gut membrane. Transport of peptides (usually di- or tri-peptides) may occur via the same systems described above, or it may require other specialized systems. Additionally, some larger peptides can be absorbed intact by endocytosis.

There is virtually no absorption of peptides longer than 3 amino acids. However, it seems that there is abundant absorption of di- and tripeptides in the small intestine. These small peptides are absorbed without dependence on Na, probably by a single transport molecule. Once inside the enterocyte, the vast bulk of di- and tripeptides are digested into amino acids by cytoplasmic peptidases and exported from the cell into blood. Only a very small number of these small peptides enter blood intact.

Lipid absorption:

As with amino acids, the majority of lipids are absorbed in the small intestine in avian species. Lipids are presented to the small intestine in much the same condition as they are consumed. The bulk of dietary lipid is neutral fat or triglyceride, composed of a glycerol backbone with each carbon linked to a fatty acid. Additionally, most feedstuffs contain phospholipids, sterols like cholesterol and many minor lipids, including fat-soluble vitamins. In order for the triglyceride to be absorbed, 2 processes (Figure 2 and 3) must occur. Firstly, large aggregates of dietary triglyceride, which are virtually insoluble in an aqueous environment, must be broken down physically and held in suspension (emulsification). Secondly, triglyceride molecules must be enzymatically digested to yield monoglyceride and fatty acids, both of which can efficiently diffuse into the enterocyte.

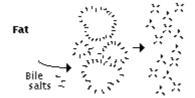


Figure 2. Emulsification of a triglyceride

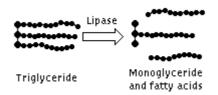


Figure 3. Enzymatic breakdown of triglyceride to monoglyceride and fatty acid

The key players in these 2 transformations are bile salts and pancreatic lipase, both of which are mixed with chyme and act in the lumen of the small intestine. Lipases in pancreatic and bile secretions hydrolyze the ester bonds of triglycerides, liberating the fatty acids of varying lengths from glycerol; generally leaving di- or monoacylglycerides. Di- and monoacylglycerides are more common end products of hydrolysis than free glycerol because as any of the 3 fatty acid chains is hydrolyzed from a triglyceride, it becomes increasingly difficult to remove the second and third chain. Any free glycerol that does result is rapidly absorbed in the small intestine by passive diffusion, as are the shorter fatty acid chains (<14 carbons) that may result.

As monoglycerides and fatty acids are liberated through the action of lipase, they retain their association with bile salts and complex with other lipids to form structures called "micelles" (Figure 4). Micelles are essentially small aggregates of mixed lipids and bile salts suspended within the ingesta. As the ingesta are mixed, micelles bump into the brush border and the lipids, including monoglyceride and fatty acids, are absorbed.

1.



Figure 4. Micelle composition

Bile salts play their critical role in lipid assimilation by promoting emulsification. Bile acids or their salts emulsify monoacylglycerides and longer free fatty acids (FFA), negating any hydrophobic properties. In the process, micelles, with hydrophobic cores and hydrophilic exteriors, are formed. Very low-density lipoproteins (VLDL), which are similar, but smaller than chylomicrons, are also formed to a lesser degree. Following the transport of lipids into the cells, bile acids are returned to the lumen, via active transport, to be reabsorbed in the ileum for later recycling back into the bile system.

Lipids absorbed and incorporated into chylomicrons are not transported directly into the circulatory system, as are carbohydrates and amino acids. Rather, the chylomicrons enter the lymphatic system by way of a small lymph channel, called a lacteal, which traverses each villus. Via the lymph system, chylomicrons can be distributed to the peripheral tissues where lipids can be extracted as needed. Eventually, chylomicrons and chylomicron remnants (what's left following extraction of some lipids) also enter the circulatory system, where further processing can occur in the blood and liver. In this process, there may be a subsequent conversion to VLDL. intermediate density lipoprotein (IDL) and high-density lipoprotein (HDL).

Mineral absorption:

Approximately 90% of water absorption occurs in the small intestine. The remaining water is absorbed in the large intestine. Along with water; these organs absorb electrolytes and minerals as well. Sodium salts are highly soluble in water. Hence

they are rapidly absorbed and circulate through all the tissues of the body.

Some minerals, such as K, are absorbed by passive diffusion. Other substances, such as water, phosphate, magnesium and Cl^- are controlled to some extent by Naconcentrations on either side of the absorptive surface. In some cases, active transport mechanisms, similar to those already described, are employed. In other cases, the concentrations of Na⁺ inside the cell cause movement of other substances based on charge. Anions such as Cl^- fall into this category.

Calcium (Ca) absorption is facilitated by Ca-binding protein in a mechanism that involves active transport to the blood. Some hormonal control is also involved in Ca absorption. Parathyroid hormone promotes formation of vitamin D_3 from vitamin D. Vitamin D_3 uphold synthesis of Ca-binding protein, which then further helps transport of Ca. This absorption process is described in the figure 5.

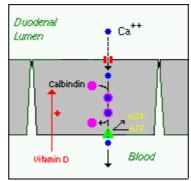


Figure 5. Calcium absorption

As it is well understood, Ca and phosphorus (P) share a somewhat antagonistic relationship. Excess P, e.g. will form insoluble complexes with Ca, thus increasing the dietary need for Ca. Proper dietary Ca: P ratios range from about 1.3:1 to about 2.3:1, depending on the species and phase of growth. A particular disorder associated with improper Ca: P ratio is nutritional secondary hyper-parathyroidism (NSH). At this point, regulation

of Ca is lost and decalcification or osteoporosis of bone occurs, leading to debilitating disorders. Phosphorus is predominantly absorbed as inorganic-phosphate in the upper small intestine.

Iron (Fe) homeostasis is regulated at the level of intestinal absorption, and it is important that adequate but not excessive quantities of Fe be absorbed from the diet. A cellular storage protein called ferritin is involved in absorption and retention in the cells. Ferrous salts are absorbed more efficiently then Fe⁺⁺⁺. Once absorbed, the body tenaciously holds on to the Fe for reuse. For example, most of the Fe released from red blood cell breakdown is used to synthesize new hemoglobin. Iron is co-transported with a proton into the enterocyte via the divalent metal transporter (DMT-1). This transporter is not specific for Fe, and also transports many divalent metal ions. This process is showed in the figure 6.

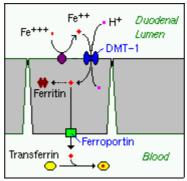


Figure 6. Iron absorption

Sulfur is also absorbed from the small intestine. Free amino acids; thiamine, pyridoxine and biotin are absorbed without decomposition, while the sulfur (S) containing amino acids are absorbed following the cleavage of protein. Absorption and assimilation of S-containing amino acids are determined by the levels of protein and energy in the feed and are affected by active transport mechanisms. Sulfate and sulfite-S taken in with the feed seems to be absorbed by simple diffusion. About 90% of the absorbed-S is excreted through the urine. When inorganic-I is consumed, it is absorbed from the GIT by 2 processes. First is common to other halides such as Cl or bromine, and second is specific for I. The stomach and duodenum not only absorb I, but also secrete it in gastric juice. In fact, gastric juice often has an I-concentration 40 times higher than blood plasma. Iodine absorbed from the blood by the thyroid gland is stored as thyroglobulin. Free-I is conserved and recycled by the body with only 20% being excreted in the urine and feces.

Infact, the dietary concentration of goitrogens is probably a more important determinant of I-status than dietary I-levels, in many cases. Cruciferous plants contain potential goitrogens of the thiouracil type, while brassicas contain cyanogeneicglycosides that are goitrogenic. Goiter can occur, therefore, even though I-level might otherwise be adequate.

Zinc homeostasis is largely regulated by its uptake and loss through the small intestine. Although a number of Zn transporters and binding proteins have been identified in villus epithelial cells but the actual mechanism is not yet well understood. Intestinal excretion of Zn occurs via shedding of epithelial cells and in pancreatic and biliary secretions.

Vitamin absorption:

Absorption of fat-soluble vitamins (A, D, E, and K) is generally a function of the medium in which they dissolve. Thus absorption of lipids is accompanied by absorption of the fatsoluble vitamins, within the micelles or chylomicrons. Likewise, impaired absorption of lipids, which can occur for a number of reasons, will ultimately affect absorption of fat-soluble vitamins with the exception of vitamin K. Some forms of this vitamin, namely the phyloquinones, are known to be absorbed via an active transport mechanism; while other forms, including the menaquinones are absorbed passively with lipid micelles.

Vitamin A and β -carotene are dispersed in micelle structure, composed of monoglycerides, long chain fatty acids

and bile salts along with vitamin D, E and K, which, facilitate absorption of vitamin A and β -carotene to the intestinal epithelial cells where most of the β -carotene gets changed into vitamin A. Dietary vitamin D₃ is absorbed primarily from the first half of the small intestine. Vitamin D₃ absorption is favored by the presence of certain organic acids, particularly lactic acid. In blood, vitamin E is transported through its β -lipoprotein fraction.

Water-soluble vitamins mainly rely on separate active transport systems. Vitamin C, riboflavin, and niacin all require energy for absorption. While thiamine and pentothenic acid can be absorbed by both passive and active mechanisms; the latter mechanism being accelerated when dietary concentrations are low.

Micro flora of both ruminants and non-ruminants synthesize many water-soluble vitamins. Ruminants absorb most water-soluble vitamins (food- and microbial-derived) in the small intestine, simply because that is the organ that deals with the majority of these compounds. In non-ruminants most of the microbial activity occurs downstream of the jejunum, therefore vitamins absorbed in the upper small intestine are mainly of food origin. While vitamins absorbed in the ileum, cecum and colon are mostly of microbial origin.

Metabolism:

Hepatocytes play critical role in synthesizing molecules that are utilized elsewhere to support homeostasis, in converting molecules of one type to another, and in regulating energy balance.

Carbohydrate metabolism:

Most of the monosaccharides occurring in the body are the D-isomers. The major product of digestion and the principal circulating sugar is glucose in the poultry. It is critical to maintain concentrations of glucose in blood within a narrow, normal range. Maintenance of normal blood glucose levels over both short (hours) and long (days to weeks) periods of time is one particularly important function of the liver.

Once glucose enters in the cells, glucose is normally phosphorylated to form glucose-6-phosphate. The enzyme that catalyzes this reaction is hexokinase. The glucose-6-phosphate is either polymerized into glycogen or catabolized. The steps involved outlined in the figure 7. The process of glycogen formation is called glycogenesis, and glycogen breakdown is called glycogenolysis. Excess glucose entering the blood after a meal is rapidly taken up by the liver and sequestered as the large polymer, glycogen

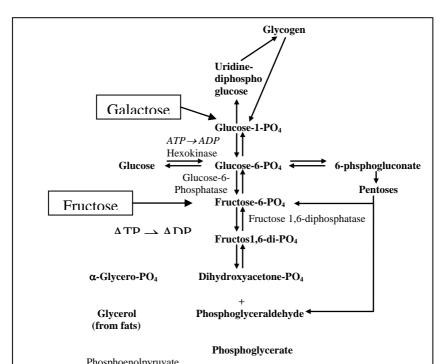
Several hormones regulate glycogen breakdown. Glycogen is synthesized from glucose-1 phosphate with the enzyme glycogen synthase. Glycogen is a branched glucose polymer with a 2 type of glucoside linkage. Cleavage of the 1-4 α linkages in the polymer chain is catalyzed by phosphorylase, whereas cleavage of the 1-6 α linkages at branching points is catalyzed by another enzyme. Phosphorylase is activated in part by the action of epinephrine on β_2 -adrenergic receptors in the liver.

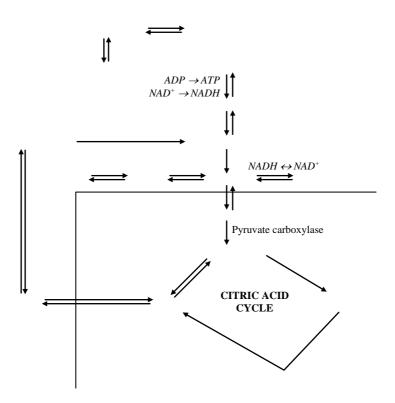
The breakdown of glucose to pyruvate or lactate is called glycolysis. Glucose metabolism proceeds via cleavage through the trioses or via oxidation and decarboxyaltion to pentoses. The pathway to pyruvate through trioses is the Embden-Meyerhof pathway and that through 6-phosphogluconate and the pentoses is the hexose monophosphate shunt.

Other hexoses that are absorbed from the intestine include galactose, which is liberated by the digestion of lactose and converted to glucose in the body; and fructose, part of which is ingested and part produced by hydrolysis of sucrose. Galactose is converted to uridine diphosphogalactose. The uridine diphosphogalactose is converted to uridine diphosphoglucose, which functions in glycogen synthesis. The latter reaction is reversible. The utilization of galactose, like that of glucose, is dependent upon insulin. Fructose is converted into fructose 6phosphate and then metabolized via fructose 1,6-diphosphate. The enzyme catalyzing the formation of fructose 6-phosphate is hexokinase, the same enzyme that catalyzes the conversion of glucose to glucose 6-phosphate.

The liver activates other pathways, when blood concentrations of glucose begin to decline, leading to depolymerization of glycogen (glycogenolysis) and export of glucose back into the blood for transport to all other tissues. After the exhaustion of hepatic glycogen reserves, as occurs when a bird has not eaten for several hours, hepatocytes recognize the problem and activate additional groups of enzymes that begin synthesizing glucose out of such things as amino acids and non-hexose carbohydrates (gluconeogenesis).

Inter-conversion between carbohydrate, protein and fat include conversion of the glycerol from fats to dihydroxyacetone phosphate and conversion of number of amino acids with carbon skeletons resembling intermediates in the glycolysis and citric acid cycle to these intermediates by deamination. In this way and by conversion of lactate to glucose, non-glucose molecules can be converted to glucose. Glucose can be converted to fats through acetyl-CoA but since the conversion of pyruvate to acetyl-CoA, unlike most reactions in glycolysis is irreversible, fats are not converted to glucose via this pathway.





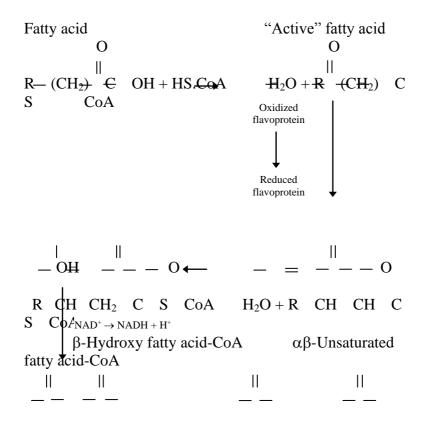
Lipid Metabolism:

In the body, fatty acids are broken down to acetyl-CoA, which enters the citric acid cycle. The main breakdown occurs in the mitochondria by β -oxidation (Figure 8). Fatty acid oxidation begins with the activation of the fatty acid, a reaction that occurs both inside and outside the mitochondria. Medium- and short chain fatty acids can enter the mitochondria without difficulty, but long chain fatty acids must be bound to carnitine in ester linkage before they can cross the inner mitochondrial membrane.

Carnitine is β -hydroxy- γ -trimethylammonium butyrate and it is synthesized in the body from lysine and methionine. The β -oxidation proceeds by several removals of 2-carbon fragments from the fatty acids. The energy yield of this process is large, e.g., catabolism of 1mol of 6-carbon fatty acids through citric acid cycle to carbondioxide (CO_2) and water generates 44 moles of ATP, compared with the 38 moles of ATP generated by catabolism of 1mol of 6-carbon carbohydrate glucose.

In the liver, acetyl-CoA units condensed to form acetoacetyl-CoA with the help of an enzyme decyclase. This keto acid is converted into β -hydroxy butyrate and acetone and diffused into circulation, because these compounds are metabolized with difficulty in liver. Acetoacetate is also formed in the liver via the formation of β -hydroxy- β -methylglutaryl-CoA. This pathway is quantitatively more important than decyclation. Tissues other than liver transfer CoA from succinyl-CoA to acetoacetate and metabolize the "active" acetoacetate to CO₂ and water via the citric acid cycle.

The major lipids in plasma do not circulate in the free form. Free fatty acids are bound to albumin, whereas cholesterol, triglycerides and phospholipids are transported in the form of lipoprotein complexes. There are 6 classes of lipoproteins namely chylomicrons, chylomicrons remnants, very low density lipoproteins (VLDL), intermediate density lipoproteins (IDL), Low-density lipoproteins (LDL) and high density lipoproteins (HDL).



Chylomicrons are formed in the intestinal mucosa during the absorption of the products of fat digestion. They enter the circulation via the lymphatic ducts. The chylomicrons are cleared from the circulation by the action of lipoprotein lipase, which is present on the endothelium of the capillaries. The enzyme catalyzes the breakdown of triglyceride in the chylomicrons to FFA and glycerol, which then enter adipose cells and reesterified. Chylomicrons depleted of their triglyceride remain in the circulation as cholesterol rich lipoproteins called chylomicron remnants. The chylomicrons and their remnants constitute a transport system for ingested exogenous lipids. There is also an endogenous system made of VLDL, IDL, LDL and HDL, which transport triglycerides and cholesterol throughout the body. Very low-density lipoproteins are formed in the liver and transport triglycerides formed from fatty acids and carbohydrates in the liver to extra-hepatic tissue. After their triglyceride is largely removed by the action of lipoprotein lipase, they become IDL. The IDL give up phospholipids and through the action of the plasma enzyme lecithin-cholesterol acyltranferase (LCAT), pick up cholesterol esters formed from cholesterol in the HDL.

Protein metabolism:

The body proteins are continuously hydrolyzed to amino acids and resynthesized. The amino acids formed by endogenous protein break down are identical to those derived from ingested protein. With the latter they form a common amino acid pool that supplies the needs of the body. In the kidneys, most of the filtered amino acids are reabsorbed. During growth, the equilibrium between amino acids and body proteins shifts towards the latter, so that synthesis exceeds breakdown.

Thyroxin (T_4), catecholamine, histamine, serotonin and melatonin are formed from specific amino acids. Methionine, cystine, cysteine provide the sulfur contained in proteins, CoA, taurine and other biologically important compounds. Methionine is converted to adenosyl-methionine, which is the active methylation agent in the synthesis of compounds such as epinephrine, acetylcholine and creatine. It is a major donor of biologically labile methyl groups, but methyl groups can also be synthesized from a derivative of formic acid bound to folic acid.

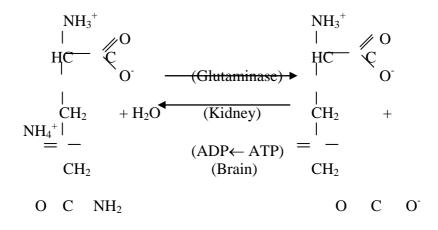
Oxidative deamination of amino acids occurs in the liver. An imino acid is formed by dehydrogenation, and this compound is hydrolyzed to the corresponding keto acid, with production of NH_4^+ (ammonium ion):

Amino acid + NAD⁺ \longrightarrow Imino acid + NADH + H⁺

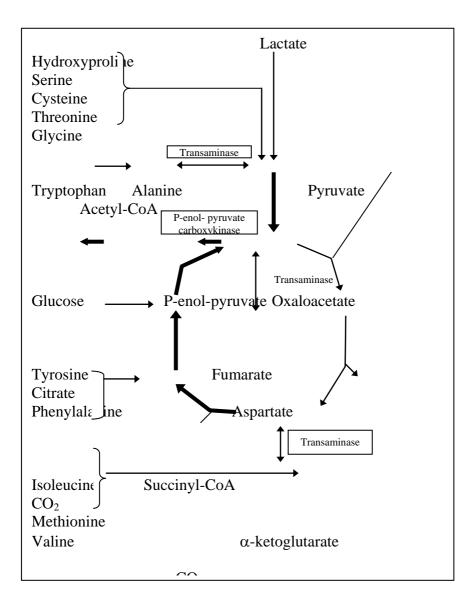
Imino acid + H_2O \longrightarrow Keto acid + NH_4^+

Ammonium ions are in equilibrium with ammonia (NH₃). Amino acids can also take up NH_4^+ forming the corresponding amide. An example is the binding of NH_4^+ in the brain by glutamate. The reverse reaction occurs in the kidney, with conversion of NH_4^+ to NH_3 (Figure 9).

Inter-conversion between the amino acid pool and the common metabolic pool is summarized in figure 10. Lucien isolucine, phenylalanine and tyrosine are said to be ketogenic because they are converted into ketone body acetoacetate. Alanine and many other amino acids are glucogenic or gluconeogenic, i.e. they give rise to compounds that can readily be converted to glucose.



The plasma proteins consist of albumin, globulin and fibrinogen fractions. The globulin fraction is subdivided into α_1 , α_2 , β_1 , β_2 and γ globulins. The albumin fraction and proteins concerned with blood clotting (fibrinogen, prothrombin etc) are synthesized in the hepatocytes. Albumin synthesis is carefully regulated. It is decreased during fasting and increased in condition such as nephrosis; in which there is excessive loss of albumin occurs. Plasma proteins in blood serve as effective buffers. Both their free carboxyl and their free amino groups dissociate:



PART TWO

Nutrition of Broilers

There are two types of broiler chickens available for meat production purposes the Cornish crosses and the dual-purpose breeds. Cornish cross birds have been bred specifically for meat production. There may be different growth rates with different crosses, but mature body weights for males are between 3.6 and 6.4kg. Crosses used commercially can reach approximately 2kg in 5-6 weeks (broiler). Females are approximately 2.7-3.6kg at maturity. Other available breeds include Rhode Island Red crossed with Barred Rock, Columbian Rock and Light Sussex etc.

Nutrient requirement of broiler:

Poultry have been produced commercially since the early 1900's, and research has been conducted for years to improve production efficiency. Poultry nutritionists employed by the industry have access to a great amount of information, to optimize the particular production parameters related with marketing such as, breast meat yield, feed conversion ratio (FCR). A good example of some of this information is supplied by the National Research Council for Poultry (NRC, 1994). The NRC has published suggested requirement estimates for many essential and non-essential amino acids, twelve minerals, thirteen vitamins and linoleic acid in chicken feed. These estimates have been developed over many years and are complicated by the fact that nutrients present in some ingredients vary with time and source. In addition the NRC provides the nutrient composition of 72 feed ingredients and regression equations to predict amino acid digestibility. The following tables depict the body weight and feed requirements (Table 1) and nutrient requirements for broiler birds (Table 2).

| Age | Body weights | | Weekl | , , | Cumulat consun | | Weekly consur | 0. | Cumulativ consun | |
|---------|--------------|-------|--------|--------|-------------------|-------|------------------|-------|---------------------|--------|
| (Weeks) | | | (8 | g) | | | (ME kcal/bird) | | | |
| | | | Female | Male | Female | Male | Female | | | |
| 1 | 130 | 120 | 120 | 110 | 120 | 110 | 385 | 350 | 385 | 350 |
| 2 | 320 | 300 | 260 | 240 | 380 | 350 | 830 | 770 | 1,215 | 1,120 |
| 3 | 560 | 515 | 390 | 355 | 770 | 705 | 1,250 | 1,135 | 2,465 | 2,255 |
| 4 | 860 | 790 | 535 | 500 | 1,305 | 1,205 | 1,710 | 1,600 | 4,175 | 3,855 |
| 5 | 1,250 | 1,110 | 740 | 645 | 2,045 | 1,850 | 2,370 | 2,065 | 6,545 | 5,920 |
| 6 | 1,690 | 1,430 | 980 | 800 | 3,025 | 2,650 | 3,135 | 2,560 | 9,680 | 8,480 |
| 7 | 2,100 | 1,745 | 1,095 | 910 | 4,120 | 3,560 | 3,505 | 2,910 | 13,185 | 11,390 |
| 8 | 2,520 | 2,060 | 1,210 | 970 | 5,330 | 4,530 | 3,870 | 3,105 | 17,055 | 14,495 |
| 9 | 2,925 | 2,350 | 1,320 | 1,010 | 6,650 | 5,540 | 4,225 | 3,230 | 21,280 | 17,725 |

Table 1. Body weights and feed requirements of broilers¹

1. Typical for broilers fed well-balanced diets containing 3,200kcal/kg metabolizable energy (NRC, 1994)

| | Weeks 0-3 | Weeks 3-6 | Weeks 6-8 |
|--|-----------|-----------|-----------|
| Energy base kcal ME/kg diet ¹ | 3,200 | 3,200 | 3,200 |
| Protein, % | 23 | 20 | 18 |
| Arginine, % | 1.4 | 1.2 | 1 |
| Glycine + serine, % | 1.5 | 1 | 0.7 |
| Histidine, % | 0.4 | 0.3 | 0.3 |
| Isoleucine, % | 0.8 | 0.7 | 0.6 |
| Leucine, % | 1.4 | 1.2 | 10 |
| Lysine, % | 1.2 | 1 | 0.9 |
| Methionine + cystine, % | 0.9 | 0.7 | 0.6 |
| Methionine, % | 0.5 | 0.4 | 0.3 |
| Valine, % | 0.8 | 0.7 | 0.6 |
| Linoleic acid, % | 1 | 1 | 1 |
| Calcium, % | 1 | 0.9 | 0.8 |
| Phosphorus available, % | 0.5 | 0.4 | 0.4 |
| Potassium, % | 0.4 | 0.4 | 0.3 |
| Sodium, % | 0.2 | 0.2 | 0.2 |
| Chlorine, % | 0.2 | 0.2 | 0.2 |
| Iron, <i>mg</i> | 80 | 80 | 80 |
| Copper, mg | 8 | 8 | 8 |
| Iodine, mg | 0.4 | 0.4 | 0.4 |
| Selenium, mg | 0.2 | 0.2 | 0.2 |
| Vitamin A, IU^2 | 1,500 | 1,500 | 1,500 |
| Vitamin D, <i>ICU</i> ³ | 200 | 200 | 200 |
| Vitamin E, <i>IU</i> | 10 | 10 | 10 |
| Vitamin K, mg | 0.5 | 0.5 | 0.5 |
| Riboflavin, mg | 3.6 | 3.6 | 3.6 |
| Pentothenic acid, mg | 10 | 10 | 10 |
| Niacin, mg | 27 | 27 | 11 |
| Vitamin B_{12} , <i>mg</i> | 0.009 | 0.009 | 0.003 |
| Thiamin, mg | 1.80 | 1.8 | 1.8 |
| Pyridoxine, mg | 3 | 3 | 2.5 |

 Table 2. Nutrient requirements of broilers as percentages or as milligrams or units per kilogram of diet

These are typical dietary energy concentrations.
 International unit.
 International chick unit

(NRC, 1994)

Broiler nutrition and feeding management in warm climates;

Warm temperature and high humidity affect performance and profitability of broiler production. In temperate climates, healthy broilers growing under comfortable conditions will undergo serious life threatening heat stress when temperatures and humidity are suddenly elevated. Mortality rates as high as 30-40% is often observed during sudden periods of hot and humid weather. In contrast, birds growing under tropical conditions have constant high daytime temperature and humidity throughout the growing period and become acclimated. In this situation, mortality is rarely over 10% unless there is a disease outbreak.

Management can improve profit in warm climates:

The most effective way to maintain production during warm weather is to keep the birds as cool as possible. Research has shown that each 3°C increase in house ambient temperature over 33°C will reduce growth by 0.9% and FCR by 2.1% (North, 1984). By considering each of the 5 method birds use to dissipate heat, best and most cost-effective strategies to keep the birds cool can be devised:

1. Evaporation:

Panting causes water to be evaporated from the respiratory tract, which removes heat. An adequate supply of water is necessary.

2. Convection:

Cool air moving past the bird will carry body heat. Maximizing the natural ventilation and provision of fan-assisted ventilation can reduce the heat in poultry house.

3. Radiation:

Heat will move from warm objects to cooler objects. When the surface of the bird is warmer than its surrounding, heat will move away from the bird and vice versa.

4. Conduction:

Heat transfers from the bird to a cooler object by direct contact. Concrete floors with thin litter are effective heat sinks during the warmest part of the day.

5. Excretion:

Large amounts of heat leave the body through excretory waste. Ample cool water should be provided.

1. Temperature and humidity:

The effect of high temperature is always much greater, when humidity is high. The normal body temperature of a broiler is about 41.5°C. Any combination of temperature; humidity and the factors above that act to increase body temperature will cause stress. Any condition above normal comfort will reduce performance and profit.

2. Water:

An adequate supply of water is absolutely essential during hot weather. Watering system must be in peak condition and management must check and maintain the water system continuously. If nipple drinkers are used, pressure regulators must be adjusted to supply maximum water. Water that gets on combs and wattles will be effective in cooling. Water-soluble electrolytes may also be useful to increase water consumption and replace minerals lost in excretion.

3. Feed management and nutrition

1. Feed restriction:

Feed may be restricted if sudden high temperatures are anticipated. This is often beneficial to prevent mortality as body heat raises 7-12%, 2-hours after feeding. In this situation, feed should be preferably removed for a period of 6 hours before the stress begins. Feed restriction is of much less value in acclimated tropical birds that have been reared under constant high daily temperatures.

Particle size, pelleting and density:

Birds spend considerable energy while consuming feed. Because of this, feeds of high bulk density and high nutrient density are advantageous in warm climates. The rate of nutrient intake per unit energy spent in consuming feed should be as high as economically possible. Dusty mash feed of small particle size is more difficult to consume and more costly to produce and should be avoided.

Nutrition:

The physiology of panting results in loss of carbondioxide and potassium (K) from the system, which should be replaced. Replacement of 50% of the added salt with an equivalent amount of sodium from sodium bicarbonate is recommended. An excess and/or unbalance amino acid induces formation of uric acid, which generates heat in the bird. Fat, as an energy source is also useful in that it produces less heat than carbohydrates during metabolism. Full fat soybean meal is an excellent ingredient as it contains 18% high quality fat. Use of phosphate sources that provide more phosphorus (P) and less calcium are also being useful in the situation. A good quality and digestible P source is especially important during hot weather (McCormick, 1981).

Feed quality:

Each 10°C increase in temperature will double the rate of oxidation and mold growth in feed. Humidity and accidental wetting of feed by rain during delivery and storage is also common. Moldy and oxidized feed will reduce performance and decrease resistance to disease.

Vitamins:

Vitamin C (ascorbic acid) is a water-soluble antioxidant vitamin that is normally produced in adequate quantities by metabolism in broilers. During heat stress its production is limited. Thus stabilized forms of Vitamin C have been found useful under certain situations.

Improving FCR in broilers:

Some of the more important factors affecting FCR are outlined and explained in broiler flocks.

Feed conversion ratio (FCR):

Feed conversion ratio is a measurement of the productivity of a bird and is defined as the ratio of feed to weight gained. For example, if 4kg of feed are used to produce a 2kg broiler, the FCR of that broiler is 2.00 (4/2=2kg). Obviously, the lower the FCR, the more efficient a bird is. Broilers convert feed to meat very effectively, and FCR of 1.8-1.9 is possible. The keys to realizing good FCR are an understanding of the basic factors, which affect it and a commitment to basic management methods that optimize these factors.

MAJOR FACTORS AFFECTING FCR:

1. Temperature:

Probably the most important factor influencing FCR is the temperature of the broilers environment. Birds are homeotherms (warm-blooded), meaning they maintain a relatively constant body temperature regardless of the temperature of their environment. Under ideal conditions of around 20-25°C, the bird uses a minimum of feed to maintain body temperature. In a cool environment, broilers will eat more feed but many of the calories they obtain from this feed will be used to sustain normal body temperature. These calories used for warmth do not convert to meat.

Feed intake will increase by about 1% for each 1°C below 20°C. Between 20-25°C, the bird will eat about 1% less per 1°C increase in temperature, and so here FCR will improve. Under these conditions, FCR further deteriorates because the bird is reluctant to eat feed, and so proportionally more feed is directed towards maintenance, and less can be used for growth.

Optimum temperatures allow the broilers to use nutrients for growth rather than temperature regulation. The ideal environmental temperatures for promoting FCR in both warm room and hover brooding situations are shown in Table 3.

| | Temperature | | | | | | |
|-------------|------------------------|-----------------------------|--|--|--|--|--|
| Week | | | | | | | |
| | Warm room ¹ | Hover brooding ² | | | | | |
| 1 | 29-31 | 32 | | | | | |
| 2 | 27-28 | 29 | | | | | |
| 3 | 24-26 | 27 | | | | | |
| 4 | 24 | 27 | | | | | |
| 5 | 21 | 24 | | | | | |
| 6 to market | 21 | 21 | | | | | |

Table 3. Ideal temperatures (°C) for different age groups of broilers

1. Measured at chick height

 $\overline{2}$. Measured at chick height, 1 foot from edge of hove

At high temperatures broilers consume less feed and inefficiently convert this feed to muscles. The biological cooling mechanisms that birds use during hot weather requires energy, just as the warming mechanisms do during cool weather. In addition, when birds consume feed, their body temperature rises as a result of the metabolic processes that occur during digestion. For this reason, it is recommended not to feed birds during the warmest part of the day (late morning to late afternoon) during very hot weather.

2. Ventilation:

Ventilation and temperature are interrelated. Under most conditions, increasing ventilation results in lower temperatures in a poultry house. Ventilating sometimes requires brooders or furnaces to operate to keep the house at the ideal temperature. Unfortunately, growers often ventilate less during cold weather to reduce fuel costs, which is improper. Clean fresh air is just as important to growing broilers as fresh feed and water.

Ammonia (NH₃) and other toxic gases build up in under ventilated broiler houses during the cooler months of the year. Studies show that FCR may be adversely affected (from 4-7 points) by NH₃ levels of just 25ppm, which is barely detectable, by the human nose. So it is strongly recommend that broiler growers ventilate to remove NH₃ during winter. Ventilation requirements will vary depending upon the tightness of housing, humidity, litter condition, etc.

3. Feed quality:

The diet that a broiler consumes greatly influences FCR. Farmers have little control over the energy level, protein or initial quality of the feed. Nevertheless, farmer must maintain the quality of the feed once it is put in feed bin. The provision of guard against oxidation, mold and contamination is necessary. The bins should be water tight, clean and disinfected after every flock. Feeding system should be checked daily to make sure it is in good working order. Closely monitor areas where feed can escape and be wasted.

4. Water quality:

Clean, fresh water is important for better FCR. Broiler performance on farms with contaminated water supplies is almost always below average. It is a common believe among some poultry experts that enclosed watering systems improve FCR (compared to trough waterers). The most important reason given for this improvement relates to water quality. Water in troughs and bells is exposed to dust, litter, feed and fecal material. However, water in the enclosed systems is protected from the contamination.

Although it takes more effort to keep water clean in trough waterers. Simply dumping the old water out of the drinkers is not enough but also scrub the drinkers with a disinfectant daily. The enclosed system does not eliminate cleaning chores completely. Mini-drinkers, frequently used in conjunction with enclosed waterers when starting chicks, require thorough cleaning at least once a day. Water is the most important nutrient for any animal; therefore, water quality cannot be over-emphasized. The effort expended to provide clean water to broilers will result in better FCR.

5. Disease and medication:

The general health of a flock also influences FCR. Sick birds do not perform well. So it is necessary to watch closely for early signs of disease, and treat sick birds quickly and properly. The careful use of vaccinations and medications is advisable since they can cause reactions by improper administration and can adversely affect weight gain. Treating only when needed reduces harmful effect on growth and FCR.

6. Timed feeding:

Research shows that timed feeding can improve FCR. In such feeding programs, broilers are fed a set amount of feed 4-6 times a day so they finish their meal and then are without feed for a short period of time (less than an hour). The short period without feed appears to stimulate feeding when the next meal is delivered. Usually the birds quiet down during the time without feed, which may enhance FCR. If birds are allowed to go without feed too long, benefits may be lost and performance may actually decreases.

7. Light:

Light levels in the broiler house can also influence FCR. Relatively bright lighting (1-2 foot candles) stimulates chick activity and helps them locate feed and water. Low light levels calm the broilers and reduce bird activity resulting in better weight gain. A lighting scheme of 1-hour of light followed by 2hours of darkness throughout the day may improve FCR. In conventional curtain houses, the same light schedule at night helps stimulate feeding.

Influence of energy on broiler performance:

With very fast growing birds a growth restriction during the first 2-3 weeks will have a positive effect on the flock. The birds are more lively and active; the development of the skeleton and legs is better. The percent flip-overs and ascites will also be lower. Such growth restriction can be achieved by using lighting programs, feed restriction or decreased energy level in the starter feed.

There is a certain negative effect of decreasing energy level on the FCR but this is of minor importance because the quantity of feed consumed in this period is small. There is compensation in FCR during the grower period. In contrast to FCR no compensation occurs for the lost growth. Only 25-50g will be lost, which is in practical terms less than one day's extra growth. When using a long dark period (e.g. 12 hours) in combination with lower energy starter, the weight loss will be larger than the 25-50g.

Broiler chickens are eating to satisfy their energy requirements. It has been suggested that the bird eats to its maximum physical capacity, and that varying the energy density of the diet can easily control the bird's energy intake. This fact may be true to some extent with the young broiler, because early growth rate can be temper by feeding lower energy diets. However as the broiler gets older it does seem to adjust its intake in relation to diet energy level. Table 4 shows the results of diluting the feed to very low levels.

| Dietry metabolizable energy | Dietary crude protein | 49-day body weight | Feed intake 35-49 day | Feed: gain 35-49 day | Energy efficiency |
|-----------------------------------|-----------------------------|--------------------------|-----------------------------|----------------------------|----------------------|
| (kcal/kg) | (%) | (| (g) | | (Mcal/kg gain) |
| | 10 | | | | U 7 |
| 3200 | 18 | 2950 | 2580 | 2.3 | 7.4 |
| 2900 | 16 | 2920 | 2760 | 2.5 | 7.2 |
| 2600 | 14 | 2880 | 2900 | 2.7 | 7.0 |
| 2300 | 13 | 2910 | 3270 | 3.0 | 6.7 |
| 1900 | 11 | 2910 | 3670 | 3.3 | 6.4 |
| 1600 | 9 | 2890 | 4300 | 4.0 | 6.4 |

 Table 4. Effect of diet dilution from 35-49 day of age on broiler performance

(Leeson et al., 1996)

The reduction in the nutrient level of the diet can results in the increased feed consumption. This means that the bird is not eating to physical capacity, because the bird was able to almost double its normal intake on the very low nutrient dense diet. This amazing ability to adjust feed intake resulted in no real difference in 49-day body weight. However, if we calculate energy efficiency, then the birds on the lowest energy feed were actually the most efficient in converting feed energy to weight gain.

Some broilers never recover from an interruption in growth early in life. In the normal growth of a broiler, nutrients

are used differently from day old to market as demonstrated in table 5.

| Age | Maintenance | Growth |
|----------------|-------------|------------|
| Age (Weeks) | (%) | (%) |
| Day old | 20 | 80 |
| 5 | 60 | 40 |
| 8 | 80 | 20 |
| | · | (Oderkirk, |

Table 5. Nutrient use by broilers

1999)

Male vs female birds:

The FCR of female broilers will usually be higher (less efficient) than male birds of corresponding weight (30-days). The reason for this is that female birds tend to deposit proportionally more fat in the carcass. Body fat takes 9 times as much feed energy to produce as does muscle. The reasons for this is that fat contains more energy than does protein per unit of weight, and more importantly, muscle is only about 20% protein by weight, the remainder being water. Likewise with heavy male birds, FCR is going to be greatly influenced by the growth of fat compared to muscle.

A very useful starting point in re-evaluating efficiency of feed use is to consider conversion of feed energy to live weight gain. Following are typical energy conversion figures for broilers up to 9-weeks of age (Table 6).

| Table 6. Energy C | Universion to live we | ight for brohers (N | (ical/kg gain) |
|-------------------|-----------------------|---------------------|----------------|
| Age | Male birds | Female birds | Mixed sex |
| (Weeks) | | | |
| 4 | - | 5.2 | - |
| 5 | 5.4 | 5.6 | 5.5 |
| 6 | 5.8 | 6.1 | 5.9 |
| 7 | 6.2 | 6.6 | 6.4 |
| 8 | 6.7 | - | - |
| 9 | 7.1 | - | - |

 Table 6. Energy conversion to live weight for broilers (Mcal/kg gain)

(Leeson, 2001)

Broiler FCR decreases dramatically as body maintenance requirements increase with age. Problems early in life that effect nutrient uptake translate to major problems in trying to get all birds to market weight at the same weight.

Manipulate feed fat deposition in birds:

Improving FCR and adapting eating habits and fat accumulation to specific needs is commercially important due to the fact that about 60% of the cost in broilers incurred on feeding. The identification of leptin and leptin-receptor in mammals, 6 years ago, the most important breakthroughs in the understanding of obesity and energy balance in mammals were discovered. Leptin was characterized in mammals as the satiety hormone and as a key element in the control of feed intake, energy expenditure and reproduction. It had been strongly suggest that the leptin-mediated control mechanism operates in avian also. Such compounds could increase profitability in poultry farming by facilitating the manipulation of feed intake, fat deposition and reproduction in commercially bred poultry, according to the specific needs of the various poultry branches.

Nutrition of Layers

Advances in genetic selection make today's commercial layers quite different from those of a decade ago. Body weight is less, age at housing and age at 5% production are earlier, total egg numbers have increased, egg mass is greater and feed conversion ratio (FCR) has improved considerably.

The period of time between housing and peak egg production is the most demanding and stressful period in the laying hen's life. During this period she is not only adjusting to her new environment, she must consume enough energy and nutrients to grow and reach a high peak in egg production. During this time a high energy/nutrient-dense diet is required. Good management of the flock during this period is critical and every attempt should be made to minimize stress.

Nutrient requirements:

A complete diet is essential for chick growth, health and development. Grains alone are inadequate for chicks because they contain only half the necessary protein and lack essential vitamins. The feed should contain chick-size grit to enhance digestion. The following tables describe the body weight and feed requirements (Table 1) and nutrient requirements for layers and breeders (Table 2).

Feeding egg production stock:

The greatest cost of raising chickens for the laying flock is for feed. It is a vital need of each bird to have a complete, well balanced diet. The amount of feed consumed is influenced by several important factors including management, health and physical conditions flock uniformity, rate of egg production, environment and feed quality. From day-1 to 6-weeks, 18% protein, starter ration should be fed. This complete balanced ration can be mash or crumbles. From 6-20 weeks of age, a 14% protein, grower ration should be fed. Again, it must be completely balanced; it may be crumbled or pelleted. From age 20-weeks on, a 16-18% protein layer mash should be fed as a complete balanced ration. These rations can be bought from local feed suppliers.

| Age | Body weight ¹ | Feed consumption ² | Typical egg production |
|---------|--------------------------|-------------------------------|------------------------|
| (weeks) | <i>(g)</i> | (g/week) | (% hen-day) |
| 0 | 35 | 45 | - |
| 2 | 135 | 90 | - |
| 4 | 270 | 180 | - |
| 6 | 450 | 260 | - |
| 8 | 620 | 325 | - |
| 10 | 790 | 385 | - |
| 12 | 950 | 430 | - |
| 14 | 1,060 | 460 | - |
| 16 | 1,160 | 460 | - |
| 18 | 1,260 | 460 | - |
| 20 | 1,360 | 460 | - |
| 22 | 1,425 | 525 | 10 |
| 24 | 1,500 | 595 | 38 |
| 26 | 1,575 | 665 | 64 |
| 30 | 1,725 | 770 | 88 |
| 40 | 1,815 | 770 | 80 |
| 50 | 1,870 | 765 | 74 |
| 60 | 1,900 | 755 | 68 |
| 70 | 1,900 | 740 | 62 |

 Table 1. Body weights and feed requirements of leghorn-type pullets and hens

<u>1</u>. Pullets and hens of leghorn-type strains are generally fed *ad libitum* but are occasionally control-fed to limit body weights. Values shown are typical but will vary with strain differences, season and lighting. Specific breeder guidelines should be consulted for desired schedules of weights and feed consumption.

<u>2</u>. Based on diets containing 2,900kcal ME/kg; consumption will vary depending upon the caloric density of the diet, environmental temperature and rate of production (NRC, 1994)

| Tuble 201 (utilient requireme | Growing | Growing | Growing | Laying | Laying, daily | Breeding |
|-------------------------------|-----------|------------|-------------|--------|----------------|----------|
| Energy base kcal ME/kg | 0-6 weeks | 6-14 weeks | 14-20 weeks | | intake per hen | _ |
| diet ¹ | 2,900 | 2,900 | 2,900 | 2,900 | $(mg)^2$ | 2,900 |
| Protein, % | 18 | 15 | 12 | 14.5 | 16,000 | 14.5 |
| Arginine, % | 1 | 0.8 | 0.7 | 0.7 | 750 | 0.7 |
| Glycine and serine, % | 0.7 | 0.6 | 0.5 | 0.5 | 550 | 0.5 |
| Histidine, % | 0.3 | 0.2 | 0.2 | 0.2 | 180 | 0.2 |
| Isoleucine, % | 0.6 | 0.5 | 0.4 | 0.5 | 550 | 0.5 |
| Leucine, % | 1 | 0.8 | 0.7 | 0.7 | 800 | 0.7 |
| Lysine, % | 0.9 | 0.6 | 0.5 | 0.6 | 700 | 0.6 |
| Methionine + cystine, % | 0.6 | 0.5 | 0.4 | 0.6 | 600 | 0.6 |
| Methionine, % | 0.3 | 0.3 | 0.2 | 0.3 | 350 | 0.3 |
| Phenylalanine+tyrosine, % | 1 | 0.8 | 0.7 | 0.8 | 880 | 0.8 |
| Phenylalanine, % | 0.5 | 0.5 | 0.4 | 0.4 | 440 | 0.4 |
| Threonine, % | 0.7 | 0.6 | 0.4 | 0.5 | 500 | 0.5 |
| Tryptophan, % | 0.2 | 0.1 | 0.1 | 0.1 | 150 | 0.1 |
| Valine, % | 0.6 | 0.5 | 0.4 | 0.6 | 600 | 0.6 |
| Linoleic acid, % | 1 | 1 | 1 | 1 | 1,100 | 1 |
| Calcium, % | 0.8 | 0.7 | 0.6 | 3.4 | 3,750 | 3.4 |
| Phosphorus available, % | 0.4 | 0.4 | 0.3 | 0.3 | 350 | 0.3 |
| Potassium, % | 0.4 | 0.3 | 0.3 | 0.2 | 165 | 0.2 |
| Sodium, % | 0.2 | 0.2 | 0.2 | 0.2 | 165 | 0.2 |
| Chlorine, % | 0.2 | 0.1 | 0.1 | 0.2 | 165 | 0.2 |
| Magnesium, mg | 600 | 500 | 400 | 500 | 55 | 500 |

Table 2. Nutrient requirements of leghorn-type chickens as percentages or as milligrams or units per kilogram of diet

| Energy base kcal ME/kg | Growing 0-6 weeks | Growing 6-14 weeks | Growing 14-20 weeks | Laying | Laying, daily intake per hen | Breeding |
|------------------------------|----------------------|-----------------------|------------------------|--------|---------------------------------|----------|
| diet ¹ | 2,900 | 2,900 | 2,900 | 2,900 | $(mg)^2$ | 2,900 |
| Manganese, mg | 60 | 30 | 30 | 30 | 3.3 | 60 |
| Zinc, mg | 40 | 35 | 35 | 50 | 5.5 | 65 |
| Iron, mg | 80 | 60 | 60 | 50 | 5.5 | 60 |
| Copper, mg | 8 | 6 | 6 | 6 | 0.9 | 8 |
| Iodine, mg | 0.4 | 0.4 | 0.4 | 0.3 | 0.03 | 0.3 |
| Selenium, mg | 0.2 | 0.1 | 0.1 | 0.1 | 0.01 | 0.1 |
| Vitamin A, IU^3 | 1,500 | 1,500 | 1,500 | 4,000 | 440 | 4,000 |
| Vitamin D, ICU^4 | 200 | 200 | 200 | 500 | 55 | 500 |
| Vitamin E, <i>IU</i> | 10 | 5 | 5 | 5 | 0.6 | 10 |
| Vitamin K, <i>mg</i> | 0.5 | 0.5 | 0.5 | 0.5 | 0.06 | 0.5 |
| Riboflavin, mg | 3.6 | 1.8 | 1.8 | 2.2 | 0.24 | 3.8 |
| Pentothenic acid, mg | 10 | 10 | 10 | 2.2 | 0.24 | 10 |
| Niacin, mg | 27 | 11 | 11 | 10 | 1.1 | 10 |
| Vitamin B_{12} , <i>mg</i> | 0.009 | 0.003 | 0.003 | 0.004 | 0.00044 | 0.004 |
| Choline, <i>mg</i> | 1,300 | 900 | 500 | - | - | - |
| Biotin, mg | 0.2 | 0.1 | 0.1 | 0.1 | 0.01 | 0.2 |
| Folacin, mg | 0.6 | 0.3 | 0.3 | 0.3 | 0.028 | 0.4 |
| Thiamin, mg | 1.8 | 1.3 | 1.3 | 0.8 | 0.09 | 0.8 |
| Pyridoxine, mg | 3 | 3 | 3 | 3 | 0.3 | 4.5 |

<u>1</u>. These are typical dietary energy concentrations
<u>2</u>. Assumes an average daily intake of 110g of feed/hen daily
<u>3</u>. International unit

 $\underline{\overline{4}}$. International chick unit

(NRC, 1994)

Once egg production begins, energy intake is the critical factor controlling egg numbers. Therefore, the diet must contain an adequate concentration of calories if small birds are going to be expected to perform to their full genetic potential at peak and as the laying cycle continues. If greater egg profits are to be realized during an entire laying cycle, it is essential that replacement pullets attain proper body weight. A bird that remains small will lay small eggs at the onset of egg laying. Since feed intake is correlated with body weight. The decreased egg size often seen in some young flocks is most likely a result of feed intake.

Egg producers will normally attempt to get the largest number of high-quality eggs of the correct size from each hen housed in the shortest period of time at the lowest cost. Some nutrients have been identified as affecting egg weight, but any adjustments in nutrient levels may reduce egg production. At the beginning of lay, increasing hen's intake of balanced protein results in increased egg size. Feeding higher levels of protein at onset of lay means that egg size will increase more rapidly. A limiting factor in the increase of early egg size is a sub-optimal energy intake.

There are numerous feeding and management programs that have an effect on investment. Feeding programs are designed to meet the nutritional needs of the hens. However, the profit margin is different with each type of feeding program.

1. Limited feeding:

It was a common belief that laying hens need to be fullfed at all times throughout the laying cycle, which is prove to be wrong. Limited feeding should not be ignored when an egg producer is looking for ways to lower feed and total production costs. Controlling in-house temperature is one-way to achieve the goal of limited consumption of feed after peak egg production in a layer flock because increased in-house temperature results in less feed intake. If a feed restriction program is implemented, it is important to formulate the diet to supply adequate amounts of critical nutrients each day to the hen. The amino acid, vitamin and mineral concentrations in the diet are more critical with limited feeding than with full feeding. In limited feeding programs, the objective is to limit only energy without limiting the intake of critical nutrients.

Laying hens fed an energy-restricted diet have a lower maintenance requirement, and a hen consuming less feed is more efficient and profitable. If a feed restriction program is used, it is usually not started until the majority of the eggs being produced fall into the large size category. A feed restriction program will result in a slight decrease in egg size, which is of less consequence once the majority of the eggs are in the large category. Initiation of a feed restriction program should commence later for layer strains of lower body weight, particularly during periods of hot weather.

2. Phase feeding:

A feeding program that uses only one feed during the entire laying period will be simple and easy to manage, but costly. Such a program has to be designed to meet the peak nutritional requirements of the hens at all times under all conditions. The feed has a high nutrient density to meet the maximum requirements at the lowest level of feed consumption expected throughout the year.

In contrast, phase feeding is used extensively in today's industry. Dr. G.F. Combs first proposed phase feeding in the 1960s. This was the term this poultry nutritionist gave to the program of reducing the protein level in the feed as the hen aged. Today, levels of other nutrients, along with protein and amino acids, are lowered as the hen ages or when egg production in the flock declines to a certain percentage.

Phase feeding reduces feed costs as egg production decreases because each change in formula is associated with a less fortified feed. The effect of temperature on total feed consumption of the flock, and thus total nutrients consumed, is not considered with a phase feeding program if no adjustments are made to the diet as feed intake changes.

3. Feeding by consumption *Ad libitum* feeding:

Feeding program that more closely meets the nutritional needs of a hen is feeding by consumption. This program, by definition, requires knowledge of the hen's feed consumption. As feed intake increases or decreases, the percentage of nutrients in the diet will decrease and increase accordingly to ensure the proper intake of the required nutrients.

Feed consumed by the layer is utilized in 2 ways. Twothirds of feed energy is used for maintenance while most of the protein is utilized in egg production. Feed consumption in layers is greater for larger birds. Increased production and cooler weather will also increase intake. For each dozen eggs the hen produces she will have eaten 1.6-2kg of feed. Feed makes up ? of the cost of producing an egg. It is therefore important that the proper feed is fed and that no wastage or spillage occurs.

Water should be cool, fresh and made available each day. One hundred layers will drink 18-27L of water/day. Hens consume double quantity of water as they do feed. Water consumption on the basis of 100 pullets/day is 2.27L for the first week of age. In hot weather water consumption may increase drastically as the bird tries to cool herself by drinking more water.

Role of calcium phosphorus and vitamin D_3 in egg shell and

bone formation:

Calcium (Ca) is the major structural element in both bone and egg shell of the layer. The bone contains 99% of total body Ca, which makes it an integral part of Ca level regulation in the body. The egg shell contains around 94% calcium carbonate (CaCO3), which by weight, an average egg would contain 2g of Ca. An average of 4g of Ca intake/day is required by a layer to maintain good shell quality since only 50-60% of dietary Ca is actually used in shell formation.

During the last 15 hours of shell formation, Ca movement across the shell gland reaches a rate of 100-150mg/hour. This process draws Ca from 2 sources, diet and bone. With a normal layer ration of 3.56% Ca or higher, most shell Ca is derived by intestinal absorption, while layers on a 2% Ca diet, 30-40% of the shell Ca is derived from bone. It is therefore important to have pullets, prior to lay, on a laying ration level of Ca.

Intestinal absorption of Ca in the diet is about 40% when the shell gland is inactive, but reaches 72% when active. This time usually coincides with late afternoon or the dark hours for the layer. Having higher Ca levels in the gut during this time is important to insure Ca is being taken from the diet and not bone.

Eighty percent of body phosphorus (P) is found in the bone. Since very little P is found in egg shell the role of P in layers has more to do with its effects on Ca metabolism. The absorption of Ca and P can be influenced by a number of factors like source and form of Ca and P, intestinal pH, Ca: P ratio and vitamin D.

The Ca and P must be in a form that is available and usable by the layer. Phosphorus absorption is optimal at pH 6.0. When the pH is higher than 6.5, absorption of P is markedly decreased. Excess free fatty acids in the diet can cause the pH to decrease and therefore interfere with Ca and P absorption. High Ca or P levels in the intestine reduce the absorption of both. Phosphorus is an integral part of the acid-base balance in the body. The proper ratio of Ca: P for growing birds is 1.5-2.0: 1.0. Vitamin D₃, in its metabolite form of 1,25-dihydroxy-D₃ is involved in Ca mobilization from bone to ensure normal plasma Ca levels. Vitamin D₃ is the major control element in stimulating Ca absorption from the intestine.

There is a complex relationship between Ca, P, Vitamin D3 and the hormonal system of the layer in Ca metabolism during lay. Practical implications of this relationship to bone development and egg shell formation are important. Layers need

4g of Ca/bird/day in the early stages of egg production. Since feed intake of young layers may be 10kg or less /100 layers/day (<100g/bird/day) the Ca level of the diet should be between 3.8-4.0% of which 50-60% is large particle (grit size or larger). High levels of Ca in the laying ration limits Ca requirements (needs) from bone for shell formation.

In growing birds, a high level of Ca in the diet is not recommended since it interferes with the absorption of P, Zn and Mn. Young birds are not able to deal with excess Ca, and it may lead to kidney damage. Most importantly, high Ca levels during the growth period will interfere with the proper development of the parathyroid gland by increasing gut pH, which will decrease absorption. Estrogen-like hormone that could be affected, acts to start the reproductive system, which triggers the body of the pullet to start laying down Ca in the bone prior to lay. This Ca store will be used during the lay cycle. Increasing Ca in the diet 2-weeks prior to lay is therefore essential to ensure available Ca is being stored. If the parathyroid is under-developed or defective this hormonal system will also be defective and optimum levels of Ca stored in bone will not be realized. Also, Ca will be immobilized when needed for lay if the parathyroid glands activities are defective.

Vitamin D_3 intake must be adequate. The function of Vitamin D_3 is related to its metabolite 1, 25-dihydroxy- D_3 which is formed in the bird's liver and kidneys. Any problem that affects the functional integrity of these organs or the parathyroid gland will have an adverse effect on the action of vitamin D_3 and thereby Ca absorption and metabolism.

Vitamin E and egg production and quality:

The effectiveness of increasing the vitamin E content of eggs through greater fortification of hen's diets has been demonstrated in numerous studies, e.g., Cherian et al. (1996) reported that as vitamin E supplementation rose, the enhancement of total tocopherols in tissues was greatest for egg yolk, followed in descending order by liver, adipose tissue, dark meat and white meat.

Meanwhile, studies such as Whitehead (1998) have shown increases of 3-16% in the number of eggs produced per hen when higher levels of vitamin E are fed during periods of heat stress. In discussing possible modes of action, Whitehead (1998) elaborated that the improvements with increased vitamin E levels could be linked to improved circulatory supply of yolk precursors, particularly vitellogenin, during heat stress. Indeed, this study showed that vitellogenin synthesis in the liver decreased during heat stress and also, more importantly, that vitellogenin accumulated in the liver at the expense of plasma concentrations for uptake by the ovaries.

In the hens receiving only 10IU vitamin E/kg of feed, liver concentrations of vitellogenin during heat stress increased by 77% compared with unstressed birds receiving the same vitamin E regimen. Plasma concentrations decreased by 49.6% during heat stress for this group. Liver concentrations of vitellogenin increased by only 10% in these hens, while plasma concentrations fell by only 28.2%. It has been suggested that because vitamin E is the principal antioxidant in cell membranes, it may protect transport mechanisms across membranes from oxidative damage induced by heat stress.

Meluzzi *et al.* (2000) reported that yolk α -tocopherol content increased linearly as fortification of the hens diet increased from 0 to 200 IU of vitamin E/kg of feed (Table 3). Eggs from the hens receiving the highest level of fortification provided 313.84µg of vitamin E/g of yolk, or more than 3 times as much vitamin E as eggs from hens with the control diet.

Table 3. The α -Tocopherol contents of egg yolk of layers fed different levels of vitamin E

| Fortification level | α Tocopherol content of yolks |
|---------------------|-------------------------------|
| (Hen's diet IU/kg) | $(\mu g/g)$ |
| 0 | 90.93 ^a |
| 50 | 130.05 ^b |
| 100 | 227.48° |
| 200 | 313.84 ^d |

(Meluzzi et al., 2000)

Chapter 12

Nutrition of Breeders

Two of the major and most costly nutrients required by the breeder are energy and protein. While meeting protein requirements have often been the major consideration of breeders, it is energy, the most costly dietary nutrient, which in many instances is limiting performance. There is lack of research on the specific nutrient requirements of meat type female breeders from hatch to maturity. Nutrient requirements data presented in table 1 and table 2 for broiler breeder are limited to that given in NRC (1994), whereas the nutritive requirements of layer breeders are given in the chapter of nutritive requirements of layers.

| | Requirements |
|--------------------------------|--------------|
| Nutrient | |
| Protein, g | 19.5 |
| Arginine, mg | 1110 |
| Histidine, mg | 205 |
| Isoleucine, mg | 850 |
| Lysine, mg | 765 |
| Methionine, mg | 450 |
| Methionine + cystine, mg | 700 |
| Phenylalanine + tyrosine, mg | 1112 |
| Threonine, mg | 720 |
| Tryptophan, <i>mg</i> | 190 |
| Valine, <i>mg</i> | 750 |
| Calcium, g | 4 |
| Chloride, <i>mg</i> | 185 |
| Sodium, <i>mg</i> | 150 |
| Biotin, μg | 16 |

 Table 1. Nutrient requirements of meat-type hens for breeding purposes as units per hen per day (90% dry matter)

(NRC, 1994)

| | Age (weeks) | |
|-----|-------------------------|--|
| 0-4 | 4-20 | 20-60 |
| - | - | 350-400 |
| 15 | 12 | - |
| 0.8 | 0.6 | - |
| 0.4 | 0.3 | - |
| 0.6 | 0.5 | - |
| 0.9 | 0.9 | - |
| | 15 0.8 0.4 0.6 | 0-4 4-20 - - 15 12 0.8 0.6 0.4 0.3 0.6 0.5 |

Table 2. Nutrient requirements of meat-type males for breeding purposes as units per rooster per day (90% dry matter)

(NRC, 1994)

Energy:

In developing feeding programs for broiler breeder consideration must be given to body size, environmental temperature and bird activity as these are the main factors influencing maintenance energy requirements. A consideration for growth is also important for the growing pullet and to a lesser extent, the mature hen. For the adult the energy requirement for egg mass output must also be taken into account.

Energy requirements for pullets:

As the bird increases in age the maintenance requirement increases, as a percentage of the total energy requirement, until by the end of the growing period over 80% of the energy consumed is used just to maintain the pullet. Growers should be aware of the high-energy cost just to maintain a bird and also to recognize that pen temperature is a significant factor influencing maintenance energy requirements. Thus, pen temperature should be monitored closely and feed intake adjusted accordingly if significant temperature changes result.

Reducing feed intake past peak production:

It is common practice to reduce feed intake shortly after peak production is attained. This may be responsible for some of the dips in production noted at this time. The hen "thinks" in terms of egg mass output and thus no reduction in feed allowance should be made until after peak egg mass output is reached, which is usually 3-5 weeks after peak production. Another error some producers make is to reduce feed allowance in relation to the drop in egg production (e.g. if production drops 5% reduce feed by a similar amount). It should be obvious that decreased feed intake, after peak production, has to be precisely calculated if decreased egg numbers and/or size is to be avoided.

Protein requirements:

Since the maintenance requirement for energy is so high, in relation to total energy requirement, changes in feed allotment are usually made in order to try and maintain an optimum intake of energy. However, often little attention is given to the amount of protein consumed. Most breeder diets contain between 16-18% protein. With daily feed allotments as high as 160g/bird/day, protein intake could be 25.6-28.8g/day.

Protein required for egg production:

Using values generated for commercial layers, the calculated protein required to produce a 65g egg (containing 7.8g of protein) should be around $7.8\div0.55$ (suggested efficiency of dietary protein utilization for egg production) = 14.2g.

Protein required for maintenance:

With an estimated total endogenous loss of nitrogen, including feather loss of 280mg/kg of body weight^{0.75}, a 3.5kg hen would require $3.5^{0.75} \times 280 = 717$ mg of nitrogen/day to meet her daily maintenance requirement. Converting this to protein would give 4.48g (0.717×6.25) of protein. With the assumption that the hen is 55% efficient in the use of dietary protein for body purposes, would give 4.48 ÷ 0.55 = 8.15g of protein intake required/day to meet her maintenance requirements.

Total daily protein requirement of a broiler breeder considering maintenance and egg production would be 14.2 +

8.15 = 22.4g/bird/day. This would be sufficient for a hen to lay a 65g egg every day. While no allowance has been made for weight gain this would be minimal after peak production and besides much of this gain would be fat deposition and thus a minimum of body protein would be deposited. Since every bird is not laying every day the flock average for protein for egg production purposes would be the average percent production times, e.g., the 14.2g of protein required for a 65g egg. Thus, the average daily protein required for a flock would be significantly less than 22.4g/bird/day.

Partitioning of the breeders protein requirement:

It was shown that approximately 80% of the energy consumed is partitioned to meet the hen's requirement for maintenance. It can be estimated, from the values generated above for protein requirements, that $(14.2 \div 22.4 \times 100)$ approximately 63% of the protein intake of the broiler breeder is going to meet it's requirement for egg mass output and thus only 37% for maintenance. Hence, the main factor influencing the protein requirement of the broiler breeder is egg mass output.

Vitamin levels in breeder diets:

Breeder hens are capable of peak egg production around 80% under commercial conditions, with sustained peaks over 75% for 12-weeks. This high egg output is only possible with superior management and nutrition, part of which is a wellfortified high-energy diet. These high persistent peaks also mean that birds have to supply adequate vitamins in the feed, not only for the hens at very active stage of reproduction, but also to ensure optimum hatchability and chick quality.

The breeder's vitamin requirements are usually met by adding all as synthetic sources. The regular feed ingredients, such as corn, soybean meal and meat meal, all contain "natural" sources of vitamins and in some situations could theoretically contain enough to meet the breeder's needs. However the concentration of vitamins contained in corn will, e.g., be affected by seed variety, growing conditions, harvesting conditions and storage conditions. Likewise the vitamins in meat meal will vary greatly depending upon the animal components used and the time and temperature of cooking and drying during processing.

The starting point used in developing vitamin requirements, is the NRC (1994) values, which are updated each 6-8 years. These NRC values are absolute requirement values for individual vitamins, and most often reflect the level of vitamin needed to prevent deficiency symptoms. In feeding breeders, the main aim is not only to prevent signs of vitamin deficiency, but also to ensure good egg production, hatchability and early chick vitality. This superior performance will only be achieved by feeding much higher levels of vitamins as part of a balanced nutritional program.

Another major loss of vitamins occurs if they are premixed with minerals and stored for any length of time prior to incorporation in feed. Also conditions within the premix and feed can cause loss of potency. For example, some vitamins are acidic whereas others break-down under acidic conditions. Finally to really cause problems to vitamin stability, we sometimes pellet feed, and here the temperature and humidity can cause vitamin breakdown.

Feeding to more precisely meet the nutrient requirements of

broiler breeders:

It is often stated that seldom is the diet at fault but rather it is the feeding program or the management conditions under which the diet is being fed which is the problem. This is especially true for broiler breeders where the nutrient intake of the birds is very much under the control of the flock manager. There appears to be a lot of evidence to suggest that many broiler breeders are being fed excessive levels of protein. Not only is such a practice detrimental to performance, but also it is uneconomical as well as resulting in a greater potential pollution problem with excreted nitrogen. Dietary protein and energy must be kept in proper balance with respect to requirements, if a deficiency or an excess of one or the other is to be avoided.

In case, if a flock is not attaining expected egg numbers or size when consuming 150-160g feed/bird/day, it should be better to look at possible management problems before changing diet composition or significantly increasing feed allowance. However, there are well-managed flocks that are peaking in excess of 85% and holding a good level of production for a sustained period of time. Such flocks may require more than the normal recommended level of feed intake.

Major changes have taken place with the feeding of broiler breeders in recent years. The most significant of these changes is separate sex feeding. With such an approach to feeding, it is now possible to more closely control the weight of the pullet coming into production and to be confident that, with challenge feeding, the additional feed is being used to maximize egg output rather than put extra weight on already over-weight cockerels. By being able to precisely control cockerel weight marked improvements in hatch of total eggs set, with reduced cockerel numbers, is now being attained.

Providing adequate vitamins in a breeding ration is very important. Deficiencies of various trace-elements and vitamins may lead to reduced hatchability and poor chick quality. Dead embryos may exhibit conditions that reveal the particular vitamin deficiencies causing their death. A deficiency of Vitamin B_{12} will cause a rapid decrease in hatchability. There's also a poorer survival rate for chickens that do hatch. Riboflavin (Vitamin B_2) deficiencies also cause poor hatchability with embryos showing clubbed down. The degree of the deficiency affects the stage at which death of the embryo takes place.

Biotin, choline and manganese (Mn) help prevent a condition known as perosis or slipped tendon. An acute deficiency of biotin causes high embryo mortality during the period of 72-96 hours of incubation. A Mn deficiency gives rise to embryos with parrot beaks and nutritional chondrodystrophy,

which is a shortening of the long bones of the embryo. A choline deficiency is unlikely, as the hen seems fully able to synthesize her own requirements.

These vitamins and minerals must be included in breeder's diet: riboflavin, pentothenic acid, vitamin B_{12} , niacin, folic acid, biotin, choline, vitamin A, vitamin D₃, vitamin E, vitamin K, Mn, phosphorus and zinc. Most commercial breeder mashes and concentrates are sufficiently fortified and contain more than an adequate amount of these essential vitamins and minerals to insure proper embryo development.

Obesity in female broiler breeders:

Broiler breeder females are reproductively unfit, when they are allowed to full-feed. The strong negative relationship between reproductive efficiency and body weight has resulted in considerable research interest in defining the "ideal" target growth curve. The reproductive symptoms associated with breeder females being over-weight are related to the birds having excessive follicular development. The problems arise when a hen is faced with being in an excessively positive energy balance. Over-weight hens frequently exhibit Erratic Ovulation and Defective Egg Syndrome (EODES). This term implies that they do not follow the normal patterns of the ovulatory cycle. Rather, EODES-afflicted hens ovulate almost at random and lay eggs at night as well as during the day. The eggs are defective for the reasons that they often have multiple yolks, are misshapen or have poor shell quality.

A normal feed restricted hen ideally should have about 8 large yolky follicles (greater than 1cm diameter). It is very important that these follicles are present in a hierarchy that is well spaced, with little evidence of a "double hierarchy". If pullets are over-fed, particularly during the time that the ovary is developing at sexual maturity, the number of large ovarian follicles can be as high as 12 or 14. In many cases, there is clearly evidence of a double hierarchy (follicles are within 1g of each other). Some hens have complete sets of "twin" yolks. These developing eggs may reside together in the shell gland. The areas where the 2 eggs are touching will not be properly calcified and neither egg will have adequate shell quality to support an embryo to hatching due to excessive moisture loss. Sometimes the 2 eggs can be separated by a greater distance in the oviduct. If the first egg is in the shell gland, it may be oviposited (laid) when the second ovulation happens. Sometimes that does not happen, and 2 eggs will be seen in the tract.

Over-weight hens can also develop some severe reproductive problems that can be fatal. In some cases, it appears that oviduct motility is impaired and the oviduct becomes impacted with developing eggs. Internal ovulation takes place when the follicles are not picked up by the oviduct, but instead fall into the body cavity. About 5-10% of all breeder hens carry some remnants of internally ovulated follicles.

Nutrition of Water Fowl

Ducks and geese are classified as waterfowl. Although they belong to the same category but we discuss them separately.

Duck:

The commercial duck industry in the various parts of world is focused around the production of young ducklings for meat purpose as broiler and turkey. In Pakistan duck farming is mainly based in Sialkot and Sheikhupura districts, but on very small scale, because of little preference towards the duck meat. The best meat yielding duck breeds include White pekin, Aylesbury, Rouen and Muscovy ducks. Whereas egg producing breeds includes Khaki Campbell and Indian Runner.

A combination of wheat and commercial turkey starter containing 25% protein is a satisfactory diet for ducks. The addition of a powdered vitamin mix to the wheat-turkey starter combination results in a diet, which adequately meets the nutritional requirements of most waterfowl species. Waterfowl tend to waste a substantial amount of feed if it is in a mash form. Therefore pellets or crumbles are recommended. A source of insoluble grit should be provided to birds, which have access to green vegetation and/or a source of whole grain. Two rations, a starter diet and a holding diet, are sufficient for all routine feeding purposes in captive ducks.

Starter ration:

Starter ration is fed to ducklings until they reach 28-days old. Initially, the starter ration will consist solely of turkey starter; after 21-days, wheat is gradually added until it constitutes 50% of the mixture by day-28. Basic starter diets can be supplemented with green feeds. Duckweed (Lemna minor) is an ideal green feed for use by ducklings.

Holding ration:

The holding ration consists of a 50-50 mixture of wheat and turkey starter with a vitamin mix added and fine granite grit offered *ad libitum*. This should be the standard diet used for feeding all captive waterfowl after 28-days of age. Oyster shell should be added during the breeding season to supply additional calcium (Ca) necessary for egg shell production.

Brooder-rearing feeding:

Ducklings should have feed and drinking water available when they are started under the brooder or hen. Feeding must initially be encouraged in newly hatched ducklings by sprinkling starter on the floor. Natural curiosity will lead them to peck at the scattered feed. Once the ducklings have learned to feed, the starter should be supplied only in dishes in order to prevent waste and maintain sanitary conditions. The young ducklings are essentially full grown by the time they reach 60-days old. Once they are fully grown, they may be placed with adult birds.

Nutrient requirements of ducks:

Ducks require the same nutrients as chickens, but in slightly different amounts, and particularly in terms of the ratio of each nutrient to the energy concentration of the diet. Suggested nutrient levels for complete duck rations are listed in table 1. These levels are set high enough to meet the requirements of all breeds of domestic ducks. For each type of ration, requirements for a high and a low energy ration are given.

| Nutrients | Starter | | Grower- | finisher | Breeder d | Breeder developer | | Breeder layer | |
|---------------------------------------|-------------|------------|-------------|------------|-------------|-------------------|-------------|---------------|--|
| | High energy | Low energy | High energy | Low energy | High energy | Low energy | High energy | Low energy | |
| Metabolizable energy, kcal/kg | 3086 | 2646 | 2646 | 3086 | 2866 | 2205 | 2866 | 2646 | |
| Crude protein, % | 22 | 19.1 | 14 | 16.1 | 17.6 | 12.8 | 17.5 | 16.2 | |
| Lysine, % | 1.2 | 1 | 0.7 | 0.8 | 0.9 | 0.6 | 0.8 | 0.7 | |
| Methionine, % | 0.5 | 0.4 | 0.3 | 0.4 | 0.4 | 0.3 | 0.4 | 0.4 | |
| Tryptophan, % | 0.2 | 0.2 | 0.2 | 0.2 | 0.2 | 0.2 | 0.2 | 0.2 | |
| Arginine, % | 1.2 | 1 | 0.9 | 1 | 1.1 | 0.8 | 0.9 | 0.8 | |
| Threonine, % | 0.8 | 0.7 | 0.5 | 0.6 | 0.7 | 0.5 | 0.6 | 0.5 | |
| Histidine, % | 0.4 | 0.4 | 0.3 | 0.4 | 0.4 | 0.3 | 0.4 | 0.3 | |
| Isoleucine, % | 0.9 | 0.8 | 0.6 | 0.7 | 0.8 | 0.6 | 0.7 | 0.7 | |
| Phenylalanine, % | 0.8 | 0.7 | 0.6 | 0.7 | 0.8 | 0.6 | 0.7 | 0.6 | |
| Phenylalanine+tyrosine, % | 1.5 | 1.3 | 1.1 | 1.3 | 1.4 | 1 | 1.1 | 1 | |
| Calcium, % | 0.7 | 0.6 | 0.6 | 0.7 | 0.8 | 0.6 | 3 | 2.8 | |
| Phosphorus, % | 0.4 | 0.4 | 0.3 | 0.4 | 0.4 | 0.3 | 0.4 | 0.4 | |
| Sodium, % | 0.2 | 0.1 | 0.1 | 0.1 | 0.2 | 0.1 | 0.2 | 0.1 | |
| Potassium, % | 0.6 | 0.5 | 0.5 | 0.6 | 0.7 | 0.5 | 0.6 | 0.6 | |
| Magnesium, <i>mg/kg</i> | 500 | 435 | 435 | 500 | 550 | 400 | 500 | 465 | |
| Manganese, <i>mg/kg</i> | 50 | 44 | 35 | 40 | 44 | 32 | 40 | 37 | |
| Zinc, mg/kg | 60 | 52 | 52 | 60 | 66 | 48 | 60 | 56 | |
| Iron, <i>mg/kg</i> | 80 | 70 | 70 | 80 | 88 | 64 | 70 | 65 | |
| Copper, <i>mg/kg</i> | 8 | 7 | 5 | 6 | 7 | 5 | 6 | 6 | |
| Vitamin A, <i>IU¹/kg</i> | 5000 | 4350 | 3480 | 4000 | 4400 | 3200 | 6000 | 5580 | |
| Vitamin D ₃ , <i>IU/kg</i> | 600 | 522 | 435 | 500 | 550 | 400 | 600 | 558 | |
| Vitamin E, <i>IU/kg</i> | 25 | 22 | 17 | 20 | 22 | 16 | 30 | 28 | |

Table 1. Suggested levels of nutrients for duck rations

| Nutrients | Starter | | Grower | Grower-finisher | | Breeder developer | | r layer |
|----------------------------------|-------------|------------|-------------|-----------------|-------------|-------------------|-------------|------------|
| | High energy | Low energy | High energy | Low energy | High energy | Low energy | High energy | Low energy |
| Vitamin K, <i>IU/kg</i> | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 |
| Choline, <i>mg/kg</i> | 1300 | 1130 | 870 | 1000 | 1100 | 800 | 1000 | 930 |
| Vitamin B ₂ , mg/kg | 4 | 4 | 3 | 3 | 4 | 3 | 4 | 4 |
| Niacin, <i>mg/kg</i> | 50 | 44 | 35 | 40 | 44 | 32 | 50 | 46 |
| Pentothenic acid, mg/kg | 12 | 10 | 9 | 10 | 11 | 8 | 12 | 11 |
| Vitamin B_{12} , <i>mg/kg</i> | 0.01 | 0.01 | 0.004 | 0.005 | 0.01 | 0.01 | 0.01 | 0.01 |
| Pyridoxine, <i>mg/kg</i> | 3 | 3 | 3 | 3 | 3 | 2 | 3 | 3 |

<u>**1**</u>. International unit

<u>I</u>. International unit <u>Note</u>. Duck rations also contain at least 0.25mg/kg of folic acid a minimum of 0.13mg/kg of biotin (Dean, 2001)

Energy:

Ducks, like chickens, have simple stomachs, and therefore cannot digest appreciable amounts of dietary fiber (cellulose, lignin). However, ducks over 4-weeks of age have an exceptional capacity to consume large quantities of feedstuffs that are high in fiber, as compare to chickens. When such feedstuffs contain even small amounts of available energy, ducks may be able to consume enough of such feedstuffs to partially or even fully meet their energy requirements. Generally, however, ducks grown for meat are more likely to attain optimal performance when their diet contains a high proportion of cereal grains that are high in available energy such as corn, wheat and sorghum grain. Such diets will also result in better FCR. However when low energy feedstuffs, such as cereal byproducts, are available at favorable prices, they can be incorporated into duck rations at fairly high levels, so long as the ration is well balanced.

Protein:

Ducks, like other poultry, require the amino acids contained in dietary proteins. The proteins in the diet are broken down during digestion to amino acids, which therefore are absorbed and used to make body proteins, such as those in muscle and feathers. Protein levels that meet the ducks amino acid requirements may vary slightly, depending upon the amino acid contents of the ingredients used in each formulation.

Minerals and vitamins:

The minerals and vitamins requirements of ducks are listed in table 1. The most critical minerals in the duck nutrition are Ca, phosphorus and sodium. If a duck producer mixes his own feed, the simplest way, and often the most economical as well, is to add vitamins and trace-minerals in the form of commercially prepared premixes. If it is not possible to use prepared premixes, the next best choice is to purchase the vitamin and mineral sources and make own premixes.

Water:

Plenty of clean drinking water should be available to ducks at least 8-12 hours/day. In some management systems it is advantageous to shut off feed and water at night to help maintain litter inside buildings in a dry condition. This applies to breeder ducks or market ducks over 3-weeks of age. This practice is not harmful and has no effect on performance during periods of moderate temperatures, if done properly. Ducks can expel excess heat through their bill and feet, when allowed contact with water that is appreciably below their body temperature (41.7°C). Water temperatures of 10-21°C are ideal for ducks.

Duck feeding:

The feed consumed must contain all essential nutrients, in an available form, that are needed for maintenance, growth and reproduction. Feeding practices will depend in part on the number of ducks raised. If a household raises a few ducks, and they have access to areas where they can forage, they may be able to survive, grow and lay eggs by consuming available feed such as green plants, insects, snails, frogs and table scraps. Under such conditions, ducks will likely grow very slowly and produce a small number of eggs. Herded ducks are an exception, but they require access to large areas where feed is available and the care of a herdsman.

A small home flocks with better growth and more eggs will require a supplemental feed. At a minimum they will have to feed some grain. With increase in size of a flock, it becomes more likely that the flock will not be able to get enough feed by foraging; therefore supplemental feeding will become necessary. In absence of a duck rations, chicken feeds will also serve as a satisfactory substitute. Under intensive rearing system, different feeding practices are followed. These include dry mash, wet mash, pellets or crumbs. Dry mash, crumbs and pellets are given at free choice but wet mash is needed to be provided at frequent intervals. Ducks have difficulty in swallowing dry mash; they will take a mouthful and swill it down at nearest water source thus wasting a great deal of nourishment in the water. To prevent this, wet mash may be given. Usually wet mash is given 4-5 times daily for ducklings up to 2-weeks of age and 3-4 times daily afterward. About 10 to 20 minutes time, at each meal of wet mash is sufficient and more time may be allowed on the last meal of the day. After each meal the residual wet mash is to be removed. Currently pellet feeding has become popular. Pellet size for ducks is approx. 3-8mm.

Feeding growing breeders:

Meat-type ducks (e.g. Pekins), which are kept as breeders can become excessively fat, if fed *ad libitum* during growing phase. It is therefore necessary to limit their daily intake of feed to an amount that will supply all the necessary nutrients that are needed for proper development, while avoiding an excess of calories. For best results, feed restriction should begin at about 2weeks of age but practically it is often begun at about 7-weeks. From the time restriction is begun, and up until the breeders are sufficiently mature (about 28-weeks of age for Pekin ducks), their daily feed intake should be limited to 60-70% of the amount they would eat *ad libitum*. Feed can be spread out in long wooden troughs, on a cement slab or on the ground if the area is dry and clean.

Feeding laying breeders:

Nutrient levels of duck breeder layer rations are listed in table 1. Layer rations contain a higher level of Ca than other duck rations because of the laying duck's need for additional Ca for egg shell formation. A level of 3% of the diet is adequate for most breeds of ducks including high egg producing breeds. When enough Ca is included in the ration, it is not necessary to feed oyster shells in addition.

Feed quality:

The most common reason of deterioration of feed quality in a duck operation is failure to dry grains and other feedstuffs properly before storage. High moisture grains without turning or aeration can cause the grains heat up and moldy and some of its nutritive value will be lost. It should be made sure that the grains and other feedstuffs used in duck feeds are properly dried and must be free of molds and other contaminants. If table scraps, bakery waste, wet mash or other feeds high in moisture are fed, feed only what ducks will clean up in a day.

Rapeseed meal is another feedstuff that is potentially toxic to ducks. Some older varieties of rapeseed meal contain erucic acid and goitrogens at levels high enough to be harmful to poultry. Ducks are much more sensitive to erucic acid than are chickens and turkeys. Genetically improved varieties of rapeseed (Canola) contain much lower levels of these toxins. However even Canola meals should first be tested in ducks before their use in duck feeds on a large scale. Mold toxins can cause damage to the duck's digestive organs, liver, kidneys, muscles and plumage, and can also reduce growth and/or reproductive performance.

Mash or pellets:

It is a well-established fact that ducks grow faster, and utilize their feed more efficiently, when fed pelleted rations than mash. The major problem with feeding dry mash to ducks is the formation of a sticky paste when mixed with saliva, which cakes and accumulates on the outer ridges of the mouth. In attempting to free their bills of caked feed, ducks make frequent trips to water to wash their bills, causing feed wastage. Feeding mash also reduces feed intake, and in the case of market ducks, reduces their growth rate. In small flocks where pellets are not available to the ducks, the solution is to feed wet mash. Water is mixed with the mash just before feeding. Enough water is added to form a thick mush without making it watery. Mix only what ducks will clean up within a day.

Pellet size:

When pelleted feeds are fed to ducks it is important to avoid feeding pellets that are too large in diameter or too long for ducklings to swallow. For newly hatched ducklings, pellets should be no larger in diameter than 4mm, and no longer than 8mm. After about 2-weeks of age, Pekin ducklings can consume pellets 4.8mm in diameter, and approximately 12.7mm in length, without difficulty.

| Nutrients | Ducks | Chicken | | Ducks | Chicken | |
|-----------------------|-------|-----------------------|-------------------------|-------|---------|--|
| (/100g of fresh eggs) | | (/100g of fresh eggs) | | | | |
| Water, g | 74.6 | 70.8 | Vitamins (mg) | | | |
| Energy, kcal | 185 | 158 | Thiamin | 0.2 | 0.1 | |
| Protein, g | 12.8 | 12.1 | Riboflavin | 0.4 | 0.3 | |
| Lipids, g | 13.8 | 11.2 | Niacin | 0.2 | 0.1 | |
| Minerals (mg) | | | Vitamin B ₆ | 0.3 | 0.1 | |
| Calcium | 64 | 56 | Vitamin B ₁₂ | 5.4 | 1.6 | |
| Iron | 3.9 | 2.1 | Vitamin A, IU^1 | 1328 | 520 | |
| Magnesium | 16 | 12 | Amino acids (g) | | | |
| Phosphorus | 220 | 180 | Lysine | 1 | 0.8 | |
| Potassium | 222 | 130 | Methionine | 0.6 | 0.4 | |
| Sodium | 146 | 138 | Cystine | 0.3 | 0.3 | |
| Zinc | 1.4 | 1.4 | Threonine | 0.7 | 0.6 | |
| Lipids (g) | | | Isoleucine | 0.6 | 0.8 | |
| Total saturated | 3.7 | 3.4 | Phenylalanine | 0.8 | 0.7 | |
| Monounsaturated | 6.5 | 4.5 | | | | |
| Polyunsaturated | 1.2 | 1.5 | | | | |
| Cholesterol, mg | 884 | 548 | | | | |

Table 3. Nutritional comparison of duck and chicken eggs

<u>1</u>. International units

| | Brown egg laying hen | White egg laying hen | Khaki campbell |
|---|-------------------------|-------------------------|-------------------|
| Hen housed average, <i>egg/bird/year</i> | 269 | 277 | 237 |
| Mean egg weight, g | 59.7 | 56.9 | 75.5 |
| Point of lay weight, kg | 1.6 | 1.3 | 1.5 |
| Feed consumed | | | |
| 0-20 weeks, <i>kg</i> | 8 | 7.1 | 8.9 |
| 21-74 weeks, <i>kg</i> | 42.2 | 41.5 | 56.8 |
| Total feed, /kg egg | 3.1 | 3.1 | 3.7 |

Table 4. Relative productivity of hens and ducks kept for egg production

Nutritive value of duck egg:

The tables 3 & 4 show the comparative nutrient contents of egg produced by chicken and duck and comparative productivity of hens and ducks kept for egg production. The nutrient content of duck egg is bit higher than the chicken, when compared on /100g of fresh eggs. The major difference is in the cholesterol contents, which in the case of duck egg is reasonably higher (884mg vs. 548mg). The total number of the eggs produced by the white egg-laying hens is higher (277) than the Khaki Campbell (237) but the ducks are producing more total mass/duck (17.89kg) than chicken (15.76kg).

Geese:

Geese are largely raised in European countries for the production of meat and feathers. Geese are the grazing birds and can be raised on the ranges. Geese are also classified as herbivores and can accept high fiber diets. However the digestibility of fiber is low in this species. The ability of geese to utilize fibrous feeds results from fast passage time that allows a high feed intake, plus the efficient manner in which gizzard breaks down plant cell walls, which allows the digestion of plant cell contents.

Nutrient requirements of geese:

Goslings should be started on 20% goose starter ration in the form of crumbles or pellets that are 2.37-4.77mm. Goslings can also be started on a crumbled or pelted chick or turkey starter, but it must be non-medicated as coccidiostats included in such feeds can cause lameness and even death among goslings. The rations for geese should be pelleted (4.8mm). It is noticed that mash or crumbles cause too much wastage. The general nutrient requirements for the commercial meat geese and some fine examples of goose rations are given in the tables 5 and 6.

| Nutrients | 0-4 weeks; | After 4 weeks | Breeding |
|---------------------------|-------------|---------------|-------------|
| | $2,900^{1}$ | $3,000^{1}$ | $2,900^{1}$ |
| Protein, % | 20 | 15 | 15 |
| Lysine, % | 1 | 0.9 | 0.6 |
| Methionine + Cystine, % | 0.6 | 0.5 | 0.5 |
| Calcium, % | 0.7 | 0.6 | 2.3 |
| Non-phytate phosphorus, % | 0.3 | 0.3 | 0.3 |
| Vitamin A, IU^2 | 1,500 | 1,500 | 4,000 |
| Vitamin D_3 , IU | 200 | 200 | 200 |
| Choline, <i>mg</i> | 1,500 | 1,000 | - |
| Niacin, mg | 65 | 35 | 20 |
| Pantothenic acid, mg | 15 | 10 | 10 |
| Riboflavin, mg | 3.8 | 2.5 | 4 |

Table 5. Nutrient requirements for geese

 $\underline{1}$. These are dietary energy concentrations expressed in kcal ME/kg diet

2. International units

Note. Values given in bold are estimated

(NRC, 1994)

Feeding:

Feed and drinking water should be available with goslings, when they are started under the brooder. For the first few days feed can be placed on egg case flats or other rough paper. The types of feeders are the same as used for chicks. After the first 2-3 weeks, a pelted chick grower ration can be fed, supplemented with a cracked grain. Geese are better foragers than ducks and a succulent pasture or lawn clippings can be provided as early as the first week.

| Ingredients (%) | Starter | Grower-finisher |
|----------------------------------|---------|------------------------|
| Ground yellow corn | 15 | 20 |
| Ground wheat/Milo | 15 | 20 |
| Ground barley | 20 | 25 |
| Ground oats | 20 | 25 |
| Meat scrap (50%) | 2 | 3 |
| Soybean meal (47%) | 21.5 | 4 |
| Dried whey | 2 | - |
| Dehydrated alfalfa meal (17%) | 3 | - |
| Dicalcium phosphate | 0.5 | - |
| Iodized salt | 1 | 1 |
| Riboflavin, <i>g</i> / <i>T</i> | 2 | - |
| Niacin, g/T | 20 | - |
| Vitamin B ₁₂ , mg/T | 6 | - |
| Total | 100 | 100 |

Table 6. Examples of goose ration

A good share of feed can be from forage; by the time the birds are 5-6 weeks old. Geese can be very selective and tend to pick out the palatable forages. An acre of pasture will support 20-40 birds, depending on the size of the geese and pasture quality. Supplemental grain feeding of goslings is often continued after they have been established on good pasture. However flocks can be raised only on green fodder during the pasture period.

The goslings can be full fed in confinement and marketed as "junior or green geese" at about 10-weeks of age. Geese can also be used for weeding purposes. Weeder geese are used with great success to control and eradicate troublesome grass and certain weeds in a great variety of crops and plantings like cotton, onion, garlic, corn and orchards etc. Geese for breeding purposes are fed holding and breeding diets for the intensive production of fertile eggs.

Breeder geese:

There is a dearth of information available on the nutritional requirements of breeder geese. A non-medicated

chicken breeder type diet fed in pellet form gives satisfactory results. It has been established that 15% protein feeds are adequate and the levels of vitamins in a chicken breeder ration appear to be sufficient. During the breeding season, feed should be available to the birds at all times. The feeding of pelleted chicken breeder rations should be started at least a month before egg production. However birds on this ration do not need supplemental hay or grass. If pasture is not available in the breeder pens, the birds can be put on pasture after they stop laying in the early summer. The breeder feed can then be restricted or stopped, depending on the quality of the pasture, until the next season.

Chapter 14

Nutrition of Turkeys

Turkey is native to the new world. Present domesticated turkeys are descendants of the Mexicans sub-species *Meleagris* gallopavo gallopavo and the eastern wild turkey *Meleagris* gallopavo silvestris. The major species of turkey are Norfolk Black, Nicolas Breed and Broad Breasted Bronze.

Nutritive requirements of turkeys:

The nutritive requirement of turkey is similar to those of chickens but vary in some respects. The turkey is quite sensitive to nutrition deficiencies. For the first 5-weeks of the poults life the birds should be fed a turkey starter mash or crumble. This should be given *ad-lib*. After 5-weeks a grower ration should be introduced and a little grain such as wheat and maize may be introduced up to 10-weeks of age. Breeders should be given pre-breeder or holding ration also.

The Nutrient requirement of turkeys may be divided into different categories according to the requirements of the birds, used as source of meat, egg production or breeding. The growth rate of turkeys has been increased greatly during the past decade, due to the efforts by commercial breeders in various parts of the world. Approximate live body weight and feed consumption data of the turkeys are shown in table 2. Nutrient Requirements for turkey are given in table 1.

| | | | | Age | (weeks) | | | |
|--|---------|---------|----------|-----------|-----------|-----------|---------|----------|
| | M: 0-4, | M: 4-8, | M: 8-12, | M: 12-16, | M: 16-20, | M: 20-24, | Breeder | Breeding |
| | F: 0-4 | F: 4-8 | F: 8-11 | F: 11-14 | F: 14-17 | F: 17-20 | Holding | hens |
| Energy base kcal ME/kg diet ¹ | 2,800 | 2,900 | 3,000 | 3,100 | 3,200 | 3,300 | 2,900 | 2,900 |
| Protein, % | 28 | 26 | 22 | 19 | 16.5 | 14 | 12 | 14 |
| Arginine, % | 1.6 | 1.4 | 1.1 | 0.9 | 0.8 | 0.6 | 0.5 | 0.6 |
| Glycine + serine, % | 1 | 0.9 | 0.8 | 0.7 | 0.6 | 0.5 | 0.4 | 0.5 |
| Histidine, % | 0.6 | 0.5 | 0.4 | 0.3 | 0.3 | 0.2 | 0.2 | 0.3 |
| Isoleucine, % | 1.1 | 1 | 0.8 | 0.6 | 0.5 | 0.5 | 0.4 | 0.5 |
| Leucine, % | 1.9 | 1.8 | 1.5 | 1.3 | 1 | 0.8 | 0.5 | 0.5 |
| Lysine, % | 1.6 | 1.5 | 1.3 | 1 | 0.8 | 0.7 | 0.5 | 0.6 |
| Methionine, % | 0.6 | 0.5 | 0.4 | 0.4 | 0.3 | 0.3 | 0.2 | 0.2 |
| Phenylalanine, % | 1 | 0.9 | 0.8 | 0.7 | 0.6 | 0.5 | 0.4 | 0.6 |
| Threonine, % | 1 | 1.0 | 0.8 | 0.8 | 0.6 | 0.5 | 0.4 | 0.5 |
| Tryptophan, % | 0.3 | 0.2 | 0.2 | 0.2 | 0.2 | 0.1 | 0.1 | 0.1 |
| Valine, % | 1.2 | 1.2 | 0.9 | 0.8 | 0.7 | 0.6 | 0.5 | 0.6 |
| Linoleic acid, % | 1 | 1 | 0.8 | 0.8 | 0.8 | 0.8 | 0.8 | 1.1 |
| Calcium, % | 1.2 | 1 | 0.9 | 0.8 | 0.7 | 0.6 | 0.5 | 2.3 |
| Phosphorus, available, % | 0.6 | 0.5 | 0.4 | 0.4 | 0.3 | 0.3 | 0.3 | 0.4 |
| Potassium, % | 0.7 | 0.6 | 0.5 | 0.5 | 0.4 | 0.4 | 0.4 | 0.6 |
| Sodium, % | 0.2 | 0.2 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.2 |
| Chlorine, % | 0.2 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 |
| Zinc, <i>mg</i> | 70 | 65 | 50 | 40 | 40 | 40 | 40 | 65 |
| Iron, <i>mg</i> | 80 | 60 | 60 | 60 | 50 | 50 | 50 | 60 |
| Copper, mg | 8 | 8 | 6 | 6 | 6 | 6 | 6 | 8 |
| Vitamin A, IU^3 | 5,000 | 5,000 | 5,000 | 5,000 | 5,000 | 5,000 | 5,000 | 5,000 |
| Vitamin D ² , ICU^4 | 1,100 | 1,100 | 1,100 | 1,100 | 1,100 | 1,100 | 1,100 | 1,100 |
| Vitamin E, <i>IU</i> | 12 | 12 | 10 | 10 | 10 | 10 | 10 | 25 |

 Table 1. Nutrient requirements of turkeys as percentages or as milligrams or units per kilogram of feed

| | | Age (weeks) | | | | | | | |
|--|---------|-------------|----------|-----------|-----------|-----------|---------|----------|--|
| | M: 0-4, | M: 4-8, | M: 8-12, | M: 12-16, | M: 16-20, | M: 20-24, | Breeder | Breeding | |
| | F: 0-4 | F: 4-8 | F: 8-11 | F: 11-14 | F: 14-17 | F: 17-20 | Holding | hens | |
| Energy base kcal ME/kg diet ¹ | 2,800 | 2,900 | 3,000 | 3,100 | 3,200 | 3,300 | 2,900 | 2,900 | |
| Vitamin K, <i>mg</i> | 1.8 | 1.5 | 1 | 0.8 | 0.8 | 0.5 | 0.5 | 1 | |
| Riboflavin, mg | 4 | 3.6 | 3 | 3 | 2.5 | 2.5 | 2.5 | 4 | |
| Pantothenic acid, mg | 10 | 9 | 9 | 9 | 9 | 9 | 9 | 16 | |
| Niacin, <i>mg</i> | 60 | 60 | 50 | 50 | 40 | 40 | 40 | 40 | |
| Vitamin B_{12} , mg | 0.003 | 0.003 | 0.003 | 0.003 | 0.003 | 0.003 | 0.003 | 0.003 | |
| Choline, mg | 1,600 | 1,400 | 1,100 | 1,100 | 950 | 800 | 800 | 1,000 | |
| Biotin, <i>mg</i> | 0.3 | 0.2 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.3 | |
| Pyridoxine, <i>mg</i> | 4.5 | 4.5 | 3.5 | 3.5 | 3 | 3 | 3 | 4 | |

<u>1</u>. These are typical metabolizable energy (ME) concentrations for corn-soy diets. Different ME values may be appropriate if other ingredients predominate.

 $\underline{2}$. These concentrations of vitamin D are satisfactory when the dietary concentrations of calcium and available phosphorus conform to those in this table.

3. International units

4. International chick units

<u>Note</u>. Estimated values are indicated in **bold** to distinguish them from the established requirements shown in Roman type (NRC, 1994)

| Age | Body | weight | Feed consumption Male Female | | Cumulative feed consumption | | ME consu | Imption |
|---------|------|--------|------------------------------------|-----------|--------------------------------|--------|----------|---------|
| | Male | Female | | | Male | Female | Male | Female |
| (Weeks) | () | kg) | (kg/v | (kg/week) | | (g) | (Mcal/v | veek) |
| 1 | 0.12 | 0.12 | 0.1 | 0.1 | 0.1 | 0.1 | 0.28 | 0.28 |
| 2 | 0.25 | 0.24 | 0.19 | 0.18 | 0.29 | 0.28 | 0.53 | 0.5 |
| 3 | 0.5 | 0.46 | 0.37 | 0.34 | 0.66 | 0.62 | 1 | 1 |
| 4 | 1 | 0.9 | 0.7 | 0.59 | 1.36 | 1.21 | 2 | 1.7 |
| 5 | 1.6 | 1.4 | 0.85 | 0.64 | 2.21 | 1.85 | 2.5 | 1.9 |
| 6 | 2.2 | 1.8 | 1.1 | 0.8 | 3.31 | 2.65 | 3.2 | 2.3 |
| 7 | 3.1 | 2.3 | 1.4 | 0.98 | 4.71 | 3.63 | 4.1 | 2.8 |
| 8 | 4 | 3 | 1.73 | 1.21 | 6.44 | 4.84 | 5 | 3.5 |
| 9 | 5 | 3.7 | 2 | 1.42 | 8.44 | 6.26 | 6 | 4.3 |
| 10 | 6 | 4.4 | 2.34 | 1.7 | 10.78 | 7.96 | 7 | 5.1 |
| 11 | 7.1 | 5.2 | 2.67 | 1.98 | 13.45 | 9.94 | 8 | 5.9 |
| 12 | 8.2 | 6 | 2.99 | 2.18 | 16.44 | 12.12 | 9 | 6.8 |
| 13 | 9.3 | 6.8 | 3.2 | 2.44 | 19.64 | 14.56 | 9.9 | 7.6 |
| 14 | 10.5 | 7.5 | 3.47 | 2.69 | 23.11 | 17.25 | 10.8 | 8.4 |
| 15 | 11.5 | 8.3 | 3.73 | 2.81 | 26.84 | 20.06 | 11.6 | 9 |
| 16 | 12.6 | 8.9 | 3.97 | 3 | 30.81 | 23.06 | 12.3 | 9.6 |
| 17 | 13.5 | 9.6 | 4.08 | 3.14 | 34.89 | 26.2 | 13.1 | 10.1 |
| 18 | 14.4 | 10.2 | 4.3 | 3.18 | 39.19 | 29.38 | 13.8 | 10.5 |
| 19 | 15.2 | 10.9 | 4.52 | 3.31 | 43.71 | 32.69 | 14.5 | 10.9 |
| 20 | 16.1 | 11.5 | 4.74 | 3.4 | 48.45 | 36.09 | 15.2 | 11.2 |

Table 2. Growth rate, feed and energy consumption of large-type turkeys

(NRC, 199

Feeding:

Turkeys are fast growing and efficient converters of feedstuffs to high quality meat. Therefore, turkeys require balanced high protein rations. Feeding programs for heavy turkey, broiler turkey, turkey breeder (Tom and Hen) is different. In order to ensure a low percentage of starve-outs, feed and water should be placed in the pens before the arrival of the poults. On arrival, it is advisable to dip a few poults beaks in water to help get them started drinking and prevent dehydration, particularly if they are more than 24 hours old when they arrive. This practice may reduce starve-outs, which peaks around 7-days of age. During the first few days, place some feed in feed trays, paper plates, box lids or egg trays. This will help to get the poults eating. A 5L water fountain per 50 poults should be used for the first several days, until the poults are accustomed to the adult drinkers. The switch-over from feeder lids and fountain drinkers should take place gradually.

Feeding by class of turkey:

The different classes of turkeys can be distinguished by weight and length of growing period (Table 3). Keeping turkeys beyond the recommended slaughter age may be costly due to poorer feed conversion ratio (FCR).

| | Slaughter age Live weight | |
|----------------|---------------------------|-------|
| | (weeks) | (kg) |
| Broiler turkey | 12-14 | < 6.2 |
| Heavy hen | 15-18 | 9.8 |
| Heavy tom | 18-23 | > 9.8 |

Table 3. Classes of turkey

The feed trough should not be filled more than half full. The birds will otherwise waste feed, which is expensive. As the birds grow, they will need larger watering and feeding equipment. For heavy turkeys (hens, toms), specialized feeding and watering equipment are required. Broiler turkeys are usually raised with chicken equipment. The starter diets should be fed as crumbles and the remaining diets as pellets. Insoluble grit may be sprinkled once a week at the rate of 1kg/100 birds.

Calorie: protein ratio:

The ratio of calories of metabolizable energy/kg of feed for each 1% protein is the calorie-to-protein ratio. In starter diets, this ratio will be narrow because of the high protein level in the diet needed to promote muscle growth. In grower, and particularly finisher diets, this ratio widens due to higher energy levels and lower protein. The purpose of this wider ratio is to improve the fattening of the birds. The comparative energy utilization values of turkey breeder and white leghorn layers are given in table 4. The average expected weights for the 3 classes of turkeys are shown in table 5.

| | Energy intake (g/day) | Egg energy (g/day) | Maintenance growth (g/day) |
|----------------|--------------------------|-----------------------|-------------------------------|
| Turkey breeder | 40 | 9.2 (23%) | 30.8 (77%) |
| Leghorn | 18 | 9 (50%) | 9 (50%) |

Table 4. Comparative energy utilization

| Table 5. Expected weights for turkeys by class | | | | |
|--|--------|----------------------|--|--|
| | Α | ge | | |
| | (Week) | (\mathbf{D}_{ana}) | | |

Table 5 Expected weights for turkeys by class

| | A | Age | |
|-----------------------------|--------|--------|------|
| | (Week) | (Days) | (kg) |
| Broilers (mixed) | 13 | 3 | 5.7 |
| Broilers (using heavy hens) | 11 | 3 | 5.6 |
| Hens | 14 | 5 | 7.4 |

Feeding pattern changes:

Toms

Changes in feeding pattern for heavy turkeys should be made depending on the season of the year the birds are being finished off. If birds are being finished off during warmer months (late summer or early fall), it is recommended to feed a higher

19

12.7

level of protein during the latter stages of the growing and finishing period. However if the birds are being finished off in the cooler months, the level of protein can be reduced approximately 2-weeks earlier.

Reducing turkey leg problems:

It has been suggested that decreasing the percentage of protein fed to turkeys during the first 6-weeks can reduce severe leg problems by as much as 50% and produce healthier birds with improved carcass quality. When fed tom turkeys at 100, 80, 70 or 60% of the NRC (1994) recommended protein levels for the first 6-weeks, followed by the full recommended levels from 7-weeks to the end of the 20-week growing period.

It has been noted that as the levels of protein decreased, body weight at 6-weeks decreased (2.23, 1.94, 1.63 and 1.39kg, respectively) and the FCR was poorer (1.63, 1.73, 1.90 and 2.16, respectively). Actually, the low protein groups consumed less feed, but their reduced weight resulted in poorer FCR to that point. Weight gain during 7-20 weeks period (on regular feed) was not affected (15.5, 15.2, 14.9 and 14.5kg, respectively), but the FCR was significantly improved as a result of the early protein restriction.

Turkeys have been bred to grow so fast that they often become too heavy for their legs to carry them. As leg weaknesses increase and the birds spend a lot of time sitting down, they tend to develop breast blisters and get cuts and scrapes from being stepped on by other birds. Turkeys without leg problems suffer less stress and fewer injuries, so they have fewer disease problems. Restricting the amount of protein during the first 6weeks decreases weight gain slightly, but at the end of 20-weeks the difference is only 6%, approximately 0.9kg/turkey.

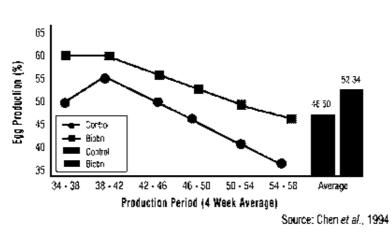
Benefits of biotin to turkey hatchability:

Biotin is an essential component of coenzymes in carbohydrate, fat and protein metabolism. The energy-producing

tri carboxylic acid (TCA) or Krebs cycle also depends on biotin. Biotin is important in thyroid and adrenal gland function, as well as in the reproductive tract and nervous system. The vitamin is essential in a number of metabolic activities, but is especially quick to make its importance known in turkey hatchability. It had been reported since long that turkey hens fed a biotin-deficient diet showed a marked decrease in hatchability and a high rate of embryonic mortality.

The need for supplemental biotin (Figure 1 and 2) has been demonstrated (Chen *et al.*, 1994). The studies compared turkey breeder performance at 2 supplemental biotin levels: 162mg and 680mg/T of complete ration in corn-soybean meal based diets starting at the time of light stimulation (31 or 30 weeks) and fed for 27-weeks.

In the first of the 2 studies, during 50 to 54 weeks of age, egg production and hatchability of fertile eggs were both significantly greater from turkey hens fed 680mg supplemental biotin/T of feed. Egg production and hatchability were increased by 22% in week-50 and by 10% in week-54, compared to hens fed 162mg/T. They reported that the higher level of dietary supplemental biotin also seemed to be beneficial to later reproductive performance.



I

Figure 1. Dietary biotin and egg production in turkeys

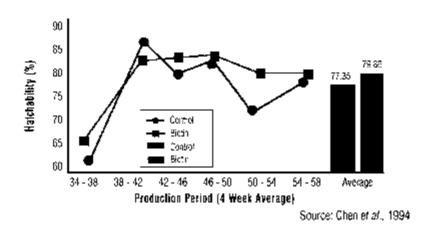


Figure 2. Dietary biotin and hatchability in turkeys

The researchers found significant benefits during 0-4 weeks and after 16-weeks of production, when both experiments were

analyzed together. Egg production was increased by 10.5% (0-4 weeks). Even greater responses were seen later, with a 33.5% increase (20-24 weeks) of production. Fertility was also increased at the higher level of biotin (45-49 weeks).

Chapter 15

Heat Stress

What is Stress?

Homeostasis mechanisms maintain the constant internal environment in the body thus keep the normal physiological function of the body of bird. Any deviation from this normal condition is called stress. Generally the term stress is used to describe the detrimental effects of variety of factors on health and performance of birds. Poultry birds have limited body resources for growth, production, reproduction, response to environmental changes and defense mechanism. So under normal conditions, there is redistribution of body resource including energy and protein at the cost of decreased growth, production and health. Under the effects of long term stress birds became fatigued and weak. These conditions lead to bird starvation and various infectious diseases. Due to stress immunity of bird depresses as a result chance to diseased attack increases. It can lead to the death of bird.

Types of stress:

Birds can be face stressed conditions keeping in house are even in the provision of the state of the art facilities. There are common sources of stress that can be grouped under one or more of the following categories;

- Climatic stress including extreme hot, cold and high humidity.
- Nutritional stress including shortage of nutrients and feed intake problems.

- Physiological stress including rapid growth and sexual maturity.
- > Physical stress including catching, injection and transport.
- Social stress including overcrowding and poor body weight uniformity.
- > Psychological stress including fear, harsh care takers.
- > Pathological stress i.e. exposure to pathological agents.

In addition to the above categories of stress, all the possible types of stressors can be classified under the,

- a). Avoidable stressors
- b). Unavoidable stressors

Avoidable stressors:

- Overcrowding
- Poor ventilation
- ➢ Wet litter
- \succ Toxin in feed
- ➤ Starvation
- High ammonia level
- > Dehydration
- Poor management

Un-avoidable stressors:

- ➢ Extreme weather
- ➤ Handling
- ➢ Vaccination
- ➢ Transportation
- ▶ Rapid growth
- ➢ De-beaking
- ➢ Lighting
- ➢ Medication

Heat stress:

Heat stress is a problem with every poultry production as high product output whether meat and egg leads to greater metabolic activity due to which greater body heat production resulted. This is especially true for the chicken since it is grow at rapid rate and while heat production is thus increasing its ability to dissipate heat is diminishing as its body surface, in relation to body weight, is decreasing.

Physiological mechanism of stress regulation:

Exposure of birds to stress is an inevitable event in poultry husbandry, when the threshold level of stress is crossed it results in stress to birds. Stress syndromes can be classified into following stages.

- > Stage of alarm reaction (Neurogenic system).
- Stage of resistance or adaptation (Endocrine system).

1. Neurogenic (sympatho- adrenal) system (Short-term

regulation of stress):

This system includes sympathetic (post ganglionic) nervous system and adrenal medullary tissue. It controls the rapid response to the animal i.e. fight or flight or alarm reaction .This reaction lasts for short time. It is characterized by increased secretion rate of the catecholamine from the adrenal medulla. These catecholamine prepare the bird for "Fight or Flight" reaction and commanding a rapid release of glucose in blood, depletion of liver glycogen, increased peripheral vasomotor activity, altered ventilation rate and increased neural sensitivity. Catecholamine also stimulates the activity of hepatic adenyl cyclase, the enzyme required for the production of cAMP. cAMP regulates the number of energy reaction (physiological processes) and directly increases the formation of antibody.

2. Endocrine system (Long-term regulation of stress):

Endocrine system in stress regulation is known as the 'stage of resistance'. This system is comprised of hypothalamuspituitary adrenal axis (HPA). It is characterized by adrenal cortical hypertrophy and increased synthesis rate and release of adrenal glucocorticoids, known as corticosterone in bird .Activation of the HPA is a longer-term adjustment by the animal to the surrounding changes.

Other hormones:

i) Glucagons:

The α cells of the pancreas produce glucagon, are stimulated in alarm response in both mammals and birds.

ii) Thyroid hormone:

It is produced by thyroid glands are also involved in stress regulation.

Nutritional manipulations to combat heat stress Water:

Water consumption is an important consideration in heat stress. However, its importance is sometimes down played by the fact that 80% of the bird's heat production during heat stress is dissipated via panting (evaporative cooling). Addition of various salts to water alters the bird's osmotic balance, resulting in increased water consumption, thus influencing water balance during heat stress. No growth response has been observed by adding salts to drinking water for non-heat stressed birds. Research has shown that increased water consumption benefits the bird by acting as a heat receptor as well as increasing the amount of heat dissipated per breath. Such thermobalance effects are principally observed when water temperature falls below 28°C. Birds in positive water balance are better able to maintain normal body temperature. This has special significance for the commercial broiler as heat stress increases urine production, independent of water intake, thus forcing birds to sustain higher water consumption levels than required to simply replace water loss due to evaporative cooling.

Acid-Base-Balance:

Enhanced respiration rate due to heat stress results in carbon dioxide loss and acid-base balance changes. However, these effects of altered acid-base balance are little understood. Weight gain has been enhanced with water carbonation or supplementation with acids such as ammonium chloride (NH₄Cl) or hydrochloric acid (HCl), suggesting that acid-base balance is critical for maximizing weight gain. While a number of additives have been added for acid/base balance all seem to act by increasing water intake. Thus, while water intake is an important consideration there is also an acid-base balance effect which should be considered.

Ration composition:

A large of number of diet strategies can be adopted to overcome heat stress. The addition of dietary fat is reported by several workers to increase the growth rate. The "fat effect" is supposed to be mediated by less waste heat produced in metabolizing fat. Thus, more energy is partitioned into productive purposes. Hence, the net affect of feeding fat is to make more dietary energy available to the bird. Research has shown that increased energy intake, during heat stress, may increase growth rate, but invariably at the cost of higher mortality. But, birds of heavier weight are more susceptibility to increased mortality.

Mineral Fortification:

During heat stress mineral excretion via the urine and feces is increased. Whether specific benefits with mineral supplementation exist, independent of their effect on water intake, is not known. However, it would appear that potassium based salt mixtures are superior to sodium when added to drinking water.

VITAMIN FORTIFICATION:

Vitamin fortification results range from the slight to magnificent response. Generally speaking, vitamin fortification will not solve a heat stress problem. However, it is critical that the bird be provided a good solid vitamin fortification premix and not has vitamins withdrawn. It has been observed that withdrawing the vitamin premix from heat stressed broilers results in a greater reduction in performance (3.2%) than withdrawing such premixes from birds housed within a thermo neutral environment (2.8%). Needless to say, such performance reductions far offset the cost of the vitamin premix.

Protein Consideration:

In this area controversy exists. Some recommend increasing dietary protein level and some recommend reduced levels with improved essential amino acid balance. There appears to be sufficient research to suggest that reduced protein is the avenue of choice as this has been shown to improve growth and enhance survivability.

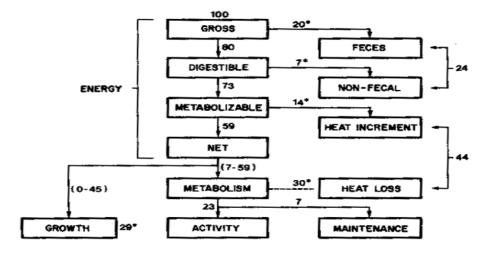
Chapter 16

Energy

In nutritional sense energy is not a nutrient. It is derived or abstracted from the nutrients. When energy forming nutrients like carbohydrates, fats and proteins are oxidized /burned or metabolized in the body, it generates energy. Energy is dissipated in the cell in the form of ADP (Adenosine Diphosphate), ATP, GDP, GTP, NAD. There is a continuous requirement of energy for different traits in the body i.e. maintenance, physical digestion. absorption. metabolism, growth, movement. production and reproduction. Energy is measured in calories. A calorie is defined as the amount of heat required for 1g of water form 15.5 to 16.5C. Energy can be expressed in Kcal, Mcal and MJ. When 1g of carbohydrates is catabolized in the body, it furnishes 4 Kcal. When 1g of protein is catabolized in the body, it furnishes 4 Kcal. When 1g of fat oxidized in the body it furnishes 9 Kcal of energy.

Gross energy (GE):

Gross energy is the total heat of combustion of a material as determined with a bomb calorimeter ordinarily expressed as kilocalories per kilogram of feed. The gross energy value of a feed has no relationship with feed's digestible, metabolizable or net energy values, except that the last can never exceed the first. Certain products such as coal, mineral oil and lignin have a high gross energy values but, because of their indigestibility, are of no energy value to the animal. Fats because of their greater proportion of carbon and hydrogen, yield 2.25 times more gross energy per kg than do carbohydrates and proteins.



Partitioning of energy in the body

Digestible energy (DE):

It is the portion of gross energy of a feed which does not appear in the feces. It includes metabolizable energy as well as the energy of the urine.

Metabolizable energy (ME):

It is the portion of gross energy consumed which is utilized by the bird for accomplishing maintenance, growth and production. It is that portion of energy that not appears in the feces and urine. It is digestible energy minus energy of urine.

Net energy (NE):

Net energy is the portion of metabolizable energy which may be used as needed by the animal for growth and production. It differs from metabolizable energy in the sense that the latter does not include the heat of nutrient metabolism. No net energy is used for heat production, to keep the bird warm.

Heat increment (HI):

It is the difference between metabolizable energy and net energy. It represents the heat unavoidably produced by the bird incidental with nutrient digestion and metabolism. It has been referred to also as work of ingestion, specific dynamic effect and thermogenic effect. This heat is useful only for keeping the bird warm during very cold weather. At other times the energy represented by this heat is not only a complete loss but also may actually interfere with production by causing the bird to be too warm.

Energy dynamics in relation to nutrients:

When body is in positive energy balance, protein / amino acids are used for synthesis of enzymes, hormones, muscles and egg production. However, in situation of negative energy balance in the body, proteins are burnt / oxidized for production of energy. Among all requirements energy is predominant in the body means that first of all energy requirements should be properly managed. When carbohydrate feeding is done for the provision of energy, excess carbohydrates are converted into fat deposition. When excess fat is fed than the requirements of energy provision, they are deposited as fats in the fat depots. Likewise when excess protein/amino acids are fed than the requirement, they are converted into glucose and eventually and fat is synthesized and deposited in the body. Chapter 17

Anti-Nutritional Factors

Compounds that interfere with the feed intake, availability or metabolism of nutrients in the bird are referred as 'anti-nutritive factors'. There are enormous numbers of naturally occurring compounds throughout the plant kingdom, which have anti-nutritional activity. Their biological effects can range from a mild reduction in animal performance to death even at low intakes. Typical concentrations for selected toxins are presented in Table 1.

| Toxin | Principal sources | Typical concentrations | |
|------------------------------|---------------------------------------|------------------------|--|
| Lectins Jack bean | | 73units/mg protein | |
| | Winged bean | 40-320units/mg | |
| | Lima beans | 59units/mg protein | |
| Trypsin inhibitors | Soybean | 88units/mg | |
| Cyanogens | Cassava root | 186mg HCN/kg | |
| Condensed tannins | Acacia spp. | 65g/kg | |
| | Lotus spp. | 30-40g/kg | |
| Saponins (steroidal) | Brachiaria decumbens; Panicum spp. | - | |
| S-methyl cysteine sulphoxide | Kale | 40-60g/kg | |
| Mimosine | Leucaena leucocephala | 145g/kg (seed) | |

Table 1. Plant toxins: sources and concentrations

(D'Mello, 1995)

I- HEAT-LABILE PLANT TOXINS:

1. Lectins (hemagglutinins):

The terms phyto-hemagglutinins, phytagglutinins and lectins are used interchangeably. Lectins comprised of large number of compounds, which are glycoproteins having 60,000-100,000 molecular weight. Haemagglutinins are compounds, which agglutinate red blood cells (RBC). They are proteins that possess a specific affinity for certain sugar molecules. Since carbon-hydrate moieties exist in most animal cell membranes, they may attach themselves to these so-called receptor groups if the specific structure of the latter is suitable, e.g. there are over 400,000 estimated binding sites for kidney bean agglutinin on the surface of each erythrocyte.

Lectins are found in most types of beans, including soybeans. The most active substances in agglutination of blood cells, and the most toxic, were found in *Phaseolus vulgaris* (phasin) and *glycine max* (soybean agglutinin). The *Vicia faba* lectin consists of 2 apparently identical sub-units and is heatlabile. The interaction of lectin components with glycoproteins on the cell surface is manifested *in vitro* by an agglutination of the cells. It has been demonstrated that fababean lectins specifically interact with D-mannose and D-glucosamine residues on the surface of erythrocytes, whereas soybean lectins react with D-galactose amine residues.

The prime example of a lectin with potent anti-nutritional and toxic properties is concanavalin-A, a component of the jack bean. Concanavalin-A enhances the shedding of brush-border membranes and decreases villus length, thereby reducing surface area for absorption in the small intestine. The overall effect is reduced nutrient absorption, but immune function may also be impaired. Raw kidney beans were found to interfere with vitamin E utilization in chicks. Raw navy beans have been found toxic for Japanese quail but not toxic for germ-feed birds. Diet rich in raw soybean has a goitrogenic effect. This is indicated by the fact that fecal loss of thyroxine from the gut is higher in animals fed raw soybeans. Raw soybean meal reduces fat and fatty acids absorption {not soybean trypsin inhibitor (TI)} in young chicks. Such meals also depress utilization of vitamin D in turkey. These effects are not found when the meals include heated soybean.

2. Proteinase inhibitors:

Legumes, such as the soybeans, lupines, field peas, fababeans and peanuts contain anti-nutritional factors, which inhibit animal performance. Proteinase inhibitors are the bestknown heat-labile anti-nutritional factors, found virtually in every legume. These are proteins in nature and frequently combine with the enzymes associated with protein digestion such as trypsin and chymotrypsin, significantly inhibiting their function. This inhibition, if not inactivated, is accompanied by moderate-to-severe depression in animal performance. Two types of soybean protein inhibitors have been identified, the Kunitz inhibitor (KSTI) and the Bowman-Birk inhibitor (BBI). It was widely accepted that KSTI was heat-labile, while BBI was heatstable, while both KSTI and BBI are inactivated during moist heat treatment. The trypsin inhibitors (TI) of soybean are now well characterized and are an important determinant of nutritive value.

Trypsin inhibitor is not the only factor in raw soybeans that inhibits growth. Removal of TI from a crude extract of raw soybeans increased the protein efficiency ratio (PER) to only about 40% of that obtained by heat treatment. Removal of the TI without the application of heat did not restore the pancreas to its normal size, but again accounted for about 40% of the total effect of unheated soybean protein. The principle cause of growth inhibition in raw soybeans for all ages of animals is a combination of the effects of the TI and the fact that the soy protein is in an undenatured state. Today, it is well accepted that moist heat treatment inactivates the heat-labile anti-nutritional factors in raw soybean products as well as denaturing the protein by making it more digestible.

3. Cyanogens:

Cyanogens occur widely in plants and in diverse forms. In sorghum and cassava (Table 1), the predominant cyanogens are, respectively, dhurrin and linamarin. The later compound is also present in linseed. Cyanogens are glycosides that readily yield hydrocyanic acid (HCN) and it is this later molecule that causes dysfunction of the central nervous system (CNS), respiratory failure and cardiac arrest. Metabolizable energy (ME) values for poultry tend to be lower in untreated cassava root meal, presumably because of its cyanogenic potential.

II. Heat stable Plant Toxins:

1. Antigenic proteins:

Certain storage proteins of legume seeds are capable of crossing the epithelial barrier of the intestinal mucosa to elicit adverse effects on immune function in farm animals. In the case of the soybean, the antigenic proteins have been identified as glycinin and conglycinin.

2. Tannins:

The word 'tannin' is very old and reflects a traditional technology. 'Tanning' (water-proofing and preserving) was the word used to describe the process of transforming animal hides into leather by using plant extracts from different plant parts of different plant species. Examples of plant species used to obtain tannins for tanning purposes are a*cacia* species, oak, eucalyptus and pine.

Tannins are complex polyphenolic compounds having molecular weights in the range 500 to 3000 daltons. They are

widely distributed in feeds of plant origin. Smith and Gayon first reported the presence of condensed tannins in 1959. Another scientist, Durkee (1971), identified cyanidin, pelargonidin and an artifact n-butyl derivative of cyanidin in the hydrolytic products of rapeseed hulls.

There are different forms of tannins. Gallic acid is derived from quinic acid. Ellagotannins are formed from hexahydroxydiphenic acid esters by the oxidative coupling of neighboring gallic acid units attached to a D-glucose core. coupling forms Further oxidative the HT polymers. Proanthocyanidin biosynthetic precursors are the leucocyanidins (flavan-3,4-diol and flavan-4-ol). Upon autoxidation, in the anthocyanidin form and absence of heat. they 3deoxyanthocianidin, which, in turn, polymerize to form PA. Hydrolyzable tannins are molecules with a D-glucose as a central core (Figure-1). The hydroxyl groups of these carbohydrates are partially or totally esterified with phenolic groups like gallic acid (gallotannins) or ellagic acid (ellagitannins). There are 2 additional classes of HT, namely taragallotannins (gallic acid and quinic acid as the core) and caffetannins (caffeic acid and quinic acid). Gallotannins belongs to the phenolic groups that esterify with the core and are sometimes constituted by dimers or higher oligomers of gallic acid. The most famous source of gallotannins is tannic acid. Ellagitannins consist of hexahydroxydiphenic acid, which spontaneously dehydrates to the lactone form, ellagic acid.

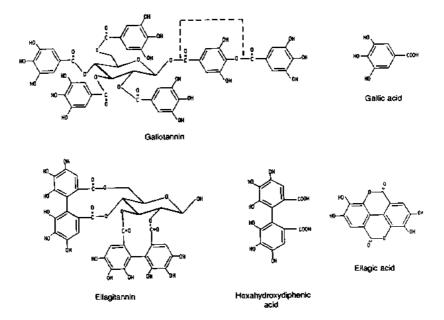


Figure 1. Hydrolyzable tannins

(Brown, 2003)

Condensed tannins are more widely distributed in leguminous forages (Table 1) than HT. They are oligomers or polymers of flavonoid units (i.e. flavan-3-ol) linked by carboncarbon bonds not susceptible to cleavage by hydrolysis. The term, proanthocyanidins (Figure 2) is derived from the acid catalyzed oxidation reaction that produces red anthocyanidins upon heating PA in acidic alcohol solutions.

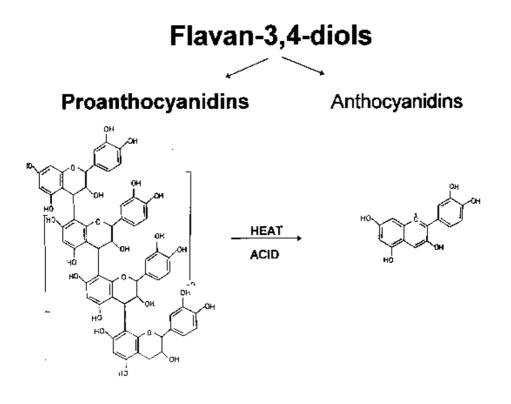


Figure 2. Condensed tannins

The tannin's phenolic group is an excellent hydrogen donor that forms strong hydrogen bonds with protein's carboxyl group. For this reason, tannins have a greater affinity to proteins than to starch. Hydrophobic bonds are stronger at higher ionic strength (higher tannin/protein ratios) and higher temperatures.

Tannins either hydrolyzable or condensed form tannin-protein complexes in similar manners. Proteins thus bound are generally resistant to attack by proteases and hence may be unavailable. However, it is hypothesized that HT may have a less damaging effect on protein digestion because these tannins may hydrolyze in the acidic gastric environment and release the bound proteins.

Animals fed diets with a level of tannins under 5% experience depressed growth rates, low protein utilization, damage to the mucosal lining of the digestive tract, alteration in

the excretion of certain cations and increased excretion of proteins and essential amino acids.

3. Glucosinolates:

Glucosinolates are thioethers. They generally consist of a sugar entity, b-D-thioglucose, with an ester bond to an organic aglycone that is an alkyl group yielding isothiocyanate, nitrile, goitrin, thiocyanate, sinapine, S-methyl cysteine sulfoxide (SMCO) or a similar compound upon hydrolysis. These compounds often contribute a bitter, 'hot' taste to condiments (mustard, horseradish) and may exhibit goitrogenic or antithyroid activity.

Rapeseed oil represents the third highest produced oil of the vegetable and marine oils. Oils high in erucic acid, a longchain fatty acid, have important industrial uses like production of nylon and mechanical lubricant etc. Rapeseed meal is an inexpensive protein supplement often used in place of soybean meal, which has similar nutritional value. Although many brassica are relatively quality feed supplements, but many of them contain anti-nutritional factors, which decrease digestibility and may be harmful to the birds.

The glucosinolates themselves are not particularly harmful, but the metabolites produced by enzymatic breakdown are known to have detrimental effects. Glucosinolases (commonly referred to as thioglucosidases or myrosinase) are enzymes found in the plant. When the plant is damaged, the enzymes are released and the glucosinolates are converted to the more toxic metabolites. At neutral pH, glucose and sulfate are broken off the glucosinolate resulting in isothiocyanates, thiocyanates and oxazolidinethiones (e.g. goitrin). Nitriles may also be produced at low pH.

Effects of metabolites:

I. Goitrin:

Progoitrin is a specific glucosinolate. It is converted to goitrin, an oxazolidine-2-thione, by the myrosinase enzymes. Goitrin inhibits thyroid function by inhibiting the incorporation of iodine (I) in the reactions leading to thyroxin particularly in the conversion of tyrosine to diiodotyrosine. It also interferes with thyroxin secretion.

II. Thiocyanates:

These can inhibit I-uptake by the thyroid, leading to reduced iodination of tyrosine and results in decreased production of important thyroid hormone thyroxine.

III. Isothiocyanates:

Isothiocyanates are usually not consumed in enough amounts to cause problems, but they may be irritating to mucous membranes and have anti-thyroid effects. However, if they are consumed as glycosinolates and then hydrolyzed to isothiocyanates in the gut, they can have powerful anti-thyroid effects and interfere with the synthesis of necessary thyroid hormones.

IV. Nitriles:

Nitriles cause reduced growth rate and increased liver and kidney size. Mixed function oxidases may metabolize the nitriles resulting in enzyme induction, thus increasing liver size. Nitriles may also be converted to thiocyanate and produce anti-thyroid effects.

V. Sinapine:

Sinapine is an ester of choline and sinapic acid. It is generally an issue for poultry because it is fermented by bacteria

in the ceca to trimethylamine. Some types of chickens do not have trimethylamine oxidase. Therefore, when they are fed diets high in canola meal, trimethylamine builds up and is deposited in the egg. If the diet contains >0.1% sinapine a fishy odor may be present in the egg. Canola meal has 2.5-3% sinapine.

vi. S-Methylcysteine sulfoxide:

S-Methylcysteine sulfoxide is an amino acid found in brassicas. It may comprise as much as 4-6% dry matter in brassicas. The dimethyl disulfide that results from these reactions is an oxidant.

vii. Indole glucosinolates:

The role played by indole glucosinolates in the alleged anti-nutritive effects of rapeseed glucosinolates is not clear. The situation regarding indole glucosinolates is complicated by the high sensitivity of these compounds to thermal degradation as approximately 70% destruction has been reported during commercial canola seed processing.

4. Gossypol:

Cottonseed meal is one of the important byproduct i.e. of great concern in animal feeding, especially poultry nutrition. Gossypol is a natural toxin present in the cotton plant that protects it from insect damage. Glandless varieties of cotton have been evolved, which have very low levels of gossypol. Unfortunately, these varieties need high levels of insecticides to yield well. Gossypol level in whole cottonseed is 0.8%.

Gossypol pigment occurs in cottonseed in free and bound forms. In whole seeds, gossypol exists essentially in the free form, but variable amounts may bind with protein during processing to yield inactive forms. Free gossypol is the toxic entity and causes organ damage, cardiac failure and death. Poultry cannot handle much gossypol before toxicity signs develop.

5. Saponins:

Saponins are glycosides, which have profuse foaming properties, producing distinctive honeycombed stable foam when an aqueous solution is shaken. They are found in many plants, but get their name from the soapwort plant (*Saponaria*), the root of which was used historically as a soap (Latin *sapo* means soap). Steroidal saponins occurs in certain pasture plants such as *Panicum* species (Table 1), whereas, triterpenoid saponins are present in soybeans. They are bitter compounds, affecting palatability and feed intake. They have growth depressing properties in poultry.

6. Phytate (phytins):

Phytins, the mixed calcium (Ca) and magnesium (Mg) salts of myo-inositol-1, 2, 3, 4, 5, 6-hexakis (dihydrogen phosphate), also known as phytic acid, are common constituents of plant tissue, especially of cereals and legumes. They are the principal form of phosphorus (P) in many seeds; 60-90% of the P in these seeds is present as phytic acid. Beans generally have a high phytic acid content ranging between 150-1800mg/100g and fababeans are relatively rich in this substance (about 250-350mg/100g).

Phytic acid is present in seeds in an almost water-soluble form (as sodium (Na) or potassium (K) salt). During processing, it becomes insoluble (as Ca, Mg or ferric phytate). Phytate also forms complexes with proteins, making them less soluble. This effect is related to its ability to form insoluble combinations with proteins in an acid medium and in a range of pH, which corresponds precisely to the optimum for the action of pepsin.

The effect of phytic acid on mineral availability is influenced by many factors, such as the mineral composition of the feed as well as its association with dietary protein, heat treatment, pH and the presence of other components reducing the mineral bioavailability such as fiber, oxalates, phenolics, tannins, saponins and histidine, which are capable of binding or interacting with minerals or phytic acid to varying extents.

7. β-Mannans:

Soybean meal is the main protein source in poultry feeds. Commercial soybean meal processing denatures heat-labile components such as TI. But in properly processed soybean meal, the amino acid availability does not exceed 90%. Soybean meal contains approximately 22% non-starch polysaccharides (NSP), which is one of highest NSP concentrations among protein meal sources. It is found mainly in the endosperm and hulls of the soybean. Its ability to hold water may help prevent dehydration of the soybean plant during growth.

In soybean meal, β -mannan occurs in the form of a galactomannan polysaccharide (GP). Guar meal is predominantly β -mannan and reduces poultry performance. Guar gum and mannan are virtually undigestible in the gastrointestinal tract (GIT) due to the absence of appropriate enzymes. Hence, β -mannan freely interferes with normal nutrient digestion and absorption processes, a problem discriminating by its water-soluble nature.

Endo- β -mannanase degrades GP in soybean meal and other ingredients. This enzyme improves the utilization of typical broiler feeds containing soybean meal at an inclusion rate of about 0.5-1kg/T of complete feed. When FCR was adjusted to a constant body weight of 2.48kg, the broilers, which consumed the enzyme-treated, feed required 0.149kg less feed/bird.

Mycotoxins:

The word mycotoxin is derived from words 'myco' and 'toxin' meaning fungus and poison, respectively (Cheeke and Shull, 1985). Mycotoxins are toxic, secondary metabolites of low molecular weight produced by naturally occurring fungi (Chu, 1992). Mycotoxins are neither infectious nor contagious, but can occur on a herd-wide basis. (Wren, 1994). It can cause toxicity

when consumed by animals. The mycotoxins of major concern are aflatoxins (B1, B2, G1 and G2), trichothecenes {including deoxynivalenol (DON), nivalenol (NIV), T-2 toxin and HT-2 toxin}, zearalenone, fumonisins (fumonisin B_1), ochratoxins (predominantly ochratoxin A) and ergot.

Diseases in animals and human beings resulting from the consumption of mycotoxins are called *mycotoxicosis* and signs of the many mycotoxicosis are diverse, numerous and often dependent on species, sex, age, stress, reproductive and health status of the animal.

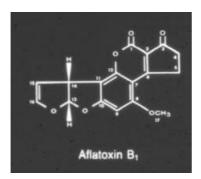
Other toxin producing fungi grow on grain at moisture contents of 17-40% and over a wide range of temperatures, from below freezing for species of *Penicillium* and *A. fumigatus* to more than 55° C. The quality of the grain and its suitability for storage are adversely affected by high moisture content; physical damage to the kernels; and the extent to which storage fungi have invaded the seed.

Aflatoxins and aflatoxicoses:

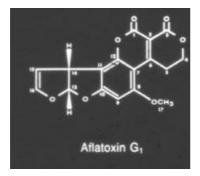
In the 1960 more than 100,000 young turkeys on poultry farms in England died in the course of a few months from an apparently new disease termed 'turkey X disease'. It was soon found that the difficulty was not limited to turkeys. Ducklings and young pheasants were also affected and heavy mortality was experienced. In fact, the toxin-producing fungus was identified as *Aspergillus flavus* (1961) and the toxin was given the name 'aflatoxin' by virtue of its origin (*A. flavis* - Afla).

Aspergillus flavus and A. parasiticus are commonly cause-stored grains to heat and decay and, under certain conditions, invade grain in the field. The problem is serious in subtropical and tropical regions of the world where cereals, peanuts, corn and copra are important crops. Aflatoxins B_1 , B_2 , G_1 and G_2 (Figure -----a,b,c,d) are produced by A. flavus and A. parasiticus in grains in both field and storage. The aflatoxigenic Aspergilli are generally regarded as storage fungi, proliferating under conditions of high RH and temperature. Infection is most

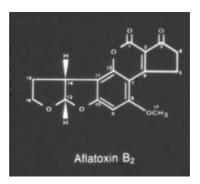
common after insects, birds, mites, hail, early frost, heat and drought stress, windstorms and other unfavorable weather have damaged the kernels. Aflatoxins M_1 and M_2 are found in milk from animals fed aflatoxin-contaminated feeds.



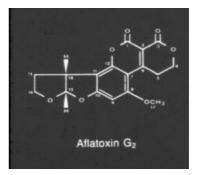
Aflatoxin B1 (C17H12O6)



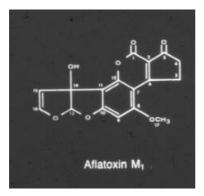
Aflatoxin $G_1(C_{17}H_{12}O_7)$



Aflatoxin B₂ (C₁₇H₁₄O₆)



Aflatoxin $G_2(C_{17}H_{14}O_7)$



Aflatoxin $M_1(C_{17}H_{12}O_7)$

Figure . Chemical structure of aflatoxins

(Brown, 2003)

Aflatoxin is present in the spores of *A. flavus*, which sometimes are produced in great abundance on the ears of fungus-infected corn. When corn is combined and unloaded at elevators or other transfer points, it generates considerable dust, and some of this dust may contain aflatoxin. Grain invaded by *Aspergillus* species is highly friable; therefore great care should be taken when feeding grain screenings. Broken grains often have very high levels of aflatoxin concentration.

Symptoms of aflatoxin poisoning:

Generally, young birds are more susceptible to aflatoxicosis than mature flocks, but there is considerable variation among species and breeds. Ducklings and turkey poults are highly susceptible to this toxin. Broilers and commercial-egg pullet chicks fed rations containing 4ppm aflatoxin may show up to 5% mortality through the first 3-weeks of the brooding period. Growth rate is markedly reduced, and abnormal feathering is attributed to a disturbance in protein deposition. Dietary levels of 2ppm aflatoxin are generally accepted as the threshold for measurable depression in growth and FCR.

In poultry, besides fatty liver and kidney disorders, leg and bone problems can develop as well as outbreaks of coccidiosis. Aflatoxins may cause vaccines to fail, increase the bird's susceptibility to disease, and result in suppression of the natural immunity to infection.

Decreased blood clotting results in a greater downgrading and condemnation of the birds because of massive bleeding and bruises. Less carcass pigmentation is exhibited and egg yolks are paler. The hatchability of eggs can drop, and reduced production may be noted as well as smaller eggs with shell problems. Growth is restricted and mortality increases, especially during growing period.

Zearalenone and zearalenol:

Zearalenone and zearalenol are produced almost exclusively by *Fusarium* species that contribute to the ear and stalk rot that occurs in the ears of corn and on the heads of cereal grains (scab) standing in the field or in stored ear corn. *Fusarium graminearum* requires a minimum of 22-25% moisture to grow in cereal grains. Generally, shelled corn stored at these moistures is likely to be colonized by a mixture of other fungi, yeasts and bacteria with which *F. graminearum* competes poorly.

Broiler chicks and laying hens are affected very little by dietary zearelenone even when fed massive doses. Pure zearalenone fed to broiler chicks and finishing broilers (10-800ppm) produced no effect on weight gain, feed consumption and FCR. The weights of the liver, heart, spleen, testicles, oviduct, comb and bursa remaining unchanged.

Fusarium toxins:

Extensive data now exist to indicate the global scale of contamination of cereal grains and animal feed with Fusarium mycotoxins. Of particular importance are the trichothecenes, zearalenone and the fumonisins. The trichothecenes are subdivided into 4 basic groups, with types A and B being the most important. Type A trichothecenes include T-2 toxin, HT-2 toxin, neosolaniol and diacetoxyscirpenol (DAS). Widespread contamination of maize and animal feed with fumonisins has recently been reported. In most instances the predominant fumonisin was fumonisin- B_1 .

Fusarium tricinctum and some strains of *F. graminearum*, *F. equiseti*, *F. sporotrichioides*, *F. poae* and *F. lateritium* produce T-2 and other toxic trichothecenes. *Fusarium graminearum* growing in the ears of corn and on the heads of cereal grains before harvest may produce other toxins besides zearalenone including DON. Fumonisin is produced by fungus *Fusarium moniliforme*.

Trichothecenes inhibit protein and deoxyribose nucleic acid (DNA) synthesis and interfere with growth, reproduction and the structural integrity of tissues. In poultry, T-2 toxin in feed contaminated with 1-3.5ppm of T-2 and 0.7ppm of HT-2 (a closely related toxicant) may produce lesions at the edges of the beaks, abnormal feathering in chicks, a drastic and sudden drop in egg production, eggs with thin shells, reduced weight gains and mortality. The same feed given to turkey results in reduced growth, beak lesions and less immunity to infection. The T-2 toxin can also cause mouth and intestinal lesions and decreased feed consumption.

With ruminants and poultry though, DON has been shown to be poorly absorbed, extensively metabolized and rapidly cleared from tissues and fluids. Therefore they are relatively insensitive to the dietary concentrations of DON likely to be found in feeds. The maximum safe level of fumonisin for all classes of poultry is 50ppm.

Tibial dyschondroplasia (TDP) is a common and economically important bone deformation in growing broiler chickens and turkeys. The lesion appears in a cone of cartilage extending distally from the proximal tibiotarsalphysis. This toxin may be largely responsible for the TDP syndrome in poultry; it also kills chick embryos in fertilized eggs.

Ochratoxins (nephrotoxins):

Ochratoxin, produced primarily by members of the *Aspergillus ochraceus* group and a number of species of *Penicillium*, especially *P. viridicatum*, have been found in some samples of feed grains. Ochratoxin-A and ochratoxin-B are 2 forms that occur naturally as contaminants, with ochratoxin-A being more ubiquitous, occurring predominantly in cereal grains especially maize, oats, barley and wheat and in the tissues of animals reared on contaminated feed. Another mycotoxin, citrinin, often co-occurs with ochratoxin.

In laying hens, decreased feed intake, egg production and egg weight, increased incidence of blood and meat spots on the eggs and characteristic yellow-brown staining of egg shells, attributed to urate diuresis, are the major signs. Immunosuppression induced by ochratoxins exacerbates intercurrent viral respiratory infection. Paler carcass results from defective hemopoiesis.

| Name | Mycotoxin | Description | Use | Remarks |
|--------------------------|-------------|---|---|--|
| Black light | Aflatoxins | Cracked grain or screenings are viewed in the dark under long- wave ultraviolet light (approx. 365nm). Samples are checked for "glowers", or starchy endosperms that fluoresce a bright greenish yellow (BGYF). The BGYF compound in not aflatoxin but a substance produced by <i>A. flavus</i> or <i>A.</i> <i>parasiticus,</i> when growing on living seed. This compound is not produced in dead seed. Grain may be cracked for testing with a cereal grain grinder | A rapid, presumptive test for the BGYF compound (kojic acid), a metabolite usually co-synthesized with aflatoxin. The minicolumn, TLC, GLC or HPLC tests should analyze positive samples. A standard should be used with each test, & fluorescing grain should be checked to see that compound is water- soluble & in starchy endosperm & peripheral parts of germ | Quick but only indicative of <i>A. flavus</i> or <i>A. parasiticus</i> . The test is neither quantitative nor qualitative. Only trained personnel should use it because many types of foreign material (glumes, cobs, some weed seeds & soybean fragments), may fluoresce but are not usually water- soluble. The training is minimal |
| Mini-column ² | Aflatoxins | Finely ground grain is extracted with solvents and purified by a precipitation procedure. Then the extract is washed through a column containing 2 absorbents. Migration and long- wave UV light are used for detection | Rapid (9-15 minutes) simple; and semi- qualitative; requires inexpensive equipment; can detect aflatoxins down to 4ppb | Quick but only qualitative. It can be used as a "go or no go" measurement above 4ppb. Short minicolumn test is not suited for mixed feeds. |
| TLC ³ | Aflatoxins, | Grain is extracted & extract | Can identify & | Slow, somewhat |

Table 3. Methods of detecting mycotoxins

| Name | Mycotoxin | Description | Use | Remarks |
|--------------------|---|---|--|--|
| | zearalenone trichothecenes | partially purified before placement on a TLC plate. UV light and migration are compared visually or densitometrically with standards used for identification of fluorescent aflatoxins or zearalenone. Trichothecenes do not fluoresce. | quantitatively determine aflatoxins B_1 , B_2 , G_1 & G_2 . The detection limit for aflatoxins is 1-3ppb. The sensitivity limit for zearalenone is 50ppb. If necessary, confirmation can be made by additional chemical tests on the TLC plate. | expensive, but precise and reasonably accurate. Detection limits for trichothecenes are relatively low. Many compounds, especially trichothecenes, cause dermal reactions. |
| HPLC ⁵ | Aflatoxins, ergopeptines, Fumonisins | Grain is extracted and the extract fractionated on either normal or reverse phase columns. The aflatoxins are detected using either UV- absorbance or fluorescence detectors. | Accurately & quantitatively identify aflatoxins B_1 , B_2 , G_1 & G_2 , and their metabolites. | The initial capital investment & technical expertise are the highest for this technique & it is potentially the most sensitive. |
| ELISA ⁶ | Aflatoxin, zearalenone, ochratoxin, DON, T-2 | Grain is extracted in methanol and placed in plastic well. Addition of antibody-enzyme conjugate and chromagen results in color, which is quantitative measure of alkaloid. | Test is specific for target alkaloid but may be cross-reactive within members of an alkaloid group. Sensitive to 5ppb (aflatoxin) and requires 10 minutes to complete. | ELISA requires a plate reader for accurate quantitation but no other specialized equipment is necessary. ELISA is a good compromise of sensitivity, speed & expense. |

1. Applied Biochem. Vol. 1

2. J. Agric. Chem. 23: 1134-36, 1975;

J. Agric. Chem. 25, 1154-50, 1975,
 Thin Layer Chromatography (Official Methods of Analysis, Chapter 26. AOAC, 12th Ed., 1975)
 Gas-liquid chromatography
 High-pressure liquid chromatography
 Enzyme-Linked Immunosorbent Assay

(Jacobsen et al., 1993)

Detecting aflatoxin:

The methods of aflatoxin analysis fall into 3 categories (Table 3). Visual inspection of the grain, which may locate lots presumed to be contaminated with aflatoxin (blacklight test). Rapid screening procedures to determine the presence of absence of aflatoxin (the fluorometric iodine rapid screening and minicolumn tests). Laboratory procedures quantifying the actual amounts of toxin present (thin-layer chromatography, gas-liquid chromatography, high-pressure liquid chromatography, fluorometric iodine, or ELISA tests).

Toxins

A poison by any other name is still a poison. It is the dose that makes the poison. Simply stated, almost anything in an overdose or a diet restricted to only one ingredient would eventually become toxic to the birds. A misplaced decimal point in medication dosage can cause toxicity. Water mixable medications can become toxic if the weather is hot and the birds are drinking more water than normal. Even high levels of certain vitamins, such as A and D, can be toxic and some doses of medication that are perfectly fine for chickens and turkeys can kill waterfowl.

Antibiotics:

Some antibiotics can be troublesome if their use is not controlled. In *turkey poults*, the subcutaneous injection of gentamicin can cause depression, edema, injection site hemorrhage and kidney damage. Streptomycin (and dihydrostreptomycin sulfate), injected intramuscularly (IM), can cause respiratory distress, paralysis and mild convulsions. Among the anti-bacterials, anti-coccidials and growth promoters added to feed or drinking water, some presenting toxicity risks are shown in table 1.

Anti-protozoals:

Some anti-protozoals such as Nitrazol and Emtryl have caused growth depression; drop in egg production, incoordination and tremors, convulsions and death in geese, ducks, pigeons and turkeys. Doses safe for other poultry may be poisonous to waterfowl. Parasitic and worming treatments should also be used with caution. Benzimidazoles (cambendazole, mebendazole and fenbendazole) and phenothiazine are all pretty well tolerated by most birds. Ivermectin is probably the safest.

| | Effects | |
|------------------|---|--|
| Drugs | | |
| Sulfonamides | Haemorrhagic syndrome, hepato- and nephrotoxicity | |
| Ionophores | Myotoxicity, anorexia, depression, weakness, paralysis | |
| Arsenicals | Colic, anorexia, depression, weakness, diarrhoea, death | |
| Imidazothiazoles | Acute toxicity and death | |

Table 1. Some poisonous drugs and their toxic effects

Levamisole (and tetramisole, which is no longer available in most countries), should be used with caution, (because of it's chemical makeup, an effective dose of levamisole is half that of tetramisole.)

Metals and minerals:

Under the group of minerals and metals, 3 stand out as the most problematic: sodium chloride or sodium bicarbonate, lead and calcium (Ca; as in the over-use of oyster shell).

Most sodium problems arise as a result of young chicks and turkey poults consuming too much saline water. Avoid using softened tap water to supply water to your birds while brooding. Some waterfowl have nasal salt glands that allow them to excrete excess, but play it safe with them too since they consume more water than chickens and turkeys. Sodium poisoning can cause kidney damage (more so in young birds than adults because their kidneys may not be fully developed when first hatched), and heart failure.

Calcium over-dose is a common occurrence, when overuse Ca supplements such as oyster shell. Oyster shell offered to any female before first egg can cause kidney damage. Never mix oyster shell into feed where young females or males of any age can free-feed. Oyster shell should be provided to laying females. A good vitamin and mineral supplement added to drinking water can sometimes provide enough Ca.

Vitamins:

Vitamins A, D_3 (cholecalciferol) and B_6 (pyridoxine), can be toxic when over-dosed. Excess Vitamin A can reduce egg production and growth rate, and cause osteoporosis. A simple top-dressing of Vitamin D_3 on feed consumed by chicks has caused kidney damage in field studies.

Disinfectants and fumigants:

Disinfectants and fungicidal fumigants such as phenolics, quats, chlorine bleach, formaldehyde, organic mercurials, thiram and captan cause toxicity when ingested or inhaled. The overexposure rates are too numerous to list here, so use them all with caution.

Insecticides:

Almost all insecticides are toxic and most attack the nervous systems of birds. The group that is relatively safe and effective to use is pyrethrum and synthetic pyrethroids. Most poultry dust and flee and tick sprays used for the control of feather mite contain a form of pyrethrum. These products are relatively safe to use.

Toxic gases:

Two common toxic gases that affect all birds are ammonia (NH_3) and carbon monoxide. Ammonia fumes can cause corneal ulceration and blindness from over-spent litter. Heart rate and breathing may be affected with bronchial hemorrhage, and egg production can drop when NH_3 levels rise. Growth rate of youngsters will be affected and they won't thrive in an environment, where there are strong NH_3 levels. Clean, dry litter will avoid this problem.

Biotoxin:

Botulism is caused by a biotoxin and is a common problem among waterfowl breeders with a pond, but can also be a problem when dead poultry are left in litter to be picked at by the live birds. Very small amounts of this toxin are deadly. Of the diverse types of naturally occurring animal toxins, the prion proteins of mammalian meat- and bone-meal have recently emerged as important feed contaminants necessitating statutory control. Prion proteins are harmless animal tissue components with the unique capability to transform themselves into agents causing fatal neurological lesions in a wide range of species.

Feed Additives

Additives are non-nutritive compounds added to poultry diets to improve performance, increase resistance to disease, enhance palatability and/or increase efficiency of nutrient uptake. Feed additives can be divided into 4 categories:

1. Supplements:

This category includes vitamins and provitamins, amino acids, trace-elements and non-protein nitrogen (NPN).

2. Auxiliary substances:

From the point of view of nutritional physiology, they are not essential. They are added to improve the quality and utilization of the feed ingredients and thus increase the level of production. Included here are antioxidants, enzymes, probiotics, flavors, emulsifiers and pelleting agents, colorants including pigments, preservatives, free-flowing agents and acidifiers.

3. Digestive enhancers:

These are the substances, which improve growth and feed conversion ratio (FCR) and decrease nutrient excretion.

4. Disease preventing agents:

These substances are added to the feed to protect the animals against diseases caused by internal parasites. Among these substances are coccidiostats for poultry. Medications are sometimes added to poultry diets (or water) to help prevent disease.

There are many others, which are often used in poultry diets. Below are the feed additives commonly included in poultry rations:

Antibiotics and anti-microbials:

Antibiotics are substances produced by microorganisms, such as bacteria and fungi, which in small quantities inhibit or kill other microorganisms. Chemobiotics have similar properties but are produced synthetically. Antibiotics were discovered in the second decade of twentieth century and used initially at high dosages to combat pathogenic bacteria that caused disease.

In 1949, Dr. Thomas Jukes stumbled on the curious observation that low level feeding of chlortetracycline to chicks improved growth and FCR. Today, more than 50% of all antibiotics produced are used in animal feeds. Antibiotics are used as growth promotants in all over the world. Table 1 gives a list of approved antibiotics for growth promotion, whereas, table 2 classifies growth-promoting antibiotics with respect to molecular type and spectrum of activity.

| Arsenicals | Chlortetracycline | Lincomycin | Spirimycin |
|------------|-------------------|-----------------|---------------|
| Avoparcin | Colistin | Olaquindox | Tylosin |
| Bacitracin | Flavomycin | Oxytetracycline | Tiamulin |
| Carbadox | Halquinol | Penicillin | Virginiamycin |

Table 1. Approved anti-microbial growth promotants

(Swick, 1997)

| Antibiotic | Activity | Туре | Molecular weight |
|-------------------|-----------------|--------------|---------------------|
| Avoparcin | Narrow g^{+1} | Glycopeptide | 1900 |
| Bacitracin | Narrow g+ | Polypeptide | 1400 |
| Chlortetracycline | Wide | Tetracycline | 515 |
| Colistin | Narrow g+ | Polypeptide | 1168 |
| Lincomycin | Narrow g+ | Lincosamide | 461 |

 Table 2. Antibiotic type and spectrum of activity

| Penicillin | Narrow g+ | β-lactam | 589 |
|---------------------------|-----------|-----------|---------------|
| Spiramycin | Narrow g+ | Macrolide | 884 |
| Virginiamycin | Narrow g+ | Peptolide | 750 |
| 1. Gram positive bacteria | 1 | | (Swick, 1997) |

Anti-microbial refers to synthesized chemicals that inhibit microorganisms. They have similar properties as antibiotics. These include arsenicals such as arsanilic acid and roxarsone (3nitro), and others such as carbadox, olaquindox, halquinol and copper sulfate. Many countries have banned sulfonamides and nitrofurans (furazolidone) as growth promotants because of problems with tissue residue and suspected carcinogenicity.

Mode of action:

Anti-microbial feed additives promote growth by altering the gut microflora to the benefit of intestinal tissue. Feeding of antibiotics to chicks has been clearly shown to improve performance, reduce illeal weight and decrease the population of *Clostridium perfringens* across different diet types. Various reports in the literature indicate that feeding of sub-therapeutic levels of antibiotics and anti-microbials result in suppression of mild but unrecognizable infection, decrease production of growth depressing toxins, lower nutrient use by micro flora and thinner gut wall capable of enhanced nutrient absorption.

In the late 1940, scientists discovered that young chickens grew faster when they were fed very low levels of antibiotics compared to chickens not fed antibiotics. None of the chickens exhibited any symptoms of a disease, nor was the level of antibiotic provided in the feed believed to be sufficient to effectively combat pathogenic bacteria if present. Subsequent experiments with chickens verified that apparently healthy birds grew faster and more efficiently when they received a low level of an antibiotic in their feed.

Alternatively, another possibility is that there may be a shift in the microorganism population causing an increase in the number of microorganisms that produce available nutrients and/or positive factors (Hathaway, *et al*, 1996; Hays, 1991). There is evidence that as feed-based antibiotics reduce the numbers of intestinal bacteria, thus a reduction of some harmful bacterial products such as NH_3 may occur. As a result, there may be lower turnover of intestinal cells and a decrease in the energy needs of the gut tissues. The energy needs of the gastrointestinal tissues are immense, and even a small decrease in energy use by these tissues will result in large amounts of energy freed up for growth and maintenance of other tissues.

Any of these scenarios, alone or in combination, could enhance growth of the host animal. To date, it is not been possible to define a direct cause and effect relationship between a specific change in the GIT microbial population and subsequent growth promotion. The mechanism, by which very low levels of anti-microbials will result in growth promotion, when given to apparently healthy birds over long periods of time, is still very much a mystery.

It has been estimated that there are several hundred different microorganisms present in the GIT, of which only a fraction may be present in sufficient numbers to play a major role in the GIT function and physiology (Salminen *et al.*, 1999). It is important to note that when there is a change in the microbial population of the GIT, e.g., in response to anti-microbial treatment, the total number of microorganisms does not change substantially in the long run. Instead, there is a shift in the balance between the various microbe populations.

Enzymes:

Enzymes are naturally occurring proteins that act, as biological catalysts for all metabolic processes in plants, animals and microorganisms. The complex biochemical reactions that form the metabolism of living organisms are regulated by thousands of enzymes, each promoting a specific reaction that takes place countless times everyday. Every enzyme has its own unique properties, like specific activity, stability, pH and temperature sensitivity and is substrate specific.

Why need enzymes in poultry feeds?

The digestive system of chicken comprises of natural enzymes to digest complex molecules in the feed like proteins, carbohydrates, lipids etc. Poultry feed involves many plant and animal origin ingredients. Plant origin ingredients include corn, oats, millet, wheat, rice, rice polish, deoiled rice bran, soybean meal, sunflower cake, ground nut cake, rapeseed meal, sesame meal, etc. Whereas animal origin ingredients include fish meal, meat meal, blood meal, etc. Chicken can easily digest animal residues; however plant material has certain inherent residues, which are not digested by chicken due to lack of endogenous Undigestible plant residues enzymes. are non-structural polysaccharides (NSP), galactosides, phytates and other antinutritional factors like lectins, tannins, trypsin inhibitors etc.

- sources of enzymes in body
 - Endogenous Enzymes
 - Exogenous Enzymes
- Exogenous enzymes are added in feed
- Used as feed additives
- Mostly digestive in nature

Classes of exogenous enzymes:

- Proteases
- Lipases
- > Phytases
- Non Starch Polysaccharidases

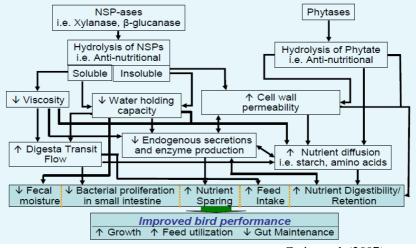
Usually, Exogenous Enzymes are used in Combination form not in single form

Enzymes for poultry:

Enzyme supplementation has the potential to improve the nutritive value of feedstuffs. The enzyme must be biologically active when reaching GIT. The structure of the enzyme is critical to its activity and can be altered by exposure to heat, extreme pH and certain organic solvents. The feed value of the grain must be known to identify what enzymes to use and in how much quantity and formulating more accurate diets to decrease variability and reduce feed costs.

For barley diets the enzyme β -glucanase is used and for wheat diets xylanase and arabinotylanase are used. Some enzyme products available may contain β -glucanase, cellulase and xylanase for improved ration digestion. Bacterial counts in the intestine are usually higher with wheat, barley and rye diets than with corn-based diets. The viscous digesta provides a stable environment for microbial growth and proliferation allowing for the establishment of bacteria in the upper small intestine. High intestinal bacteria populations irritate and thicken the gut lining by damaging microvilli and decreasing nutrient absorption.

Broilers respond well to feed enzymes, as their rate of feed intake is more than other poultry. This consumption of feed overwhelms the broilers ability to produce sufficient enzyme either from its own tissue or the micro flora in the gut. Feed enzymes used in wheat and barley diets improve energy availability, improve nitrogen retention and significantly effect protein digestion. The greatest benefit is seen with low quality feeds. By adding 0.1% enzyme to barley-based diets, less feed will be used (20%) and wet litter would not be a problem.



Craig *et al*, (2007)

Effects of exogenous enzymes:

- Improve digestibility and performance.
- Enhance the health status of flock is enhanced.
- Improve Litter Quality by reducing moisture content of feces.
- Cost effective feed formulation due to supplementation.
- Environmental benefits due to reduction in nutrient losses.

Advantages of Exogenous Enzymes:

- ↑ Digestibility
- ↑Feed Efficiency
- $\blacksquare \downarrow \text{Nutrient Losses}$

- Mitigate Anti nutritional Factors
- Make the poultry operations economical.

(Fernando et al., 2008)

Storage conditions for exogenous enzymes:

- Sensitive to high temperature and high humidity
- Heat stability should be considered during diet pelleting as they are denatured at high temperature.
- Popper handling procedure should be adopted to avoid hazards.

SUMMERY:

Non-starch polysaccharides increase viscosity of digesta due to their water holding capacity.

Non-starch Polysaccharidases results in better quality litter by reducing the viscosity of digesta. Due to which moisture content of feces is reduced. So incidence of pododermatitis decreased in broilers.

Phytase improve phytic phosphorus usage, thus reduce pollutant potential of excreta.

Reduce feed cost due to increase in nutrient availability especially protein and metabolizable energy.

Environmental pollution decrease by addition of exogenous enzymes.

Use of exogenous enzymes reduce the amount of excreta due to better nutrient utilization in body.

In Conclusion exogenous enzymes potentiate endogenous enzymes, improve feed intake, digestibility, gut health, litter quality and FCR. In addition they decrease environmental pollution. Caution regarding expiry date, shipping, storage conditions and usage in diet (pelleting) must be kept in mind. Their use in feed is cost effective.

1. Non-structural polysaccharides:

The term NSP covers a large class of polysaccharides excluding starch. These inherent residues trap (lock) valuable nutrients. A customized blend of enzymes unlocks these trapped nutrients and improves the feed nutritive value and FCR.

Soluble NSP can increase gut viscosity thus reducing effective contact of substrates and digestive enzymes. They can modify gut physiology to reduce internal secretion of water, proteins, electrolytes and lipids and can bind bile salts, lipids and cholesterol thereby changing digestive and absorptive dynamics of the gut. It can also increase retention time of digesta in the intestine thus decreasing oxygen tension to favor growth of anaerobic toxigenic microflora causing deconjugation of bile.

2. Phytates:

All feed grains, their byproducts and oilseed meals used contain phosphorus (P) in organic form as phytate-P and nonphytate-P. The non-phytate-P is easily digestible and hence bioavailable for the chicken and other monogastric animals. However phytate-P is largely unavailable to the chicken due to negligible amounts of phytase enzyme in the intestine, which hydrolyses the phytate bond. Moreover phytate-P is not assimable by chicken so additional organic or inorganic-P source has to be added to the feed.

3. Galactosides:

These are short chain carbohydrates usually found in legumes like soybean. Galactose being the main subunit is crosslinked to raffinose, stachyose and verbascose. These interfere with the gut physiology leading to flatulence and poor assimilation of nutrients.

4. Other anti-nutritional factors:

This class includes chemically varied type of residues, which usually occur in low concentrations. Trypsin inhibitors are proteins where as tannins and lectins are long chain NSP. They affect digestion by way of interfering with the action of endogenous enzymes, e.g., trypsin inhibitors inhibit action of trypsin like enzymes.

| Enzyme | Activity | Application | | |
|---------------|---|------------------------------------|--|--|
| Amylase | Splitting starch into dextrins | Cereals, starch | | |
| β-glucanase | Splitting β-glucans into oligomers | Cereals, like barley and rye | | |
| Cellulase | Splitting cellulose into oligosaccharides | Plant cell walls | | |
| Phytase | Splitting phosphate from phytate complex | Phytate containing plant materials | | |
| Hemicellulase | Splitting hemicellulose chains into oligomers | Plant tissue, seeds | | |
| Pectinase | Splitting pectin chains | Legume seeds, such as soybean | | |

 Table 3. Some important enzymes and their activities

Enzymes for breaking down carbohydrates:

Starch is a very important energy carrier in plants. The bird's enzyme system usually breaks down these molecules into simple sugars that are then absorbed. Cereals such as wheat, rye and barley, e.g., contain long, complex carbohydrate molecules known as NSP, for which poultry do not produce the necessary digestive enzymes. The major NSP in wheat are called arabinoxylans, and those in barley are β -glucans. It is now well recognized that these components are anti-nutritional in behavior.

Not only do NSP increase the viscosity of digesta, which means that the birds own enzymes have a harder time locking onto nutrients and the absorption of these nutrients is reduced, but they also encapsulate nutrients, thus making them unavailable to the birds. The addition of NSP enzymes to bird's diet allows the breakdown of these anti-nutritional factors and thus faster and more complete digestion of the feed, leading to improved nutritive value. Feed enzymes are also able to upgrade sources of vegetable protein (such as soybeans, rapeseed, sunflower seed and legumes) in poultry diets.

Enzymes for reducing environmental pollution:

Apart from contributing to improving nutritive value, feed enzymes can also have a positive impact on the environment

by allowing better use of natural resources and reducing pollution by nutrients. In areas with intensive livestock production, the P output is often very high. This can lead to environmental problems such as eutrophication. This is the process by which a body of water becomes, either naturally or by pollution, rich in dissolved nutrients (such as phosphates), causing algae blooms and deficiencies in oxygen.

Most (50-80%) of the P contained in feedstuffs of plant origin exists as the storage form phytate, or phytic acid, and is indigestible for non-ruminant animals such as poultry. They cannot digest the P contained within these complex phytate structures, since they lack the enzyme to break down the phytate and free the P. Phytase enzyme is essential for the release of phytate-bound-P. Therefore, sufficient phytase needs to be added to the feed.

Phytate also forms complexes with proteins, digestive enzymes and minerals, and as such is considered to be an antinutritional factor. Phytase frees the P contained in cereals and oilseeds, and by breaking down the phytate structure also achieves the release of other minerals such as Ca and magnesium (Mg), as well as proteins and amino acids, which have become bound to the phytate. Thus, by releasing bound P in feed ingredients of vegetable origin, phytase makes more phosphorus available for bone growth, and reduces the amount excreted into the environment.

Probiotics:

A probiotic is a culture of one or more microorganisms, which benefit the host by stimulating the positive properties of its natural occurring microflora in the gut. The use of probiotics as a daily supplement has become a popular routine in the commercial poultry industry in various parts of world, particularly following antibiotic treatment. The common example is Lactobacillus acidophilus. Introducing probiotics into the digestive system everyday to ward off bacterial infection is also known as 'selective exclusion'. Selective exclusion is very good way to keep birds healthy and disease resistant throughout their life. There is constant competition in the gut between disease causing bacteria and desirable bacteria. During times of stress or the use of antibiotics, hormonal changes can occur, causing the pH of the small intestine to rise. This causes an imbalance between bad and good bacteria and allows existing bad bacteria to take a foothold in the lining of the intestine because of the deterioration of the protective mucus lining. Because of this, the villi can be lost. Unfortunately, E. coli, a disease causing bacteria, can double its population every 15 minutes during ideal conditions while the desirable Lactobacillus requires 20 minutes.

Prebiotics:

Prebiotics have been defined as "non-digested feed ingredients that beneficially affect the host by stimulating the growth and activity of one or a limited number of bacterial species already residing in the colon" (Gibson and Roberfroid, 1995). A prebiotic would ideally alter the microbial balance such that the potentially beneficial microorganisms become predominant. There are a wide variety of non-digestible carbohydrates (resistant starches, plant cell wall polysaccharides, pectins, gums, pentosans, etc.) that are capable of bypassing enzyme degradation in the upper GIT and reaching the large intestine where they undergo fermentation by the resident microorganisms.

The role of oligosaccharides appears to vary by type. It may serve as a selective nutrient source and/or as a competitive attachment site for pathogenic bacteria. The pathogenic bacteria preferentially attach to the mannose rich oligosaccharides rather than to the intestinal wall.

Antioxidants:

Antioxidants limit oxidation in byproducts such as poultry and fish meal, as well as preserve the vitamin quality and

pigmentation potential of commercial feeds. All fats do not show the same propensity for oxidative attack. Oxidation occurs most readily in fats with multiple points of unsaturation (double bonds) in their fatty acid chains. Table 4 lists the most commonly used fats in animal feeds. For comparison, their fatty acid profile, expressed as the percentage of each fatty acid, is also shown.

To facilitate handling, it is quite common for oils to be hydrogenated during manufacture. This process reduces the degree of unsaturation (double bonds) contained in these oils, and theoretically, reduces their potential for oxidation. Unfortunately, the hydrogenation process is quite harsh, and may actually reduce the feeding value of these oils when they are subsequently incorporated into animal byproduct meals. Also of growing concern is the fact that the hydrogenation process greatly increases the concentration of *trans*-fatty acids in these products. *Trans* fatty acids have a negative effect on both animal health and immune status, and have different absorptive efficiencies than their "*cis*" counterparts.

| Source | C _{8:0} | C _{10:0} | C _{12:0} | C _{14:0} | C _{16:0} | C _{16:1} | C _{18:0} | C _{18:1} | C _{18:2} | C _{18:3} |
|--------------------|------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| Animal/poultry fat | - | - | 0.1 | 1.6 | 19.9 | 2.8 | 13.7 | 46.4 | 11.8 | 1 |
| Animal tallow | - | - | 0.9 | 3.7 | 24.9 | 4.2 | 18.9 | 36 | 3.1 | 0.6 |
| Animal-vegetable | 1 | 0.9 | 6.6 | 3.1 | 16.5 | 2.8 | 11.9 | 30.2 | 20.6 | 2.6 |
| Canola oil | - | - | - | - | 4.8 | 0.5 | 1.6 | 53.8 | 22.1 | 11.1 |
| Coconut oil | 7.5 | 6 | 44.6 | 16.8 | 8.2 | - | 2.8 | 5.8 | 1.8 | - |
| Corn oil | - | - | - | - | 10.9 | - | 1.8 | 24.2 | 58 | 0.7 |
| Cottonseed oil | - | - | - | 0.8 | 22.7 | 0.8 | 2.3 | 17 | 51.5 | 0.2 |
| Palm oil | - | - | 0.1 | 1 | 43.5 | 0.3 | 4.3 | 36.6 | 9.1 | 0.2 |
| Peanut oil | - | - | - | 0.1 | 9.5 | 0.1 | 2.2 | 44.8 | 32 | - |
| Poultry fat | - | - | 0.1 | 0.9 | 21.6 | 5.7 | 6 | 37.3 | 19.5 | 1 |
| Safflower oil | - | - | - | 0.1 | 6.2 | 0.4 | 2.2 | 11.7 | 74.1 | 0.4 |
| Soybean oil | - | - | - | 0.1 | 10.3 | 0.2 | 3.8 | 22.8 | 51 | 6.8 |
| Sunflower oil | - | - | - | - | 5.9 | - | 4.5 | 19.5 | 65.7 | - |

 Table 4. Fatty acid composition (%) of selected fats and oils

<u>**1**</u>. Number of double bonds in fatty acid chain (NRC, 1984)

The handling of feed grade fat can significantly alter its subsequent feeding value. The addition of liquid fat to storage tanks and transport vehicles can cause aeration if "free fall" distance is too great. Consequently, holding tanks and tank trucks should be bottom loaded when possible. If impractical, care should be taken to limit aeration by adding the material slowly, and as close to the bottom of the tank as possible. Once applied to a feed, the liquid fats surface exposure to oxygen is greatly increased and potential for oxidation enhanced.

Formulations may contain mineral chelators such as citric acid or EDTA that limit oxidation catalyzed by minerals. Sodium bicarbonate may be added as a buffer, and carriers such as hydrated alumino silicate may be used. Synergistic combinations of chelators and antioxidants are claimed to contribute to greater stability of ingredients and finished feed than using a single antioxidant alone. Most products are recommended at an addition rate of 125g/T of feed although blended products often provide much less than 125g of actual antioxidant.

Combinations of antioxidants are usually more efficient in maintaining the potency of vitamins A and E and suppressing the formation of free fatty acids than an equivalent quantity of ethoxyquin. Antioxidant supplementation suppresses the thiobarbituric acid (TBA) value in poultry feed compared to controls at 4 and 6 weeks after manufacture. The TBA value is a measure of antioxidation as it quantifies the evolution of aldehydes, which are byproducts of free radical formation.

Synthetic antioxidants are generally more effective, and far more economical than natural antioxidants in preventing feed oxidation. Synthetic antioxidants are also superior to natural products in that they are generally more stable, and show better survivability, when added during the rendering process, and through feed processing and pelleting. Vitamin E is the vitamin most typically added for this purpose, generally as α -tocopherol, or in the form of mixed tocopherols.

Chemistry of oxidative rancidity:

Oxidative decomposition is a chain reaction, which, once begun, proceeds at ever increasing speed. Rancidity of fat components is most occurring results of oxidation but raw materials of animal origin such as fish meal and meat meal may also be damaged, as may fat-soluble vitamins and carotenoids. Poultry feed, spoiled by oxidation develops a characteristic, pungent smell, which is caused by the products of fat decomposition.

Long-chain polyunsaturated fatty acids (PUFA) are extremely susceptible to peroxidation. Animal tallow and poultry fat have a high proportion of C_{18} fatty acids incorporating 1-3 double bonds. In addition, some vegetable oils, including canola, cottonseed, peanut and safflower, contain $C_{18:2}$ fatty acids in proportions ranging from 20-70%.

Oxidative degradation of fatty acids is initiated at the carbon-hydrogen bond located adjacent to a double bond. The higher the level of saturation, the more susceptible fatty acid is to oxidative rancidity. The process occurs in three phases. In the initiation phase, the bond undergoes an abstraction of hydrogen (effectively oxidation) to produce a labile hydrogen atom and a free radical. In the subsequent propagation phase, the free radical, formed as a result of initiation, combines with an oxygen molecule producing peroxide. Hydrogenation of the peroxide forms a hydro-peroxide with evolution of a second free radical. This process is autocatalyzed and the intensity of propagation increases as a cascade of free radicals is produced by the oxidation of PUFA. The termination phase occurs when free radicals combine to form polymerized fatty acids. Introducing an antioxidant into a system undergoing oxidative rancidity will terminate the reaction since free radicals are neutralized by the antioxidant molecule (Table 5). Table 6 depicts the reaction of nitrogen or phenolics containing antioxidants with free radical species.

| Table 5. Three | phases of the oxidation reaction | | | |
|----------------|----------------------------------|----------|------------------|--|
| Phase | Reaction ¹ | | | |
| Initiation | RH | Catalyst | R● + H● | |
| Propagation | $R \bullet + O_2$ | Catalyst | $RO_2 \bullet$ | |
| | $RO_2 \bullet + RH$ | Catalyst | R● + ROOH | |
| Termination | $R \bullet + R \bullet$ | Catalyst | R:R | |
| | A-H + R● | Catalyst | $RH + A \bullet$ | |

Table 5. Three phases of the oxidation reaction

1. RH = a fatty acid molecule; H = a labile hydrogen atom; O_2 = molecular oxygen; • = the unpaired electron; R• & RO₂• = free radicals; R:R = 2 fatty acids which has been cross linked (polymerization); ROOH = an organic peroxide; A-H = antioxidant; Catalyst = Cu, Fe and other free radicals

Table 6. Termination of the oxidation reaction with antioxidants

| R● + H-N< | ► R-H + •N< |
|-------------------------------|--|
| R● + H-O- | ► R-H + •O- |
| Note Re - free redicals: H N. | z = nitrogan containing antioxidant: U O = |

<u>Note</u>. $R \bullet =$ free radicals; H-N< = nitrogen containing antioxidant; H-O- = phenolic containing antioxidant

Free radicals vary in their stability; some exist only for nanoseconds. Others with large cyclic structures and conjugated double bonds are relatively stable and their biological activity is responsible for damage to cell systems. Free radicals damage cell membranes, especially the endothelium (lining cells) of blood vessels. Free radicals also act as initiators and promoters of mutagenesis, resulting in neoplasia. It is now widely accepted that oxidation *in vivo* is involved in the process of aging.

The presence of highly reactive free radicals in feed will also result in degradation of nutrients such as the vitamins A and D, biotin and the tocopherols (vitamin E), which act as intracellular and biological antioxidants. Despite acceptable rates of incorporation of these nutrients into feed, oxidative rancidity can result in deficiency. The initiation of free radical formation is stimulated by high ambient temperature and the presence of metallic catalysts. Addition of copper sulfate to mineral premixes, or the presence of iron (Fe) from blood in animal byproducts and from cooking vessels and conveyors, can stimulate the onset of lipid peroxidation with deleterious results. Synthetic and biological antioxidants function by donating electrons to neutralize free radicals. Although antioxidants effectively become free radicals in the process, they are extremely stable because of their cyclic structure and the propagation phase of rancidative oxidation is effectively suppressed. Autoxidation can be prevented by chelation of metallic ions, which are responsible for catalyzing the initiation phase of autoxidation or scavenging the free radicals by synthetic antioxidants, which serve as electron donors.

Fat-soluble vitamin (A, D, E and K) destruction may also lead to a number of disease/deficiency syndromes, as these vitamins are essential to animal nutrition and health. Table 7 represents a partial list of the various functions of the fat-soluble vitamins.

| Vitamin | Impaired function |
|---------|--|
| А | Vision, immune response, feathering, hatchability |
| D | Bone and joint formation, shell thickness, hatchability |
| E | Immune response, nervous and circulatory function, embryonic development |
| K | Production of blood coagulation factors, blood clotting |

 Table 7: Partial function of the fat-soluble vitamins in poultry nutrition

The impact of oxidative rancidity under practical conditions is exacerbated by a number of factors common to tropical countries. Mycotoxicosis is responsible for uncoupling of oxidative phosphorylation, resulting in depressed growth rate and an interference with immune function. Immunosuppressive viruses, such as Marek's disease, infectious bursal disease, chick anemia agent and, in the case of turkeys, adenoviral hemorrhagic enteritis, will interact synergistically with suboptimal nutrient quality to accentuate mortality and depress growth rate and reproductive efficiency.

Recent research has suggested that endogenous free radical reactions increase the likelihood of tumor formation, by serving as a readily available source of tumor initiators and promoters. This hypothesis is supported by experiments involving the induction of malignancy by dietary fat, increasing age and its prevention with mega-doses of antioxidant vitamins. Molecular oxygen readily reacts with polyunsaturated compounds in blood serum and the lipid fraction of the arterial wall. The resulting free radicals, peroxides and secondary/tertiary metabolites serve as an available pool of compounds having the capability of damaging membrane integrity, leading to atherogenesis (plaque formation).

Prevention of oxidative rancidity:

A number of practical measures can be implemented to reduce the impact of autoxidation on performance. The use of non-stabilized tallow and blended oils sources must be limited in manufacturing feeds or stabilize them at point of manufacture. Vitamin and mineral supplements and premixes should be prepared separately with using antioxidants as a stabilizing agent Ingredients must be stored in well-ventilated insulated areas especially high fat containing ingredients to inhibit bacterial degradation, which results in evolution of heat. Effective synthetic antioxidants should be incorporated in feed to achieve protection from oxidative rancidity.

Biological antioxidant systems:

Biological antioxidant systems help prevent oxidative damage by metabolizing organic and superoxide free radicals. Superoxide dismutase, a metal-containing enzyme found in aerobic cells, controls the superoxide free radical. Catalase, peroxidase and glutathione peroxidase are then able to break down hydrogen peroxide and other peroxides to water and molecular oxygen (Table 8).

| I. 2O ₂ ` + 2H2 | Superoxide dismutase | • | $H_2O_2 + O_2$ |
|------------------------------------|----------------------|---|----------------|
| II. 2H ₂ O ₂ | Catalase | • | $2H_2O + O_2$ |

How Antioxidants work?

To understand how antioxidants work, it's important to start with the molecules they neutralize: free radicals and singlet oxygen. Free radicals are oxygen-derived molecules that contain one or more unpaired electrons and react with other molecules, acquiring or giving up an electron to achieve stability. Singlet oxygen contains two electrons, but it is highly reactive and also capable of inducing the formation of free radicals.

The body produces these destructive molecules as normal byproducts of oxygen metabolism. In addition, immune cells generate free radicals with which they kill antigens. This is usually a beneficial action, but any abnormal activation of the immune cells can lead to the constant generation of free radicals that will damage the body's own cells. There are also environmental sources of free radicals, including ultraviolet light, ozone and various carcinogens.

Virtually all major constituents of a cell are at risk of oxidative damage, but the principal targets are DNA, ribose nucleic acid (RNA) and the membranes that make up about 90% of each cell. Besides causing direct damage, each free radical can also induce chain reactions that form new free radicals and lead to further damage. These chain reactions are perhaps best shown with the peroxidation of the PUFA present in cell membranes. As a free radical reacts with a PUFA molecule, a hydrogen atom is first abstracted from the PUFA and then an oxygen molecule is inserted. This turns the PUFA into a peroxy radical, which will then abstract a hydrogen atom from a previously non-radical PUFA. Once the process begins, the chain reaction continues.

Ultimately, this peroxidation will affect vital membrane functions. For example, Bendich (1993) noted that lipid peroxidation reduces the fluidity of cell membranes, and that loss of fluidity has been directly related to the decreased ability of lymphocytes to respond to immune challenges. To counteract these assaults, each of the antioxidants works in specialized ways. Vitamin E, e.g., is the primary fat-soluble antioxidant in the body and tends to concentrate within the membranes both inside and surrounding the cell.

Besides neutralizing free radicals and singlet oxygen, vitamin E also interrupts the chain reaction that leads to the conversion of membrane PUFA into peroxy radicals (Table 9). The vitamin E itself becomes deactivated (oxidized) during this reaction, but vitamin C or glutathione will reduce it back to an active molecule with renewed antioxidant properties.

| Tuble 71 Hitto | sidant functions of vitamin E, vitamin C and p-carotene |
|----------------|---|
| Vitamin E | 1. Chain reaction breaking antioxidant |
| | 2. Free radical scavenger |
| | 3. Singlet oxygen quencher |
| | 4. Efficient at high oxygen pressure |
| β-Carotene | 1. Chain reaction breaking antioxidant |
| | 2. Free radical scavenger |
| | 3. Singlet oxygen quencher |
| | 4. Effective at low oxygen pressure |
| Vitamin C | 1. Free radical scavenger |
| | 2. Singlet oxygen quencher |
| | 3. Regenerator of vitamin E |

Table 9. Antioxidant functions of vitamin E, vitamin C and β -carotene

Vitamin C, which is water-soluble, is itself one of the most important antioxidants in extracellular fluids. It also scavenges free radicals and quenches singlet oxygen in the water compartment of the cells.

The β -carotene is the most effective naturally occurring quencher of singlet oxygen. Because of the chemical structure of β -carotene, the energy in singlet oxygen can be dissipated throughout the carotenoid molecule and then released as a small amount of heat. It has been estimated that one molecule of β -carotene can quench up to 1,000 molecules of singlet oxygen.

Although β -carotene and vitamin E are both fat-soluble antioxidants, they appear to be complementary rather than redundant. For example, the reaction with β -carotene takes place in tissues with low partial pressure of oxygen, while vitamin E appears most active when oxygen pressure is high. Such differentiation under-scores the importance of having a full complement of the right antioxidants to protect against oxidative damage. Another comparison that emphasizes this point would be between vitamin E and ethoxyquin, an antioxidant that is highly effective in preventing rancidity in poultry feeds.

Stabilizing animal byproducts:

To ensure the nutrient quality of byproduct meal and fat, antioxidants should be added at appropriate points and at efficacious levels in the rendering facility. Since oxidation is a rapid reaction, the selected antioxidant should be added as early in the rendering process as is physically practical. Generally, this can quite easily be accomplished in the raw offal stream as the material enters the rendering plant cookers.

Fat-soluble antioxidants like ethoxyquin will be proportionately distributed in the liquid fat and fat portion of byproduct meal after pressing. Normally, it is convenient to add a sufficient level of an antioxidant like ethoxyquin to the raw offal (approximately 115g/T) to adequately stabilize the liquid fat segment of the plants output.

Since the majority of the antioxidants will be incorporated with the liquid fat (400-500ppm), the byproduct meal should be top-dressed with additional product either before or after the material enters the hammer mill for grinding (340-450g/T). Figure 1 depicts a typical rendering flow-chart, with the suggested points of antioxidant application identified.

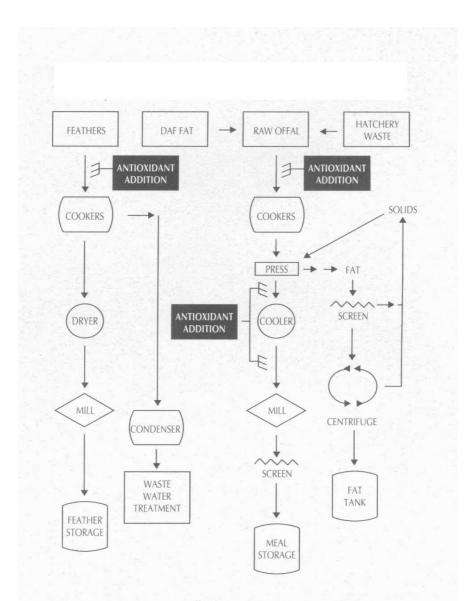


Figure 1. Flow chart of a rendering plant and proposed sites for the addition of antioxidants

Chapter 20

Feed Ingredients

Introduction

The key for successful poultry production is dependant on the provision of balanced and economical ration. Good quality feed ingredients are prerequisites for balance diets for poultry. As feed cost accounts 77% of the total poultry production. Formula feed for different classes of poultry is orchristrated by the combination of diverse type of feeding ingredients. So the selection of feed ingredients for poultry rations depends upon the quality and cost effectiveness. The basis of feed formulation for different classes of poultry / game birds at cellular level is amino acids and metabolizable energy. There are three main classes of commonly used feed ingredients in poultry rations.

- ➢ Cereal grains
- Protein supplements
- Mineral and vitamin supplements
- Synthetic amino acids and feed additives

Cereal Grains:

Commonly used cereal grains and their by-products used in poultry diets are;

Corn

Sorghum

Barley

Rice Tips

Rice Polish

Molasses

Corn:

Corn is high in energy content but low in fiber. Crude protein in corn varies from 9 to 13%. It contains 4280 Kcal /kg gross energy Kcal and 3100 / Kg metabolizable energy. Two types of maize are usually available i.e. white and vellow maize. The main



difference lies in the amount of yellow, orange pigments in the pericarp but both have an extremely high content of starch with very little indigestible organic matter. The oil content is relatively high, thus maize is one of the richest sources of energy. Corn though an excellent source of digestible energy, it is low in protein and deficient in tryptophan. Yellow maize is the only grain with 0.5 ppm carotene and around 5 ppm xanthophylls imparting the high degree of pigment in egg yolks and body fat of chickens and is extremely low in calcium and fiber, deficient in vitamin B12. maize kernel contains two main types of proteins i.e. Zein occurring in the endosperm, in quantitatively the most important but this protein is deficient in the indispensable amino acids, lysine and tryptophan and second is maize gluten, occurring in lesser amounts in the endosperm and also in the germ, is a better source of tryptophan and lysine.

Sorghum:

There are different varieties of sorghum which are known by different names e.g., Kaffir, Milo, Darso, Feterita etc, of these, the white varieties are preferable for poultry since



they contain little of bitter principle (tannin) found richer in the darker grains. Sorghum is similar to maize grain in its composition. The kernel of sorghum is very similar to that of maize, although smaller in size. Whole grain can be fed to poultry but cracked or ground grain give better feed efficiency. Crude protein varies from 9 to 14%. Metabolizable energy is 3000 Kcal /Kg. it is low in calcium and moderately high in phosphorus. Yellow sorghum contains carotenoid pigments; thus have some vitamin A activity.

Barley:

It is palatable but fibrous feed. Barley is medium energy protein ingredient. Its protein content varies from 11 to 12%. The metabolizable energy level is highly correlated to the bulk density and negative correlated to the fiber.



Rice Tips:

Rice is very commonly used ingredient of poultry diet. It can be used in diet up to 20%.

Its protein content is about 9%. Energy is 3000 Kcal /Kg. fiber content is also low. Calcium content varies from 0.04 to 0.06% and phosphorus content is 0.26 to 0.48%. Rice contains



quite high level of trypsin inhibitor that will be destroyed by normal plating temperatures. Brown rice is high in starch, low in fat in fat and indigestible organic matter and when obtainable is an excellent poultry feed.

Rice Polish:

Rice polish is the by-product of rice dehulling and cleaning of rice. It is high in fat content. Crude protein content is about 11%. Due to high fat it is content verv susceptible to oxidative rancidity. Its metabolizable energy varies from 2800 to



3000 Kcal /Kg. Phosphorus content in rice polish ranges from 1.32 - 1.48%.

Molasses:

It is the byproduct of sugar refining industry, whatever the raw material should be; sugar cane or sugar beet. It contains approximately 50 % sugar. It is liquid feed ingredient. Usually its inclusion level in feed is not more than 4%. It



reduces the dustiness of feed. Its CP is about 4% and energy is 2200 Kcal /Kg. The potassium content is high which leads to problems with wet dropping if excessive levels are used. It has the binding effect in the pellet formation or protein blocks for animals.

Protein Supplements:

There are two main classes of protein supplements

- 1. Vegetable origin (Soybean meal, Canola meal, Sunflower meal, Cotton Seed meal corn gluten meal)
- 2. Animal origin (Fish meal, blood meal, Feather meal, meat meal)

VEGETABLE ORIGIN:

Soybean meal:

This is derived from the seed of the soybean plant. Raw soybeans contain sovin. a toxic protein which acts s a depressant to the appetite and a trypsin inhibitor, both of which can be destroyed heat. The trypsin bv inhibitor reduces the availability of the



methionine and cystin and modern methods of manufacture include heat treatment. The protein is probably the best balanced of any vegetable protein, with methionine as the limiting amino acids. Soybean meal is a good source of choline and glycine and is fairly rich in riboflavin. Properly processed soybean meal is an excellent ingredient for poultry. All commercial soybean meal undergoes a toasting process. During the toasting process antinutritional factors (trypsin inhibitors, antigenic proteins, lectins and lipoxygenase) present in the soybean meal are destroyed or reduce to tolerable levels. Poorly processed soybean meal reduces nutrients availability to the birds. If active, protease to the digestive enzymes trypsin inhibitors bind and chymotrypsin, rendering them inactive, residual anti-nutritional factors reduce growth and feed efficiency. Over toasting is also detrimental, valuable protein is denatured making it less soluble and digestible. The high levels of lysine contained in soy proteins are extremely sensitive to over toasting. Thus soybean meal must be processed within a narrow range of time and temperature to ensure high quality. Essential amino acids are properly processed soybean meal will be over 90% digestible. Variation in total amino acid content and other nutrient in soybean meal is typically lower than observe in fish meal.

Canola meal:

Canola meal has low glucosinolate and erucic acid. It is new variety of rapeseed (CP 33%). Canola was created from specially selected rapeseed by Canadian Plant Scientists in 1970s. The old rapeseed was high in glucosinolate compounds.



When this was fed to the animals at high levels, created palatability problems and lowered performance due to goitrogenic action, canola changed this. The new canola meal is more yellow in color than the dark brown color of rapeseed meal. Canola is low in glucosinolate in the meal and low in erucic acid (a long chain fatty acid) in the oil. Its amino acid composition is comparable with soybean meal and has 33-34 % crude protein. When the price is favorable, canola meal may be used as a protein supplement for all classes of livestock and poultry. It may be used at maximum levels of 20 % in most rations, but the amount in total ration should be limited to about 10 % for layer. Both meals (canola and rapeseed) are high in sulfur that may cause leg abnormalities in poultry.

Sunflower meal:

It is the product produced from what remains following the extraction of oil from dehulled sunflower seed. The pericarp of hard the sunflower seed accounts for about 40 % of the weight, hence decorticated sunflower meal is a much



better feed than un-decorticated meal. It is good source of nicotinic acid but the protein is deficient in lysine. Expeller sunflower meal has a higher energy level than solvent extracted meal because of higher residual oil content. The crude fiber of partially de-hulled or non de-hulled sunflower meal exceeds 20 % and thus is a major limiting factor for used in poultry feed. The high variability in quality of sunflower meal due to hulls is the most important limiting factor with the use of this ingredient. Processing temperature is also important. Low temperature processing is desirable to prevent denaturation of lysine and other valuable amino acids. Sunflower meal contains high levels of chlorogenic acid, a tannin like compound that inhibits activity of digestive enzymes including trpsin, chymoptrypsin, amylase and lipase. Additional supplementation with methionine, lysine and choline are required to counteract the effect of chlorogenic acid. Unlike soybean meal, a sunflower meal is high in methionine but low in lysine and threonine. Therefore, these two ingredients, when used together, improve the amino acid balance of the feed. In broiler diets, it is recommended that only high quality de-hulled sunflower meal be sued in poultry rations.

Cotton seed meal:

The meal results from the kernels being removed from the leathery hulls, crushed, and much of the oil possible is removed by one of the extraction process. For poultry feeds, cotton seed meal is prepared from the de-hulled seeds of the cotton plant.



The fresh kernels contain two toxic principles, gossypol and gossypurpurin. These are destroyed by steam cooking the seeds before the oil is removed. When unheated meal is fed in amounts too low to produce the toxic signs, yolk color is adversely affected (olive yolk) as is keeping quality of the egg. Addition of iron salts in the feed can help alleviate the harmful effects of gossypol. Cottonseed also contains the cyclopropenoid fatty acids, malvalic and sterculic acid. These cause pink discoloration of the egg albumin when fed to layers and are also known to alter liver metabolism and possibly increase aflatoxin damage. Cottonseed meal is deficient in methionine, threonine, and tryptophan and has low digestibility of lysine for poultry. It is used up to 8 % in poultry rations.

Corn gluten meal:

It is product of corn milling industry having very high crude protein content (57-60 %) and is high in metabolizable energy content 2.5 MJ/kg DM. it is commonly used in poultry rations, which is obtained in the wet milling



of corn to produce starch and syrup or by enzymatic treatment of the endosperm. It contains low amounts of lysine and tryptophan. It is rich in carotene and in the yellow pigment xanthophylls.

ANIMAL ORIGIN:

Fish meal:

Fish meal is produced by cooking, drying and grinding fish. Fish meal is a high quality protein feed, rich in all essential amino acids, vitamin B₁₂ and choline. It is a good rather than a rich source of calcium and phosphorus.



Adverse effects on hatchability have been reported when fish meal has comprised more than 10 % of the diet. These were only found with some fish meals and appear to have been due to overheating during manufacturing, with the production of toxic products which were transmitted by hen to the egg. These toxins increased the stress on the developing embryo and resulted in mortality, late in the incubation period. Some fish meals, when incorporated into the diet, have caused gizzard erosion in young birds. Again, faulty manufacturing may be the real cause. Product whose chloride content expressed as sodium chloride is less than 2% may be referred to as low in salt.

Protein may vary from 40-62 % and fat from 2-12 % or more in non extracted fish. Salt content may vary from 1-4 %. The fatty Acid composition of fish meal varies depending on the variety of fish used. The unsaturated oils in fish meal are highly susceptible to oxidation that creates toxic free radicals and lowers energy content. Oxidation may also cause heating during storage that reduces amino acid digestibility and sometimes cause spontaneous combustion to occur. Fish meal is also prone to contamination with biogenic amines. Substances such as gizzerosine and histamine are formed during heat processing of fish that have been allowed to spoil or putrefy. These substances increase stomach acid secretion and have been documented to cause gizzard erosion and other lesions in poultry. Fish meal should be avoided in diets in the period before slaughter to avoid fishy taint in the meat caused by amines that re abundant in fish meal. More than 7-8 % fish meal in layer diets may result in a fishy taint in eggs. Depending on quality and composition, fish meal should be limited to between 4-8 % of the diet of growing broilers Limitations are necessary to prevent over supplementation with minerals.

Blood meal:

Blood meal is a dark chocolate colored powder with а characteristic smell. It is the product obtained when whole blood is cooked. dried and ground to a meal. It is an extremely rich protein supplement but the quality of the protein



varies with the degree of heating during the drying process. Blood meal is one of the richest sources of lysine. It is a rich source of arginine, methionine, cystine and leucine but low in isoleucine and contains only a trace of glycine. Owing to the proper balance of amino acids its biological value is low and in addition it has a low digestibility. It has the advantage in certain situation, that its protein has a low degradability and is unpalatable. The use of blood meal results in reduced growth rate in poultry and it is not recommended for young stock. The normal rate of inclusion for older birds is 1-3 % which did not affect the growth rate.

Feather meal:

Following the broiler growth of industry, large quantities of feathers have to be disposed off and the production of feather meal by steam processing method is now in operation. Feather meal is a rich source of fat and



protein, which is high in luccine and cystine but deficient in lysine, tryptophan and methionine.

Meat meal:

Meat unfit for human consumption whether because of disease or parasitic infestation, cooked, dried and ground is called meat meal, and when sold as an animal food must contain not less than 55 % crude protein and not more than



4 % salt. It is a good source of lysine, methionine, cystine, leucine, tryptophan and vitamins like riboflavin, choline and vitamin B_{12} .

Meat meal is obtained from the rendering and drying of waste products from abattoirs (slaughter house) and meat processors. Meat meals should be considered highly variable in protein, fat, calcium, phosphorus and sodium. Meat meals are, however, prone to being overcooked which reduces availability of amino acids. They may also contain biogenic amines if spoilage is allowed to occur before processing. Rancidity is another problem as residual fat is highly susceptible to oxidation because of a high surface area of the meal particles. Meat meal prepared locally (Pakistan) is poorly processed, adulterated, thus may act as a source of contamination for the chicken.

Due to risk of bacterial infections and zoonotic viral diseases like mad cow disease, the use of blood meal, meat meal is almost diminished in poultry diets in advanced countries. However, fish meal is considerably used as a protein source due to better profile of amino acids in Pakistan and other countries. However, due to high salt and urea impurities its use is getting minimal especially in Pakistan.

Feedstuff or Feed Ingredient:

Any product natural or artificial origin that has nutritional value in the diet when properly prepared. Feed ingredients are constituents of ration.

Feed (or Food):

Edible materials which are consumed by bird s a part of daily ration contributing energy and/or nutrients to the bird's diet. Feed is commonly refers to birds and animals rather than man. It is synonymous with nutriment.

Forms of Feed:

1. Mash:

The ration and feed in mixture all of the ingredients are ground or reduced in particle size.

2. Pellets:

Agglomerated feed formed by compacting and forcing through die openings by a mechanical process.

3. Crumbles;

Pelleted feeds reduced in irregular size are called as crumbles.

Balanced ration:

Such a combination of feed stuffs which will provide the essential nutrients in proper amounts and proportions to the bird.

Basal feed:

A feed common to all groups of experimental birds to which the experimental substance is added.

| | Composition (%) | | | | | | | | |
|-------------|-----------------|------|------|------|------|------|------|------|------|
| Corn | 9 | 3400 | 2.49 | 0.26 | 0.81 | 0.36 | 0.08 | 0.02 | 1.55 |
| Wheat | 12 | 3100 | 2.56 | 0.37 | 0.21 | 0.51 | 0.13 | 0.05 | 1.92 |
| Rice | 8.5 | 3000 | 1.11 | 0.22 | 0.22 | 0.43 | 0.03 | 0.08 | 1.27 |
| broken | | | | | | | | | |
| Sorghum | 10 | 3200 | 2.28 | 0.22 | 0.15 | 0.26 | | 0.04 | 2.48 |
| Rice polish | 11 | 3000 | 12.7 | 0.57 | 0.22 | 0.32 | 0.14 | 0.05 | 10.2 |
| Wheat | 15 | 1300 | 12.8 | 0.61 | 0.23 | 0.55 | 0.2 | 0.14 | 4.77 |
| bran | | | | | | | | | |
| Cotton | 40 | 2400 | 11 | 1.59 | 0.55 | 1.14 | | 0.2 | 6.83 |
| seed meal | | | | | | | | | |
| Rape seed | 30 | 2200 | 8.7 | 1.62 | 0.59 | 1.34 | 0.28 | 0.71 | 11.2 |
| meal | | | | | | | | | |
| Canola | 33.5 | 1850 | 12.8 | 1.94 | 0.71 | 1.57 | 0.3 | 0.68 | 6.1 |
| meal | | | | | | | | | |
| Guar meal | 40 | 2200 | 11 | 1.4 | 0.51 | 1.05 | 0.4 | 0.17 | 5.06 |

Nutrient profile of feed ingredients:

| Sunflower | 32 | 1500 | 14.5 | 1 | 0.5 | 1 | 0.14 | 0.21 | 7.65 |
|------------|------|------|------|------|------|------|------|------|------|
| | 52 | 1300 | 14.3 | 1 | 0.5 | 1 | 0.14 | 0.21 | 7.05 |
| meal | 4.4 | 2200 | 7 | 0.01 | 1.00 | 1.04 | 0.24 | 1.00 | 5.6 |
| Sesame | 44 | 2300 | 7 | 0.91 | 1.22 | 1.94 | 0.34 | 1.99 | 5.6 |
| meal | | | | | | | | | |
| Corn | 27 | 2400 | 8.56 | 0.51 | 0.51 | 0.89 | 0.14 | 0.18 | 7.74 |
| gluten 30% | | | | | | | | | |
| Corn | 55 | 3600 | 1.01 | 0.95 | 1.7 | 2.74 | 0.13 | 0.02 | 1.39 |
| gluten 60% | | | | | | | | | |
| Soybean | 44 | 2300 | 8.5 | 2.69 | 0.62 | 1.28 | 0.27 | 0.29 | 7.14 |
| meal | | | | | | | | | |
| Fish meal | 45 | 2900 | 0.7 | 3.24 | 1.17 | 1.57 | 2 | 5 | 11 |
| Feather | 45 | 2900 | 2.7 | 2.33 | 0.74 | 1.47 | | 3 | 9 |
| meal | | | | | | | | | |
| Blood | 70 | 2800 | 0.6 | 6.06 | 0.47 | 0.92 | 0.18 | 0.55 | 4 |
| meal | | | | | | | | | |
| Bone meal | | | | | | | 15 | 21 | |
| DCP | | | | | | | 18 | 23 | |
| Limestone | | | | | | | | 33 | |
| (ground) | | | | | | | | | |
| Soybean | | 8800 | | | | | | | |
| oil | | | | | | | | | |
| Molasses | 4 | 2200 | | | | | 0.04 | 0.8 | |
| Lysine | 96.5 | 3990 | | 78.4 | | | | | |
| Methionine | 58.7 | 5020 | | | | | | | |
| Premix | | | | | | | | | |
| (vitamin, | | | | | | | | | |
| Mineral) | | | | | | | | | |

Chapter21

Glossary

Acid:

A compound yielding a hydrogen ion in a polar solvent, such as water; any chemical compound that has a sour taste.

Acid-base balance:

The Dynamic state of equilibrium with regard to hydrogen ion concentration in the body.

Aciduria:

Presence of acid in the urine.

Acquired indispensable amino acids:

May become indispensable in immaturity, in states of metabolic disorder, and/or during severe stress. It includes cysteine, tyrosine, arginine, citrulline and taurine.

Active immunity:

Immunity acquired when an individual produces immune products, e.g., antibodies in response to an antigenic stimulus; OR, immunity produced in the bird either by natural exposure or by vaccination.

Active site:

That part of the enzyme surface on which the reaction takes place.

Active transport:

The movement of substances (particularly electrolyte ions) across cell membranes, usually against a concentration gradient. Unlike diffusion or osmosis, active transport requires the expenditure of metabolic energy.

Additives:

An ingredient or combination of ingredients added to the basic feed mix to fulfill a specific need. Usually used in micro quantities and requires careful handling and mixing. Ingredients are antibiotics, anti-oxidants, coloring agents, binders, flavorings and veterinary prescriptions etc.

Adenyl-pyrophosphatase:

An energy-releasing enzyme, which acts upon adenosine triphosphate (adenyl-pyrophosphate).

Adipose:

Related to animal fat; the fat found in adipose tissue.

Adrenal glands:

Glands located at the upper end of each kidney and are responsible for the secretion of many hormones including glucocorticoids, mineralocorticoids, adreneline and more. The adrenal cortex produces estrogen, androgen, progesterone, aldosterone and cortisone; the adrenal medulla produces epinephrine and norepinephrine.

Alkalosis:

Excessive accumulation of base, or the loss of hydrogen ions or acid from the body.

Allergen:

A substance that is capable of producing an allergic response in the body.

Allergy:

A hypersensitive state caused by the interaction of an allergen with an antibody.

Anti-coccidial:

Drug used to control coccidial infections. Coccidia are microscopic protozoa that live in the intestinal lining of livestock and poultry, causing severe diarrhea or unthriftiness.

Anti-toxin:

A specific antibody capable of neutralizing a specific toxin.

Anti-vitamin:

A substance that interferes with the synthesis or metabolism of vitamins.

Apo-enzyme:

An enzyme before attachment of its coenzyme or prosthetic group.

Apparent metabolizable energy (AME):

It refers to the traditionally determined energy values, which include metabolic and endogenous fractions.

Arachidonic acid:

An unsaturated 20-carbon fatty acid that is a precursor of prostaglandin synthesis.

Atrophy:

Wasting away or decreasing in size of cells, organs or entire body; due to disuse, disease or severe malnutrition.

Auto-immunity:

A situation in which the body produces an immune response against its own organs or tissues.

Avian:

Pertaining to aves or birds.

Avidin:

A protein in egg albumin, which can combine with biotin to render the later unavailable to the animal.

Avitaminosis:

A disease or malfunction caused by a vitamin deficiency.

Bacteria:

A group of very small, microscopic, unicellular microorganisms to which belong to a large number of disease producing germs. DNA (via methylation) from damage.

Bicarbonate:

An alkaline secretion of the pancreas that helps to neutralize the acidic chyme.

Bile:

A bitter, alkaline brownish-yellow or greenish-yellow liquid that is secreted by the liver, stored in the gallbladder, and discharged into the duodenum and that aids in digestion, chiefly by saponifying fats.

Bioavailability:

The degree to which a drug or other substance becomes available to the target tissue.

Biochemistry:

The chemistry of living things (plant and animal life).

Bioflavonoid:

Naturally occurring flavone or coumarin derivatives having the activity of the so-called vitamin P. It is found in brightly colored fruits and vegetables.

Biological value (*BV*):

The efficiency with which a protein furnished the proper proportions of the essential amino acids.

Biomass:

Living as well as dead material of plant or animal origin.

Biosynthesis:

The formation of chemical substances from other chemical substances in a living organism.

Biotechnology:

The use of current technologies such as DNA technologies for the modification and improvement of biological systems.

Blood meal:

Produced from clean, fresh animal blood and exclusive of all hair, stomach products and urine.

Blood-brain barrier:

A barrier composed of the cells lining the blood vessels in the brain, which are highly selective in what they allow to pass into the brain.

Bomb calorimeter:

An instrument used for determining the gross energy content of a material.

Calcification:

The process in which calcium, phosphorus, and other minerals crystallize on the collagen matrix of a growing bone, to harden it.

Calcitonin:

A hormone that opposes the action of parathyroid hormone in regulating blood calcium levels and bone mineralization.

Calcitriol:

Metabolically active form of vitamin D produced by the kidney and functions as a hormone.

Calcium (*Ca*):

It is needed for general health and bone and shell formation. Limestone and shell grits supply sources of the mineral.

Calcium chelates:

These forms of calcium are chelated (bonded) to amino acids, proteins, or peptides. This allows the calcium to enter through the intestinal wall as a protein/ amino acid rather than a metal cation.

Calmodulin:

A protein that relays calcium's messages.

Calorie:

One calorie is the amount of energy required to raise the temperature of 1g of water through 1°C from 14.5°C to 15.5°C. It is a measure of energy and usually expressed as kilocalorie (kcal) or mega calorie (Mcal).

Carcinogen:

A cancer-causing agent.

Catabolism:

The breaking down in the body of complex chemical compounds into simpler ones often accompanied by the liberation of energy.

Celsius:

Same as Centigrade.

Centigrade:

A thermometer scale in which water freezes at 0° C and boils at 100° C.

Central nervous system (CNS):

Brain and spinal cord.

Cereal:

Edible grain.

Chaff (part):

Husk or other seed coverings together with plant parts separated from seeds in the cleaning process.

Chelate:

A complex formed between a metal ion and a polar molecule.

Chelated mineral:

A compound formed between an organic molecule and a mineral.

Cholesterol:

Fat soluble essential nutrient required by the body as a part of all cell membranes and helps in the synthesis of sex and adrenal hormones.

Choline:

A water-soluble vitamin closely associated with fat metabolism and transport.

Chondrodystrophy:

This is a disorder of the growth plates in broiler with an enlargement and malfunction of the tibial and metatarsal joint (hock joint) resulting in the slipping of the gastrocnemus tendon from its condyle. It is due to inadequate manganese or choline in the broiler diet.

Chondroitin sulfate:

Chondroitin sulfate is a glycosaminoglycan, or sometimes a mucopolysacharide, which is added to various proteins to make up proteoglycans. It is the major building blocks of cartilage, bone and aorta tissues and can be synthesized by glucuronic acid and N-acetylgalactosamine.

Chopping (process):

Particle size reduction by cutting with sharp-edged instruments. i.e., knives.

Chromium (Cr):

It is an essential trace-element involved with proper metabolism of carbohydrates and lipids.

Chylomicrons:

Droplets consisting of triglyceride, cholesterol, phospholipids and protein. It is the form by which absorbed long-chain triglycerides and cholesterol are transported from the intestine into the intestinal blood or lymphatic system.

Classification:

The whole of the classes set out in a show schedule.

Cleaned:

Undesired materials removed using screens, magnets or forced air.

Clinical malnutrition:

Changes in skin, hair, membranes or growth that can be attributed to an excessive or deficient intake of a nutrient or nutrients.

Coagulation:

The change from a fluid state to a thickened jelly, curd or clot.

Coarse product:

A mixture of products, which are not ground.

Coccidiostat:

See anti-coccidial.

Cock:

A cock or rooster (of either sex) is a mature chicken with coarse skin, toughened and darkened meat and hardened breastbone tip, above 1 year of age.

Cockerel:

A young male chicken from day of hatch to approximately 1 year of age.

Collagen:

A glycoprotein from which gelatin is made. It is the main supportive protein of connective tissue.

Comfort zone:

The thermoneutral zone or temperature range in which animals maintain their body temperature within the thermal range without expending any extra energy. Comfort zone for poultry is between 18-25C.

Commercial feed:

The term "commercial feed" means all materials, which are distributed for use as feed or for mixing in feed, for animals other than humans.

Complete feed:

By specific formula, it is compounded to be fed as the sole ration and is capable of maintaining life and/or promoting production without any additional substance being consumed except water.

Complete ration:

See complete feed.

Complex carbohydrates:

Polysaccharides (starch, glycogen and some fibers).

Cortisol:

The major adrenal cortical steroid influencing carbohydrate metabolism. It increases the release of glucose from the liver, stimulates gluconeogenesis from amino acids and decreases peripheral use of blood glucose.

Cottonseed hulls:

The outer covering of the cottonseed.

Cottonseed meal:

It is the product obtained by finely grinding the cake, which remains after removal of most of the oil from cottonseed by a mechanical or solvent extraction process. It must not contain less than 36% crude protein.

Cracking (process):

Particle size reduction by a combined breaking and crushing action.

Creatinine:

A nitrogenous compound arising from protein metabolism and secreted in the urine.

Crop:

An enlargement of the gullet where food is stored and prepared for digestion.

Crude fat:

The part of a feed, which is soluble in ether.

Crumbling (process):

A process of reduction of pellets to granular form.

Cryptoxanthin:

One of the carotenoid pigments that may be converted into vitamin A.

Cyanocobalamine:

It refers to a group of cobalamine compounds, of which the most frequently used is cyanocobalamine and is required as a cofactor for various enzymes. It transfers a methyl group from methylfolate, helping to convert homocysteine to methionine.

Cyanosis:

A blue discoloration of the skin reflecting excessive concentration of reduced hemoglobin in the blood due to poor oxygenation.

Cytochrome oxidase:

An iron-containing oxidase present in various tissues and cells; a constituent of the respiratory enzyme system.

Deficiency:

Lack of an essential nutrient.

Defluorinated:

Having had the fluorine content reduced to a level, which is non-toxic under normal use.

Dehulled:

Grains, fruit or nuts whose seed coat or outer shell has been removed.

Dehulling (process):

Having removed the outer covering from grains or other seeds.

De novo:

Starting from the beginning; a new.

Digestible nutrients:

The part of each feed material that is digested or absorbed by bird.

Dispensable amino acids:

These are extensively synthesized in the body and not essential components of the diet. It may includes alanine, glutamine, aspartinine, glycine, serine, proline, glutamic acid, asparagine etc.

Drake:

Mature male duck.

Dried beet pulp:

The dried residue from sugar beet. Dark in color, in strip or pellet form.

Duckling:

Young duck of either sex from day of hatch to 6-weeks of age.

Duodenum:

The upper portion of the small intestine that extends from the stomach to the jejunum.

Edema:

Swelling of a part or the entire body due to abnormal accumulation of an excess of water in the intercellular tissue spaces or body cavities.

Eicosanoid:

A biologically active substance, derived from arachidonic acid, eicosatetraenoic acid and eicosapentaenoic acid, including the prostaglandins, thromboxanes and leukotrienes.

Eicosapentaenoic acid (EPA):

An omega-3 fatty acid found in fish.

Electrolyte:

A substance in solution with a positive electrical charge (sodium, potassium, calcium, magnesium) or a negative charge (chloride, carbondioxide, phosphorus, sulfate, lactate etc).

Electron transport chain:

It along with the process of oxidative phosphorylation enables the production of ATP, the energy "currency" of the body.

Embryo:

A fertilized ovum (egg) in the earlier stages of pre-natal development usually prior to development of body parts.

Encephalomyelitis:

A disease of the central nervous system of chicks caused by a filterable virus; sleeping sickness. It results in lack of balance, head retraction and walking in circles.

Endocrine gland:

Any of the ductless glands, such as the thyroid or adrenal, the secretions of which pass directly into the bloodstream from the cells of the gland.

Endocrine system:

A regulatory system in the animal's body that operates by means of chemical messengers, hormones produced by ductless or endocrine glands.

Entero-hepatic circulation:

The recurrent cycle in which bile salts and other substances excreted by the liver pass through the intestinal mucosa and become reabsorbed by the hepatic cells and re-excreted.

Environment:

Sum total of all factors, which effect the growth and the adaptation of an organism.

Ergocalciferol:

Plant derived vitamin D₂.

Ergosterol:

One of the plant sterols, which upon exposure to ultra violet light are converted to vitamin D.

Esterify:

To combine an acid and an alcohol with elimination of a molecule of water, forming an ester.

Ether extract:

See crude fat.

Evaporating:

Reduced to a denser form; concentrated as by evaporation or distillation.

Evaporation (*process*):

Heat loss resulting from the latent heat of vaporization, which removes heat from a wetted surface.

Exploded (process):

Grain kernels swollen to several times their original size by first steaming under pressure to force moisture into the kernels, and then exposing to air.

Extracellular water:

The water in the plasma, lymph, spinal fluid and secretions.

Extracted, solvent (process):

Having removed fat or oil from materials by organic solvents.

Extrusion (process):

A process, by which feed has been pressed, pushed or protruded through orifices under pressure.

Exudative diathesis:

Increase in capillary fragility leading to accumulation of fluid (edema) under the skin in various parts of the body, mainly under the abdominal tissue.

Feed additive supplement:

An article for the diet of an animal which contains one or more food additives and is intended to be: (1) Further diluted and mixed to produce a complete feed; or (2) Fed undiluted as a supplement to other feeds; or (3) Offered free choice with other parts of the rations separately available.

Feed additives:

Products added to basic feed mixes to improve the rate and/or efficiency of gain, prevent disease or preserve feeds.

Feed conversion ratio (FCR):

Units of feed consumed per unit of weight gained.

Feed grade:

Term to describe the quality of feedstuffs suitable for animal, but not human, consumption.

Feed processing:

Physical or chemical changes in feedstuffs that influence their nutritional value.

Fish meal:

The clean, dried, ground tissue of undecomposed whole fish or fish cuttings, either or both, with or without the extraction of part of the oil.

Flakes (physical form):

An ingredient rolled or cut into flat pieces with or without prior steam conditioning.

Gall bladder:

A membranous sac lying next to liver of all farm livestock, except the horse, in which bile is stored.

Gastric glands:

Glands in the stomach wall that secrete gastric juice into the stomach.

Gastroenteritis:

Inflammation of the stomach and the intestines.

Gastrointestinal tract (GIT):

See alimentary tract.

Gelatinizing (process):

Having had the starch granules completely ruptured by a combination of moisture, heat and pressure and in some instances by mechanical shear.

Germ (part):

The embryo found in seeds and frequently separated from the bran starch during milling.

Gizzard:

Muscular stomach in poultry. Its main function is grinding food and partial digestion of proteins.

Gland:

An organ that produces and secretes a chemical substance in the body.

Glucagon:

A hormone produced by the α -islets of the pancreas that stimulates the conversion of glycogen to glucose and gluconeogenesis in the liver to bring about a rise in plasma glucose levels.

Glucocorticoid:

The group of corticosteroids predominantly affecting carbohydrate metabolism through promotion of gluconeogenesis and liver glycogen deposition and elevation of blood glucose levels.

Gluten (part):

The tough substance remaining when the flour or wheat or other grain is washed to remove the starch.

Glycerol:

An alcohol containing 3 carbons and 3 hydroxyl groups.

Glycine:

A non-essential amino acid. It is a part of many proteins in plants and animals.

Glycogen:

A polysaccharide with the formula $(C_6H_{10}O_5)_n$. It is found in the liver and depolymerized to glucose to serve as a ready source of energy when needed by the animal. It is also known as animal starch.

Glycogenesis:

The synthesis of glycogen.

Glycogenolysis:

The splitting up of glycogen in the body tissues, yielding glucose.

Gossypol:

A substance present in cottonseed and cottonseed meal, which is toxic to non-ruminant animals.

Gout:

A group of disorders of purine and pyrimidine metabolism characterized by hyperuricemia and deposition of urate crystals in joints.

Grain:

Seed from cereal plants e.g., oats, corn and wheat.

Gram-negative bacteria:

Those, which retain a violet color even in the presence of alcohol or acetone.

Gram-positive bacteria:

Those, which lose their color in the presence of alcohol or acetone.

Grinding (process):

Reduced in particle size by impact, shearing or attrition.

Grit:

Angular, hard crushed rock, preferably from granite, used by the chickens in place of "teeth".

Groat:

Grain from which the hulls have been removed.

Hemoglobin:

The oxygen carrying, red pigmented protein of the red corpuscles.

Hemolysis:

Disruption of the integrity of the red blood cell membrane causing release of hemoglobin.

Hemolytic anemia:

Anemia caused by shortened survival of mature red blood cells.

High-density lipoprotein (HDL):

A plasma lipid/protein complex rich in phospholipid and cholesterol; considered to be of benefit in reducing the risk of cardiovascular disease.

Histamine:

A chemical in the body tissues that constricts the smooth bronchial tube muscles, dilates small blood vessels, allows fluid leakage to form itchy skin and hives, and increases secretion of stomach acid.

Histidine:

One of the essential amino acids.

Homeostasis:

Maintaining stable internal body conditions.

Homoeothermic:

It means that birds maintain a relatively constant deep body temperature.

Hormone:

A chemical messenger secreted by ductless glands into the blood stream, in the body, which exerts a profound effect on physiological function of another organ e.g. thyroxin, estrogen, cortisone etc.

Hormone-sensitive lipase:

An enzyme within the adipose cell that catalyzes the release of free fatty acids from the cell.

Hulls:

The hard, outer protective covering of seeds (grains) obtained as a byproduct during seed processing.

Husks:

Usually refers to the fibrous covering of an ear of grain.

Hydraulic process:

A process for the mechanical extraction of oil from seeds, involving the use of a hydraulic press. Sometimes referred to as the old process.

Hydrochloric acid (HCl):

An acid secreted by the parietal cells in the lining of the stomach that helps in protein digestion.

Hypothalamus:

A brain center that integrates signals about the blood's temperature, glucose content and other conditions.

Ileum:

The lower portion of the small intestine extending from the jejunum to the cecum.

Immunity:

Developing resistance to a specific pathogenic microorganism.

Immunoglobulin:

A globulin protein produced by lymphocytes in response to antigenic stimulus.

Indispensable amino acids:

See essential amino acids.

Infection:

Entry of pathogenic microorganisms into the body.

Ingredient:

A component, part or constituent of any combination or mixture making up a commercial feed.

Intestine, large:

The tube like part of the digestive tract lying between the small intestine and the anus. It is large in diameter but shorter in length than small intestine.

Intestine, small:

The long, tortuous, tube like part of the digestive tract leading from the stomach to the cecum and large intestine. It is smaller in diameter but longer than the large intestine.

Intracellular:

Within the cell.

Intrinsic factor:

A chemical substance (glycoprotein) secreted by the gastric glands in normal stomach juice. It is necessary for the absorption of vitamin B_{12} . Its secretion is impaired in pernicious anemia.

Ion:

An atom or molecule that has acquired a net electric charge by gaining or losing electrons.

Iron (*Fe*):

It is one of the most important mineral. It is complexed with hemoglobin and is essential for oxygen transport in the blood.

Kernel:

A dehulled seed.

Kibbled corn (*physical form*):

It is obtained by cooking cracked corn under steam pressure and extruding from an expeller or other mechanical pressure device.

Kibbling (process):

Cracking or crushing of an extruded feed that has been cooked prior to or during the extrusion process.

Kilocalorie (kcal):

One thousand (1,000) calories.

Krebs cycle:

See citric acid cycle.

Labile:

Likely to undergo chemical change; unstable; labile nutrients are affected by light, oxygen, heat, etc.

Lactalbumin:

Protein found in the whey component of milk.

Lactase:

The intestinal enzyme that hydrolyzes lactose to glucose and galactose. It is required for digestion of milk and milk products.

Lactic acid:

A product from glucose metabolism in anaerobic condition.

Lameness:

It may be due to physical cause i.e. injury to pad by sharp stone or a symptom of a disease such as paralysis.

Layer:

A hen, which is kept specifically for the production of eggs.

Lecithin:

A choline-containing phospholipid that is found in all plant and animal tissues, and frequently functions as an emulsifier.

Lecithinase:

An enzyme that disintegrates lecithin.

Lesion:

A wound or injury; a pathological alteration of tissue.

Leucine:

One of the essential amino acids.

Leukocyte:

Usually referring to white blood cells.

Lignin:

A non-carbohydrate material sometimes included in fiber determination that is a major component of the woody portion of plants.

Limiting amino acid:

The essential amino acid found in the shortest supply relative to the amounts needed for protein synthesis in the body.

Lipoprotein lipase:

An enzyme that catalyzes the hydrolysis of fats present in chylomicrons and lipoproteins. It is present in various tissues and important in mobilization of fatty acids from depot fats.

Lipoprotein:

A combination of a lipid and protein, possessing the solubility of proteins. They can act as agents of lipid transport in the lymph and blood, e.g., chylomicrons, HDL, VLDL, LDL.

Lipotropic:

Pertaining to substances preventing or correcting the fatty liver of choline deficiency.

Liver:

One of the most complex organs of the body. It is responsible for the metabolism of fats, proteins and carbohydrates. It is also involved in the body's detoxification, digestion and the regulation of blood sugar.

Long chain fatty acid (*LCFA*):

A fatty acid containing 13-27 carbons; 16-18 is most common.

Low-density lipoprotein (LDL):

A lipid-protein complex circulating in the plasma, transporting most of the blood cholesterol.

Lumen:

The inner open space of a tubular organ, as of a blood vessel or an intestine.

Lymph:

A clear fluid that circulate through lymph vessels and is collected from the tissues throughout the body. Its function is to nourish tissue cells and return waste matter to the blood stream.

Lymphatic system:

It is generally a concentrated area of immune cells, which can interact with the blood stream to help fight infections, and control overall body fluids. The spleen and thymus are major lymphatic organs.

Lysine:

An essential amino acid for protein synthesis. It is the first limiting amino acid in corn-soybean based poultry diets.

Lysozyme:

It is a mucolytic enzyme with natural antibiotic properties and is excreted in the tears, nasal mucus, milk and saliva in most animals. It is a part of the body's first natural defense against bacteria and viruses.

Macro-minerals:

Minerals required in relatively large amounts by livestock. Includes calcium, phosphorus, magnesium, potassium, chlorine, sulfur and sodium.

Macrocytic anemia:

Anemia characterized by larger than normal red blood cells, increased mean corpuscular volume and mean corpuscular hemoglobin.

Malting:

To convert cereals into malt using the steeping process.

Maltose:

A disaccharide having the formula $C_{12}H_{22}O_{11}$ composed of 2 glucose units. It is obtained from the partial hydrolysis of starch.

Manganese (Mn):

It is widely distributed metal, making up nearly 0.1% of the earth's crust. It is required in several metalo-enzymatic reactions such as superoxide dismutase, an enzyme used to resolve highly damaging superoxide free radicals.

Mega calorie:

One thousand (1,000) kilocalories or one million (1,000,000) calories.

Megaloblastic anemia:

Anemia characterized by the presence of large, immature, abnormal red blood cell progenitors in the bone marrow; characteristic of a folic acid or vitamin B_{12} deficiency.

Melanin:

A dark pigment found in the skin, retina and hair.

Melatonin:

It is a key hormone, produced in the pineal gland, in the body's regulation of daily, monthly, yearly and possibly even life-long bodily functions.

Menadione:

Synthetic form of vitamin K.

Menaquinone:

Vitamin K synthesized by bacteria.

Metabolic body size:

The weight of the animal rose to the 3/4 power (W^{0.75}); a figure indicative of metabolic needs and of the feed required to maintain a certain body weight.

Metallothionein:

An abundant non-enzymatic zinc-containing protein.

Methionine:

It is one of the essential amino acids and one of amino acids that contains a sulfur group. It acts as a methyl donor via Sadenylmethionine, one of the principle sources of methyl groups in the body.

Methyl (*CH*₃):

It is derived from methane and occurring in many important organic compounds.

Methylation:

A process by which methyl groups are added to a compound.

Micelle:

A particle containing lipids and bile salts that moves fatty acids from the intestinal lumen to the intestinal mucosa for absorption.

Middling:

A byproduct of flour milling, consisting of varying proportions of small particles of bran, endosperm and germ.

Mill byproduct:

A secondary product obtained in addition to the principal product in milling practice.

Mineralocorticoid:

Adrenocortical hormones that regulate electrolyte balance. Aldosterone is the most potent.

Minerals:

Inorganic feed elements essential for life.

Mitochondria:

Organelles that are the principal energy source of the cell, and contain the cytochromic enzymes of terminal electron transport, and the enzymes of the Krebs cycle, β -oxidation and oxidative phosphorylation.

Mixing (process):

To combine by agitation 2 or more ingredients to a specific degree of dispersion.

Molasses:

A thick viscous, usually dark colored, liquid product containing a high concentration of soluble carbohydrates, minerals and certain other materials.

Molt:

To shed old feathers and regrow new ones.

Molting:

It is a natural process of all birds in an endeavor to renew their feathers prior to migration, shorter days or cooler weather.

Myopathy:

Any disease of the muscle.

Nephritis:

Kidney disease resulting in some loss of function.

Net energy (*NE*):

The amount of feed energy actually available for animal maintenance and production. It represents the energy fraction in a feed left after fecal, urinary, gas and heat losses.

Net protein utilization (NPU):

A measure of protein quality; the amount of protein nitrogen that is retained from a given amount of protein nitrogen eaten.

Neuritic:

Pertaining to the nerves.

Neuropathy:

Non-inflammatory lesions related to functional disturbances in the peripheral nervous system.

Nitrogen free extracts (NFE):

That parts of feed dry matter, which is not crude protein, crude fat, crude fiber or ash. It consists mostly of sugars and starches.

Nitrosamines:

Carcinogenic derivatives of nitrites that may be formed in the stomach when nitrites combine with amines.

Non-essential amino acids:

See dispensable amino acids.

Non-heme iron:

The form of iron found in eggs, grains, vegetables and fruits, and which is less well absorbed than heme iron.

Nutrients:

Any chemical compound having specific function in the nutritive support of animal life. It can be obtained from feed and used in the body to promote growth, maintenance and/or repair. The 6 classes of nutrients are carbohydrate, fat, protein, vitamins, minerals and water.

Nutrition:

Nutrition is a branch of science which deals with the nourishment of body properly, that is providing adequate nutrients for maintenance, growth, repair, production of the bird.

Oleic acid:

An 18-carbon, monounsaturated fatty acid.

Olein:

The fat formed from the reaction to oleic acid with glycerol.

Oligosaccharide:

A carbohydrate that upon hydrolysis yields 3-10 monosaccharide units.

Organelle:

A specialized part of a cell that resembles and functions as an organ.

Osteomalacia:

A condition of impaired mineralization of the bones caused by vitamin D and calcium/Phosphorus deficiencies.

Osteoporosis:

A loss of bone density to the point that the skeleton is unable to sustain ordinary stresses and fractures develop.

Oxidant:

The substance that is reduced and that, therefore, oxidizes the other component of an oxidation-reduction system. It may cause the production of free radicals.

Parathyroid:

Any one of 4 small glands situated beside the thyroid gland concerned chiefly with calcium and phosphorus metabolism.

Particle Size:

Refers to the diameter of granular feed materials. (e.g., grains, pellets, mineral particles). Particle size can affect mixing of feed ingredients and digestion rate.

Parts per million (*ppm*):

A measurement used for nutrients present in very small quantities, e.g., micro minerals; ppm = milligrams per kilogram (mg/kg) or milliliter per liter (ml/L).

Peptide:

Any compound of low molecular weight that yields 2 or more amino acids on hydrolysis.

Peristalsis:

The movement of the intestine, or other tubular structure, characterized by waves of alternate circular contraction and relaxation of the tube by which the contents, such as feed, are propelled onward.

Pernicious anemia:

A macrocytic, megaloblastic anemia caused by a deficiency of vitamin B_{12} .

Perosis:

See chondrodystrophy.

Phylloquinone:

Vitamin K from plants.

Physiology:

The study of functions of parts of body.

Phytic acid (phytate):

A phosphorus-containing compound found in the outer husks of cereal grains that binds with calcium and inhibits its absorption.

Pica:

An abnormal craving to consume unusual substances such as clay, chalk, laundry starch and dirt.

Pituitary:

A small endocrine gland in the lower parts of the brain, which produces a number of hormones. It controls many different aspects of physiology, like, thyroid stimulating hormone, adrenocorticotropic hormone, follicle stimulating hormone, leuteinizing hormone and prolactin.

Plasma:

The clear, yellowish fluid portion of blood, lymph or intramuscular fluid in which cells are suspended.

Plasmid:

A small circle of bacterial DNA that functions independently of the bacterial chromosome. These are used to transfer genes from one microorganism or plant to another, because they are capable of replicating independently in a host cell.

Poikilotherms:

The animals whose body temperature fluctuates with external environmental temperature.

Potassium (K):

It is one of the major minerals in the body. Its intake has been associated with lowering blood pressure.

Poult:

Young turkey of either sex, from day of hatch to 8-10 weeks of age.

Poultry science:

It is the study of principles and practices involved in the production and marketing of poultry and poultry products. It includes breeding, incubation, brooding, housing, feeding, disease, marketing and poultry-farm management.

Poultry byproduct meal:

Ground dry rendered parts of slaughtered poultry, such as heads, feet, undeveloped eggs and intestines etc. It should be exclusive of feathers.

Poultry:

A term designating those species of domesticated birds, which can be raised under human care, reproduce freely, used for food and fiber and economical for human beings. This includes all chickens, turkeys, ducks and geese, as well as swans, guineas, pigeons, peafowl, pheasants, partridge, quails and ostriches.

Probiotics:

A live microbial feed supplement that beneficially affects the host animal. It can reduce early mortality, increase growth rate, and improve feed conversion, egg quality and animal health.

Proline:

It is one of the non-essential amino acids.

Prosthetic group:

A coenzyme that is physically part of its enzyme.

Protease:

An enzyme that breaks peptide bonds found in protein.

Pyruvate:

It is the end product of glycolysis and can be converted into lactate or acetyl-CoA.

Radiation:

The transfer of heat by electromagnetic waves passing between 2 separated objects without any heating of the intervening space between emitter and absorber.

Ration:

The amount of total feed or any specified portion of a provided to bird over a 24 hours period.

Ribonucleic acid (RNA):

A nucleic acid molecule, consisting of ribose, phosphate and the bases adenine, guanine, cytosine and uracil, found in all cells. It translates the instructions encoded in DNA to build proteins.

Ribosome:

A part of a cell that builds proteins by linking amino acids according to the sequence on a strand of messenger RNA.

Rice bran:

The pericarp or bran layer and germ of the rice, with only such quantity of hull fragments and calcium carbonate as is unavoidable in the regular milling of edible rice. It must contain not more than 13% crude fiber.

Safflower meal:

The ground residue obtained after extracting the oil from whole safflower seed, by mechanical or solvent extraction process.

Salmonellosis:

Any disease caused by a salmonella infection, which may manifest as food poisoning with acute gastroenteritis, vomiting and diarrhea.

Saponification:

The process of hydrolyzing fats into soaps and glycerol by the addition of alkali.

Saturated fat:

Completely hydrogenated fat. Saturated fats are solid at room temperature.

Saturated fatty acid (SFA):

A fatty acid with the formula $C_nH_{2n}O_2$ that has no double bond and that contains all the hydrogen it can hold.

Screenings:

It may include light and broken grains, small imperfect kernels, hulls, chaff, weed seeds, agriculture seeds and other foreign material obtained from the cleaning of grain.

Selenium (Se):

It is considered as a trace-mineral and found in minute amounts in soil. It closely interacts with vitamin E in the body. The antioxidant properties of selenium are related to this interaction as well as its active selenoprotein involvement in glutathione metabolism.

Sensible heat:

Heat that causes the temperature of the surroundings (air or physical medium) to increase.

Serotonin:

A neurotransmitter produced from the amino acid, tryptophan, which assists in relaxation and sleep.

Serum:

The cell-free fluid that remains after the fibrin clot and blood cells are removed.

Specific pathogens free (SPF):

It is used to designate a flock that is certified free from certain specific pathogenic organisms.

Spleen:

It is a small lymphatic organ located on the left side of the body between the diaphragm and the stomach. It is involved in destroying worn-out blood cells and is involved in helping the immune systems during infections.

Starch:

A polysaccharide, composed of glucose, found only in plants. It occurs in both amylose and the amylopectin form.

Steaming (process):

Having treated ingredients with steam to alter physical and/or chemical properties.

Sterilization (process):

The complete destruction of microorganisms.

Sterol:

A compound composed of carbon, hydrogen and oxygen atoms arranged in rings like those of cholesterol.

Straw:

Remaining portion after the removal of the seed by threshing or combining, usually of cereals.

Supplements:

A feed or feed mixture used to improve the nutritional value of a basal feeds. Supplements are usually rich in protein (more than 25%), minerals and vitamins or feed additives.

Taurine:

It is an amino acid, synthesized in the body from methionine and cysteine.

Thermal stimulus:

Changes in temperature or other weather related factors that cause a response.

Thermal stress:

Changes in temperature or other weather related factors that produce a response.

Thermoneutral zone:

It is a range of environmental temperature to which the body temperature remains constant.

Thermoregulation:

The regulation of body temperature.

Thiamin:

It is an essential B-complex vitamin, found in, meat products, vegetables and grain husks etc. It functions as a cofactor for several enzymes in the form of thiamine triphosphate (TPP).

Trans-fatty acids:

Stereoisomers of the naturally occurring *cis*-fatty acids. These are the artifacts of the hydrogenation process and found mainly in margarines and vegetable shortenings.

Transferrin:

A protein synthesized in the liver that transports iron in the blood to the erythroblasts for use in heme synthesis.

Transketolase:

An enzyme essential in carbohydrate metabolism that requires thiamin (B_1) as a coenzyme.

Triacylglycerol:

See triglyceride.

Tricarboxylic acid cycle (TCA):

See citric acid cycle.

Triglyceride:

A lipid consisting of 3 fatty acid chains esterified to a glycerol molecule.

Tripeptide:

Three amino acids bonded together by peptide bonds.

True metabolizable energy (TME):

It is the gross energy of the feed minus the gross energy of the excreta of the feed origin.

Vitamin:

A group of organic substances, which in relatively small amounts are essential for normal growth and metabolism, and that cannot be synthesized by the body.

Vitamins, fat-soluble:

It includes vitamins A, D, E and K, which are soluble in organic solvents.

Vitamins, water-soluble:

Vitamins, which can dissolve in water. It includes B-complex vitamins and vitamin C.

Wafering (process):

Having agglomerated a feed of a fibrous nature by compressing into a form usually having a diameter or cross section measurement greater than its length.

Wafers (physical form):

A form of agglomerated feed based on fibrous ingredients in which the finished form usually has a diameter or cross section measurement greater than its length.

Wheat bran:

The coarse outer covering of the wheat kernel as separated from cleaned and scoured wheat in the usual process of commercial milling.

Zinc (Zn): It plays a vital role in over 50 different proteins or enzymes, which are critical for the immune system, overall cellular metabolism, control of protein synthesis and protection of membrane lipid peroxidation.

Literature Cited

Acar, N., et al., 1991. Poult. Sci., 70: 2315.

Adams, A.W., et al., 1975. Poult. Sci., 54: 707.

Adams, C.R., 1972. Nat. Acad. Sci., Washington, D.C., pp. 142.

Adams, R.L. and J.C. Rogler. 1968. Poult. Sci., 47: 579.

Ahn, D.U., et al., 1995. Poult. Sci., 74: 1540. Montreal, Canada.

Ananymous. 1994. World Poult. Misset, 10:20.

Anderson, J. and R. Warnick, 1964. Poult. Sci., 43:1091.

Anderson, J.O., et al., 1984. Poult. Sci., 63: 311.

Andrews, N.C., 1999. New Eng. J. Med. 341: 1986.

Angkanaporn, K., et al., 1996. Poult. Sci., 75: 1098.

Anonymous, 1974. National Academy Press, Washington, D.C.

Anonymous, 1981. National Academy Press, Washington, DC.

Anonymous, 1983. World's Poult. Sci., 39: 75.

Anonymous, 1984. National Academy Press. 8th ed. Washington, D.C.

Anonymous, 1987. Saskatchewan Agriculture and The University of Saskatchewan.

Anonymous, 1990. Assoc. of Off. Anal. Chem., 15th Ed., Arlington, VA.

Anonymous, 1990. Univ. of Saskatchewan, Saskatoon, Canada. S7N 0W0.

Agriculture and Food.

Anonymous, 1999. National Academy Press, 10th Ed., Washington, D.C.

Appelquist, L.A. and R. Ohison, 1972. Elsevier publishing Company.

Araba, M. and N. Dale, 1990. Poult. Sci., 69: 76.

Armstrong, H., 1993. Feed Mix, 1: 26.

Ash, W.J., 1976. Farmer Bulletin No. 2215, U.S.D.A.

Balloun, S.L., 1980. Amer. Soybean Assoc., St. Louis, Missouri, USA.

Balnave, D and T. Scott, 1986. Nutrition Reports International, 34: 29.

Balnave, D., 1970. World's Poult. Sci. J., 26: 442.

Balnave, D., 1971. Comp. Biochem. Physiol., 40A: 1097.

Balnave, D., 1988. Proceedings of the Poultry Research Foundation Symposium, p. 02.

Balnave, D., et al., 1992. Poult. Sci., 71: 2035.

Barbour, G.W. and J.S. Sim, 1991. Poult. Sci., 70: 2154.

Barton, T.L., 1989. Zootecnica Intern., March 1989, pp44-46.

Baziz, H.A., et al., 1996. Poult. Sci., 75: 505.

Bedford, M. and H. Classen, 1992. J. Nutr. 122: 560.

Bedford, M., 1996. J. Appl. Poult. Res., 5: 370.

Begin, J.J. and T.H. Johnson, 1976. Poult. Sci., 55: 2395.

Behnke, K.C., 1996. 57th Minnesota Nutr. Conference, pp305-320

Bell, J.M. 1984. J. Anim. Sci., 58: 996.

Blakely, R.M., et al., 1963. Brit. Poult. Sci., 4: 261.

Blaxland, J.D., 1946. Veterinary J., 102: 157.

Boling, S.D. and J.D. Firman, 1998. Poult. Sci., 77: 547.

Bolton, W., 1962. Proc. XIIth World's Poult. Congress, pp. 3842.

Bowmaker, J.E. and R.M. Gous, 1989. Brit. Poult. Sci., 30: 663.

Branton, S.I., et al., 1986. Poult. Sci., 65:1659.

Brenes, A., et al., 1989. Brit. Poult. Sci., 30: 81.

Brenes, A., et al., 1993. Can. J. Anim. Sci., 73: 605.

Brennenkmeiyer, C. 1996. Proc. of the World Poultry Science Society, Vol II, pp119. New Delhi.

Brewer, D.E. and P.R. Ferket, 1989. Poult. Sci., 68: 18.

Brister, R.D., Jr., et al., 1981. Poult. Sci., 60: 2648.

Britton, W.M. and R.D. Wyatt, 1978. Poult. Sci., 57: 163.

Britton, W.M., 1990. Proc. 1990 Georgia Nutr. Conference., pp. 152.

Bronner, F., 1998. J. Nutr., 128: 917.

Bryden, W.L., et al., 1996. In: Protein Metabolism and Nutrition,

Estacoa Zooteecnica Nacional, Vale de Sanatarem, Portugal.

Budowski, P., et al., 1987. Brit. J. Nutr., 58: 511.

Bundy, C.E. and R.V. Diggins, 1960. Poult. Prod., Prentice-Hall, Inc, New Jersey.

Bureau, D.P., et al., 2000. Aquaculture, 181: 281.

Burns, R., 1995. Feedstuffs, 67: 11.

Butler, E.J., et al., 1982. J. Sci. Food Agric., 33: 866.

Cabel, M.C., et al., 1988. Poult. Sci., 67: 1725.

Cabrera, M., 2000. Proc. of the Southeastern Comm. Egg Producers Forum, Tybee Island, GA.

Cambell, R.D., *et al.*, 1986. Res. Rep. AL-1986-5, Dept. Anim. Sci., Florida Agric. Exp. Station, Gainsville, Fla.

Campbell, G.L. and M.R. Bedford, 1992. Can. J. Anim. Sci., 72: 449.

Campbell, L.D. and B.A. Slominski, 1991. *Proc.* 8th Int. Rapeseed Cong., Vol. 2, pp. 442.

Campbell, L.D. and F. Schöne, 1998. EAAP Publ. No. 93, Wageningen, The Netherlands, pp. 185.

Caston, L. and S. Leeson, 1990. Poult. Sci., 69: 1617.

Cheeke, P.R. 1991. 1st ed. Macmillan Publishing co., New York, NY.

Chesson, A., 1993. Anim. Feed Sci. Technol., 45: 65.

Chesson, I., 1987. Haresign and Cole, eds. Oxford Univ. Press, Oxford, UK.

Chew, B., 1993. J. Dairy Sci., 76: 2804.

Chibowska, M., et al., 1997. Poult. Sci., 76: 80.

Choct, M., 1992. Proc. of 13th Western Nutrition Conf., p.41.

Choi, J.H., et al., 1986. Poult. Sci., 65: 594.

Chu, T.K. and F.A. Kummerow, 1950. Poult. Sci., 29: 846.

Classen, H.L., 1996. Anim. Feed Sci. Techn., 62: 21.

Classen, H.L., 1996. Feed Mix., 4: 22.

Cleophas, G.M.L., et al., 1995. World Poult. Misset, 4: 12.

Clunies, M. and S. Leeson, 1983. Proc. Guelph Nutr. Conf., pp8796, Guelph, Ontario, Canada.

Coates, B.J., et al., 1977. Can. J. Anim. Sci., 57: 209.

Coelho, M.B., 1994. Feed Management, 45: 10.

Coelho, M.B., 1994. Proc. Arkansas Nutr. Conf., Fayetteville, pp. 52-61.

Colnago, G.L., et al., 1984. Poult. Sci., 63: 1136.

Cook, M.E., 1991. Crit. Rev. Poult. Biology., 3: 176.

Cook, M.E., 1991. Proc. BASF Tech. Symp., pp106, Bloomington, MN.

Cook, M.E., et al., 1993. Poult. Sci., 72: 1301.

Coon, C.N., 1998. Amer. Soybean Assoc., Brussels, Belgium.

Coon, C.N., et al., 1990. Poult. Sci., 69: 787.

Corrier, D.E., et al., 1998. J. Appl. Poult. Res., 7: 132.

Couch, J.R., et al., 1967. Brit. Poult. Sci., 8: 243.

Cowman, O.J. and W. Michie, 1978. Brit. Poult. Sci., 19: 601.

Craig, et al., 2007. Mechanisms of Action for Supplemental NSP

and Phytase Enzymes in Poultry Diets. Carolina Feed Industry

Association, 35th Poultry Nutrition Conference.

Croom, J, et al., 1996. Feed Mix, 4: 2.

Cruickshank, E.M., 1934. Biochem., .28: 965.

Cuervo, C., et al., 1972a. Poult. Sci., 51: 281

Cuervo, C., et al., 1972b. Poult. Sci., 51: 813

Dale, N. and M. Araba, 1991. Zootechnica International, March.

Dale, N., 1996. J. Appl. Poult. Res., 5: 105.

Dale, N.M., 1997. J. Appl. Poult. Res., 6: 169.

Dale, N.M., et al., 1987. Georgia Nutr. Conf., Atlanta, Georgia.

Dale, N.M., et al., 1990. Poult. Sci., 69: 72.

Dale, N.M., et al., 1993. J. Appl. Poult. Res. 2: 4042.

Dalvi, R.R. and C. McGowan, 1984. Poult. Sci. 63: 485.

Damron, B.L., 1998. Poult. Sci., 77: 1488.

Davidson, J. and S. Graham, 1981. J. Agric. Sci. Camb., 96: 221.

DeLuca, H.F., 1992. Ann. N.Y. Acad. Sci., 669: 59.

Devendra, C. and G. Raghavan, 1978. World Rev. Anim. Prod., 14: 11.

Deyhim, F. and R.G. Teeter, 1991. Poult. Sci., 70: 2551.

Deyhim, F. and R.G. Teeter, 1993. J. Applied Poult. Sci., 2: 347.

Dhand, N.K., et al., 1998. Ind. J. of Anim. Sci., 68: 1095.

Dibner, J.J., et al., 1995. J. Appl. Poult. Res., 5: 70.

Dibner, J.J., *et al.*,1995. Proc. of the Nutritional and Tech. Symp., pp. 45, Atlanta GA.

Dilworth, B.C. and E.J. Day, 1964. Poult. Sci., 43: 1039.

Douglas, M.W., et al., 1999. Poult. Sci., 78 (Supplement 1): 12.

Dowman, M.G. and F.C. Collins, 1982. J. Sci. Food Agric., 33: 689.

Ehrich, M., et al., 1986. Avian Diseases, 30: 802.

Elkin, R.G., 1987. World's Poult. Sci., 43: 84.

Englyst, H.N., et al., 1992. Eur. J. of Clinical Nutr., 46: S33.

Enos, H.L. and A. Monsi, 1977. Poult. Sci., 56: 1373.

Ensminger, M.E., *et al.*, 1990. Feeds and Nutr. Digest. 2nd ed. Ensminger Pub. Comp. California, USA.

Erf, G.F. and W.G. Bottje, 1996. Proc. Ark. Nutri. Conf.

Evans, R.J. and J. McGinnis, 1946. J. Nutr., 31: 449.

Fairchild, F. and D. Greer, 1999. Feed International, Aug. 32.

Fairfield, D., 1994. In: Feed Manuf. Tech., IV. pp111, Amer. Feed Ind. Assoc., Arlington.

Fancher, B., et al., 1996. JAPR, 5: 386.

Farrell, D.J. and P. Stapleton, 1986. University of New England, Armidale.

Farrell, D.J., 1980. Feedstuffs, November 3, pp2425.

Farrell, D.J., et al., 1983. Anim. Feed Sci. and Techn., 9: 99.

Ferket, P.R., et al., 1993. Proc. Carolina Poult. Nutr. Conf, p1.

Fernandez, S.R., et al., 1994. Poult. Sci., 73: 1887.

Fernando, *et al.*, 2008. Economic and Environmental Impact of Using Exogenous Enzymes on Poultry Feeding. Int. J. Poult. Sci., 7 (4): 311-314.

Firman, J.D., 1994a. BioKyowa Tech. Rev., 7:2, NutriQuest, Inc., Chesterfield, MO.

Firman, J.D., 2001. Tech. Bullet., Amer. Soybean Assoc., AN32-2001. MITA (P) No. 070/10/2001. www.asasea.com

Firman, J.D. and S.D. Boling, 1998. Poult. Sci., 77: 105.

Fraps, G.S., et al., 1940. Texas Agr. Exp. Sta. Bull., 589.

Furuya, S., 1980. JARQ 14: 5255.

Gadient, M. and R. Fenster, 1994. Aquaculture, 124: 207.

Gadient, M., 1986. Proc. Maryland Nutr. Conf., pp73.

Gadient, M., 1994. Zootecnica Intern., 58.

Gatel, F., 1994. Anim. Feed Sci. and Tech., 45: 317.

Graham, H. and P. Aman, 1991. Anim. Feed Sci. Tech., 32: 191

Gregory, J.F. and G.T. Edds, 1984. Poult. Sci., 64: 1678.

Gross, W.B., 1988c. Avian Dis., 32: 561.

Guenter, W., et al., 1995. Proc. of the 9th Intern. Rapeseed Congr., Cambridge, U.K., pp. 164.

Hagemeister, H. and H. Erbersdobler, 1985. Proc. Nutr. Soc., 44: 133A.

Hakanson, R., et al., 1994. Yale J. Biol. Med., 67: 123.

Hamilton, P.B., 1971. Poult. Sci., 50: 1880.

Hamilton, P.B., 1974. Poultry Digest.

Hamilton, P.B., 1975. Poult. Sci., 54: 1706.

Hamilton, P.B., et al., 1974. Poult. Sci., 53: 871.

Hamilton, P.B., et al., 1982. Poult. Sci., 61: 1832.

Han, Y. and D.H. Baker, 1993. Poult. Sci. 72: 701.

Han, Y. and D.H. Baker, 1994. Poult. Sci., 73: 1739.

Herrman, T. and K. Behnke, 1994. Bulletin MF-1172 Revised,

Kansas State Univ. Cooperative Ext. Service, Manhattan, KS

Herrman, T.J. and G. McClure, 1995. Feed Management, 46: 23.

Hesselman, K. and P. Aman, 1986. Anim. Feed Sci. Tech., 15: 83.

Heuser, G.E., 1995. Feeding Poult., 2nd Ed. Wiley Poultry Science Series.

Heuser, G.F. and M.L. Scott, 1953. Poult. Sci., 13: 137.

Hilliker, J., 1992. Proc. of the broilerpelleting seminar for Asia. California Pellet Mill Co., San Francisco, CA.

Hoehler, D. and R.R. Marquardt, 1996. Poult. Sci., 75: 1508.

Holzapfel, W.H., et al., 1998. Int. J. Food Microbiol., 41: 85.

Hopkins, W.I., 1991. Avian Pathology, 20: 403.

Huff, W.E., et al., 1974. Poult. Sci., 53: 1585.

Huff, W.E., et al., 1992. Poult. Sci., 71: 64.

Humphreys, P.N., 1976. J. Small Anim. Pract., 17: 607.

Inborr, J and S. Foder, 1996. Feed Intern.

Isshiki, Y., et al., 1989. Jap. J. Zootech. Sci., 60: 1082.

Ivusic, S.I., et al., 1994. Anim. Feed Sci. Tech., 45: 205.

Izat, A.L., et al., 1989. Poult. Sci., 68: 651.

Jacobsen, B. J., *et al.*, 1993. Mycotoxins and Mycotoxicoses. Ext. Serv., Auburn Univ. Department of Physiology and Pharmacology.

Jackson, S. and L.M. potter, 1984. Poult. Sci., 63: 2391.

Jackson, S., et al., 1983. Poult. Sci., 62: 1117.

Janssen, W.M., *et al.*, 1979. 2nd Ed. Beekbergen, Netherlands. Spelderholt Center for Poultry Research and Information Services.

Jensen, L.S., 1975a. J. Nutr., 105: 769.

Jensen, L.S., 1975b. Proc. Soc. Exp. Biol. Med., 149: 113.

Jensen, L.S., 1990. Feed Intern., 11: 14.

Jensen, L.S., et al., 1962. Poult. Sci., 41: 1414

Jensen, L.S., et al., 1965. Poult. Sci., 44: 1435.

Jiang, Y.H., et al., 1994. Poult. Sci., 73: 1137.

Jiang, Z., et al., 1992. Poult. Sci., 71: 378.

Jin, S.H., et al., 1998. World Poult. Sci. J., 54: 335.

Johnson, R.J. and H. Karunajeewa, 1985. J. Nutr., 115: 1680.

Kennedy, D.G., et al., 1992. Brit. Poult. Sci., 33: 1015.

Ketels, E. and G. DeGroote, 1989. Poult. Sci., 68: 1506.

Khajarern, J., et al., 1987. Dhornhvaj Co., Ltd. Bangkok, Thailand.

Khan, N., 2000. Proc. 3rd Europ. Symp. on Feed Enzymes, Noorfwijkerhout, The Netherlands.

Kiiskinen, T., 1989. Annales Agriculturae Fenniae, 28: 385.

Kimivac, A, 1978. Archiv fur Geflugelkunde, 42 : 238.

Kino, K. and J. Okumura, 1986. Poult. Sci., 65: 1728.

Klasing, K.C., 1992. Proc. of the Multi-State Poultry Meeting, May 20-21.

Klinkert, W., et al., 1981. Toxicol. Eur. Res., 3: 185.

Klosterman, H.J., et al., 1967. Biochemistry, 6: 170.

Knowles, T.A. and L.L. Southern, 1998. Poult. Sci., 77: 564.

Kofi Y., 1994. World Poult. Misset, 10: 40.

Kondo, A.K. and E. Ross, 1962. Poult. Sci., 41: 1126

Krapu, G.L., 1976. J. Wildl. Manage., 40: 180.

Kratzer, F., et al., 1967. Poult. Sci., 46: 1489.

- Kratzer, F.H., 1946. Poult. Sci., 25: 541.
- Kubena, L.F., et al., 1993a. Poult. Sci., 72: 51.
- Kubena, L.F., et al., 1993b. Poult. Sci., 72: 651.
- Kuhl, H.J., Jr., et al., 1977. Poult. Sci., 56: 605.
- Kurbel, S. and Kurbel, B., 1995. Medical Hypotheses, 45: 539.
- Lacey, J., 1985. John Wiley & Sons, New York.
- Lacy, M.P. and L.R. Vest, 1999. Ext. Poult. Lit., GA 30602-4356,
- Dept. of Poult. Sci., Univ. of Georgia Coop. Ext. Serv.
- Lanz, G.T., 1992. Novus Nutrition Update, Vol 2(1).
- Larsen, C., et al., 1985. Poult. Sci., 64: 2287.
- Lee, H. and J.D. Garlich, 1992. Poult. Sci., 71: 499.
- Lee, H., et al., 1991. Poult. Sci., 70: 2509.
- Leeson, S. and J.D. Summers, 1991. University Books, Guelph, Ontario, Canada.
- Leeson, S., 2001. Ext. Poult. Lit., Dept. of Animal and Poult. Sci., Univ. of Guelph, Guelph, Ontario.
- Leeson, S., et al., 1987. Can. J. Anim. Sci., 67: 151
- Leeson, S., et al., 1995. University Books, Ontario, Canada.
- Leske, K.L., et al., 1993. Poult. Sci., 72: 664.
- Leske, K.L., et al., 1995. Anim. Feed Sci. Tech., 54: 275.
- Lessire, M. and B. Leclercq, 1983. Arch. Geflugelk., 47: 13.
- Levielle, G.A., et al., 1970. Proc. Soc. Exp. Biol. Med., 135: 483
- Lilburn, M.S., 1996. J. Appl. Poult. Res., 5: 78.
- Lonnerdal, B., 2000. J. Nutr., 130: 1378S.
- Maenz, D.D., et al., 1997a. Anim. Feed. Sci. Tech., 81: 177.
- Maenz, D.D., et al., 1997b. Poult. Sci., 76: 71.
- Mahmoud, K.Z., et al., 1996. Poult. Sci., 75: 1555.
- Makled, M.N. and O.W. Charles, 1987. Poult. Sci., 66: 705.
- March, B.E. and J. Biely. 1793. Can. J. Anim. Sci., 53: 569.
- Martland, M.F., et al., 1984. Res. Vet. Sci., 36: 298.
- Mateos, G.G. and J.L. Sell, 1981a. Poult. Sci., 60: 1509.
- Mateos, G.G. and J.L. Sell, 1981b. Poult. Sci., 60: 1925.
- Mateos, G.G. and J.L. Sell, 1981c. Poult. Sci., 60: 2114.
- McDowell, L.R., 2000. Academic Press, San Diego, California.
- McKee, J.S. and P.C. Harrison, 1995. Poult. Sci., 74: 1772.
- McLelland, J., 1979. Academic Press, New York, NY, pp69.

McNab, J. 1993. Feed Mix, 1: 24.

McNab, J.M., 1995. In Recent Adv. in Anim. Nutr. in Austr., pp7. Univ. of New England, Armidale.

McNaughton, J.L. and F.N. Reece, 1980. Poult. Sci., 59: 2300.

Meluzzi, A., et al., 2000. Poult. Sci., 79: 539.

Menge, H., 1968. J. Nutr., 95: 578.

Menge, H., 1971. Poult. Sci., 50: 261.

Mercier, C., et al., 1989. Amer. Assoc. of Cereal Chemists, St. Paul, MN.

Moir, K.W. and J.K. Connor, 1977. Anim. Feed Sci. Tech., 2: 197.

Mommer, R.P. and D.K. Ballantyne, 1991. Hess and Clark, Inc., Ashland, OH.

Mongin, P.J., 1981. Proc. Nutr. Soc., 40: 285.

Moran, E.T., 1989. Butterworths, London, pp87.

Moran, E.T., Jr. and S.F. Bilgili, 1990. Poult. Sci., 50: 1753.

Moran, E.T., Jr., et al., 1992. Poult. Sci., 71: 1687.

Moreng, R.E. and J.S. Avens, 1985. Poult. Sci. and Prod., Reston Publishing Company Inc. Virginia.

Morrison, F.B., 1984. Feeds and Feeding. 22nd Ed. CBS Publisher and Distributors, Delhi, India.

Muir, F.V., et al., 1976. Poult. Sci., 55: 1046.

Mullin, J., 1987. Arrowhead Hunting & Conservation Club, Goose Lake, Iowa 52750.

Nagpal, M.L., et al., 1971. Ind. J. Anim. Sci., 41: 283.

Nernberg, L., et al., 1997. Poult. Sci., 76: 80.

Nesheim, M.C., et al., 1964. J. Nut., 84: 361.

Nichols, T.E., 1983. Southem Cooperative Scries Bulletin 279.

pp67, Aubum University, Aubum, Alabama.

Nitsan, A., et al., 1991. Poult. Sci., 70: 2040.

Nitsan, Z., et al., 1991. Brit. Poult. Sci., 32: 515.

Nixey, C., 1994. Feed Mix, 2: 19.

Noble, R.C. and K. Connor, 1984. World Poult. Sci. J., 40: 114.

Noll, S.L., et al., 1995. Gobbles, 52: 6.

Noll, S.L., et al., 1996. Gobbles, 53: 5.

Norris, K.H., 1964. Trans. Am. Soc. Agric. Eng., 7: 240.

North, M.O., 1978. 2nd Ed. AVI Publishing, Westport CN, U.S.A.

Noy, Y. and D. Sklan, 1995. Poult. Sci., 74: 366.

Nurmi, E. and M. Rantala, 1973. Nature, 241: 210.

Oderkirk, A., 1999. Poult. Fact Sheet, Sep. 1, 1999. Ref. # 990018.

Parr, J.F. and J.D. Summers, 1991. Poult. Sci., 70: 1540.

Parsons, C.M., 1996. Anim. Feed Sci. Tech., 147.

Parsons, C.M., et al., 1991. J. Anim. Sci., 69: 2918.

Parsons, C.M., et al., 1992. Poult. Sci., 71: 133.

Parsons, C.M., et al., 1996. Poult. Sci., 75: (Suppl. 1)

Parsons, C.M., et al., 1997. Poult. Sci., 76: (Suppl. 1).

Payne, W.L., et al., 1971. Poult. Sci., 50: 143.

Patel, M.B. and J. McGinnis, 1985. Poult. Sci., 64: 1148.

Pearson, R.A. and K.M. Herron, 1981. Brit. Poult. Sci., 22: 227.

Pesti, G.M., et al., 1986. Poult. Sci., 65: 2258.

Pfost, H., 1962. Feed Production School Proc., KSU.

Phillips, T.D., et al., 1988. Poult. Sci., 67: 243.

Pimentel, J.L. and M.E. Cook, 1987. Poult. Sci., 66: 2005.

Pipa, F. and G. Frank, 1989. Advances in Feed Technology, 2: 22.

Potchanakorn, M. and L. M. Potter, 1987. Poult. Sci., 66: 505.

Potter, L.M., et al., 1995. Poult. Sci., 74: 813.

Ravidran, V., et al., 1995. Poult. & Avian Biology Rev., 6: 125.

Ravidran, V., et al., 1998. Poult. Sci., 77: 873.

Reddy, C.V., et al., 1962. J. Nut., 77: 428.

Reece, F.N., et al., 1985. Poult. Sci., 64: 1834.

Reece, F.N., et al., 1986. Poult. Sci., 65: 1257.

Renner, R., et al., 1953. Poult. Sci., 32: 517.

Robblee, A.R., *et al.*, 1986. Canola Council of Canada, Winnipeg, Manitoba.

Roberts, J.R. and D. Balnave, 1992. J. Anim. Physiol. Anim. Nutr., 68: 197.

Roland, D.A., Sr. and R.H. Harms, 1973. Poult. Sci., 52: 369.

Roland, D.A., Sr., et al., 1974. Poult. Sci., 53: 662.

Roos, N., et al., 1994. J. Nutr., 124: 2404.

Said, N.W., et al., 1979. Poult. Sci., 58: 1557.

Sainsbury, D., 1984. Poult. Health & Mgt. 2nd Ed. Granada Publisher London.

- Salminen, S., et al., 1998. Brit. J. Nutr., 80: Supp. 1, S147.
- Salmon, R.E., et al., 1984. Poult. Sci., 63: 1994.

Salunke, D.K., et al., 1992. pp59, Van Nostrand Reinhold, New York.

Samarajeewa, V., et al., 1990. J. Food Prot., 53: 489.

Schaible, P.J. 1981. Poult. Feeds and Nutr., 2nd Ed. AVI Pub. Comp., INC. Westport, Connecticut.

Scheideler, S., 1998. Proc. 1998 Multi-State Poultry Meeting.

Scheideler, S.E., 1993. Poultry Sci., 72: 282.

Schutte, J.B., et al., 1997. Poult. Sci., 76: 321.

Scott, M.L., et al., 1960. J. Nutr., 71: 282.

Scott, M.L., *et al.*, 1982. 3rd Ed. M. L. Scott and Associates, Ithaca, NY.

Shingari, B.K., et al., 1995. Pak. Poult., April: 5-8.

Shlosberg, A., et al., 1984. Vet Hum. Toxicol., 26: 384.

Sibbald, I. and M.S. Wolynetz, 1986. Poult. Sci., 65: 98.

Sibbald, I.R., 1982. Can. J. Anim. Sci., 62: 983.

Sibbald, I.R., 1987. Can. J. of Anim. Sci., 67: 221.

Sibbald, I.R., et al., 1963. Poult. Sci., 42: 486.

Sibbald, I.R., et al., 1980. Poult. Sci., 59: 808.

Silvest, O., 1994. World Poult. Misset, 10: 36.

Simbaya, J., et al., 1996. Anim. Feed Sci. Tech., 61: 219.

Simon, J., 1999. Worlds Poult. Sci. J., 55: 353.

Singh, K.S. and B. Panda, 1988. Poult. Nutr., 1st Ed. Kalyani Publisher New Delhi Ludhiana.

Siriwan, P., et al., 1994. Brit. J. Nutr., 71: 515

Skinner, J.T. and P. W. Waldroup, 1992. J. Appl. Poult. Res., 1: 273.

Skinner, J.T., et al., 1991. Poult. Sci., 70: 1223.

Skinner, J.T., et al., 1992a. J. Appl. Poult. Res., 1: 167.

Skinner, J.T., et al., 1992b. J. Applied Poult. Res., 1: 42.

Skinner, J.T., et al., 1992c. J. Appl. Poult. Res., 1: 280.

Skinner, J.T., et al., 1992d. J. Appl. Poult. Res., 1: 367.

- Skinner, J.T., et al., 1992e. Poult. Sci., 71: 1207.
- Skinner, J.T., et al., 1992f. Poult. Sci., 71: 1364.
- Congress, Saskatoon, Canada, pp. 396-401.
- Slominski, B.A. and L.D. Campbell, 1991. Proc. of the 8th Intern. Rapeseed Congr., Saskatoon, Canada, pp1402.
- Slominski, B.A., 1994. J. Sci. Food Agric., 65: 323.
- Slominski, B.A., *et al.*, 1992. Proc. of the World's Poult. Congress, Amsterdam, pp241.
- Slominski, B.A., et al., 1993. J. Agric. Food Chem., 41: 2304.
- Slominski, B.A., *et al.*, 1999. Proc. of the 10th Intern. Rapeseed Congr., Canberra, Australia.
- Slominski, B.A., *et al.*, 2000. Proc. of the 3rd European Symp. on Feed Enzymes, Noordwijerhout, Netherlands.
- Sluis, W.V.D., 1994. World Poult. Misset, 10: 10.
- Smith, J.W., et al., 1971. Poultry Sci., 50: 768.
- Smith, P.A., et al., 1995. Poult. Sci., 74: (abst.) 145.
- Smith, T.W., 1997.URL: http://www.msstate.edu/dept/poultry/bwq feed.htm. Mississippi State University.
- Summer, J.D., et al., 1988. Can. J. of Anim. Sci., 68: 241.
- Summers, J.D., et al., 1988a. Can. J. Anim. Sci., 68: 907.
- Summers, J.D., et al., 1988b. Can. J. Anim. Sci., 68: 1315.
- Summers, J.D., et al., 1989. Can. J. Anim. Sci., 69: 469
- Summers, J.D., et al., 1990. Poult. Sci., 69: 615.
- Summers, J.D., et al., 1992. Can. J. Anim. Sci., 72: 127.
- Svihus, B., et al., 1997. Brit. Poult. Sci., 38: 390.
- Swick, R.A., 1994. Amer. Soybean Assoc. Tech. Bull., No. 071/12/93. ASA, Singapore.
- Swick, R.A., 1997. Amer. Soybean Assoc. NO. 195/11/95 (Vol. AN04-1996) ASA, Singapore.
- Tabib, Z., et al., 1981. Poult. Sci., 60: 1392.
- Tabib, Z., et al., 1984. Poult. Sci., 63: 70.
- Tadtiyanant, C., et al., 1991. Poult. Sci., 70: 44.
- Tannock, G.W., 1999. Horizon Scientific Press, Norfolk, England. pp1.
- Teeter, R.G. and M.O.Smith, 1986. Poult. Sci., 65: 1777.
- Tengerdy, R.P. and C.F. Nockels, 1973. Poult. Sci., 52: 778.

Terpstra, K., 1997. In Proc. Vth Intern. Symp. of Amino Acids, pp221 Budapest.

Thompson, J.N. and M.L. Scott, 1969. J. Nutr., 97: 335.

Trevis, J., 1979. Feedstuffs, pp20, November.

Turk, D.E., 1982. Poultry Sci., 61: 1225.

Um, J. S. and I.K. Paik, 1999. Poult. Sci., 78: 75.

Underhill, L., 1996. Mycotoxin Inspection Program.

Underwood, E.J. and N.F. Suttle, 1999. 3rd Ed. CABI Publishing, New York.

Valdes, E.V. and S. Leeson, 1992a. Poult. Sci., 71: 1179.

Valdes, E.V. and S. Leeson, 1992b. Poult. Sci., 71: 1396.

Valdes, E.V. and S. Leeson, 1992c. Poult. Sci., 71: 1493.

Valdes, E.V., et al., 1985. Poult. Sci., 64: 213.

Van Barneveld, R.J., 1999. Aust. J. of Agri. Res., 50: 807.

Vanbelle, M., et al., 1990. Arch. Anim. Nutr. Berlin, 40: 543.

Vandeputte-Poma, J., 1980. J. Comp. Physiol., 135: 97.

Veltmann, J.R., et al., 1981. Poult. Sci., 60: 1748 (Abstr.)

Waibel, P., et al., 1992. Poult. Sci., 71: 1059.

Waibel, P.E., et al., 2000a. Poult. Sci., vol. 79.

Waibel, P.E., et al., 2000b. Poult. Sci., vol. 79.

Waldie, G.A., et al., 1991. Poult. Sci., vol. 70.

Waldroup et al., 1982. Feedstuffs, USA.

Waldroup, P., 1986. Part I and II. Nutrition Updates, 3:2 and 4:1,

Monsanto Company, Feed Ingredients Division.

Waldroup, P.W. and M.H. Adams, 1994. J. Appl. Poult. Res., 3: 209.

Waldroup, P.W., et al., 1993. Poult. Sci., 72: 816.

Waldroup, P.W., et al., 1998. Poult. Sci., 77: 702.

Wallach, J.D., 1974. E.P. Dutton, Co, New York NY, pp203.

Watkins, B.A., 1988. Brit. J. Nutr., 61: 99.

Weiser, H., et al., 1990. Proc. 2nd Symp. Kartause Ittingen, Switzerland.

Wessling-Resnick, M., 2000. Annu. Rev. Nutr., 20: 129.

West, J.R., 1984. North Carolina Agric. Ext. Serv., Deptt. of Poult. Sci., North Carolina State University, Raleigh.

Whitehead, C.C., 1984. Butterworths, London, England.

Whitehead, C.C., et al., 1985. Brit. Poult. Sci., 26: 73.

Whitehead, C.C., et al., 1995. Brit. Poult. Sci., 36: 113.

Whittle, E. and M. Araba, 1992. J. Appl. Poultry. Res., 1: 221.

Wicker, D.L. and D.R. Poole, 1991. Feed Manage., 42: 40.

Wilcox, R.A. and J.L. Balding, 1976. Bulletin C-555 Revised,

Kansas State Univ. Cooperative Ext. Service, Manhattan, KS

Wilson, K.J. and R.S. Beyer, 1997. Poult. Sci. Annual Meeting, Athens, GA.

Wilson, K.J., *et al.*, 1999. Southern Poult. Sci. Soc., Atlanta, GA. Wylie, P.W., *et al.*, 1972. Poult. Sci., 51: 1695.

Yaharjo, Y.C. and D.J. Farrell, 1984. Austr.J. Exp. Agri. and Anim. Husb., 24: 516-521.

Yalda, A.Y. and J.M. Forbes, 1996. Brit. Poult. Sci., 37: 797.

Yamazaki, M., et al., 1982. Bull. Nat. Inst. An. Husb., 38: 93.

Yoselewitz, I. and D. Balnave, 1989a. Brit. Poult. Sci., 30: 273.

Yoselewitz, I. and D. Balnave, 1989b. Brit. Poult. Sci., 30: 715.

Yoselewitz, I. and D. Balnave, 1989c. Austr. J. of Agri. Res., 40.

Yoselewitz, I., et al., 1988. Nutr. Rep. Intern., 38: 697.

Zamora A.F., *et al.*, 1988. Procd. of the 8th Intern. Conf. on Global Impacts of Appl. Microb. and Intern. Conf. on Appl. Biol. and Biotec., (Hong Kong), Chinese Univ. Press, pp497.

Zapata, L.F. and A.G. Gernat, 1995. Poult. Sci., 74: 1049.

Zatari, I. and M. Schiedler, 1990. Poult. Sci., 69: 198.

Zuprizal, M. L., et al., 1993. Poult. Sci., 72: 289.

Zuprizal, M.L. and A.M. Chagneau, 1992. Poult. Sci., 71: 1486.