CHAPTER

3

NUTRITION

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The feeding of companion birds has become a true art form with as many theories and practices suggested as there are those feeding birds. This feeding "art" has evolved out of necessity brought about by a lack of valid scientific information on the nutritional needs of these birds. Most current nutritional beliefs stem from years of "trial and error" feeding practices that are perceived as successful for the individual. A number of these practices have gradually been passed on, modified and eventually accepted as status quo by aviculturists. Their endorsement has come through the realization of certain improvements over previous feeding standards (such as the addition of fruits and vegetables to an all-seed diet), with the conclusion being that this small degree of improvement represents an end. These feeding practices may be deeply instilled in the bird enthusiasts' anthropomorphic views (ie, "humanizing" the pet and perceiving all of its needs through the eyes of the owner). There is often a belief that nothing can be too good for the bird, and it is provided with an incredible variety of often not-so-nutritious foods. Theory in companion bird nutrition has also been inundated with self-proclaimed experts, trying to achieve personal gain or recognition through their emphatic and frequently unsupported recommendations of certain feeding programs.

As aviculture has advanced over the past decade through the efforts of truly dedicated aviculturists, sound feeding practices that are based on the eating habits of long-lived birds or on sustained reproductive successes have begun to emerge. Although most of this information is still anecdotal, there appear to be valid principles to support many of these practices. Some of these dietary theories are based on what a particular species of bird is perceived to eat in the wild. Placing too much emphasis on this rationale can be deleterious. There is only a moderate understanding of what free-ranging birds eat, partly because their diets vary widely with the seasons. The majority of companion and aviary birds are considered opportunistic omnivores; that is, they will eat a large number of the foods that are available to them at any specific time. In most cases, this includes a wide array of vegetative material and a variety of animal products, as well as the consumption of soil and mineral deposits. Even a relatively accurate analysis of 90% of a bird's intake may not be truly reflective of the total nutrient profile of the diet,

because items consumed in trace amounts are difficult to quantitate and can have a significant impact on the bird's overall nutritional status. Additionally, most free-ranging birds do not live to their full genetic potential. This is due not only to predation and disease exposure, but also to the frequency of malnutrition caused by seasonally insufficient supplies of nutrient-adequate foods.

Nutritional Research Potential

The science of feeding companion birds has lagged behind that of most other pet species, due largely to historic perception that the diets available were not nutrient-deficient. The lack of financial incentives for either university or industry to employ nutritionists to study these species, and the expense and difficulty of studying nutrient requirements in a variety of species and metabolic conditions have further delayed avian nutritional research.

It has been only in the last decade that nominal research has begun on the nutritional needs of companion bird species, and it will take several decades to establish a partially accurate picture. Fortunately, however, general nutritional principles apply to nearly all vertebrates, with a few notable exceptions. Additionally, the most studied living species from a nutritional standpoint has been the domestic chicken. Although they are obviously not identical to each other in all ways, the domestic chicken does share similar physiologic parameters with popular companion Psittaciformes and Passeriformes. The greatest difference among these species is the fact that Galliformes are precocial (the neonate is mobile and generally self-sufficient at only a few hours of age). In addition, domestic poultry have been genetically selected and modified over the course of several hundred generations of domestication to thrive on commercially produced diets. Nonetheless, domestic poultry provide a starting point for the study of companion bird nutrition. It is at this point that the anecdotal nutritional information that pervades aviculture becomes of great significance. Subtle differences among the species have become obvious, as several species on a dietary regime will perform adequately, while another species on the same diet will do poorly. These observations suggest that species-specific nutritional requirements exist, but because many species of a genus or family perform similarly on a certain diet, it can be assumed that the variations in nutritional requirements are minor.

Poultry Adaptations

Current nutrient recommendations for companion birds are derived from an extrapolation of the nutritional requirements for commercial poultry, the application of general nutritional principles that are fairly constant among all vertebrates, an evaluation of ornithological information (eating habits in free-ranging birds, the role of ecological niches, any known anatomic or physiologic differences) and information that has been generated through the years of trial and error feeding, which has resulted in certain species-specific or family-specific feeding practices. The culmination of this multifaceted approach has resulted in a general estimation of the nutrient needs for companion birds that can be shown to be successful in growth studies and long-term feeding trials. It does not, however, determine the specific requirement of an individual nutrient or necessarily produce a diet that is totally optimized. It also fails to elucidate any species-specific problems, but rather attempts to compensate for them.

To optimize health, longevity and production of companion bird species, a great deal of nutritional research will be required. It is doubtful that the nutritional needs of either the Psittaciformes or Passeriformes, not to mention of a specific individual species, will ever be fully known. Even today, after almost a century of research in chickens and rats, the entire nutritional picture has not been completely elucidated for these species. There are substantial data on the nutrient requirements for the growing animal, but there are still many questions as to the requirements for optimal reproduction, optimal health and maximal longevity. The latter tends to be of little concern in any commercial species, but does have eminent importance for companion animals. The knowledge base of canine and feline pet nutrition is well over 50 years ahead of its companion bird counterpart. Although research in this area involves only two species and is strongly supported by universities and hundreds of competitive manufacturers, the science of canine and feline nutrition is still rather limited and is rapidly evolving.

Role of Nutrition in Bird Health

Nutrition itself is a critical link between the management practices provided for a bird and the bird's good health. Figure 3.1 illustrates a simple building block approach to the final goal of bird health. The foundation of the entire pyramid is the genetic background of the individual, which is largely responsible for the

FIG 3.1 Factors contributing to bird health.

nutrient needs. It can also predispose the individual to health problems and may even have implications for management techniques. Pet owners, nutritionists and veterinarians seldom have any impact on genetic background because it is predetermined at conception. The breeder, however, can impact this area through selection of breeding stock. Regrettably (especially in larger birds), the genetically poorer individual is frequently kept for breeding. These individuals may be physically, emotionally or behaviorally abnormal and are abandoned as companion birds and relegated to breeding. This is a counterproductive process, because the breeder may unknowingly be selecting for undesirable traits. The selection of certain unusual traits or the practice of heavy breeding within a very small gene pool will ultimately accentuate both desirable and undesirable characteristics represented in the original genotype. This has created considerable problems in budgerigars, cockatiels, canaries and finches.

Without good, sound management techniques (see Chapter 2), an otherwise genetically strong and nutritionally sound bird will not maintain its good health. Finally, a properly balanced diet and a professionally administered health care program must be provided to ensure the long-term health of a bird.

Just as providing complete, thorough veterinary care is impossible without proper training, so is the formulation of a properly balanced, complete diet. The formulation, development and production of a diet is surprisingly complex due to the large number of nutrient interactions, the differing bioavailabilities of nutrients from different ingredients and the difficulty of procuring and administering micronutrients into the diet. A well formulated, properly balanced diet represents a precise combination of over 40 nutrients, sometimes provided by just as many different ingredients.

Water

Although not a nutrient per se, water is essential to the body for cooling and for the maintenance of intracellular and extracellular fluids. It is the medium in which digestion and absorption take place, nutrients are transported to cells and metabolic waste products are removed.

The quality of water provided to companion birds should be of utmost concern to both the client and the veterinarian. Water and "soft foods" (foods containing high moisture content over 20%) are frequently implicated in exposures to high concentrations of bacteria. An open water container that becomes contaminated with fecal material or food will promote rapid bacterial proliferation. In water containing added vitamins, there can be a 100-fold increase in the bacterial count in 24 hours. Changing the water and rinsing the container will obviously decrease the bacterial load, but an active biofilm remains on the container walls unless it is disinfected or washed thoroughly. Contamination in the water container, in addition to the aqueous medium and compatible environmental temperatures, provide all the requirements for microorganisms to thrive. Likewise, high-moisture foods such as egg foods, nestling foods, cooked foods, sprouts, fruits and vegetables provide excellent growth media for microorganisms. At warm environmental temperatures, these types of foods can become contaminated in as little as four hours.

Water intake will be greatly influenced by the type of diet provided. Most birds can derive the majority of their water requirement from foodstuffs when the diet consists primarily of fruits, vegetables or moist foods. Processed diets tend to increase the bird's water intake over that typical for a seed diet because they generally are dry, lower in fat and tend to have overall higher nutrient levels. Slightly moister feces are often observed in birds on a formulated diet.

Nutrient Interrelationships

There exists a vast array of interrelationships between the different nutrients. Ideally, these must all be evaluated to protect against nutrient imbalances and interferences, and to ensure that the proper amounts of nutrients are being both consumed and absorbed by the bird. One of the most frequent misinterpretations is to judge the nutrient adequacy of a diet strictly on the total amount of a nutrient in the food. It is critical to go beyond this quantitative approach and evaluate both the quality of the nutrient and the animal's actual intake of the nutrient. By evaluating the intake level and the quality (bioavailability), the total body uptake can be determined. A simplistic example of nutrient intake miscalculation is the baby bird being hand-fed recommended volumes of a well balanced, high-nutrient diet that is prepared excessively dilute. In this situation, the nutrient uptake is insufficient to support growth.

The Effective Energy Content of Food

It is important that the individual nutrient levels be balanced with respect to the energy content of the food, because the food intake by the animal is largely dependent on the total caloric density of that food. In the case of very low caloric density foods, the gastrointestinal tract capacity can become a limiting factor for adequate caloric intake. Conversely, if the dietary caloric density is extremely high, the appropriate feedback systems that regulate satiety may not have time to respond before the caloric needs are exceeded, resulting in overconsumption.

Energy content of the diet, or specifically fat content, also has an influence on the rate of food passage through the system. As the fat content of a diet increases, the rate of passage is slowed. This not only has an effect on the bird by prolonging satiety, but also improves digestibility of most nutrients in the food by increasing the length of exposure to digestive enzymes and the time for absorption. This improved absorption, however, is generally not very dramatic. An example of this relationship is given in Table 3.1. This shows how some moderate increases in dietary protein and rather small increases in dietary calcium are required to balance the daily intake levels between a low and a very high fat diet. Although there is a substantial difference in the metabolizable energy values of these two diets, the daily intake of protein and calcium is identical with respect to the energy content of the diet. Consumption of 30 grams and 22.5 grams of diets A and B, respectively, both provide 90.5 kilocalories to the bird. This example illustrates how some seemingly dramatic differences in nutrient levels can actually give very similar results in the animal.

TABLE 3.1 The Effect of Dietary Energy Level on Intake and Proper Nutrient Density

	Diet A Approx. 4% Fat	**Diet B** Approx. 22% Fat
Energy Content, kcal/kg	3,015	4,020
Intake, grams	30.0	22.5
Energy Intake, kcal	90.5	90.5
Protein Content, %	15.0	20.0
Protein Intake, g	4.5	4.5
Calcium Content, %	0.5	0.7
Calcium Intake, g	0.15	0.15

Mineral Interrelationships

There are a vast number of different mineral interrelationships, with every mineral affected by at least one other. The most critical in companion bird nutrition, and in most species, is the relation between calcium and phosphorous. For proper growth, bone maintenance and health, a ratio of calcium to available phosphorous should be 1.5:1 to 2:1. In these proportions, both minerals are most effectively absorbed in the gastrointestinal tract as well as metabolized within the body. The widest tolerable range of calcium to phosphorous ratio should be considered to be 0.8:1 to a maximum of 3.0:1 (3.3:1 produces rickets and leg abnormalities) Additionally, excess levels of calcium can precipitate deficiencies of magnesium, iron, iodine, zinc and manganese if these are only marginally supplied.

Vitamin Interrelationships

The most obvious example of vitamin interrelationship is the effect of the absorption of fat-soluble vitamins, in which an excess of one would decrease the absorption of the others due to competition for binding sites in the intestinal mucosa. For this reason, it is necessary that all the fat-soluble vitamins be balanced with respect to one another to assure proper absorption of them all.

There is also an interrelationship in the metabolism of folic acid and choline (and the amino acid, me-

thionine) as they relate to the metabolism of single carbon units (ie, methyl groups). This metabolic role is also dependent on vitamin B_{12} as part of the enzyme system.

Vitamin and Mineral Interactions

Although there are many cases of interactions between vitamins and minerals, certainly the most significant metabolically is the relationship of calcium, phosphorus and vitamin D_3. It is obligatory for adequate vitamin D_3 to be available for the proper absorption of both of these minerals to take place. Inadequate vitamin D_3 levels in the body can cause calcium deficiency symptoms in an otherwise calcium-adequate diet. Conversely, excess levels of dietary vitamin D_3 can produce hypercalcification even in a diet normally considered to be marginally sufficient in calcium.

The other critical vitamin/mineral interaction is that between vitamin E and selenium, in which their biologic functions are essentially the same, but occur in different parts of the cell (lipid-based and aqueous, respectively). Even though they act in different parts of the cellular structure, a generous supply of one tends to spare a marginal supply of the other by quickly scavenging the additional free radicals that are produced (because of the lack of one nutrient) as they migrate throughout the cell structure, coming into contact with both the aqueous and lipid phases of the cell.

Another example of a mineral and vitamin interrelationship is the increased absorption of iron in the presence of ascorbic acid.

Amino Acid/Vitamin Interactions

In addition to several of the vitamins' direct roles in enzyme systems that are involved in protein synthesis and metabolism, there are also interactions between amino acids and vitamins that may have an effect on the absolute requirements of each other.

The most notable interrelationship between a vitamin and an amino acid is the relationship of niacin and tryptophan. In fact, a significant portion of the niacin requirement can be spared by an excess of tryptophan in the diet over what is required for necessary protein biosynthesis. This bioconversion is most efficient when levels of both niacin and tryptophan are low in the diet. The ultimate efficiency of this conversion is determined by the liver enzyme, picolinic acid carboxylase, which catalyzes the breakdown of the immediate precursor of niacin. This enzyme activity is species-dependent, affecting the animal's potential use of tryptophan to satisfy the niacin requirement. Additionally, several of the reactions in the bioconversion require riboflavin and pyridoxine-dependent enzyme systems. Protein, energy and hormonal status also play roles in this series of reactions.

Choline is an example of a vitamin that can directly spare the requirement of an amino acid, namely methionine. This occurs through its ability to act as a methyl donor in a fashion similar to methionine, thereby limiting the specific role that methionine would serve if an otherwise insufficient level of methyl donors existed.

Nutrient Antagonists (Anti-nutritional Factors)

There are a number of nutrient antagonists that can be present in foodstuffs. Many of these are natural compounds within the food, some of which can be tolerated in limited amounts. Others can be treated commercially to minimize their impact on the animal. Some compounds, most notably mycotoxins (the toxic metabolic byproducts of molds) can be produced when field or storage conditions are less than ideal.

Enzyme Inhibitors

Enzyme inhibitors are present in a large variety of foods, and most can be largely inactivated by thorough cooking. The largest group of enzyme inhibitors are the protease inhibitors, which inhibit the digestive enzymes trypsin and chymotrypsin and others. Fortunately, these inhibitors are thermosensitive and readily inactivated by cooking. Ingestion of a diet high in active inhibitors results in poor protein digestion and pancreatic hypertrophy, stimulated by the direct inactivation of digestive enzymes or the effect of limited bioavailability of methionine (decreasing the synthesis of digestive enzymes).[35] Protease inhibitors are present to some degree in all plants, with significant levels found in all of the legumes (mature beans), barley, beets, buckwheat, corn, lettuce, oats, peas, peanuts, potatoes, rice, rye, sweet potatoes, turnips and wheat. Potatoes are extremely high, with a large percentage (15%) of protein comprised of inhibitors.

Tannins, found in a variety of plant sources, can bind protein, inhibit digestive enzymes and reduce the bioavailability of iron and vitamin B_{12}. At high levels, they can cause liver and epithelium damage. These

polyphenolic compounds found in most plants are associated with an astringent taste and cause the normal browning on fruits and vegetables when they are cut or bruised. Tannins are found at high levels in acorns, carrots, rape seed, milo, grape seeds, tea, coffee, chocolate, bananas, grapes and raisins, lettuce, spinach, rhubarb and onions.

Some of the other enzyme inhibitors include amylase inhibitor in beans, wheat, rye and sorghum; plasmin inhibitor (inhibiting blood clotting) in some beans; kallikrein inhibitor in potato (decreases antibody formation); and cholinesterase inhibitors in asparagus, broccoli, carrots, cabbage, celery, radishes, pumpkin, raspberries, oranges, peppers, strawberries, tomatoes, turnips, apples, eggplant and especially potatoes.

Mineral Antagonists

Oxalate (oxalic acid) is an organic acid that efficiently binds calcium and other trace minerals, making them unavailable to the animal. The highest levels of oxalate is found in tea, spinach and rhubarb, with lower levels found in peas, beets and beet greens, lettuce, turnips, carrots and berries. Potentially toxic levels are found in the leaves of rhubarb and the common house plant, diffenbachia. High levels of oxalates can cause vomiting, diarrhea, poor blood clotting and convulsions. Lower levels can result in decreased growth, poor bone mineralization and kidney stones.

Phytate or phytic acid is a complex of phosphoric acid and sugar, and is very effective at chelating minerals such as zinc, iron and calcium, resulting in an unavailable complex. Phytates are most commonly found in nuts, legumes, cereal grains (germ and bran) and, in lesser quantities, in green beans, carrots, broccoli, potatoes, sweet potatoes and berries.

Vitamin Antagonists

Thiaminase is a naturally occurring enzyme that destroys thiamine. Thiaminase is most often associated with raw fish, but it can also be found in a number of fruits and vegetables such as beets, brussel sprouts, red cabbage and berries, some organ meats and as a product of certain microorganisms that can inhabit the gastrointestinal tract.

A compound found in flax seed (and therefore linseed meal) acts as an antagonist to pyridoxine (vitamin B_6). This compound apparently is an amino acid-type compound that forms an unavailable complex with pyridoxine.

Natural Plant Toxins

Although not nutrient antagonists as such, lectins or phytohemagglutinins can cause kidney, liver and heart damage, destruction of gastrointestinal epithelium, red blood cell agglutination and cell mitosis interference. These compounds occur in legumes, especially the castor bean and black bean, and in lower levels in other plant seeds.

When saponins are consumed in high amounts, diarrhea and vomiting can occur. They are found in soybeans, alfalfa, spinach, asparagus, broccoli, potatoes, apples and eggplant.

There are several foods that have goitrogenic properties that could cause symptoms mistaken for iodine deficiency, or that could exacerbate a marginally iodine-deficient diet. Goitrogens are contained in soybean, peanuts, pine nuts and the entire brassica family (turnips, rutabaga, broccoli, brussel sprouts, cabbage, cauliflower, kale, kolrabbi and mustard). They are also found to a lesser degree in carrots, peaches, pears, radishes, strawberries and millet. Low-protein diets increase the effects of goitrogens (anti-thyroid effects).

Other natural toxins or nutrient antagonists present in foods include gossypol, cyanogenic glycosides, photosensitizers and a variety of alkaloids and phenolic compounds. The significance of these compounds in most species is questionable. Many have shown beneficial effects in the body when provided at low amounts, but at higher amounts they may be toxic or carcinogenic. Low exposure to these items should never be considered to be dangerous, but the inclusion of any of them at high amounts in the diet, particularly in the raw form, should be avoided.

Mycotoxins

Mycotoxins are compounds that are produced under certain conditions as metabolic by-products of molds. There have been nearly 100 mycotoxins identified since their initial recognition in the 1960's. They possess varying degrees of toxicity, some of which are carcinogenic. Mycotoxins are not associated with all molds, nor are they always produced by mycotoxin-producing species. The difficulty with mycotoxins is that they are totally undetectable by sight, smell and taste. Any product that is known to be moldy should not be fed due to the possibility of mycotoxins, as well as nutrient degradation and decreased palatability. Toxins, sources and pathology are shown in Table 3.2.

TABLE 3.2 Sources of Exposure and Pathology Related to Mycotoxin Ingestion

Mycotoxins	Common Feed Sources	Agent	Pathology
Aflatoxins	Corn, peanuts, cottonseed	Aspergillus flavus A. parasiticus	Liver damage Hepatomegaly Immunosuppression Kidney damage
Ochratoxin	Corn, barley, oats, wheat	A. ochraceus Penicilliumviridicatum	Kidney and liver damage Hemorrhaging
Zearalenone	Corn, wheat	Fusarium roseum F. graminearum	Production of estrogen-like compounds
Trichothecenes (T_2 toxin)	Corn, wheat, barley, oats, forages	F. tricinctum F. roseum F. graminearum	Oral inflammation and lesions Neural disturbances Immunosuppression Hemorrhaging
Vomatoxin (2-deoxynivalenol)	Corn, sorghum, wheat	F. roseum F. graminearum	Gastrointestinal inflammation Vomiting
Ergot	Rye, barley, wheat, oats	Claviceps purpurea	Tissue death Kidney and liver damage

Mycotoxins can have a broad range of effects on the body ranging from a toxic dose with mortality in two to three days to chronic exposure of moderate levels where decreased disease resistance is encountered along with lesions in the liver, kidneys, nervous system, reproductive system and integument. Carcinogenic, mutagenic or teratogenic effects may also be exhibited. The type of effect and response is related to the exposure level and duration.

Mycotoxins are some of the most carcinogenic compounds known, with chronic exposure of levels in parts-per-billion causing cellular transformation. Species differ considerably as to their susceptibility, with ducklings being among the most susceptible. The LD_{50} ranges from 0.5 mg/kg (duckling) to 60 mg/kg (mouse).[13]

Aflatoxin levels in food must be controlled by good harvesting, handling and storage procedures. Peanuts and corn are considered to be the human population's largest source of aflatoxin. The United States Food and Drug Administration does not allow any peanuts to be used in human food products with levels greater than 20 parts per billion of aflatoxin. This is also the level used by the livestock industry as the safe, allowable level in grain products. Certain crops, depending on the climatic conditions during the growing season and at harvest (peanuts, hay, corn, wheat), may be considered the most common sources of aflatoxin.

Mycotoxin contamination usually occurs when fungus is able to penetrate a seed hull or protective coating and reach the kernel. Because molds are ubiquitous, spores will always be present on untreated crops. Plant damage such as drought, stress and insect damage will increase the incidence of mold penetration into the seed and the possibility of mycotoxin production. After inoculation, warm and humid conditions help promote the mold growth and toxin development. Unfortunately, mycotoxins are very stable to heat and typical processing methods.

Some of the mycotoxins (trichothecene or T_2 toxin) are among the most potent protein synthesis inhibitors known. It has also been found that T_2 toxin reduces the plasma level of vitamin E by affecting micelle formation in the gastrointestinal tract.[11] Similarly, aflatoxin increases the dietary requirement for vitamin D_3 and lowers the vitamin A stores in the liver. In addition, many of the mycotoxins, particularly aflatoxin, the trichothecenes (T_2 toxin) and ochratoxin, have metabolic effects in the body that impair the defense mechanisms.[32,33]

Methods for Determining Nutrient Requirements

Growth Requirements

There are a number of approaches for determining the requirement of a specific nutrient in a bird. The simplest and probably most effective way is to examine the nutrient's influence on growth. Diets that are identical in all aspects, except the experimental nutrient, are provided to groups of experimental birds. By feeding specific diets (each of which contains an incrementally larger level of the test nutrient), growth and other parameters are measured. The point at which no further statistically significant increase in growth is observed would be considered to be the requirement of that particular nutrient in that particular diet, under those specific experimental conditions. If this result is consistently reproducible, it can be considered valid. This method is relatively accurate, and a single study can be performed rather quickly. This experimental design is most often used to evaluate nutrient requirements for growth, but it

may also be used to evaluate a nutrient's influence on egg production, antibody production and bone strength.

Because growth is the period in which most nutrients are required at their highest levels, this type of study can establish the upper end of the suggested nutrient range. The use of these levels for adults would certainly provide a level far greater than the true metabolic need but, in most cases, these would still be within the safe range. The determination of the requirements for adults is very difficult, complex and in many cases impractical. Additionally, the differing requirements for each separate strain within a species is often different. Because of this, the accepted practice in humans has been to establish a minimum daily allowance, which is designed to meet or exceed the estimated requirement of 97.5% of the entire population, or approximately two standard deviations above the mean. This approach compensates for the great degree of biological variability within the entire species, as well as bioavailability in foods, variability of absorption efficiency, health status, environmental conditions and genetic background. *(Editor's note: The correlation of growth rate and health has not been established for companion birds.)*

Evaluating Nutrient Status

The nutrient status of an individual is most easily accessed by carefully evaluating the adequacy of the diet provided. Considering the current feeding practices of many bird owners, it is likely that basic deficiencies can be discovered with very little effort. If simple dietary evaluation is not possible, or seems inconclusive, further testing is possible (however, somewhat difficult and inconclusive). The only practical method for further testing is through serum or plasma samples. These samples are ideally taken after a fast to reduce the presence of nutrients that were recently absorbed from a meal. Additionally, the circulating levels of many nutrients are tightly controlled, and, therefore, only show levels outside the normal range when body stores are severely depleted or exceeded. The matter is further complicated by the lack of reliable normal ranges (or in some cases, no information at all) and the high cost of certain nutrient assays. Many laboratories, however, are equipped to run plasma retinal or carotene levels (for vitamin A), plasma alkaline phosphatase (an indicator of vitamin D status), prothrombin time or clotting time (indicator of vitamin K status), serum calcium, phosphorous, electrolytes, trace minerals (although they may inaccurately reflect status) and parameters for the evaluation of lipids and proteins.

Estimation of Nutrient Requirements

There is a severe need to set dietary guidelines to serve as a reference point that can be used as a standard for testing. Safe guidelines are needed to help aviculturists and companion bird owners who choose to feed a widely varied diet, to guide the commercial food manufacturers in producing diets that can assure longevity and good health, and to help veterinarians assess a patient's diet and educate the client in proper feeding methods. Because of the extreme difficulty in accurately determining the requirement of all nutrients, even for a single species, documented studies and specific requirements will not be available for decades, if ever. It is therefore necessary to derive these nutrient recommendations from other species that are better understood. Extrapolation from known species, if done wisely, can provide a reasonable starting point from which to base diets and efficacy studies. With subsequent evaluation of this derived nutrient profile and long-term monitoring to assess overall nutrient status, recommendations or allowances can be generated for a particular genus, species or strain that may be unique with regard to dietary requirement, digestive efficiency or other physiologic differences. This methodology does not look at "minimums" but rather at nutrient levels that would attempt to optimize all experimental parameters by providing more generous nutrient allowances. Table 3.3 lists the possible minimum requirements (an extrapolation from poultry species) and the dietary recommendations for companion bird species.[7]

Nutrient Needs During Different Life Stages

Embryonic

An egg produced by a hen fed a nutrient-adequate diet is normally a rich source of the essential amino acids, energy, linoleic acid and all of the required vitamins and minerals for normal cell division, growth and maturation. If a hen is fed a nutrient-deficient diet that will allow production, embryo development may progress, but will be abnormally affected. This most often is observed as early embryonic death, usually with the formation of a blood ring after approximately three days of development (vitamin A deficiency), losses immediately prior to hatch due to an embryo with insufficient strength to complete the hatching process (riboflavin, biotin, folic acid and vitamin B_{12} deficiencies) or embryonic malformation (zinc and manganese deficiencies).

TABLE 3.3 Recommended Nutrient Allowances for Companion Bird Diets[7,29]

These allowances can be used as general dietary guidelines for most psittacines and the commonly kept passerines. Species differences do occur, but have not been listed due to insufficient research. The anticipated minimum requirement (as extrapolated from other species) is included for comparison. These values do not compensate for nutrient bioavailability, genetic variability and other conditions.

Nutrient	Anticipated Minimum Requirement	Recommended Allowance for Maintenance[1]
Protein, %	10.00	12.00*
Fat, %	–	4.00*
Energy, kcal/kg	–	3000.00
VITAMINS		
Vitamin A, IU/kg	2500.00	5000.00*
Vitamin D_3, IU/kg	500.00	1000.00*
Vitamin E, IU/kg	15.00	20.00*
Vitamin K, ppm	0.80	1.00
Thiamine, ppm	2.00	5.00
Riboflavin, ppm	4.00	10.00
Niacin, ppm	40.00	75.00
Pyridoxine, ppm	4.00	10.00
Pantothenic acid, ppm	12.00	15.00
Biotin, ppm	0.15	0.20
Folic acid, ppm	1.00	2.00
Vitamin B_{12}, ppb	5.00	10.00
Choline, ppm	750.00	1000.00*
Vitamin C	No requirements demonstrated*	
MINERALS		
Calcium, %	0.30	0.50*
Phosphorus (available), %	0.15	0.25*
Phosphorus (total) approx., %	0.30	0.40*
Sodium, %	0.10	0.15
Chlorine, %	0.10	0.15
Potassium, %	0.30	0.40
Magnesium, ppm	500.00	600.00
Manganese, ppm	60.00	75.00
Iron, ppm	60.00	80.00
Zinc, ppm	40.00	50.00
Copper, ppm	6.00	8.00
Iodine, ppm	0.30	0.30
Selenium, ppm	0.10	0.10
AMINO ACIDS		
Lysine, %	0.45	0.60
Methionine, %	0.20	0.25
Tryptophan, %	0.10	0.12
Arginine, %	0.50	0.60
Threonine, %	0.35	0.40
Other essential amino acids are sufficient in common diets.		

1. The recommended allowances will support normal maintenance of companion birds and have been demonstrated to be adequate during long-term feeding. These levels, however, may not be sufficient for optimized health under varying conditions and will not be adequate for breeding and growth, which may require higher levels of certain nutrients.
* Increased levels are suggested for growth/breeding diets due primarily to high requirements for adequate chick growth as opposed to increased demands for low-level breeding.

Growth

Shortly before hatch, the embryo absorbs the remaining portion of the yolk sac into its abdominal cavity. At hatch, the absorbed yolk sac serves as a temporary energy reservoir. This may be adequate to supply the chick with nutrients for the first one to three days, depending on the species. As the chick's digestive system becomes fully functional, the period of rapid growth begins. Due to the high metabolic rate and the rapid division and growth of cells, the amino acid, energy, linoleic acid, vitamin and mineral requirements are at the highest point of the animal's normal life. Furthermore, if brooding temperatures are not sufficient, there is a further increase in the energy demand to maintain adequate body temperature. The requirement for amino acids are further increased during the period of feather development. These feathers, which are comprised of more than 90% protein (on a dry matter basis), can approach up to 10% of the total body weight in the young bird.

Under normal situations, the absolute nutrient requirements decrease throughout the growth phase, since the level of growth proportional to body weight declines with age. If optimal nutrient levels are not present at an earlier growth phase, but are present in excess of requirement towards the end of the growing cycle, the bird will display compensatory growth (compensating for an earlier lack of normal growth). Compensatory growth is characterized by both the flattening and extension of the normal growth curve, with the end result of a chick that reaches normal adult weight, but requires a longer time to do so. This is often observed when a baby is fed a nutritionally marginal diet (see Chapter 30). As the chick advances through the growth period, at some point the once marginal diet becomes adequate and eventually may even provide a generous proportion of nutrients relative to the requirement at that time. The compensatory growth phase is generally marked by a temporary increase in feed efficiency and rate of gain when compared to normal chicks of the same age.

Maintenance

Requirements for the maintenance of an adult bird are the lowest for the entire life cycle. The bird's greatest need at this time is to provide adequate energy to maintain body temperature, metabolic functions and the appropriate activity level. Protein requirement is minimized, because the primary need is for the replacement of dead cells or of amino acids used in various metabolic systems (ie, enzymes). Similarly, the need for vitamins and minerals is to replace those that were lost through metabolic proc-

esses. In nearly all cases, these needs are considerably lower than for the growth period (or any other stage of production) due to the lower rate of cell formation and overall metabolic rate. Any increase in activity level, ambient temperature outside of the thermoneutral zone, molting and the exposure to any type of stress will alter the minimum nutrient levels required for maintenance.

Breeding

The increased requirements by the hen for breeding can be divided into two general categories: those required for egg production and those required for maximum hatchability of the embryo. On a dry matter basis, the egg (without the shell) consists of approximately 45% fat and 50% protein. Additionally, the shell, which comprises approximately 10% of the total egg weight, is approximately 94% calcium carbonate (38% calcium). These three constituents represent the largest increase in nutrient needs in order for the hen to produce eggs. Because birds generally eat to meet their energy demands, increasing the energy content of the diet is not generally necessary. The diet does, however, require higher levels of protein, particularly of the sulfur amino acids (eg, methionine) and lysine. Calcium levels in the diet should be increased to minimize the decalcification of the bone and to prevent the formation of soft egg shells. Other nutrients that improve egg production (in poultry) when present at levels higher than the minimum maintenance requirement are vitamins A, B_{12}, riboflavin and zinc. Vitamin D_3 levels slightly over the requirement will tend to improve egg shell characteristics, with larger amounts having no additional benefits.[19] To maximize hatchability of the embryo, increased levels of vitamin E, riboflavin, pantothenic acid, biotin, folic acid, pyridoxine, zinc, iron, copper and manganese are required over what is adequate for egg production.

Much of the reason for dramatically increasing the nutritional plane of a breeding bird's diet is to provide adequate dietary components for the chick to be fed. Psittacine and passerine birds are relatively low egg producers and their increased demand for nutrients required for egg production is transient. With adequate body stores through proper daily feeding, a diet designed specifically for egg production is not necessary (such as a diet that will meet the immediate need for calcium during the days of production). Instead, a moderately high plane of nutrition that will optimize body stores, allow ready repletion of depleted stores and provide adequate nutrition for chick growth is probably the simplest and safest means of dietary management. This will allow for adequate chick growth and satisfactory levels of all nutrients for egg production. Calcium can be quickly repleted without the risk of over-supplementing by providing an "egg production" diet during the breeding season. Feeding for optimal chick growth not only decreases the duration in the nest of parent-raised chicks, but also promotes rapid recycling of the hen (repletion of body stores and physiologic preparation for returning to nest).

Geriatric Nutrition

To date, there has been no research on the nutritional needs of geriatric psittacine birds. This is due largely to the relative scarcity of geriatric birds in aviculture or as companion animals. Because of the historically poor diets offered to these birds and their subsequent shortened life-span, the mean population age of companion birds is low with respect to the potential. As the husbandry and veterinary care of these species continue to improve, proper geriatric nutrition will become a concern. Based primarily on geriatric research (in humans, rats, dogs and cats), it can be assumed that the geriatric bird should be provided with a highly digestible diet that maintains proper weight while providing slightly reduced levels of proteins, phosphorous and sodium, and levels of other vitamins and minerals similar to those received earlier in life. Slight increases in vitamins A, E, B_{12}, thiamine, pyridoxine, zinc, linoleic acid and lysine may be helpful to overcome some of the metabolic and digestive changes accompanying old age.

Stress

Companion and aviary birds are possibly subjected to more stresses than any other animals maintained in captivity. Stresses are both psychological and physical. Whether the bird is imported from the wild or is one of the most "domesticated" species, captivity alters its innate behaviors. The caretaker is often viewed as a threat, and the natural social interactions (flocking, mate selection) are inhibited. Crowding, handling, exposure to unusual pathogens, unsanitary conditions and malnutrition may all be considered stress factors. Stresses tend to be cumulative, and a single stress often has very little clinical effect on the bird. However, when one or more additional stress is applied, the bird may be weakened to the point of clinical illness or death. Stress in young birds results in a decrease in weight gain and, if left uncorrected, weight loss and morbidity may occur.

The body's response to stress is the "flight or fight" syndrome, and the immediate response is to mobilize

and produce glucose for the increased energy need. After carbohydrate stores are depleted (within approximately 24 hours), protein and fat stores are broken down, with the breakdown of skeletal muscle supplying amino acids for gluconeogenesis. The changes in metabolism also affect the normal metabolism or levels of vitamin A, C, calcium, zinc, iron, copper and magnesium. Attempts to restore these nutrients through special dietary modifications are probably futile. Instead, adequate diets should be provided to ensure the normal presence of sufficient body stores, which will also allow for satisfactory repletion of stress-depleted stores.[44]

Disease

There has been very little research done on the specific effects of diseases on the requirement and metabolism of each nutrient, and how these might affect the total requirement of individual birds. As the body enters the disease state, it rapidly begins to conserve nutrients in order to maintain needed functions. The most critical nutrient for the body to maintain during illness is water (see Chapter 15).

Secondly, the necessary energy supplies to the body must be maintained. Because of the increased metabolic rate during illness, there is a higher energy need. In humans, it has been found that the basal energy requirement will be exceeded by 50-120%, depending on the severity of the stress response. Although much of this energy demand still falls within the normal maintenance requirement, it is critical to maintain or exceed the typical energy intake, which can be provided via carbohydrates, fats or protein.

Dietary protein is the third most critical component to be provided to the debilitated patient. With the increased metabolic rate, there is a subsequent increase in body protein turnover, much of which is recycled by the body and not lost. Because this degradation and resynthesis is not completely efficient, an increase in metabolic rate results in an increased amino acid requirement. There is also increased demand for amino acids because of the need for additional immune components and tissue repair. Without adequate amino acid intake, labile protein stores (plasma, liver, muscle) are degraded for the process of gluconeogenesis. There may also be a decreased efficiency in the utilization of proteins, thereby further increasing the needs and importance of an adequate protein diet. The exceptions to increasing the protein in the diet are during the acute phase of liver or renal disease.

TABLE 3.4 Changes in Need for Nutrients During Periods of Debilitation

Vitamin C	The debilitated animal may not be able to adequately synthesize enough vitamin C, especially in the case of hepatic damage. Increased vitamin C in other species exposed to a number of different types of stresses has shown to improve production and health criteria.
Vitamin D	In diseases affecting the liver and kidneys, the enzymes required to produce the metabolically active form of vitamin D_3 will be impaired. In these situations, or in the case of a marginally deficient animal, it may be beneficial to provide vitamin D_3 therapy.
Vitamin K	For animals that have undergone extensive antibiotic therapy and are being maintained on an unsupplemented or marginally supplemented diet, it may be necessary to provide vitamin K because of its decreased synthesis by normal intestinal flora.
Vitamin B complex	In the case of an anorectic animal, it may be beneficial to supply additional B vitamins, especially thiamine. Other water-soluble vitamins such as riboflavin, pyridoxine and folic acid are particularly important in protein and energy metabolism; therefore, these vitamins have increased importance in the disease state.
Zinc	In a nutritionally compromised animal, zinc will improve healing and is an important component in protein synthesis; therefore, zinc is necessary for the maintenance of the immune system and phagocytic activity.

There is a lack of consistent studies in the literature indicating increased vitamin or mineral requirements in the debilitated animal. Supplying nutrients at recommended levels is probably sufficient in most cases; an increase in certain vitamins and minerals may be prudent, however (Table 3.4).

Current Nutritional Knowledge

Protein Needs

There have been few scientific studies conducted to investigate the nutritional needs of companion and aviary birds. Most of the beliefs on nutrition stem from observations in clinical and avicultural settings. Two of the best scientifically conducted studies that have been published investigated the total protein requirement and lysine requirement of the growing cockatiel. Chicks performed best and reached the

weaning stage earliest on a 20% crude protein diet. Those fed a 10% or 15% crude protein diet grew considerably slower, with stunting and slightly increased mortality occurring in the group fed 10% protein. On 5% crude protein diets, chicks were severely stunted, with subsequent mortality. Those fed a 25% crude protein diet performed similarly to the 20% group, but developed behavioral problems marked by meal refusal and increased aggressiveness. Those provided with a 35% protein diet displayed slight growth depression and further increased signs of aggression.[21,40,41]

Lysine Needs

The requirements for lysine were estimated by providing purified diets that were equal in all respects except lysine levels. In two experimental trials, diets supplying 0.1, 0.4, 1.0 or 2.0% lysine and 0.2, 0.4, 0.6, 0.8 or 1.2% lysine were provided. Cockatiel chicks showed the best growth responses when given diets in the range of 0.8 to 1.2% lysine. At lower levels, growth was proportionately depressed, displaying a typical nutrient-to-growth-response curve. The two lowest levels of dietary lysine resulted in little growth and high mortality. Performance on the 2% lysine diet was slightly poorer than the 1% diet, most likely due to the creation of a marginal amino acid imbalance at the higher level. Unlike poultry species, which exhibit feather depigmentation (the formation of feathers lacking melanin pigment) during a lysine deficiency, all cockatiels, even those on the most severely deficient diet, had normal feather pigmentation. This suggests a metabolic difference between poultry and altricial birds (at least the cockatiel).[38,39]

Energy

Energy requirements have been estimated in a variety of companion bird species. The approximate daily metabolizable energy (ME) needs for budgerigars appear to be between 12 and 16 kilocalories (kcal) per day in a normal maintenance situation.[7,17,47] Canaries require approximately 12 kcal/day[7,24] if maintained at 70°F. A 350 g Amazon parrot would require an intake of 100 kcal/day, and a 1000 g macaw would require 220 kcal/day. Temperatures above or below 70°F would result in lower or higher needs, respectively.[9]

Current Beliefs on Nutrient Requirements

Based on avicultural and clinical observations, there have been a number of hypotheses developed regarding species-specific differences in nutrient requirements. These have not been scientifically tested, but many have been substantiated by repeated reports in a variety of situations. It is difficult, however, to distinguish between the actual increased requirement of a nutrient in a specific species and species-specific differences in the manifestation of clinical deficiency signs. That is, on a marginal diet, one species may not display overt deficiency signs, while another on the same diet (same nutrient intake) could possible show distinct clinical changes. In a clinical situation, the overall adequacy of the diet should be evaluated before additional supplementation is suggested for the species (Table 3.5).

Vitamin Differences

It has been suggested that several species may have increased needs for vitamin A over most other commonly kept species. Those most frequently seen to respond to "higher" levels are Eclectus Parrots, conures[20] and certain Amazon parrots, most notably the Blue-fronted Amazon. The increased need for vitamin A in Amazon species is often linked to increased immunity against viral disease (poxvirus). This could well be an example of the variation in needs to maximize specific metabolic functions. Generally, the amount of a nutrient required to maximize a production parameter (such as growth or egg production) is often not sufficient to maximize immune response or other parameters. Limited research on vitamin A requirements indicates a need of 7,000 IU/kg feed in budgerigars. Clinically, a level equivalent to 5000 to 10,000 IU per kg in the diet has proven successful in preventing deficiency symptoms.[15]

Certain neonatal macaw species, especially the Blue and Gold Macaw and Hyacinth Macaw, seem more prone to the development of hypervitaminosis D_3 than other psittacine chicks. When a cross section of large psittacine babies was fed a moderately high level of vitamin D_3 (2500 International Chick Units [ICU]/kg *dry mix*; 1.0% Ca), Blue and Gold Macaws were the only species to develop mild signs of hypervitaminosis D_3, characterized by enlarged kidneys and mild, early calcification of the renal tubules.[5] Similar findings have been reported on a hand-feeding diet containing between 1000 and 4000 ICU/kg (the range due to the variable addition of vitamin supplementation), which resulted in crop stasis, increased serum uric acid levels and the presence of articular gout and regurgitation after feeding. Radiographically, the kidneys were found to be enlarged, with areas of calcification in the kidneys and proventriculus. Subsequent necropsy showed widespread soft tissue calcinosis.[46] In both reports, other species fed similarly on the same diets were not affected.

TABLE 3.5 Potential Toxic Effects of Nutrients

VITAMIN A
(20-100 times required)[43]
- Weight loss
- Decreased food intake
- Swelling/crusting eyelids
- Inflammation of mouth
- Inflammation of nares
- Decreased bone strength
- Dermatitis
- Hepatopathy
- Hemorrhaging

VITAMIN D
(4-10 times required)
- Increased calcium absorption
- Increased bone resorption
- Hypercalcemia
- Decreased PTH
- Mineralization of soft tissues
- Nephrocalcinosis
- Polyuria

VITAMIN E
(100 times required)
- Decreased growth
- Anemia
- Increased prothrombin time
- Decreased bone mineralization
- Decreased liver storage of vitamin A

VITAMIN K (Menadione)
(1000 times required)
- High mortality
- Anemia
- Hyperbilirubinemia
- Toxicity unlikely
- Thiamine (rapidly excreted by kidneys)
- Riboflavin (rapidy excreted by kidneys)
- Pantothenic acid
- Folic acid
- Cyanocobalamin
- Vitamin C

NIACIN
(10 times required)
- Flushing - vasodilation
- Pruritus
- Gastroenteritis

PYRIDOXINE
(50 times required)
- Decreased egg production
- Infertility

CHOLINE CHLORIDE
(2 times required)
- Increased mortality
- Decreased use of vitamin B_6

CALCIUM
(2.5% of diet)[45,48]
- Nephrosis
- Visceral gout
- Renal gout
- Hypercalcemia
- Hypophosphotemia
- Decreased food intake

MAGNESIUM
(20 times required)
- Decreased growth
- Decreased egg production
- Decreased egg quality

MANGANESE
(20-50 times required)
- Iron deficiencies

SELENIUM
(50 times required)
- Decreased weight gain
- Weight loss

(100 times required)
- Decreased egg weight
- Decreased hatchability
- Dermatitis

(Severe excesses)
- Blind staggers
- Pulmonary congestion/edema
- Liver cirrhosis

COPPER
(50 times required)
- Decreased growth
- Hepatopathy
- Accumulates in liver
- Death

ZINC
(10-20 times required)
- Gastroenteritis
- Decreased food intake
- Anemia
- Decreased bone mineralization
- Depression

See text for toxic effects associated with excesses of phosphorus, potassium, sodium, chloride, iron and iodine.

High levels of vitamin D_3 frequently result in the occurrence of gout.[18]

It has also been suggested that conures have a higher requirement for vitamin K, due to the bleeding disorder often seen in this species. This theory may not be valid because incidence of the syndrome has not been reported on a nutritionally adequate diet. This bleeding syndrome has also been alleviated by calcium supplementation and a generally improved diet, empirically verifying the importance of calcium status for blood clotting and suggesting that the syndrome is not dependent on vitamin K alone.[37]

Minerals

Cockatiels have been noted to be particularly sensitive to high calcium or high calcium and vitamin D_3 levels in the diet. Adult diets containing over 1% calcium, particularly when accompanied by generous levels of vitamin D_3 (over 2000 ICU/kg dry diet) have been found to be excessive in long-term feeding studies.[2] Normal egg production criteria have been satisfied at dietary calcium levels as low as 0.3 and 0.35%.[6,41]

Research in adult poultry has indicated that normal bone mineralization, plasma calcium and alkaline phosphatase levels can be maintained at below 0.05% calcium in the diet.[28,42] This is supported by a similar observation in cockatiels,[41] and is consistent with dietary levels of unsupplemented seeds, which have sustained birds for decades, although poorly. Levels for optimal health would seem to be considerably higher.

Energy

Large macaws, particularly the Hyacinth, appear to perform better on a higher fat diet than other species. This does not seem surprising considering the predominance of oil-based foods in the native diet of some of these species. An increase of approximately 25% fats over that adequate for other species has been found to be necessary to support maximum growth.[8]

A number of species are more prone to obesity than others. This can be a result of lower metabolic needs (ie, more energy efficient), better energy absorption, lower energy expenditures (ie, more sedentary in nature) or poor satiety biofeedback to the hypothalamus (overeating). Rose-breasted Cockatoos (galahs) and budgerigars are very prone to obesity and are probably examples of birds with slightly lower energy requirements. Amazon parrots frequently become obese due to their sedentary behaviors. In all birds, the likelihood of becoming overweight is increased as the bird ages and its metabolic rate decreases. Reducing the caloric density of the diet or limiting intake (by reducing food quantity or feeding duration) and

encouraging additional activities are essential in these cases.

Differences in Nutrient Metabolism and Requirements Based on Evolutionary Diversion

There is no generic companion bird with respect to nutritional requirements. It is highly likely that there will be distinct species' differences verified as the base of nutritional knowledge of companion birds increases. Based on the ecological diversity in which species have evolved, differences can be expected. For instance, budgerigars, cockatiels and a number of the grass parakeets and finches range into the vast, arid interior of Australia. These birds are expected to have developed biological adaptations allowing them to conserve both nutrients and water for existence in this sparse habitat. The sensitivity of cockatiels to calcium and vitamin D_3 levels that apparently have no negative impacts on other psittacines may be an example of such an adaptation. In the wild, these birds exist on a diet composed primarily of seeds,[50] which tend to be only a moderate source of many nutrients. Conversely, psittacines of the neotropics tend to consume a wide variety of foodstuffs, including an abundance of fresh vegetative matter, providing a less seasonally dependent, higher plane of nutrition. Birds in this environment have not had the need to develop any nutrient-conserving mechanisms, and may, therefore, have somewhat higher needs.

Nutritional Labeling of Commercial Products

Commercial labeling is frequently misunderstood, particularly with respect to the guaranteed analysis. All pet foods are required by law to list levels of crude protein, crude fat, crude fiber and moisture. These are not precise numbers, but rather guarantees of either the minimum or maximum amounts contained in the product. Protein and fat are listed as minimums, because they are of specific nutritional value and are among the most expensive components of food. The food should not contain less than the guaranteed level, but may contain any amount in excess of this minimum. In a processed food, these levels are generally close to the guarantee because of the significant added cost in oversupplying these nutrients.

The protein guarantee is analytically quantitative, being determined from the amount of nitrogen in the product (usually calculated as % crude protein = % nitrogen x 6.25). It provides no estimation of protein quality (ie, the product's amino acid profile). Indeed, non-protein nitrogenous sources will be reflected in the crude protein value.

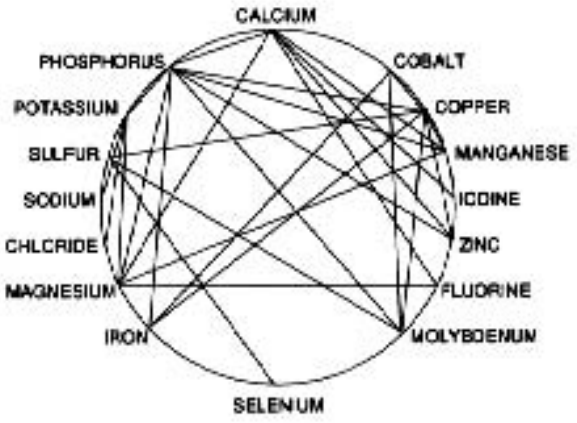

FIG 3.2 A well formulated, properly balanced diet represents a precise combination of over 40 nutrients, sometimes provided by just as many different ingredients.

Fiber and moisture are required to be listed as maximum amounts in the product, because both are traditionally considered of little nutritional importance and can, at higher levels, create quality problems. Manufacturers will often list the highest possible value that might occur in order to avoid violations, unless of course there is a negative consumer impression associated with the high value, in which case the manufacturer will guarantee a value with a narrower margin of safety.

Other nutrient guarantees are optional, except when the product specifically states that it is supplemented with certain nutrients (or category of nutrients), in which case those nutrients must be guaranteed. This law exists to ensure that all nutrient supplements are adequately labeled and the consumer is protected and informed about the product that they are buying. In the case of products that claim "vitamin-enriched" or other similar statements, those nutrients must be guaranteed so that a knowledgeable consumer can differentiate among the class of products (eg, diet, supplement, concentrate). In general, regulatory officials discourage the guaranteeing of vitamins, minerals and amino acids due the difficulty of ensuring compliance. Unless a product expiration date is listed on the package, the guarantees are stated for the life of the product. Due to normal loss of vitamin potency, a product that is not sold quickly may eventually fall

below the levels guaranteed. These nutrients often are expensive to analyze; therefore, regulatory officials are limited in the extent to which they can verify product compliance.

Complete nutrient listings may encourage the purchase of a product with unnecessarily high nutrient levels. Because of the typical philosophy that "more is better," number comparison between products will often result in the decision to purchase the product with the highest level. This may be particularly dangerous, especially in light of the relative lack of information regarding companion bird diets. Because of the lack of reference values, incorrect decisions can be expected to be the norm and should not be encouraged by listing numbers that may be relatively "meaningless."

Ingredient Statement

Companies are required to list the ingredients contained in the food in their order of dominance, (eg, in order from highest to lowest concentrations). This order is very difficult to police and is essentially left to the manufacturer to, in good faith, provide accurate information to the consumer. While still maintaining accuracy in labeling, manufacturers may opt for labeling techniques that become vague or "hide" ingredients that have poor consumer perception. Instead of listing each ingredient by its full, approved term, "collective" terms can be used to group similar products together under an umbrella term. Therefore, the collective term "grain products," can be used to describe the product's total content of cereal grains (corn, wheat, oats, barley), regardless of its form (whole, ground, heat processed). Likewise, the term "animal protein products" can be used to reflect a wide variety of ingredients such as meat meal, blood meal, dried milk, hydrolyzed feathers or fish residue. If the manufacturer chooses this method of terminology, all major ingredients must be listed in the collective manner. This gives manufacturers the opportunity to make major changes within general ingredient categories in order to take advantage of least-cost formulation.

Multiple ingredients serving the same functional purpose are sometimes used so that each ingredient can be listed in a lower position on the ingredient statement. For example, corn syrup, honey, sugar, dextrose or fructose could all be used as sources for natural sweeteners and could be combined in a product to make an individual ingredient appear very low on the ingredient listing, while maintaining a very high level of the functional compound (ie, sugars). This type of ingredient selection can make the product, through the labeling, more consumer-appealing.

TABLE 3.6 Control of Product Oxidation

1. **Environmental control** - Lowering the product temperature to decrease the rate of oxidation (refrigerating), or modifying the atmosphere to remove the available oxygen (packing in nitrogen), minimizes the amount of oxidation.
2. **Rapid product use** - Oxidation is minimized by using the product as quickly as possible after the ingredients are mixed and processed. This is particularly critical with a complete, processed food that does not contain antioxidants, because the presence of trace minerals acts as a catalyst for the oxidation process.
3. **The use of antioxidants** - Either natural or chemical antioxidants can be used. Natural antioxidants such as vitamin E (and other tocopherols) and vitamin C tend to have a limited antioxidant life and do not give the product the length of protection that is possible with chemical antioxidants. Chemical antioxidants (ethoxyquin, BHT, BHA) provide the longest period of protection. There are no scientific studies detailing the effects of any preservatives on the long-term health of companion birds.

   Chemical antioxidants are assumed to be safer than the carcinogenic compounds that are known to be produced through oxidative rancidity.

Antioxidants

Some form of protection against product oxidation is essential to maintain nutritional adequacy of the product, to ensure a high level of palatability and to prevent the formation of oxidative by-products, some of which are carcinogenic. Oxidation of formulated diets can be minimized through a number of techniques (Table 3.6).

Grit

Grit is not required in the normal, healthy psittacine or passerine bird. Grit, defined as a granular, dense, insoluble mineral material (generally granite or quartz) is required in birds that consume whole, intact seeds. Examples of birds that require grit are pigeons, doves, free-ranging gallinaceous species and Struthioniformes. These species naturally eat whole grains as a varying portion of their diet. Because of the inert nature of the fibrous coating of many seeds (particularly corn, peas), digestive enzymes are relatively ineffective against them. Grit in the ventriculus acts to grind the whole seeds, thereby providing a substrate on which the digestive enzymes can act. Psittacine and passerine birds normally remove this fibrous hull, allowing the ingested portion to be easily acted upon by the digestive enzymes. It is likely, however, that in the case of a bird with a pancreatic

dysfunction or other problems involving the physical digestion of food, grit could provide a benefit by enhancing the surface area for digestive enzymes to act. There have been numerous examples of birds not having grit for 15 to 20 years and still not showing any signs of decreased performance or poor digestion. Amazon parrots that did not receive grit for over five years still maintained high digestibility of ingested sunflower seeds, showing the unimportance of grit in the healthy bird.[1] There have been numerous reports of birds, especially with health problems and depraved appetites, consuming copious quantities of grit and developing crop or gastrointestinal impactions. Considering the small chance of benefit and the potential risk, ad libitum feeding of grit should be avoided.

Food Selection

Psittacines, in particular, have individual preferences for foods based on previous experience (or habit), food placement (position in the cage), particle size, fat content, texture, shape, color and taste. These preferences can be strong, and most clients encourage them by providing what the bird is most likely to readily eat. Some owners even interpret these avid habits as an "addiction" to a certain food (often sunflower seeds or peanuts) because the bird refuses to eat anything else by its own volition. This type of limited feeding pattern can result in severe nutrient deficiencies if the selected food is not nutrient-complete and balanced. This is especially likely if the poor eating habits are left unchecked for an extended length of time. It must be emphasized that these preferences are individualized, especially in the larger psittacines, with some individuals having very distinct preferences. This can be illustrated by the choice of food based on color. Some individuals have no color preferences whatsoever, while others have distinct biases for certain colors (eg, red, yellow, brown).[3] Birds must be trained to eat new foods. This is best accomplished by providing limited portions, or meals, to encourage consumption of everything offered, as opposed to a virtual ad libitum feeding program where the bird can reach satiety by eating only one or two of its favorite ingredients. Providing a large variety of foods immediately pre- and post-weaning is a very effective way to develop good eating habits that will tend to persist throughout life. This will result in a healthier, less finicky companion bird.

Essential Nutrients and Their Biological Functions

Essential nutrients are those that are required to properly drive biochemical reactions within the body. These nutrients may be required as a specific energy source, as structural components or as factors and cofactors in specific biochemical reactions or processes (Table 3.7).

Energy

The total amount of energy, or the gross energy contained within the feed, is broken into several fragments as it is metabolized in the body. During the process of digestion, potential energy sources are lost through the feces, urine and urates. What remains is the metabolizable energy (ME), or what is available for the body's metabolic processes. A portion of the ME is lost as heat (the heat increment). The remaining energy (net energy value of the food) is available for maintenance of the bird. Any energy that remains after satisfying the basic maintenance requirements is available for production activities such as growth of body mass and feathers, deposition of fat, production of eggs and for exercise.

The bird derives energy from proteins, fats and carbohydrates in the diet. Of these, protein is the least efficient source of energy, because the body must deaminate the amino acid, excrete the nitrogen as uric acid and then use the remaining carbon skeleton for glucose or fat synthesis. The average gross energy of protein is 5.65 kilocalories/gram. After the losses through deamination and subsequent metabolic reactions, protein yields a net of 4.1 kcal/g.

Carbohydrates are the most important energy source for the body because they are the only energy form that the brain can use. Of the carbohydrate family, energy is derived from starches (digestible polysaccharides), disaccharides (sucrose, maltose) and the simple sugars or monosaccharides (glucose, fructose, mannose, galactose). Lactose, the disaccharide contained in milk, is a very poor energy source for avian species because of an inefficient supply of lactase in birds to hydrolyze lactose into its components of glucose and galactose. Carbohydrates are efficiently metabolized with an ME value of 4 kcal/g.

TABLE 3.7 Relative Nutrient Content of Commonly Used Food Sources[4,30]

NUTRIENT	EXCELLENT (Over 20 times requirement)	GOOD (Over 2 times requirement)	ADEQUATE (1/2 - 2 times requirement)
VITAMINS			
Vitamin A	Fish liver oil Liver Alfalfa meal Carrots Sweet potato Greens (spinach, parsley, kale, dandelion, turnip greens) Red peppers	Dried milk Cheese Egg	Fish meal Corn Peanuts
Vitamin D_3	Fish liver oil Liver (depending on levels fed) Fish oil	Eggs (especially yolk)	Dried milk
Vitamin E	Safflower oil Sunflower oil	All vegetable oils Alfalfa meal Sunflower seeds Safflower seeds Soybeans Wheat germ meal Corn gluten meal and germ	Cereal grains Dried milk Fish products
Vitamin K		Parsley Cabbage Brussel sprouts Spinach Cauliflower	Lettuce Broccoli Carrots Liver Turnip greens Milk Eggs Fish meal
Thiamine	Dried brewer's yeast	Wheat germ meal Rice bran Sunflower seeds Soybeans Wheat middlings Corn germ (and by-products) Peas and beans Dried whey Wheat Oats Peanuts Millet Carrots	Soybean meal Eggs Alfalfa meal Dried milk Fish meal Liver Most whole grains Potatoes
Riboflavin		Brewer's yeast Dried whey Dried milk Wheat germ Liver Eggs Fish and fish by-products Alfalfa meal	Millet Peas Beans Wheat Corn
Pyridoxine (B_6)		Brewer's yeast Eggs Whey Liver Alfalfa meal Black strap molasses Peanuts Sunflower and safflower Peas Soy products Alfalfa meal Wheat germ Fish by-products	Flax Millet Milo Buckwheat Wheat Other whole grains
Niacin		Yeast products Sunflower seeds Meat & fish by-products	Peanuts Corn by-products Wheat germ Alfalfa meal Wheat Barley Corn
Pantothenic Acid	Royal jelly	Yeast products Eggs Whey and dried milk Liver Alfalfa meal Peanuts Sunflower and safflower seeds Wheat germ meal	Peas Millet Wheat Oats Corn Other whole grains
Folic Acid		Yeast products Alfalfa Soybeans Wheat germ Liver	Beans Wheat Oats Peanuts Other whole grains Beets Spinach
Biotin		Safflower Liver Eggs Molasses Dried milk and whey Soybean products Alfalfa meal Milo Oats Peas Peanuts Corn gluten meal	Barley Beans Flax Wheat
B_{12}	Fish and meat by-products	Eggs Dried milk Yeast products	
Choline		Fish and meat by-products Yeast products Rape seed Dried whey Wheat germ Sunflower and safflower seeds Soybean products Peanuts	Alfalfa meal Most whole grains Beans Peas Eggs

TABLE 3.7 Relative Nutrient Content of Commonly Used Food Sources (cont.)[4,30]

NUTRIENT	EXCELLENT (Over 20 times requirement		GOOD (Over 2 times requirement)		ADEQUATE (1/2 - 2 times requirement)	
MINERALS						
Calcium	Calcium carbonate (incl. cuttle-bone, egg shell)	Bone meal Dicalcium phosphate	Fish and meat meals Kelp	Alfalfa meal Whey	Dried milk Cheese	Oil-type seeds Most nuts
Phosphorous	Dicalcium phosphate	Bone meal	Fish and meat meals Brewer's yeast Dried whey Wheat germ meal	Peanuts Pumpkin seeds Most oil seeds Nuts	Corn gluten meal Cereal grains	Egg
Magnesium					Kelp Bone meal Sunflower, safflower and other oilseeds	Nuts Alfalfa meal Brewer's yeast Wheat germ meal
Sodium	Salt	Bone meal	Dried whey and milk	Dried parsley Fish meal	Dried leafy vegetables Dried carrots	Alfalfa meal Eggs
Chlorine	Salt		Molasses Meat and fish products Dairy products	Alfalfa meal Dried parsley Carrot	Egg Green leafy vegetables	Broccoli Cereal grains
Potassium			Dried peppers Whey Dried carrot Alfalfa meal Molasses Soybean products Dried apricots	Bananas Brewer's yeast Oil seed products Legumes Oil seeds Wheat germ meal	Nuts Dried fruits and vegetables	Oil-type seeds Cereal grains
Manganese			Dicalcium phosphate	Calcium carbonate Wheat germ meal	Hemp seed Wheat products Soy products	Oat products Nuts
Iron	Bone meal		Dried parsley Fish and meat meals Calcium carbonate Corn gluten meal	Alfalfa meal Dried whey Soybean meal Brewer's yeast	Most cereal grains (especially millet, barley, oats, canary grass seed)	Oil-type seeds Nuts Dried carrots
Copper			Dried whey Molasses Brewer's yeast	Oil seeds Corn gluten meal Nuts	Fish and meat products Peas	Alfalfa meal Cereal grains
Zinc			Fish and meat meals Wheat germ meal	Wheat middlings Bone meal	Oil-seed products Soybean meal Nuts	Wheat Oats Corn gluten meal
Iodine	Dried whey		Fish and meat meals	Molasses	Egg Cheese	Brewer's yeast
Selenium	Fish meal		Brewer's yeast Corn gluten meal Wheat middlings Oil seeds Alfalfa meal	Wheat germ meal Rice Dried parsley and spinach Oats	Cheese Egg Soybean meal	Cereal grains (depending on soil)
AMINO ACIDS						
Lysine			Fish and meat meals Soybean meal Dried parsley and spinach	Brewer's yeast Wheat germ meal Peas Dried whey	Oil-type seeds (especially sunflower and safflower) Corn gluten meal Alfalfa meal	Nuts Oats Canary grass seed Barley Buckwheat Millet
Methionine			Corn gluten meal Fish and meat meals Brewer's yeast Soybean meal	Canary grass seed Sunflower Wheat germ meal Dried whey	Oil seeds Millet Peas Alfalfa meal	Nuts Wheat, oats, barley & other cereal grains

Carbohydrates also form the fiber fraction of the diet, broadly classified as undigestible carbohydrate. This fraction consists mainly of cellulose, which is essentially undigested because of the bird's lack of the enzyme cellulase. Also included are the hemicelluloses and lignin, all of which are poorly digested. These fibrous agents generally minimize the absorptive space in the gastrointestinal tract. The hemicellulose, psyllium, is an exception, as it acts to increase absorption. The required dietary fiber intake of varying species of companion breeds is undetermined.

Dietary fat is not only an important source of energy but it is the primary storage form of energy in the body. The ME in fat is concentrated with a value of 9 kcal/g, 2.25 times greater than that of either carbohydrates or protein. Fat is also easily absorbed into the body via the gastrointestinal tract, with its digestibility being dependent on the fatty acid composition.

Essential Fatty Acids

Animals and birds have no requirement for fat per se, but they do have a requirement for the individual fatty acids that make up fat. Fatty acids are characterized based on their length (ie, the number of carbon atoms contained in the chain), the degree of saturation (the number of double bonds in the chain, commonly referred to as saturated, unsaturated or polyunsaturated) and the location of the initial double bond.

The primary essential fatty acid for animals and birds is linoleic acid. This compound cannot be synthesized in the body so it must be provided through the diet. Arachidonic acid is sometimes considered to be an essential fatty acid; however, it can be synthesized from linoleic acid.

The predominant fatty acid compounds in bird tissues are oleic acid, palmitic acid and linoleic acid. Body fat composition will be somewhat influenced by dietary fatty acid content because of the absorption and subsequent deposition of some intact fatty acids. Common vegetable oils are generally high in linoleic acid (eg, corn oil, soybean oil, peanut oil = 50%; sunflower oil = 60%; safflower oil = 75%). Tropical oils, such as coconut oil, contain substantial amounts of medium chain fatty acids, and are therefore poorer sources of linoleic acid.

Absorption of these fatty acids varies depending on the type, the form (free or as part of a triglyceride), the ratio of unsaturated to saturated fatty acids in the diet, other dietary constituents and the intestinal microflora. Generally, oleic and linoleic acids are the most efficiently absorbed by the bird. This occurs because of the ease with which these fatty acids form mixed micelles with the bile salts, thereby improving their digestion by pancreatic lipase. In this manner, they will also enhance the absorption of other less efficiently absorbed fatty acids when they are present together.

The essential fatty acids are used as structural components in the cell with particular importance in the cell membranes. They are also precursors of prostaglandins.

Based on the general requirements for most other species, it can be safely predicted that the linoleic acid requirement for companion and aviary birds is 1.0 to 1.5% of the diet. In seed-based diets, this would rarely fall short, but in a processed, low-fat diet there could be a marginal deficiency.

Amino Acids and Protein

Amino acids are the building blocks of the protein chain. The type of protein synthesized depends on the complex genetic process of transcription and translation between the DNA and RNA of the body. A specific protein is created by the shaping of the polypeptide chain into its unique three-dimensional structure based on interactions between the individual amino acids of the chain.

The protein chain can contain up to 22 different amino acids. Of these, ten cannot be manufactured by the body, so they must be routinely provided by the diet (essential amino acids). They are lysine, arginine, histidine (basic amino acids), methionine (sulfur-containing), tryptophan (heterocyclic), threonine, leucine, isoleucine, valine (aliphatic) and phenylalanine (aromatic). Three other amino acids (cystine, hydroxylysine and tyrosine) are formed through modification of an essential amino acid (methionine, lysine and phenylalanine, respectively). These are not considered essential per se, but they may affect the total amount of the essential amino acid required, depending on their level in the diet. There are nine additional amino acids that are nutritionally nonessential because they are manufactured from other compounds in the body.

The quality of a protein is determined by two primary factors. The first is the balance of amino acids within

that protein. To be optimally utilized, the protein should have an amino acid profile similar to that of the animal's body. If this occurs, each individual amino acid will be present in approximately the right proportion that the body needs with no major excesses or deficiencies of any one amino acid. This profile is achieved only in a few foods, most notably in eggs and in milk. It seems obvious that these two protein sources would fit the profile of the body, because they provide the only source of food during early periods of rapid growth.

Very few ingredients have an amino acid profile that approaches ideal; therefore, it is preferable to choose individual ingredients for the diet that complement each others' amino acid profile. With proper selection, the ingredients work together in a synergistic manner to enhance the overall performance of the mixed diet. By dividing the percentage of a specific amino acid in the protein of an ingredient by the percentage of that amino acid in an ideal protein, an evaluation of the degree of amino acid adequacy can be determined. Doing this to all the essential amino acids for an ingredient will determine the limiting amino acid, or that essential amino acid that is present in the lowest proportion of ideal. This amino acid will have to be supplemented by either adding an ingredient that is particularly high in this amino acid or by supplying the specific amino acid in a purified form. Similarly, this kind of evaluation can be performed on the entire diet to determine the adequacy of the amino acid profile. These values would be reflected in the amino acid requirement of the animal at its particular stage of life.

The second criteria that affects protein quality is the availability of the amino acids within the foodstuff. Certain ingredients have structural characteristics or contain chemical compounds that will decrease the bioavailability of an amino acid. A typical example of this would be the interaction between lysine and dietary simple sugars resulting in a chemical complex that makes lysine unavailable to the animal. Another example would be the trypsin and chymotrypsin inhibitors in unprocessed soybeans that prevent normal proteolytic activity of these digestive enzymes, thereby decreasing digestibility. The specific structure of an amino acid chain can also render a protein undigestible. This occurs due to secondary and tertiary structural characteristics preventing the enzymatic hydrolysis of the amino acid chain in the body. An example of this is the extremely poor digestibility of keratin and the other fibrous proteins.

After a protein source is consumed, it is initially processed by the combination of pepsin and hydrochloric acid secreted by the glandular stomach (proventriculus). The resultant polypeptide chains are then further degraded by a series of enzymes from the pancreas (trypsin, chymotrypsin, carboxypeptidases), aminopeptidases and finally dipeptidases. The individual amino acids that result from this series of enzymatic hydrolyses are then absorbed in the small intestine, predominantly in the jejunum, although all sections of the small intestine are involved in absorption.

Vitamins

The vitamins are chemically unique but share similar metabolic roles and modes of action and are therefore grouped together.

Generally, vitamins are defined as natural food components that are present in minute quantities, are organic in nature and are essential for normal metabolism and health. They will cause specific, characteristic deficiency symptoms when they are severely limited in the diet. Metabolism will generally be affected to a degree proportional to the level of the deficiency; therefore, in the case of mild deficiency, the symptoms are usually vague and nonspecific, such as poor performance or compromised health. Vitamins are generally not synthesized by the body in amounts sufficient to meet the physiologic requirement.

Vitamins are now subcategorized into two general groups based on their solubility characteristics. The fat-soluble vitamins are comprised of vitamins A, D, E and K. The water-soluble vitamins include thiamine (vitamin B_1), riboflavin (vitamin B_2), niacin, pyridoxine (vitamin B_6), pantothenic acid, biotin (vitamin H), folic acid (vitamin M), vitamin B_{12} (cyanocobalamin), choline and ascorbic acid (vitamin C). Other vitamin compounds that are generally not considered to be required by higher animals include lipoic acid (occurs widely in natural foodstuffs), inositol (synthesized by higher animals and widely distributed in most foodstuffs), and para-aminobenzoic acid (required by microorganism for the synthesis of folic acid).

Vitamin A

Vitamin A occurs in several forms: retinol (alcohol), retinal (aldehyde) and retinoic acid, all having different metabolic activity. Plants do not contain active vitamin A, but instead contain vitamin precursors.

These exist in the form of carotenoid plant pigments, with the carotenes being the most important of the pro-vitamin A compounds. In the avian species studied, beta carotene is the most active of the carotenoid compounds, yielding the equivalent of 1667 international units (IU) of vitamin A activity per milligram. The sum of the vitamin A content (expressed in retinol equivalents or IU) and the contribution from carotene represents the total vitamin A activity of the food.

The most well understood function of vitamin A is its role in vision, but the most impactful action of vitamin A in avian medicine is its effect on the growth and differentiation of epithelial tissues, with deficiencies resulting in keratinization of the tissue. It is in this function that vitamin A is obligatory for normal disease resistance because it is required for the maintenance of adequate mucous membranes and for the normal functioning of secretory tissues (eg, the adrenal glands for the production of corticosteroids).

Vitamin A is also required for normal mucopolysaccharide formation and apparently affects the stability of cell membranes and of the subcellular membranes (such as the mitochondria and lysosomes). A major metabolic function of vitamin A may be the maintenance of the structural integrity and the normal permeability of the cell membrane. Vitamin A also functions in the proper growth of bones and in the maintenance of normal reproduction.[34]

It is generally accepted that vitamin A improves the immune function of the body; however, its mode of action has not been totally elucidated. Vitamin A apparently acts by the increased production and differentiation of immune related cells, while the carotenoids possibly improve the activity of lymphocytes. Obviously, this function is also significantly influenced by the importance of vitamin A in maintaining healthy mucosal membranes.

The liver will typically contain over 90% of the total body stores of vitamin A with the preferential storage form being retinyl palmitate. Additional supplies are also contained in the kidneys, lungs, adrenals and blood. As vitamin A is required by the body, it is mobilized from the liver by the hydrolysis of the retinyl esters to free retinol by the enzyme retinyl ester hydrolase.

Vitamin A is usually considered safe up to approximately ten times the requirement in monogastrics (including poultry). Experimentally, vitamin A toxicities have been achieved by feeding over 100 times the daily requirement for extended periods of time. Probably an excess of 1000 times requirement would be necessary to induce an acute intoxication. Carotenoids in the diet do not contribute to potential vitamin A toxicity, because they are not converted to retinol unless there is a metabolic need for vitamin A. At excessive levels, they may result in a temporary yellow pigmentation of the skin and fat.

Vitamin D

There are two predominant forms of vitamin D: ergocalciferol (vitamin D_2), a plant derivative, and cholecalciferol (vitamin D_3), produced exclusively in the bird's body. In all of the birds studied, vitamin D_3 is considered to be 30 to 40 times more potent then vitamin D_2 as a source of vitamin D activity. Therefore, plant sources of vitamin D are essentially disregarded when providing vitamin D to birds. Vitamin D_3 levels are quantified in International Chick Units (ICU) as a way to differentiate it from vitamin D_2 or total vitamin D. Unlike most other vitamins, the active form of vitamin D_3 can be synthesized in the body by the conversion of 7-dehydrocholesterol in the skin and sebaceous secretions by irradiating with ultraviolet rays. Early studies in poultry showed that sufficient Vitamin D_3 could be formed to prevent rickets in growing chickens and maximize growth with 11 to 45 minutes of sunshine (not filtered by glass) each day.[22]

The cholecalciferol formed in the skin is then transported by the blood to the liver, where it is hydroxylated by a liver microsomal enzyme (to a lesser extent, the reaction may also occur in avian kidneys). This new compound is then transported to the kidney, where it is again hydroxylated to the metabolically active form. When the renal levels of calcium and phosphorus are normal and parathyroid hormone (PTH) is being secreted, an inactive form is produced. Unlike other vitamins, the active metabolite actually acts as a hormone in the body being transported to the intestines, bones and other target organs where it exerts its role in the metabolism of calcium and phosphorus (see Chapter 23).

The most important physiologic role of vitamin D is the homeostasis of calcium and phosphorus levels in the body. There is also evidence that the active form has additional roles, eg, induction of cell differentiation and immune system regulation.[12,36]

The active metabolite also acts in the body in a manner similar to a steroid hormone, acting on a specific receptor protein in the target organ. The

vitamin D receptor is located in the nucleus of the intestinal mucosal epithelial cells.

Hypervitaminosis D_3

In a prolonged feeding study with cockatiels on a diet containing 1.0% Ca, 0.5% P and 4000 ICU vitamin D_3 (18% crude protein and 3150 kcal/kg), high egg production for approximately one year was followed by a rapid decline in reproductive performance, concurrent with the onset of polyuria in all birds. Most had signs of anorexia and lethargy, with some exhibiting signs of diarrhea or lameness. Radiographs indicated the presence of nephrocalcinosis. These signs were exacerbated with the onset of subsequent reproduction. Several females were lost, with necropsies showing extensive soft tissue mineralization, especially of the kidneys. The onset of reproduction and subsequent increasing hormonal activity (presence of prolactin) and related increases in calcium uptake in females were found to enhance the problem. Males were affected to a much lesser extent, with all clinical signs disappearing after the birds were removed from the experimental diet.[2]

Vitamin E

Vitamin E is a compound of plant origin with eight active forms derived from four tocopherols and four tocotrienols. The compound of the greatest biologic importance in the avian species is alpha-tocopherol. Vitamin E is essentially a biologic antioxidant that functions at the intercellular and intracellular level by preventing the oxidation of saturated lipid compounds in the cell, thereby maintaining membrane integrity.

Free radicals, the highly reactive breakdown products from reactions such as the oxidation of polyunsaturated fatty acids to fatty hydroperoxides, can be extremely damaging to the cell. Free radicals occur in the body through normal oxidative metabolism, cytochrome activity and from stimulated phagocytes. These free radicals can then attack the polyunsaturated fatty acids of membranes, creating additional radicals, producing a chain reaction that can continue until all of the polyunsaturated fatty acids in the membrane are oxidized. Vitamin E acts to scavenge these radicals, thereby preventing the initiation as well as interrupting propagation of peroxidation.

Working in conjunction with vitamin E are several metalloenzymes, which block the initiation of peroxidation in the aqueous phase of the cell. These enzymes incorporate manganese, zinc, copper, iron and selenium as active components. Glutathione peroxidase (GSHp) is probably the most important of these metalloenzymes because of its integral relationship with vitamin E. This selenium-containing enzyme is very active in the destruction of peroxides before they cause membrane damage. Because of their similar activity, selenium and vitamin E tend to have a sparing effect on each other. Exudative diathesis, the condition observed in poultry, generally appears only when both selenium and vitamin E are limited in the diet. Additionally, sulfur-containing amino acids can exhibit a similar sparing effect on vitamin E because they are precursors of GSHp.

Vitamin E has been suggested to be active in several other metabolic systems: 1) cellular respiration; 2) normal phosphorylation reactions (eg, ATP metabolism); 3) cofactor in the synthesis of ascorbic acid; and 4) sulfur amino acid metabolism.[43] There is also considerable evidence in poultry that levels higher than those required for optimum growth can increase immunity, as evidenced by decreased mortality after challenge of treated birds by *E. coli*.[27] This protective effect occurs by increasing phagocytosis and antibody production as well as stimulating the activity of macrophages and lymphocytes.

Vitamin E is absorbed through passive diffusion and is dependent upon normal lipid digestion requiring proper micelle formation and the presence of bile salts and pancreatic juices. Any malabsorption syndrome will decrease uptake. Vitamin E is absorbed predominantly as a free alcohol in the small intestine. Vitamin E enters the portal circulation in association with chylomicra, but is readily transferred to plasma lipoproteins for transportation to the liver. Initial storage occurs in the liver, being released primarily in the high density lipoproteins, and to a lesser degree, the low density lipoproteins and very low density lipoproteins. Liver and plasma stores of vitamin E are the most readily accessible to the body in times of need. Vitamin E stores of the body tend to be relatively stable and may not be effective in preventing a vitamin E deficiency from occurring. It appears that lipolysis of fatty stores may be required for vitamin E to be released.

Vitamin E is abundant in plant materials (particularly those high in oil) and in plant leaves. In cereal grains, vitamin E is concentrated in the germ. Alfalfa leaves are a particularly high source of vitamin E.

Vitamin K

Vitamin K actually represents a large number of related compounds that possess widely varying de-

grees of anti-hemorrhagic characteristics, all being forms of the compound naphthoquinone. Vitamin K comes from three sources: 1) green plants (phyloquinones - K_1 series), 2) bacteria (menaquinones - K_2 series) and 3) synthetic forms (menadione - K_3). The microbial synthesis of vitamin K_2 is significant in most species. It is generally difficult to produce a vitamin K deficiency without the use of germ-free animals, the use of antibiotics to kill intestinal flora or the prevention of coprophagy (the ingestion of excreta).

Natural vitamin K compounds require the presence of dietary fats and bile salts for proper absorption from the gastrointestinal tract; therefore, altered micelle formation (eg, decreased pancreatic and biliary function) will impair the normal absorption of vitamin K. Menadione salts are fairly water-soluble so they are less reliant on micelle incorporation. Absorption of the K_2 and K_3 forms occurs by passive diffusion throughout the intestines and also in the colon, while K_1 is absorbed via an active transport process in the proximal small intestine. Vitamin K then enters the portal circulation and, in association with a chylomicron, is transported to the liver. Generally, vitamin K is stored only briefly in the liver before it is released into the body and transported to all tissues via lipoproteins. It is believed that menadione is well absorbed but poorly retained, while phylloquinone is rather poorly absorbed but retained much longer in the body. Vitamin K absorption has been observed to range from 10 to 70%, depending on the form of vitamin.

A number of plasma clotting factors (eg, prothrombin) are dependent on vitamin K for their synthesis. This occurs by activating inactive protein precursors that occur through the action of an enzyme; this is found predominantly in the liver, but also in lung, spleen, kidney, bone and skin. The bone also contains a vitamin K-dependent protein (osteocalcin), which acts in the regulation of calcium phosphate incorporation into bone.

Thiamine (Vitamin B_1)

Thiamine is fairly common in food sources, but generally at only low concentrations. In plants, thiamine exists as the free vitamin, while in animal tissue it is present in its phosphorylated form, thiamine pyrophosphate. Several compounds in nature possess anti-thiamine activity, many of which exhibit competitive inhibition with thiamine based on their structural similarities. An example of this is amprolium, which inhibits thiamine absorption from the intestine and prevents thiamine phosphorylation.[26] Another well known compound is thiaminase, a thiamine-splitting enzyme contained in some raw fish and produced by certain types of bacteria. Other thiamine antagonists include caffeic acids, chlorogenic acid and tannic acid, (often found in deeply pigmented fruits and vegetables such as blueberries or beets as well as coffee and tea). These compounds react with thiamine to prevent its absorption. Sulfites, a frequently used food preservative, can also destroy thiamine under certain conditions.

Thiamine is readily available from natural sources when normal amounts of gastric hydrochloric acid are present. Thiamine is absorbed both by an active transport system and at high luminal concentrations, by passive diffusion. After absorption, thiamine is transported via the portal vein to the liver, predominantly bound to serum albumin. Thiamine is not stored for any length of time in the body. It is excreted primarily through the urine and in lesser amounts through the feces. About 80% of thiamine in the body is present as thiamine pyrophosphate. The remaining fraction exists as the triphosphate, monophosphate and free forms.

Riboflavin (Vitamin B_2)

In foods, riboflavin is generally bound to proteins in the form of flavin mononucleotide (FMN) or flavin adenine dinucleotide (FAD). Riboflavin contained in plant materials is generally less available than from animal sources because of decreased digestibility of the flavin complexes in plants.

In the gastrointestinal tract, the phosphorylated forms of riboflavin are hydrolyzed. The free riboflavin enters the mucosal cells via an active transport system in the proximal small intestine. In the intestinal mucosa, riboflavin is rapidly phosphorylated, producing FMN. Both free riboflavin and FMN then enter the portal circulation, predominantly bound to plasma albumin (and to a lesser degree to globulins and fibrinogen). These compounds are then transported to the liver and other tissues, where riboflavin enters the cell in the free form.

Very little riboflavin is stored in the body; the highest concentrations are found in the liver, kidney and heart. Unlike other tissues, the egg contains predominantly free riboflavin. Laying chickens have been found to have specific riboflavin-binding proteins in the plasma. These are produced in the liver under the influence of estrogen and are believed to be involved in the transovarian passage of free riboflavin.

Riboflavin as part of the coenzymes FMN or FAD (flavoproteins) act in a large number of enzyme complexes that are responsible for essential reactions in the utilization of carbohydrates, fats and proteins. The flavoprotein enzyme complexes often contain a metal ion (eg, iron, molybdenum, copper) and function to help regulate cellular metabolism, the metabolism of carbohydrates, the breakdown of amino acids, the formation of uric acid, the formation of ascorbic acid, fatty acid biosynthesis and degradation, oxidation of various substrates in drug metabolism and other functions.

Riboflavin toxicity is very unlikely due to the fact that it is rapidly excreted, and when fed at high levels, the transport system across the gastrointestinal mucosa becomes saturated, thereby limiting the amount absorbed.

Niacin

Niacin exists in two major forms, nicotinic acid and nicotinamide. Niacin is widely distributed in foods, but that found in plants has low bioavailability. It is also not uniformly distributed within the feedstuff so milling often removes the fraction with the highest content. Therefore, in diet formulation, the natural content of niacin in plant materials is generally ignored. Bioavailability in animal products tends to be very high. Niacin can also be synthesized from the essential amino acid tryptophan; however, the amino acid's preferential use is for protein synthesis, so only tryptophan in excess of the animal's needs will be available for bioconversion to niacin.

Plants generally contain protein-bound nicotinic acid while animal sources are present as NAD and NADP. These forms are digested by the body, releasing nicotinamide that is then absorbed by diffusion. The greatest concentrations of niacin compounds are in the liver, but no true storage occurs.

The coenzymes NAD and NADP are important components in carbohydrate, fat and protein metabolism, being especially important in the energy-yielding reactions of the body. These functions are critical to the generation of energy for the body as well as for normal tissue integrity, especially of the skin, alimentary tract and the nervous system.

Pyridoxine (Vitamin B_6)

Vitamin B_6 refers to the group of three compounds: pyridoxal, pyridoxamine and pyridoxal phosphate. Pyridoxal is the form predominantly found in plants, the other two are found mainly in animal tissues. Large amounts of vitamin B_6 in foods are bound to proteins or complexes, some of which have very low bioavailability. After digestion to free the vitamin from these protein complexes, vitamin B_6 is absorbed by passive diffusion throughout the entire small intestine and is transported to the liver. The various forms are then converted and phosphorylated to the predominate tissue form, pyridoxal phosphate, which requires both niacin (as NADP) and riboflavin (as FMN) for the enzyme systems. Pyridoxal phosphate and lesser amounts of pyridoxal are found in the circulation associated with plasma albumin and erythrocyte hemoglobin. Minimal amounts of the vitamin are stored in the body, primarily as pyridoxal phosphate and secondarily as pyridoxamine phosphate. Storage occurs predominately in the liver, brain, kidney, spleen and muscle.

The metabolically active form of vitamin B_6, pyridoxal phosphate, is involved in a number of enzyme systems as a coenzyme. It is required in essentially all major areas of amino acid utilization, the synthesis of niacin from tryptophan and in the formation of antibodies. It is required in the decarboxylation of glutamic acid to form gamma-aminobutyric acid (GABA), the lack of which has been shown to cause seizures. A deficiency of pyridoxine creates a deficiency of many other important metabolites and hormones such as serotonin and histamine. Evidence also suggests that it may play a role as a modulator of steroid hormone receptors.

Pantothenic Acid

Pantothenic acid is a structural component of coenzyme A (CoA). Pantothenic acid is present in feeds in both the bound form (predominantly CoA) and free forms. During the digestive process, the free form is liberated prior to absorption. Pantothenic acid is then absorbed via a saturable transport system and at high levels, simple diffusion also occurs. The free form is then carried via the plasma to the rest of the body. Tissues convert pantothenic acid to coenzyme A (predominantly), with the greatest concentrations found in the liver, adrenals, kidneys and brain. The majority of the pantothenic acid in the blood is found as CoA in the erythrocytes. CoA is one of the most critical coenzymes in tissue metabolism, forming the compound acetyl CoA. Acetyl CoA acts as the entry point into the citric acid cycle for carbohydrate metabolism, a point of entry for amino acid degradation and as an essential component in fatty acid biosynthesis and degradation, the synthesis of triglycerides and phospholipids, as well as in the formation of

compounds such as acetylcholine, mucopolysaccharides, cholesterol, steroid hormones and many more.

Biotin

Biotin is widely distributed in foods but generally at low concentrations. A relatively large portion of naturally occurring biotin is present in a protein-bound form with varying degrees of biological availability. There is evidence that suggests that the synthesis of biotin by intestinal microflora is important in an animal. Microbial-derived biotin would be manufactured and absorbed in the large intestine.

Intestinal proteases help free the bound biotin prior to absorption. Free biotin is then absorbed, apparently both by facilitated and simple transport systems. It is carried to the tissue through the plasma, possibly in conjunction with a biotin-binding protein (identified in both yolk and plasma of laying chickens). The largest concentrations of biotin in the body are found in the liver; however, this storage site seems to be poorly mobilized during times of biotin deprivation.

Biotin is an active part of four different carboxylase enzymes in the body, and is responsible for the fixation of carbon dioxide (carboxylation). These enzymes have important functions in the metabolism of energy, glucose, lipids and some of the amino acids.

Folic Acid (Folacin)

Folic acid is the compound pteroylmonoglutamic acid. Additionally, there is a large group of modified folic acid compounds, referred to collectively as folates. At one time, PABA was believed to be essential in the diets of vertebrates, but it has since been determined that if the requirement for folic acid is met, PABA provides no additional benefit.

Folates are generally widely distributed in foods and are present as the polyglutamic derivatives of folic acid. These are converted by hydrolysis to free folic acid and absorbed by both an active transport system and passive diffusion in the duodenum and jejunum. The absorption process is only moderately efficient (<50%).

Folic acid's primary metabolic role is in the transfer of single-carbon moieties in a wide variety of reactions. This function is particularly important in amino acid metabolism, in the bioconversion of amino acids and in the biosynthesis of nucleotides.

Because of folic acid's requirement in the synthesis of three of the four nucleic acids, a deficiency results in impaired cellular division and an alteration of protein synthesis. This is particularly noticeable in the young growing animal. Additionally, due to impaired cell mitosis in a deficient bird, females do not physiologically prepare for breeding, as noted by a lack of oviduct hypertrophy in the presence of estrogen. Further, there is an effect on normal red blood cell maturation, resulting in the characteristic macrocytic anemia. Similarly, deficiencies result in immune system impairment due to the effects on cell replication and protein synthesis. Folic acid is involved in the formation of uric acid, so there is an increased requirement when high-protein diets are provided. Folic acid is required for the production of white blood cells and a severe deficiency can reduce immunologic response through decreased WBCs or reticuloendothelial cells.

In some species, a deficiency of zinc has been found to impair the utilization of dietary sources of folic acid. A zinc deficiency decreases the absorption of folic acid because of impaired activity of the mucosal enzyme that creates an absorbable form of folic acid. Enzyme inhibitors are present in a number of foods such as cabbage, oranges, beans and peas (in the seed coat) and brewer's yeast. These inhibitors are generally destroyed by processing since they are heat-labile. Sulfa drugs (eg, sulfanilamide) may increase the requirement of folic acid since they will compete with structurally similar PABA in the bacterial synthesis of folic acid.

Vitamin C and iron may improve the bioavailability of folates in food.

Vitamin B_{12}

Vitamin B_{12} or cyanocobalamin is a product of bacterial biosynthesis and therefore must be obtained by consuming a bacterial source or animal tissues that accumulate the vitamin. The only exceptions are a few plants, such as peas, beans, spirulina and kelp, that may be able to synthesize minute amounts of this vitamin, although this accumulation is likely due to their close symbiotic association with bacteria.

Naturally occurring vitamin B_{12} occurs in the coenzyme form bound to protein. This complex is broken, primarily through the normal action of pepsin and trypsin. Free vitamin B_{12} is absorbed by the intestinal tract via an efficient active transport system involving a vitamin B_{12} specific-binding protein. At very high levels, simple diffusion occurs throughout the small intestine.

Most of the vitamin B_{12} in the body is found in the liver with secondary stores in the muscle. Lesser

amounts (but high concentrations) are contained in the pituitary gland, kidney, heart, spleen and brain. Vitamin B_{12} is stored efficiently, with a long biological half-life (approximately one year in humans).

Vitamin B_{12} is a critical component of a large number of metabolic pathways. It interacts with several other nutrients such as folic acid, pantothenic acid, choline and methionine. Similar to folic acid, most of the metabolic reactions of vitamin B_{12} involve single carbon units and are very important in the synthesis of nucleic acids and protein as well as carbohydrates and fats.

Like folic acid deficiencies, vitamin B_{12} deficiencies result in an impairment of protein synthesis causing failure or delay of normal cell division. This affects growth rate and feed intake, may result in nervous disorders and poor feathering, perosis, anemia, ventricular erosion and fat accumulation in the heart, liver and kidneys. Deficiency of vitamin B_{12} can also create a folic acid deficiency.

Some research indicates that vitamin B_{12} absorption is decreased in the presence of protein, iron or vitamin B_6 deficiencies or by dietary tannic acids.[23]

Choline

Natural sources of choline are widely distributed and occur primarily in the form of phosphatidylcholine (lecithin). It is also present as free choline, acetylcholine and in other phospholipids, such as sphingomyelin.

Phosphatidylcholine is readily hydrolyzed in the intestinal lumen and is absorbed by the mucosa via both active transport and passive diffusion, depending on luminal concentrations. Of the free choline that is ingested, up to two-thirds may be metabolized by intestinal microorganisms. The remainder is absorbed intact. Choline is found in all tissues as a part of the membrane phospholipids, with the greatest concentrations in organs such as the brain, liver and kidney (as phosphatidylcholine and sphingomyelins).

Choline can be synthesized in the body but in the avian species tested to date, it cannot be synthesized at high enough levels to meet the needs of the young bird. It appears that with age, the synthetic abilities improve, thereby meeting most of the body's needs. This is especially true when choline-sparing compounds such as methionine, betaine and myo-inositol are present in the diet. Dietary sulfates can also have a sparing effect on choline by helping to spare methionine.

Choline has four general metabolic functions: 1) As a component of phospholipids, choline is an essential part of the cell membrane and is required for maintaining cell integrity; 2) Choline is required for maturation of the cartilage matrix of bone; 3) Choline is involved in fat metabolism of the liver by promoting fatty acid transport and utilization, and is therefore necessary to prevent hepatic lipidosis in the normal bird; 4) Choline is acetylated to form the neurotransmitter acetylcholine.

Because of their interrelated functions, the requirement for choline is dependent upon the levels of folic acid and vitamin B_{12} available to the animal. Excess protein increases the choline requirement, as do diets high in fat. Dietary levels of choline chloride (the normal supplemental form) should not exceed twice the requirement.

Vitamin C (Ascorbic Acid)

Vitamin C has not been demonstrated to be a required nutrient for any of the avian species, except for a few highly evolved, largely frugivorous species (Willow Ptarmigan and Red-vented Bulbul).[10] Vitamin C is easily manufactured in birds with the enzyme L-gulonolactone oxidase. This enzyme works on a substrate generated from glucose producing an intermediate that is then converted to L-ascorbic acid. This process occurs in the liver in most passerine species, and in the kidneys of psittacines and other older phylogenetic orders of birds. Biosynthesis of ascorbic acid can be inhibited by deficiencies of vitamin A, E and biotin.

Vitamin C occurs in the forms of ascorbic acid and dehydroascorbic acid, with both forms having similar biological activity. Vitamin C is found in the highest concentration in fruits, vegetables (but not seeds) and organ meats (particularly the liver and kidney).

Dietary sources of vitamin C are absorbed by passive diffusion in those species that do not have a specific dietary requirement. Absorption appears to be relatively high when fed at normal levels. Decreased absorption occurs as the physiologic dose is exceeded. The highest concentrations of vitamin C are found in the pituitary and the adrenal glands followed by the liver, spleen, brain and pancreas. Vitamin C also tends to accumulate around healing wound sites. The metabolic functions of vitamin C are related to its ability to act in oxidation and reduction reactions. Its best understood role is in the synthesis of collagen, where it is involved in the hydroxylation of procollagen residues. Collagen, the major component of skin

and connective tissue and also the single most abundant protein in the body, is critical for proper cell structure and integrity. In species requiring vitamin C in their diet, the breakdown of this function produces the classic deficiency symptoms (scurvy, capillary fragility, gum and bone alterations and poor healing).

Vitamin C is also an excellent antioxidant, acting to neutralize free radicals that are produced in the body. Ascorbic acid can also regenerate vitamin E (the active lipid antioxidant).

Based on their scientific orders, evolutionary status and limited testing, psittacine and passerine birds appear to have no requirement for vitamin C. In other species with no specific requirement (eg, domestic poultry), there have been documented benefits of providing a dietary source of vitamin C to birds at certain stages of life or under certain conditions. Stressful conditions that have been shown to improve with supplemental vitamin C are: 1) dietary deficiencies of energy, protein, vitamin E, selenium or iron; 2) high production or high growth rates (the newly hatched chick has a slower rate of ascorbic acid synthesis); 3) management stresses, eg, handling, insecure environment, transportation, crowding; 4) extreme temperature variations from normal; 5) health stresses: fever and infection reduce blood ascorbic acid and diseases with liver involvement decrease synthesis while increasing overall requirement for ascorbic acid.[25] Supplemental ascorbic acid has been shown to increase total sperm production in turkeys[14] and improve broiler fertility and hatchability, due to decreased early embryonic mortality.[31]

Considering the normal stresses that companion and aviary birds experience, it seems reasonable that a supplemental source of vitamin C may be of some benefit during certain situations. This may be even more important considering that many birds lack proper diet and health care. Fresh food sources should be considered as the most important way to supplement the diet because of the vitamin's general instability in manufactured products.

Minerals

Minerals are essentially classified in one of two groups: macro minerals and trace or micro minerals. The macro minerals can be classified based on their use in the body. Calcium and phosphorus act primarily in the body's skeletal structure, while sodium, potassium and chlorine (along with phosphates and bicarbonates) function to maintain homeostasis in the body (acid/base balance and proper osmotic pressures). The required trace minerals are magnesium, manganese, zinc, iron, copper, iodine, selenium and, in certain situations, cobalt and molybdenum. These trace elements have their primary function as parts of enzymes, hormones or as enzyme activators. Additionally, in purified diets, there have been beneficial effects achieved by the addition of some of the other trace elements such as fluoride, nickel, silicone, tin, vanadium and chromium. These benefits have usually been seen only in sterile conditions with extreme environmental controls. At this time, they should not be considered as dietary essentials because of a lack of conclusive evidence regarding their essentiality and the poor understanding of their metabolic function.

As the normal digestion process breaks food into its components, the minerals are liberated, and the cationic elements are converted to chloride salts in the presence of gastric hydrochloric acid. Once in the intestinal tract, they are able to easily dissociate and be absorbed. There is also considerable complexing with other minerals or chelating agents. An example of this is the calcium and phosphorus precipitate that is formed by excess levels of these minerals while in the alkaline conditions of the small intestine. This complex can then adsorb manganese or zinc, causing excretion of the trace mineral, and subsequently, an increased requirement.

Mineral (particularly trace mineral) concentrations of foodstuffs are largely dependent on the original mineral source. Concentrations in plant products are dictated by the soil mineral content, while those of animal products are dependent on the diet consumed.

Calcium

Calcium is the predominant mineral in the body (approximately 1.5% of body weight) with primarily skeletal system containment. Calcium is also contained in the body fluids, where it plays an essential role in blood coagulation and membrane permeability, and maintains normal excitability of the heart, muscles and nerves. Several enzyme systems are also activated by calcium. Ionic calcium (Ca++) is the physiologically active form. Low Ca++ concentrations result in a decrease in electrical resistance and an increase in membrane permeability (to sodium and potassium) of nerve tissue, which causes hyperexcitability of neural and muscle tissue and can result in spontaneous fiber discharge.

Calcium absorption occurs predominantly in the upper small intestine by an active transport system involving a calcium-binding protein. This is regulated by the active metabolite of vitamin D_3 in response to low plasma calcium levels. A lesser amount of absorption also occurs in the lower small intestines through passive diffusion. High-protein diets and acidification of the intestines aid calcium absorption. Compounds such as phytate (in cereal grains), oxalates (in spinach, rhubarb and related vegetation) and phosphates will decrease absorption of calcium due to the formation of complexes. Similarly, high intestinal concentrations of free fatty acids (from very high-fat diets or because of impairment in fat digestion) will result in the formation of insoluble calcium soaps. Once absorbed, calcium is carried by the plasma as ionized calcium, protein-bound calcium and a small amount of chelated calcium (chelated with citrate and phosphate). Regulation of calcium metabolism involves parathyroid hormone, calcitonin and vitamin D_3 (see Chapter 23).

The calcium content of dried, fat-free bone is approximately one-third of the total weight, predominantly present in the form of calcium phosphate, with lesser amounts of calcium carbonate. In egg shells, calcium carbonate is the structural compound. For maintenance of proper bone tissue, the calcium to available phosphorus ratio should be approximately 2 to 1. A range of 0.5:1 to 2.5:1 can be tolerated. The further this ratio deviates from the ideal level, the more critical proper vitamin D_3 levels become. Vitamin D_3 is essential to regulate absorption and metabolism of calcium and phosphorus, especially when dietary levels are unbalanced. During growth of most species, ratios of approximately 1:1 are required to support adequate growth, 1.5:1 to maintain normal serum calcium and phosphate and alkaline phosphatase values, and 2:1 to achieve maximum bone density. High egg-producing hens (poultry) may be provided with dietary ratios in excess of 10:1 in order to support daily shell production. This must not be confused with the significantly lower needs of a hen (most companion birds) that produces a periodic clutch of eggs. This ratio is based on the amount of phosphorus available to the bird, not the total phosphorus content of the diet. As much as 70% of the phosphorus in certain ingredients can be present in a form that is unavailable to the bird. Therefore, an estimation of the diet's available phosphorus is essential in order to balance these two minerals.

Levels of over 1.0% calcium in the diet have been observed to decrease the utilization of proteins, fats, vitamins, phosphorous, magnesium, iron, iodine, zinc and manganese. Where there are marginal intakes of one or more of these nutrients, increased calcium intake can induce a deficiency state.

Phosphorus

In addition to being an important bone constituent, phosphorus is also a component of proteins, carbohydrates and lipid complexes that perform vital functions in the body. Phosphorus has a wider range of biological functions than probably any other element.

Phosphorus is widely distributed in nature, occurring as phosphates, orthophosphoric acid salts and organophosphates. Absorption of phosphorus in the orthophosphate form takes place primarily in the duodenum, with efficiency of adsorption being dependent on the metabolic requirement and affected by a number of factors such as its source, calcium:phosphorus ratio, intestinal pH and dietary levels of vitamin D, potassium, magnesium, manganese, iron and fat. Once absorbed, it is readily incorporated into bone and other tissues, with bone acting as the metabolic reservoir. Like calcium, circulating levels are regulated by parathyroid hormone and calcitonin, with plasma levels being inversely related to plasma calcium levels. Excretion of excess amounts of phosphorus takes place primarily through the kidneys.

In plant sources, phosphorus is often complexed with phytin, making it unavailable to all monogastric animals because of their lack of the enzyme phytase. When the diet consists predominantly of high-phytin foods, phytase-producing microorganisms may colonize the gastrointestinal tract and provide a modest improvement in the phosphorus availability. This is low, however, because the amount of phytate hydrolysis is limited by the rapid transit time through the avian gut, with poor absorption of the liberated phosphorus due to hydrolysis occurring primarily in the distal portion of the tract. As a general rule, phosphorus from animal products or inorganic supplements is almost completely available, while that from plant sources is generally considered to be approximately 30% available. These typical values can be used to generate an estimation of the available phosphorus in the diet.

When kept within the range of acceptable calcium:phosphorus ratios, moderately higher phosphorus does not create a significant problem. Amounts of phosphorus outside these acceptable ratios, however, will cause decreased performance and will interfere with the absorption of calcium from the gastrointes-

tinal tract. Additionally, high serum phosphorus levels can induce nutritional secondary hyperparathyroidism by suppressing serum calcium, resulting in stimulation of the parathyroid. In some species, increased excretion results in the development of urolithiasis. It is estimated that the level of available phosphorus, when balanced with calcium and vitamin D, can be supplied at approximately two times the requirement without adverse effects. Amounts greater than this level have resulted in increased mortality in a number of species.

Magnesium

Most of the body's magnesium is present in the bone, complexed with calcium and phosphorus. In the body fluids, the majority of magnesium is found in the blood cells, whereas calcium is predominantly associated with the plasma. Magnesium (like potassium) is found at the highest concentrations in soft tissue cells (intercellularly) such as liver, striated muscle, kidney and brain. In these tissues, magnesium serves as an activator for many of the enzymes involved in phosphate transfer and metabolism.

Magnesium is absorbed in a manner similar to calcium and phosphorus, with the efficiency of absorption dependent on the concentration in the gastrointestinal tract. With low levels, absorption tends to be very efficient, with decreasing efficiency as levels become higher. Most of this mineral appears to be absorbed in the small intestine. Levels of calcium and phosphorus in a diet affect the magnesium requirement, with high levels of either of the former tending to increase the requirement of the latter.

Magnesium generally functions in enzyme systems by catalyzing the reaction through the formation of a metal-enzyme complex, where the magnesium ion is loosely associated with the enzyme.

Potassium

Potassium is widely distributed in most foods, making deficiencies unlikely in adult animals. Unlike sodium, potassium is located primarily intracellularly, and is found at the highest levels in muscle, erythrocytes, brain and liver. Potassium is the primary intracellular cation, affecting acid-base balance and osmotic pressure. It is also involved in protein biosynthesis, cellular uptake of amino acids and as a cofactor in a number of enzyme systems. In the extracellular fluids, potassium reduces muscle contractility and induces relaxation, therefore having the opposite effect of calcium.

Potassium is absorbed predominately in the upper small intestine by passive diffusion, although absorption occurs to a lesser extent throughout the entire intestinal tract. Excess potassium is excreted through the kidneys under the influence of sodium and aldosterone levels. Severe stress can create hypokalemia because of an increase in renal potassium excretion caused by elevated plasma proteins. This hypokalemia can be extended during the adaptation to the stress as potassium stores are replenished in the muscle and liver.

The minimum requirement of potassium is influenced by the dietary levels of sodium, total chlorides, the energy content of the food and possibly the protein content.

Potassium toxicity is not likely due to the capacity of the unimpaired kidney to excrete large concentrations of the mineral. Excesses of three times the required amount have presented no problems in avian species.

Sodium

Sodium is the primary cation of the extracellular fluid, and is predominantly responsible for the regulation of the body's acid-base equilibrium by associating with either chloride or bicarbonate. Sodium is critical in the maintenance of the proper osmotic pressure in the body, protecting against excessive fluid losses. It is also involved in the transmission of nerve impulses, the permeability of cells and acts to inhibit mitochondrial enzyme systems that are otherwise activated by the intercellular ions, potassium (K+) or magnesium (Mg++).

Sodium salts are readily and efficiently absorbed by the body (primarily in the ileum), and can be efficiently conserved when the dietary supply is limited. Excess sodium, on the other hand, can be efficiently excreted through the kidneys by an increase in water consumption. Sodium retention is regulated by the adrenal hormone, aldosterone, which maintains proper plasma sodium levels and regulates sodium excretion.

Depending on the species, bone will contain between 25 and 50% of the total body sodium, which is bound to the inorganic matrix of the bone. The rest of the sodium is predominantly found in the extracellular fluid of the body, with highest concentrations in plasma, nervous tissue and muscle tissue. Nearly all the species investigated show a sodium plasma content of 3.3 to 3.4 g/l. Total body sodium content is similar in all animals, ranging from 0.11 to 0.13%.

The body has a specific mechanism for concentrating sodium in the extracellular fluid while concentrating potassium in the intracellular fluid. This high concentration gradient is maintained by the sodium-potassium/ATPase pump system. This system transports Na+ out of the cell, while transporting K+ in. This is an energy-requiring process that uses intracellular ATP as an energy source. Intracellular sodium activates the enzyme system, which uses Mg++ as a cofactor.

In the presence of chronic renal disease, especially when the animal is in acidosis, sodium levels are depleted because of poor tubular resorption and the use of sodium for the buffering of acids. Both renal disease and diarrhea may cause sodium depletion. This will often be followed by a rapid loss in weight due to dehydration.

Moderate increases in dietary sodium are relatively nontoxic providing adequate (low sodium) water is provided for renal excretion. Levels of five to ten times the requirement can be provided before there is a decrease in growth and loss of appetite in a young bird. At all stages of life, there will be a considerable increase in water intake resulting in looser droppings. Higher levels of sodium intake result in poor feathering, polydipsia, polyuria, nervousness, edema, dehydration and mortality.

Chlorine

Chlorine, metabolically active as the chloride ion, is closely associated to sodium in foods, in the body and in metabolic processes, and both will be excreted under the same conditions. Chloride is also essential in maintaining the body's acid-base balance, osmotic pressure and water balance. It is a component of the hydrochloric acid that is produced by the body as a primary gastric secretion. In the body, chloride is concentrated in spinal fluid and blood.

It is critical to evaluate the overall dietary sodium, chlorine and potassium levels together. In the diet there must be a balance of the total sodium and potassium content with the total chloride and sulfate content in order to maintain the proper acid-base balance in the blood. This becomes particularly important with the addition of relatively high levels of dietary supplements that are complexed with one of these ions (such as high levels of choline chloride or lysine hydrochloride), especially when the chloride or sulfate form increases the acidity of the diet.

Toxicity of chloride alone is seldom a problem, but excess dietary chloride (in conjunction with unbalanced cations) can result in cartilage anomalies in chicks. Correction of the acid-base balance alleviates this symptom.

Essential Trace Minerals

Iron

The functions of iron in the body are almost entirely related to the cellular respiration processes. In the body, iron exists as heme iron (which is chelated with a porphyrin group) and non-heme iron (which is found bound to proteins). Iron is present in the body at approximately 50 to 100 parts per million.

Iron is unique in that body reserves are conserved and recycled very efficiently with negligible excretion. The primary method of iron depletion is through bleeding. Any iron found in the feces is generally a result of unabsorbed iron from the diet. Because the body has no normal pathway for the excretion of excess iron, intestinal absorption is carefully controlled to prevent accumulation. Under normal situations, the absorption of iron from the gastrointestinal tract is poor, however, if the body becomes marginally deficient, the absorption is improved until the situation is corrected.

Normally, heme iron (from animal sources) is considered to be approximately 20-25% available to the animal, while nonheme, vegetative sources are usually less than 5% available. Additionally, the nonheme iron present in most foods is in the ferric form (Fe^{+++}), which is poorly absorbed. This can be present either as the free ferric ion or loosely associated with an organic compound. In order for proper absorption to take place, ferric iron must be reduced to the ferrous state (Fe^{++}). In the ferrous form, iron becomes more soluble and therefore absorption is improved. This can be accomplished by any reducing compound in the food, with ascorbic acid being one of the more efficient agents. Proteins also enhance absorption, probably by forming soluble amino acids chelated with the iron. Additionally, absorption may be improved by dietary organic acids (eg, citrate, lactate), fructose and vitamin E, as well as by diets low in phosphorus. Normal gastric secretion is necessary to solubilize iron and increase its availability. Total iron absorption from a variety of mixed diets has been observed to range from 2 to 20% across a number of species. The deficient state can increase these efficiencies by over three-fold.

In the normal, healthy animal there should be no toxicity symptoms from moderate excesses of dietary

iron because of the efficient controls the body has over iron absorption and metabolism. Excess iron can reduce performance, however, by creating interactions with a number of nutrients. Examples of this would be reducing phosphorous absorption through the formation of an insoluble iron phosphate compound or the adsorption of vitamins or other trace minerals, preventing absorption into the body. Chronically high iron intake can result in elevated blood levels, increased tissue concentrations (especially of the liver and spleen) and the eventual development of hemosiderosis and possibly hemochromatosis (skin pigment changes). Liver damage and sometimes pancreatic fibrosis occur in this condition, which in other species is most often due to a genetic anomaly (extremely efficient absorption). Iron storage diseases have been predominantly seen in mynahs and toucans, possibly being caused by a combination of genetic and dietary factors.[16,49]

Copper

The copper content in the bodies of most species is approximately two parts per million. The largest concentrations are in the liver. Copper is a component of several proteins, enzymes and certain natural pigments. It is required for hemoglobin synthesis, proper collagen (bone), elastin and keratin formation and maintenance of the nervous system.

Copper is well distributed in normal feedstuffs, so the likelihood of a copper deficiency on a mixed, practical diet is not great. Availability can be affected by the chemical form as well as the copper status of the animal, with more efficient absorption occurring when the animal is deficient or when the dietary concentration is low.

Zinc

Zinc is critical to the animal for growth, reproduction and normal longevity because of its involvement in tissue repair and wound healing. It functions in a number of reactions in protein and carbohydrate metabolism, cell division and mucopolysaccharide formation. It also functions in the mobilization of vitamin A from the liver. Zinc is required in a large variety of enzymes, either as an enzyme activator or as a component of certain metalloenzymes.

Zinc is widely distributed in foodstuffs, but generally is not present in adequate supply to fill the needs of the young or producing animal. In plant sources, phytate can actively bind with inherent zinc, producing varying degrees of zinc availability. Some high-phytate ingredients, such as wheat bran or buckwheat, may also bind zinc from other dietary sources. Additionally, zinc requirements are increased with added calcium in the diet.

Manganese

Manganese is present in most plant sources at moderate to poor levels. Compounding the problem of marginal levels is its relatively poor availability. The formation of chelates appears necessary for the proper absorption of manganese, which occurs throughout the intestinal tract. Bile salts are important in the absorption, excretion and reabsorption of this mineral. Recycling appears to occur several times before the mineral is finally excreted in the feces. In addition to the constantly recycling pool in the intestines, the primary storage sites for manganese are bone, kidney and liver. High concentrations are also seen in the pituitary and pineal glands. At the cellular level, the mitochondrion is the principal site for manganese uptake. With high dietary intakes, the skin and feathers will accumulate large quantities of this element.

Manganese has several functions in the body. It is essential for normal bone structure, being required for the formation of the organic bone matrix through involvement in the synthesis of chondroitin sulfate (at two separate points in its synthesis).

Iodine

Iodine's sole metabolic function is for the biosynthesis of the thyroid hormones. Thyroid hormone functions to control the rate of energy metabolism in cells. In this way it influences growth and tissue differentiation or maturation, with resultant effects on other endocrine glands, neuromuscular function, skin and tissue development and nutrient metabolism.

Iodine is easily absorbed from the gastrointestinal tract in the reduced iodide state. Iodide is transported by loose attachments to plasma proteins. A large portion of the ingested iodide is excreted by the kidney, while the remaining amounts are taken up primarily by the thyroid gland. Small amounts can also be found in the salivary glands, stomach and other locations. The iodide uptake by the thyroid is stimulated by thyroid stimulating hormone (TSH) produced by the pituitary (see Chapter 23).

Moderate increases in dietary iodine do not present a problem because of the efficient excretion process in the body. Prolonged intake of high dietary levels of iodine causes reduced iodine uptake by the thyroid with antithyroidal or goitrogenic effects. Levels of

about 1000 times requirement are required before effects on growth, egg production or hatchability are seen in poultry.

Selenium

To a greater degree than other trace minerals, selenium content in foods is largely dependent upon the soil selenium content in which they were grown. Fortunately, some of the most productive agricultural states (for livestock foods) are in regions with adequate-to-high selenium soils (namely, the Great Plain states).

Because the consumption of "accumulator" plants by grazing animals caused blind staggers and death within a few days, this mineral was originally considered to be only toxic. Its essentiality was not recognized until 1957, when it was accidentally found to prevent liver necrosis in rats and exudative diathesis in chicks when studies were being conducted to determine minimum toxic intake levels.

The absorption of selenium is dependent upon its chemical form. The bioavailability of selenium in most plant products ranges from 60 to 90%, while in animal products it is less than 25%. Of the different chemical forms of selenium, selenite has the highest availability followed by selenomethionine, selenide and lastly, elemental selenium. The efficiency of absorption is also dependent upon the levels in the diet, with absorption higher during a deficiency situation. Once absorbed, selenium is carried in association with plasma proteins and transported to all tissues. Although selenium is distributed throughout the body, it is found in the highest concentration in the kidneys, pancreas, pituitary and liver. Other then the enzymatic form, there are no stores of selenium, making the selenium pool quite labile.

Selenium's metabolically active form is as a component of glutathione peroxidase. This enzyme is located in the aqueous phase of the cell and is responsible for oxidizing reduced glutathione, allowing it to act as a biological antioxidant. Reduced glutathione serves to protect membrane lipids and other cellular constituents by preventing oxidative damage by neutralizing any hydrogen peroxide and fatty acid hydroperoxides that are formed in the body.

Vitamin E and selenium are interdependent, each having the ability to spare the other. Selenium is important to the vitamin E status by preserving pancreatic integrity, maintaining normal fat digestion, micelle formation and vitamin E absorption. Selenium, as a part of glutathione peroxidase, destroys peroxides and prevents them from attacking the polyunsaturated fatty acids in cell membranes. This reduces the amount of vitamin E that is required to maintain the integrity of these membranes. Finally, selenium helps retain vitamin E in the blood plasma.

Conversely, vitamin E spares selenium by helping to prevent selenium loss from the body through its own antioxidant properties. By limiting the chain reaction destruction of membrane lipids, vitamin E minimizes the production of hydroperoxides, which would later require glutathione peroxidase to neutralize.

It is through these methods of sparing one another that selenium and vitamin E work together in the prevention of exudative diathesis. This disease is characterized by generalized edema (first appearing on the breast, wing and neck) due to abnormal capillary permeability, resulting in the leaking and accumulation of fluid. This is accompanied by decreased growth, leg weakness and mortality. Exudative diathesis has not been shown to occur except when both vitamin E and selenium are deficient.

The protection of lipid membranes from exposure to free radicals is not only important for the cell membrane, but also for the membranes of the mitochondria and microsomes. Because these act to both fuel and protect the cell, it is necessary for adequate vitamin E and selenium to be present for the cell to maintain its defense mechanisms.

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