CHAPTER
4

Nutritional Considerations

Section I: Nutrition and Dietary Supplementation

Section II: Nutritional Disorders

Nutritional Considerations Section I

Nutrition and Dietary Supplementation

DEBRA McDONALD, PhD, BSc (HONS I)

Selection or formulation of appropriate diets for companion and aviary birds is based on wild feeding ecology, digestive anatomy and physiology, and nutritional requirements of related species. Research indicates that requirements of some key nutrients for psittacines vary from those of poultry. Apart from vitamin E, there is no evidence to suggest that vitamin and trace mineral requirements for psittacines are greater than those recommended for poultry.[54] While there are substantial differences between production species and companion bird species, dietary requirements of poultry remain the standard for estimating the needs of companion birds. Individual nutrient classes will be discussed with particular focus on recent research into the nutritional requirements of companion birds. See Nutritional Disorders, Section II of this chapter for aspects of malnutrition and nutritional diseases commonly diagnosed in companion birds.

Water

Calculated water intake of adult Australian parrots does not correlate with observed water intake (Table 4.1.1).[37] Desert-adapted birds require less water intake than tropical birds. Changes in diet or environmental temperatures can alter water intake.

Table 4.1.1 | Water Intake Per Day of Various Birds

Species	Body Weight (g)	Calculated Water Intake (ml)	Actual Water Intake (ml)
Budgerigar[86]	30-35	0.7-0.8	4
Canary[85]	18-24	0.4-0.6	4
Lovebird[86]	55	1.3	10
Cockatiel[86]	100	2.4	13.6
Cockatoo[86]	300-900	7-22	15
Amazon/Grey[86]	350-600	8-14	17-35

WATER REQUIREMENTS OF PSITTACINE NEONATES

The ratio of feed to water to maximize survivability in the growing chick is dependent on age; insufficient water within the first few days after hatch leads to high mortality, and insufficient solids result in slow growth rates.[31] Studies of cockatiels indicate changes in the proportions of solids to water from 7:93 for the first 4 days after hatch, increasing to 30:70 thereafter.[65]

NUTRITIONAL SUPPLEMENTATION OF DRINKING WATER

Supplementation of vitamins and minerals via the drinking water is not recommended. Water intake can vary inter- and intraspecifically and is influenced by environmental temperatures and diet. The high redox potentials of minerals, such as zinc, iron and copper, can destroy vitamins, and some vitamins are light-sensitive. It is impossible to standardize intake of vitamins via drinking water. Vitamin A and D toxicoses have been reported in macaws and conures being supplemented with liquid vitamins.[7,67,78] Dehydration may result if the additives decrease water intake due to unpleasant taste or unfamiliar coloration.

Energy

When calculating energetic requirements of birds, the following equations are used:

Passerine BMR

$\text{kcal/day} = 114.8 \times \text{kg}^{0.726}$

$\text{kJ/day} = 480 \times \text{kg}^{0.73}$

Non-passerine BMR

$\text{kcal/day} = 73.5 \times \text{kg}^{0.734}$

$\text{kJ/day} = \text{kg}^{0.73}$

Basal metabolic rate (BMR) is calculated from energy expended when a bird is sleeping. Perching can increase energy expenditure in budgies 2-fold; preening, eating and shuffling locomotion cause 2.3-fold increase and flight increases as much as 11 to 20 times over BMR.[10] Energy requirements of free-living birds are greater than those of their captive counterparts due to the increased energy required for thermoregulation, food procurement and territorial defense. However, the daily needs for amino acids, minerals and vitamins are relatively constant regardless of energy expenditure.

Climate can influence BMR. Psittacines from temperate climates have BMRs approximately 20% higher than those of tropical species, with seasonal changes in thermoregulation varying from 3.07 times BMR in winter (5.9° C) to only 2.77 times BMR in summer (20.7° C).[10]

ENERGY REQUIREMENTS

Daily consumption of calories must exceed daily energy expenditure for a sustained period in order for overweight or obese body conditions to develop. A diet for weight loss should be replete in all nutrients so that protein, essential fatty acids, vitamins and minerals are present in amounts sufficient to support normal physiological processes and to retain lean body tissue. Reducing fat content of the diet too quickly or too far has led to obsessive eating behaviors in obese double yellow-headed Amazons.[22] Formulated calorie reduction diets generally contain lower levels of fat with simultaneous increase in indigestible fiber, air or moisture. Increased levels of dietary fiber slow gastric emptying. Insoluble fibers have a greater effect on slowing gastrointestinal tract (GIT) transit time than soluble fibers. Fiber also increases excretion of bile acids and fat.

ENERGETIC COSTS OF MOLT

Feather replacement requires energy and specific nutrients, as well as metabolic and physiological adaptations.[24] Energy costs of the molt include the caloric content of the new feathers and feather sheath, the energy required in their synthesis, and the energy required to produce and maintain feather pulps. Approximately 3 to 10% of total body mass (20 to 30% total lean body mass) of passerines is replaced during a complete molt.[24] Daily energy expenditure of passerines undergoing a rapid complete molt can increase from 3% (early and late in the molt) to 20% (at peak molt),[52] with BMR doubling in some passerines at peak molt.[34] Increases in energy expenditure due to rapid molt can be partly offset by reductions in other activities such as locomotion or singing.[28,34]

Feathers grow throughout the day and night at similar rates,[50] but the feather material deposited at night when most birds are fasting is of a slightly different quality. Feather synthesis at night requires complex and costly modifications to the metabolism of amino acids, compared to daytime synthesis, so the overall costs of molt may be lower in areas with relatively longer day length.[51]

Carbohydrates

Carbohydrates (Fig 4.1.1) are used to produce energy in the form of adenosine triphosphate (ATP) from glycolysis

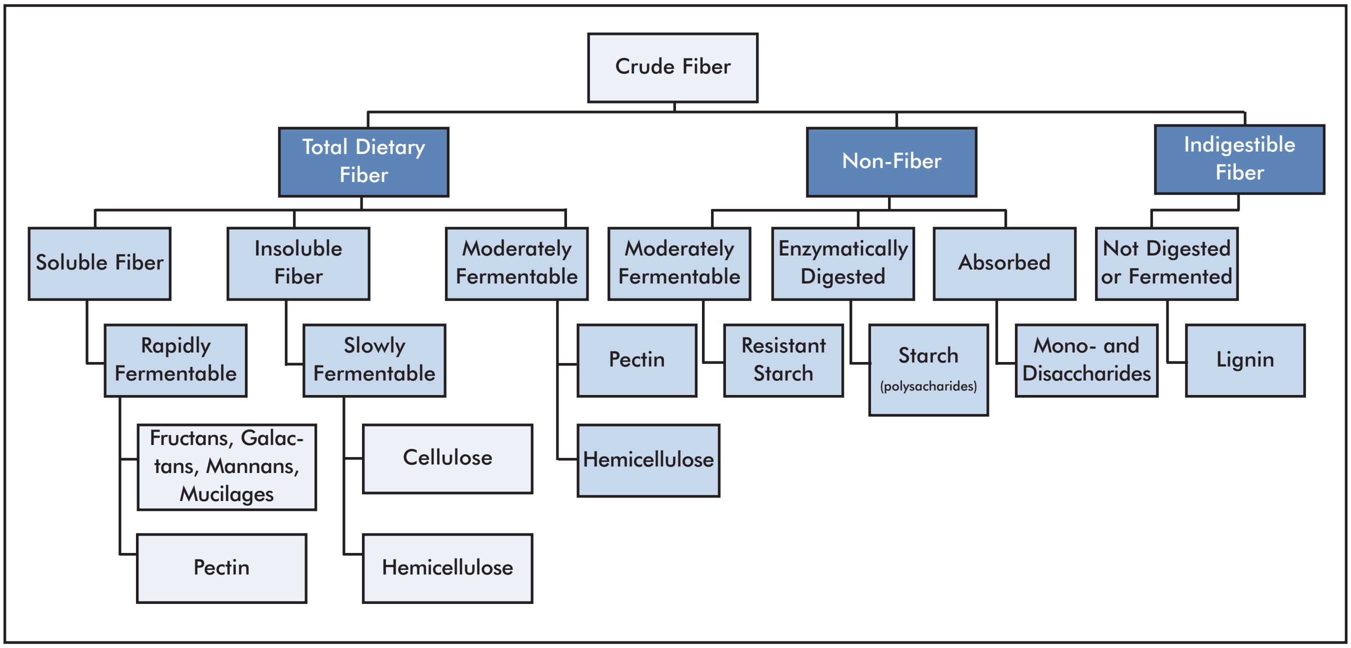

Fig 4.1.1 | Classification of carbohydrates.

Table 4.1.2 | In the Absence of Adequate Dietary Carbohydrate, Glucogenic Amino Acids are used to Manufacture Carbohydrates

- Alanine
- Arginine
- Asparagine
- Aspartic Acid
- Cysteine
- Glutamic Acid
- Glutamine
- Glycine
- Histidine
- Methionine
- Phenylalanine
- Proline
- Serine
- Threonine
- Tryptophan
- Tyrosine
- Valine

and the TCA (tricarboxylic acid) cycle, and produce heat from the oxidation of glucose to CO_2 and H_2O. They are also used to produce precursors of other nutrients, synthesize glycogen or fat from glucose, decrease luminal pH through production of short-chain fatty acids, and increase the population of anaerobic flora. The antibacterial properties of short-chain fatty acids may decrease pathogenic intestinal bacteria and may be important in prevention of, and recovery from, intestinal disorders.

The central nervous system and erythrocytes require glucose for energy, in contrast to muscles that can utilize substrates such as fatty acids. In the absence of adequate dietary carbohydrates, amino acids (glucogenic amino acids via the gluconeogenic pathway) are shunted away from growth and production to be used for glucose synthesis (Table 4.1.2).

SUCRASE AND FRUIT SUGARS

Sucrose, one of the predominant disaccharides of fruit sugars (Figs 4.1.2, 4.1.3), is easily digestible. However, some insectivorous passerines, such as thrushes that feed on diets high in protein/fat and low in carbohydrate, lack the sucrase enzyme necessary for the digestion of these simple sugars. The differences in proportions of fruit mono- and disaccharides are important for species that lack the sucrase enzyme. Avoid feeding these birds fruits high in disaccharides such as mango, apricot, nectarine and peach.

POLYSACCHARIDES

While simple sugars such as the monosaccharides and disaccharides are readily absorbed, the α-bonds of starches require further digestion. Heat from extrusion processes gelatinizes the starch molecule, increasing its digestibility. The ß-bonds of some of the complex carbohydrates, such as those comprising the fibrous fraction of foods, require microbial degradation that may not be sufficient in psittacines and passerines. Cellulose is composed of ß-bonds and is generally unavailable as a source of energy, while hemicellulose consists of varying proportions of α- and ß-bonds, depending on the source. Hemicellulose is therefore partially digestible without microbial breakdown, but its close association with the polyphenolic lignin often makes it indigestible. The carbohydrate content of psyllium fiber consists predominantly of hemicelluloses, so is partially digestible.

Fermentation of Polysaccharides

Postgastric microbial fermentation of polysaccharides occurs in nectarivorous, frugivorous and florivorous species. The process of enzymatic digestion taking place prior to fermentation benefits species that feed on easily digested foods such as nectar and fruit, as fermentation of nutrient-rich foods is not energetically efficient. In contrast, pregastric fermentation occurs in the hoatzin, which feeds on mature leaves, stems and branches.[18] Such bulk foods are generally incompatible with flight.

Chitin

Chitin is a naturally occurring polysaccharide similar in structure to cellulose with an additional amino group. It is the principal polysaccharide of cell walls of fungi and

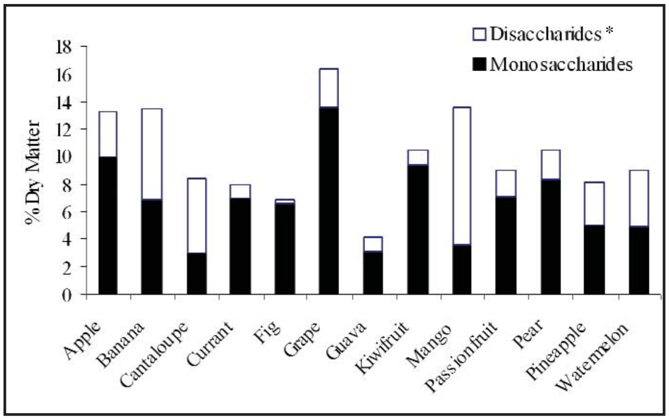

Fig 4.1.2 | Sugar content of fruits commonly fed to birds.
*Require sucrase for digestion

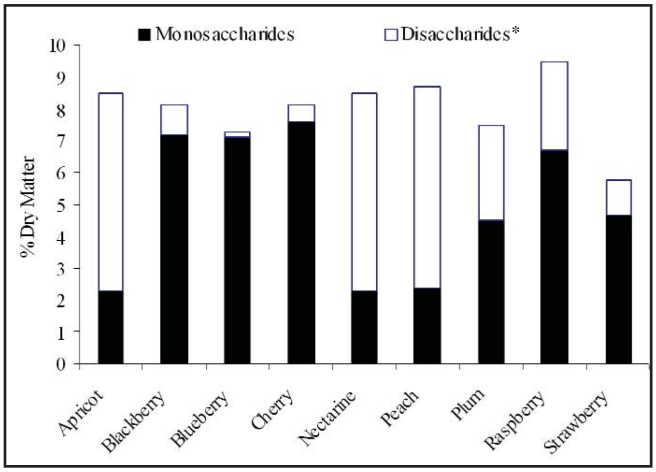

Fig 4.1.3 | Sugar content of stone fruits and berries.
*Require sucrase for digestion

Table 4.1.3 | Chitin Content of Various Invertebrates and Fungi

Food Item	Chitin Content
Fungi	5-20%
Worms	20-38%
Squid	3-20%
Scorpion	30%
Spider	38%
Cockroach	35%
Crab	70%

the primary constituent of the exoskeletons of crustaceans and invertebrates (Table 4.1.3). The digestion of chitin is considered low, but it still presents a useful energy source for some species. While chitinase activity has been identified in starlings, raptors and a variety of seabirds, it is low in chickens and absent in African grey parrots and pigeons. Measurements of crude protein may overestimate protein availability for birds that lack chitinase enzymes.

Protein

Proteins are composed of the nitrogen-containing molecules, amino acids. They can be manufactured from dietary precursors (non-essential) or are required as dietary constituents (essential). Amino acids that are deemed to be essential for birds include arginine, isoleucine, leucine, lysine, methionine, phenylalanine, valine, tryptophan and threonine. In addition, glycine is known to be essential for budgerigars (*Melopsittacus undulatus*),[79] and histidine and proline are essential for chickens. It is presumed that the digestion of proteins by psittacines reflects that of poultry. Lorikeets digest only 13.3% of complete protein (egg white).[16] Digestion of proteins is more efficient in nestlings than in adult birds.

MEASUREMENT OF PROTEIN CONTENT OF FOODS

Protein values of food (crude protein) are measured using the Kjeldahl technique with the assumption that all nitrogen in the food sample is present as protein and that all proteins contain 16% nitrogen. Crude protein is calculated as follows:

$$\text{CP } (\%) = \text{g N/kg} \times 6.25$$

While this value is commonly used for converting nitrogen values to protein, it is not applicable to all foods, especially some seeds.

DIETARY REQUIREMENTS FOR PROTEIN AND AMINO ACIDS

The dietary requirement for protein varies with age and physiological state, being highest in hatchlings and females laying large clutches, and lowest in adults at maintenance. In granivorous birds protein requirement is correlated with body size,[29] with larger species such as macaws possibly requiring more protein than smaller birds. Preliminary studies with African grey parrots (*Psittacus erithacus erithacus*) indicate a protein requirement of 10 to 15% (dry matter).[27] Protein requirement for the adult budgerigar is 6.8% (balanced protein diet).[14] Sunflower seeds, safflower seeds and peanuts are deficient in the amino acids required by poultry.[54]

High fiber diets can increase fecal nitrogen content due to bacterial digestion, which can confound the results of a protein digestion study.[63] Nectarivorous and frugivorous species have lower obligatory protein losses. Rainbow lorikeets (*Trichoglossus haematodus*) may require as little as 2.9% protein when fed high-quality, readily digested protein.[16]

While high dietary protein has been implicated in renal dysfunction and gout in psittacines, studies of male cockatiels (*Nymphicus hollandicus*) indicate that protein levels of 20, 30 or even 70% do not result in renal insufficiency.[32] However, 70% dietary protein is not recommended, as excessively high protein is strongly correlated with a significant increase in liver lesions.[32] Excess protein has been associated with overgrowth of beaks and nails.[35] Introduction of birds to new diets with varying levels of protein should be undertaken gradually, as sudden changes in dietary protein levels may result in nephritis and gout.[32] Since birds are uricotelic, an overload of the excretory ability of the kidneys, caused by excessive intake of proteins or nucleic acids, may lead to hyperuricemia. Dehydration exacerbates this problem and may produce visceral and articular gout even without excessive dietary protein.[35]

PROTEIN REQUIREMENTS FOR REPRODUCTION

Protein requirements during egg production are influenced by clutch size/frequency and the protein composition of eggs. There is little taxonomic variation in amino acid composition of avian eggs.[39,49] Protein requirements for species that lay only single eggs are similar to maintenance requirements, but that may increase if essential amino acids are lacking. Birds laying eggs require dietary amino acids for maintenance, growth of the oviduct and accretion of egg proteins, at least a week prior to the first oviposition.[29,49] Budgerigars can maintain breeding performance on 13.2% protein. However a diet of white millet, canary seed and hulled oats (13.4% protein) containing only half the necessary lysine, methionine and cysteine is not sufficient to support reproduction.[1]

PROTEIN REQUIREMENTS FOR CHICKS

Protein requirements for growth are highest at hatch and decrease over time as growth rate slows. Altricial chicks have a higher fractional growth rate and may have higher total amino acid requirements. Protein requirement for growth of cockatiels has been estimated at 20%. In addition, the protein must include 0.8 to 1.5% lysine.[64,65] Many birds in the wild supplement their diets with insects, which will often provide additional protein; however, some wild birds just increase their food intake. While there is generally sufficient crude protein, lysine and arginine in commercial hand-rearing mixes for psittacines, most lack sufficient quantities of the sulphur amino acids, methionine and cysteine, which leads to stunted feather growth.[85]

PROTEIN REQUIREMENTS FOR FEATHERS

Feathers comprise a large percentage of total body protein (22% lovebirds; 28% budgies) with approximately 15% by mass contained in the sheath.[50] However, the amino acid composition of whole plumage differs from that of the sheath and calamus.

Dietary deficiencies in some of the sulphur amino acids cause a pronounced curvature and periodic restriction of the rachis, abnormal persistence of the basal sheath, and misshapen vanes.[50] Feather strength is correlated with adequate dietary lysine (0.203%).[80] Cysteine is abundant in the epidermal structure and feather barbs.[51] While cysteine is frequently cited as a potentially limiting nutrient in the growth of plumage, cysteine reserves may be sufficient for keratin synthesis during overnight periods of fasting and short-term sulphur amino acid deficiencies. Dietary deficiencies of methionine result in dark, horizontal “stress lines” on feathers,[38] while threefold excesses are correlated with soft, weak feathers.[80] Tyrosine is an important factor in melanogenesis, and a deficiency in phenylalanine also impairs melanogenesis.[83]

Production of sheaths during molt can increase protein requirements 4 to 8% per day above maintenance requirements.[51] The additional energy required for thermoregulation may increase food intake to provide sufficient protein for feather growth without increasing the proportion of protein in the diet. A number of products on the market promoted as supplements during periods of molt are deficient in the required amino acids.[87]

Lipids

Lipids supply energy, essential fatty acids and facilitate the absorption of fat-soluble vitamins. In addition, they are precursors of many hormones and eicosanoids.

CLASSIFICATION OF FATS

Fats are described by the length of the carbon chain, the number of double bonds and the exact position of the double bonds. Short-chain fats contain 2 to 4 carbons, medium-chain fats 6 to 10 and long-chain fats 12 to 24 carbons. Fatty acids with 4 to 12 carbons are found in milks; those with 10 to 12 carbons are found in certain seed oils. Longer chain fatty acids are common in plants of marine origin.

ESSENTIAL FATTY ACIDS

Saturated fatty acids (SFA) are those where all carbons of the fat are satisfied with a single bond to another element. If one double bond is introduced, they are monounsaturated fatty acids (MFA). Those with two or more double bonds are polyunsaturated fatty acids (PUFA). The preferred nomenclature for fatty acids is based on the position of the double bond from the methyl end **(Fig 4.1.4)**. For example, linoleic acid 18:2 (*n*-6) contains 18 carbons with two double bonds, placed six carbons from the last double bond with reference to the terminal methyl end. Higher evolved animals are unable to manufacture fatty acids of the *n*-3 or *n*-6 families and must obtain these from dietary sources. Fatty acid composition of seeds and nuts that are available for companion birds may not resemble that of wild diets **(see Fig 4.1.6)**.

PUFA profiles are not amenable to dietary manipulation. Grains and seeds are generally rich in linoleic acid (*n*-6), grasses and leaves in α-linolenic acid 18:3 (*n*-3) and docosahexaenoic acid (*n*-3) (DHA). Fish and other aquatic insects are rich in 20:5 (*n*-3) or 22:6 (*n*-3).

FATTY ACIDS AND EICOSANOIDS

Certain membrane fatty acids have specific roles in the regulation of cell functions. Arachidonic acid (AA), γ-linolenic acid and eicosapentaenoic acid (EPA) act as precursors for the synthesis of eicosanoids, an important group of immunoregulatory molecules that function as local hormones and mediators of inflammation. Changes in characteristics of fatty acids available to cells modify the fatty acid composition of the membrane phospholipids of those cells and may influence inflammatory processes **(Fig 4.1.5)**.

Cis and *Trans* Fatty Acids

While most natural fatty acids occur in the *cis* form (carbons on same side), processing such as extensive heat or partial hydrogenation can convert fats to the *trans* form

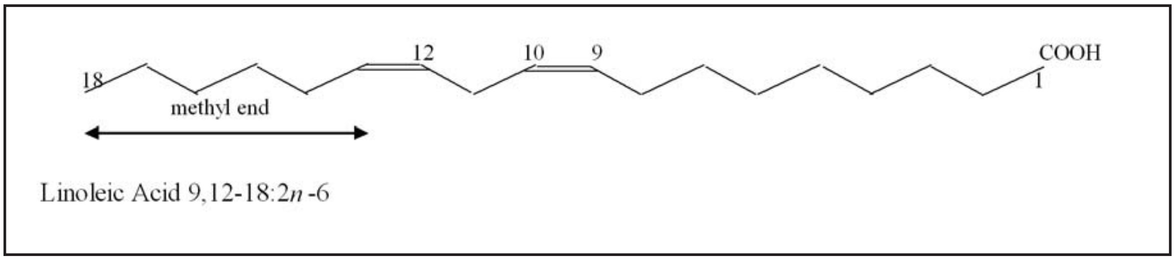

Fig 4.1.4 | Structure of *n*-6 fatty acid.

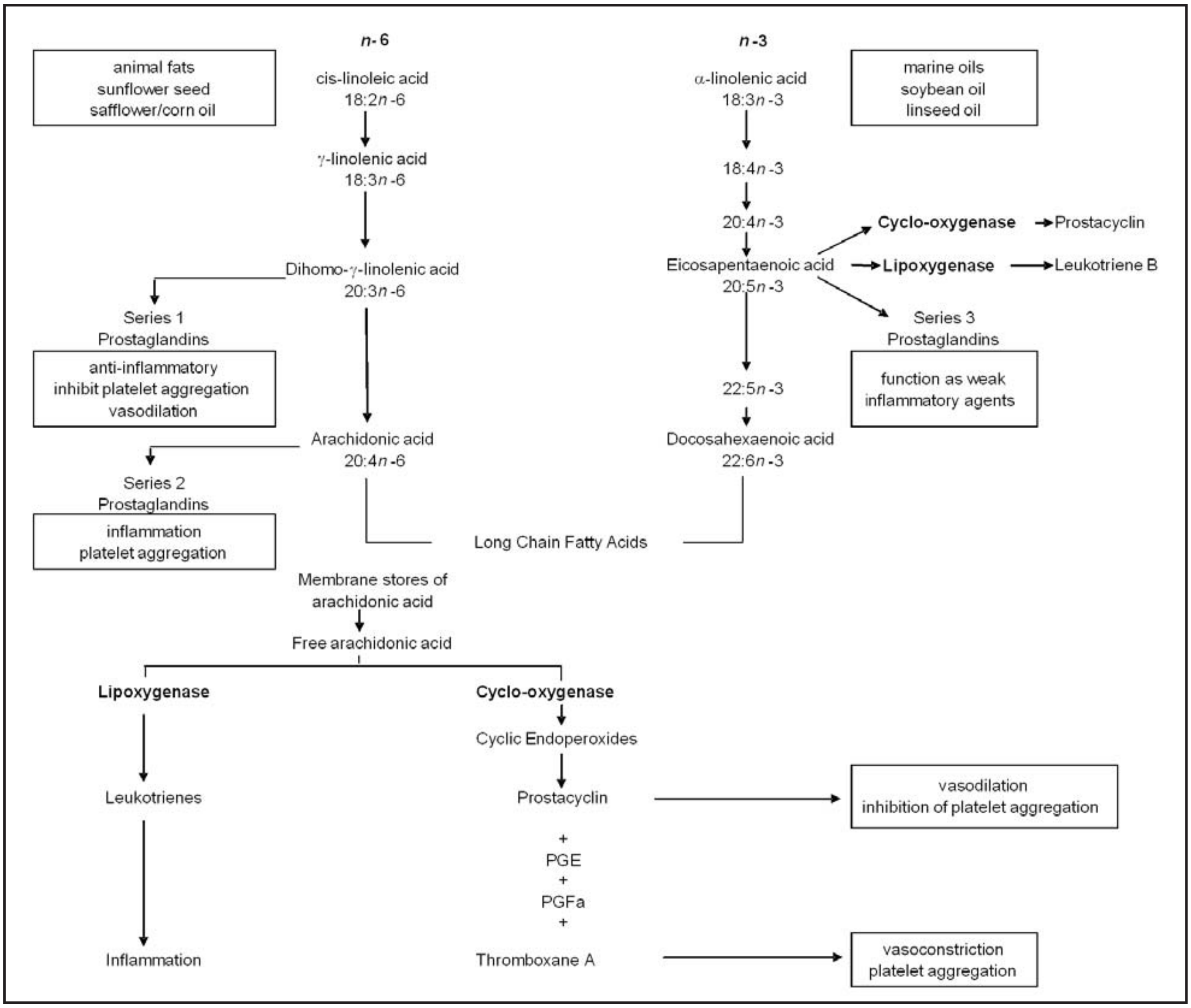

Fig 4.1.5 | Production of eicosanoids from essential fatty acids.

(carbons on opposite sides), which affects their biological activity. Although both *cis* and *trans* forms are metabolized for energy, *trans* isomers cannot function as essential fatty acids.

Antioxidants and Lipid Peroxidation

Insufficient antioxidants such as vitamin E in the feed also may enhance lipid peroxidation during storage.[48] Diets high in PUFA require additional antioxidant protection to prevent rancidity. There are a number of naturally occurring substances in food that have antioxidant properties including vitamins A, C, E, and yellow-colored carotenoids such as ß-carotene.

Antioxidants help to counter the detrimental effects of oxygen-free radicals. Oxygen-free radicals have been implicated in the development of cancer, inflammatory conditions and heart disease. A deficiency of antioxidants may promote peroxidation of membrane phospholipids.

OBESITY

Obesity can lead to congestive heart failure or hepatic lipidosis and may predispose a bird to diabetes mellitus or exacerbate this illness. Body weight relative to a bird's

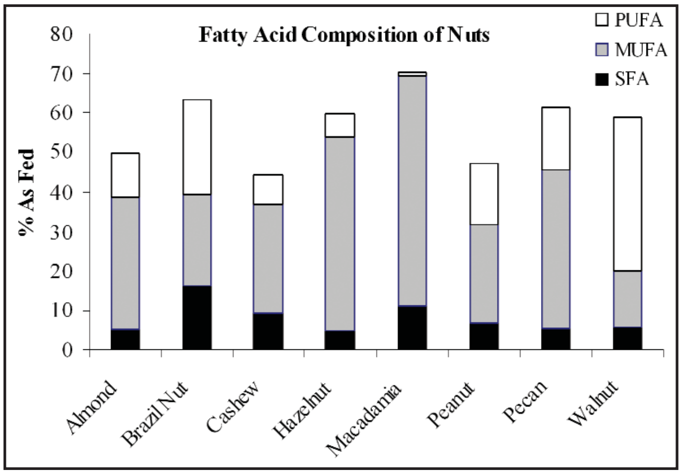

Fig 4.1.6 | Fat composition of nuts commonly fed to birds.

Table 4.1.4 | Lipid Content of Egg Yolk of Various Birds

Species	Chicken	Gull	Pigeon
Mode	Precocial	Semi-precocial	Altricial
Triacylglycerol	71.4%	35.1%	58%
Phospholipid	20.7%	24.6%	30.7%
Cholesterol Ester	0.8%	1.1%	5%
Free Cholesterol	5.6%	5.9%	4.7%

Table 4.1.5 | Fatty Acid Composition of Seeds of Wild Food Plants of the Orange-bellied Parrot (*Neophema chrysogaster*)[44]

	Linoleic Acid C18:2n-6	α-linolenic Acid C18:3n-3	AA C20: 4n-6	EPA C20: 5n-3	DHA C22: 6n-3
Introduced Mainland					
Atriplex prostrata	0.04	0.13	0.27	0	0.30
Cakile maritima	0.06	23.8	0	0	1.23
Chenopodium glaucum	0.06	5.28	0.14	0.02	0.06
Indigenous Mainland					
Halosarcia pergranulata	0	1.6	0	0	0
Samolus repens	0	1.53	0	0	0
Sarcocornia quinqueflora	0	2.0	0	0	0
Suaeda australis	0.23	2.39	0	0	0.04
Indigenous Tasmania					
Baumea tetragona	0	4.48	0	0	0
Gahnia grandis	0	0.37	0	0	0
Restio complanatus	0	3.55	0	0	0
Commercial	0	1.42	0	0	0.08

optimal weight has been used as a defining criterion for obesity because body weight is easier to measure than body fat. Body weight in excess of optimal body weight of 1 to 9% is acceptable, 10 to 19% is considered overweight and greater than 20% is defined as obese.

DEPOSITION OF FATS IN EGG YOLK

The yolks of precocial and altricial birds vary in lipid composition (Table 4.1.4). However, determination of specific fatty acid dietary requirements has been undertaken only on granivorus species. In contrast to fatty acid profiles of commercial grains, fatty acid composition of wild seeds on which the orange-bellied parrot (*Neophema chrysogaster*) feeds is characterized by a distinct lack of *n*-6 fatty acids (Table 4.1.5).[44]

BRAIN LIPIDS

There is a surge of brain growth in the second half of the embryonic/early neonatal stage, with specific uptake of docosahexaenoic acid (DHA) by embryonic brain tissue. The selective depletion of yolk phospholipid DHA results in a range of cognitive, behavioral and visual impairments. The high proportions of amino acids in brain tissues imply a requirement for adequate levels of both *n*-3 and *n*-6 fatty acids in yolk lipids. While the avian embryo may be able to synthesize DHA from α-linolenic acid, this ability may be species-specific. Yolk lipids of the domestic chicken maintained on formulated diets are almost devoid of α-linolenic acid, while those of the goose, pheasant and ostrich on similar diets have high levels of this fatty acid, providing sufficient precursors for conversion to DHA.

SPERMATOZOA LIPIDS

The high PUFA content of avian semen predisposes them to lipid peroxidation. Susceptibility is increased in membranes high in DHA, which is present in duck spermatozoa and can be exacerbated by low vitamin E. However, similarities in lipid peroxidation of chicken and ducks suggest the presence of high levels of antioxidant enzymes. Age-related decreases in sperm output are reduced with supplementation of 200 mg/kg vitamin E. Supplementation with longer chain essential fatty acids is also beneficial.

Vitamins and Supplementation Requirement

WATER-SOLUBLE VITAMINS

Water-soluble vitamins include the B complex and vitamin C. As dietary requirements for B vitamins have not been evaluated for companion birds, further discussion will be confined to disease entities that specifically implicate imbalances in the B vitamins (see Section II Nutritional Disorders).

Vitamin C

Vitamin C (ascorbic acid) is involved in the syntheses of collagen, carnitine and catecholamine; in tyrosine, histamine, steroid, fatty acid and drug metabolism; and in the prevention of peroxidation. Birds under stress, including the stresses associated with high temperatures, growth and reproduction may have increased requirements for vitamin C.

Sources of Vitamin C

Most birds synthesize vitamin C in the kidney, liver or both. Evolutionarily, enzymatic activity occurs in the kidneys of birds in older orders and becomes localized in the liver of more advanced Passeriformes. Some passerines such as the vented bulbul (*Pycnotus* sp.) are unable to synthesize vitamin C. Vitamin C is concentrated in fresh fruits, green leafy vegetables and animal organs, with only small amounts in skeletal muscle.

Vitamin C Deficiency

Reproduction and growth increase the demand for collagen. Supplementation of 100-200 mg/kg vitamin C improves growth, egg production and eggshell strength of young chicks and hens exposed to high environmental temperatures.[68] Dietary requirements also may vary with age, as willow ptarmigan (*Lagopus lagopus*) adults are able to synthesize sufficient vitamin C, whereas chicks require supplementation with 750 mg/kg.[21] Berries that form part of the diet of the willow ptarmigan during the breeding season can contain up to 5000 mg/kg vitamin C.[21]

Vitamin C is susceptible to destruction with handling and processing. While it is stable when exposed to boiling water for short periods, a greater proportion is destroyed when heated at low temperatures for long periods. Any form of processing that ruptures tissue (such as freezing and thawing) exposes vitamin C to air losses due to oxidation, but vitamin C is generally stable during normal pelleting processes.

Vitamin C Toxicity

Metabolites of L-ascorbic acid such as oxalic acid can bind calcium. Excesses of vitamin C also can bind copper, resulting in growth deficiencies and increases in the incidence of aortic rupture and decreases in elastin content of the aorta if diets are also deficient in copper. It is important to minimize dietary vitamin C content for species that are susceptible to iron storage disease, as vitamin C improves the absorption of iron by facilitating the reduction of the ferric form to the more absorbable ferrous state.

FAT-SOLUBLE VITAMINS

It is important to maintain an appropriate balance with the fat-soluble vitamins as they all compete for sites of uptake. Dietary excess of one vitamin can diminish uptake and availability of another, despite adequate dietary intake.

Vitamin A

Vitamin A is involved in vision, reproduction, immunity, membrane integrity, growth, embryogenesis and the maintenance of epithelial cells. Vitamin A is of animal origin and does not occur in plant tissues. Some carotenoids can be converted to vitamin A in the intestinal wall via a specific enzyme.

Forms of Vitamin A

The most active form (retinol) is susceptible to moisture, heat and light. The retinal aldehydes are incorporated in rhodopsin and influence dim light vision. Retinoic acid supports growth and tissue differentiation but not vision. Although retinoic acid appears to play a role in testosterone synthesis, it does not support the production of sperm.

Dietary Requirement

Vitamin A requirements vary among production species **(Table 4.1.6)**. Dietary requirements of cockatiels (*Nymphicus hollandicus*) at maintenance are 2000-4,000 IU/kg.[30] Levels below 10,000 IU/ kg do not significantly influence plasma levels in cockatiels.[30] Dietary deficiencies of vitamin A may not impact immunocompetence for up to eighteen months in birds previously maintained on sufficient dietary Vitamin A. Dietary vitamin A can be adequately provided from 2.4 mg/kg ß-carotene in cockatiels. Recessive white canaries (*Serinus canaria*) are unable to convert ß-carotene to vitamin A and require three times as much vitamin A as colored canaries.[88]

Sources of Vitamin A

The vitamin A content of animals varies. Vitamin A levels in invertebrates are extremely low **(Table 4.1.7)**.[4] Fish store large amounts of vitamin A in the liver and fatty tissue. Supplementation with cod liver oil is not recommended. Seeds and nuts are generally low in carotenoids **(Table 4.1.8)**, while some fruits can provide large quantities **(Table 4.1.9)**.

The vitamin A content of many formulated products, most parrot foods and nectar-replacement products **(Fig 4.1.7, Table 4.1.10)** exceed dietary recommendations for poultry[54] and cockatiels.[30] While the vitamin A content of some products is higher than data reported by manufacturers, poor packaging may result in breakdown of the vitamin.[41]

Table 4.1.6 | Vitamin A Requirements of Birds, Expressed as IU/kg of Feed Dry Matter

	Growing	Laying
Chicken[54]	1,670	4,440
Turkey[54]	5,550	5,550
Quail, Coturnix[54]	1,830	3,670
Duck, Pekin[54]	2,780	4,440
Goose[54]	1,670	4,440
Cockatiel (Maintenance)[32]	2,000	

Table 4.1.7 | Fat-soluble Vitamin Content of Various Invertebrates Commonly Fed to Birds

Invertebrate	Vitamin A (IU/kg)	Vitamin E (mg/kg)
Mealworm, larvae	811	30
Cricket, adult[3]	811	80
Cricket, juvenile[3]	471	70
Earthworm, wild-caught[4]	2400	70
Earthworm, commercial[4]	328	230
Fruit Fly[4]	0	23
Waxworm[4]	150	500

Table 4.1.8 | Carotenoid Content of Nuts/Seeds

Nut/Seed	Carotenoid (RE/g)
Flax	0
Safflower	0.53
Sesame	0.09
Sunflower	0.53
Almond	0
Brazil	0
Hazel	0.71
Macadamia	0
Peanut	0
Walnut	1.29

Table 4.1.9 | Carotenoid Content of Fruits[41]

Nut/Seed	Carotenoid (RE/g)
Apple	3.3
Banana	3.15
Cantaloupe	315.0
Grape	3.76
Honeydew	3.87
Kiwi Fruit	10.32
Mango	212.9
Raspberry	9.68
Strawberry	3.2
Watermelon	43.11

Data reprinted with permission from Elsevier Science[41]

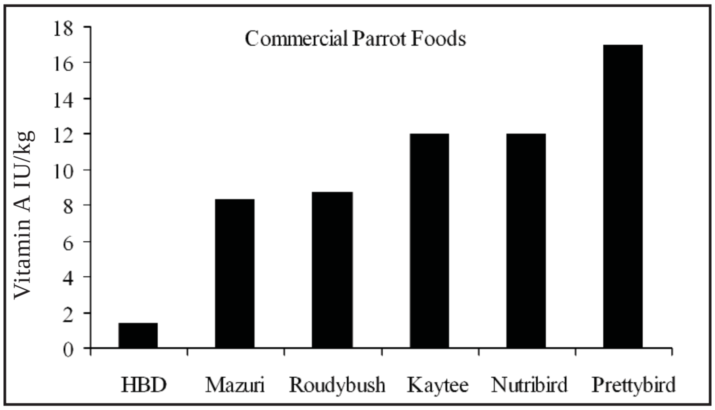

Fig 4.1.7 | Vitamin A content of various commercial parrot foods.

Table 4.1.10 | Vitamin A Content of Various Commercial Nectar Products[41]

Product name	Vitamin A (IU/kg)	Vitamin E (mg/kg)
Aristopet*	5,994	6
Aves Nectar†	24,150	22
Avione*	4,296	20
Elliots Dry*	666	2.8
Elliots Wild nectar*	666	1.3
HBD Adult Lifetime Fine (Maintenance)‡	1,400	215
HBD High Potency Fine (Breeding)‡	1,500	240
Lory Life Nectar§	52,900	54
Lory LifePowder§	10,130	11
Marion Lory¶	8,500	250
Nekta Plus§	12,470	12
Nekton Lori†	60,550	34
Nekton Lori and Gelb†	244,820	136
Noah's Kingdom§	330	25
Passwells*	9,990	29
Quicko Nectar§	400	5
Rainbow Landing Lorikeet Nectar§	22,467	45
Roudybush 15% Protein§	19,500	33
Roudybush 9% Protein§	18,860	81
Sheps Lori dry*	167	1.7
Sheps Wet*‖	12,500 (333)	25 (1.8)
Wombaroo Nectar†¶	26,640 (28,740)	89 (27)

Data reprinted with permission from Elsevier Science[41]

*Independent laboratory analyses, Australia 2002

†Author's laboratory analyses, 1999

‡Independent laboratory analyses, USA 2002

§Laboratory analyses (Graffam, 1999)

¶ Manufacturer's data, 1999

‖Manufacturer's data, 2002

Measuring Vitamin A

Approximately 90% of total-body vitamin A is contained in the liver.[46] Storage levels of 2-5 IU vitamin A/g liver are deemed to be adequate.[46] Plasma retinol does not change dramatically with marginal vitamin A status, and reduced levels are generally not detected until animals reach a severe deficiency. It is not adequate to evaluate vitamin A status from blood samples. Liver biopsies are recommended.

Interpreting Laboratory Data

Units are reported in *international units* (IU) for vitamin A or *retinol equivalents* (RE) for provitamin A.

The equivalencies are as follows:

0.6 µg ß-carotene is equivalent to 1 IU provitamin A activity

1 RE = 1 µg retinol = 6 µg ß-carotene or 12 µg of other provitamin A carotenoids

1 RE = 3.33 IU retinol = 1.818 IU vit. A palmitate = 10 IU ß-carotene

Data that is reported as µg/dl retinol can be converted to SI units (µmol/L) as follows:

µg/dl x 0.0349 = µmol/L

Approximately 10% of the more than 500 carotenoids have provitamin A activity.

Signs of Vitamin A Deficiency

Clinical signs of vitamin A deficiency often resemble those of toxicity, and distinguishing between the two requires careful evaluation of dietary intake and other influencing factors. While turkey chicks exhibit signs of deficiency after only five weeks[2], cockatiels can be maintained on a diet devoid of vitamin A for up to two years before demonstrating clinical signs.[30] Signs of vitamin A deficiency can generally be classified into five categories:

Vision

Retinol is utilized as the prosthetic group in iodopsin (cones). Vitamin A deficiency results in a loss of opsin, the protein that converts vitamin A to rhodopsin, from the outer segments of the rods, leading to their eventual degeneration. Even in the late stages of vitamin A deficiency, it is possible to regenerate rods, but cones eventually disintegrate and result in blindness. Vitamin A deficiency also results in decreased secretions of tear glands.[40]

Bone

A deficiency results in reduced activity of osteoclasts, leading to excessive deposition of periosteal bone by the unchecked function of osteoblasts.

Maintenance of Epithelial Tissue

Dietary deficiencies of vitamin A can alter the permeability of lipoprotein membranes of cells and intracellular particles. Low levels of liver vitamin A are correlated with symptoms of focal metaplasia of the excretory duct as well as the glandular epithelium of salivary glands. Loss of membrane integrity also interferes with water retention, and renal uric acid deposits can result. Coccidiosis can lead to the destruction of vitamin A in the gut and injure microvilli of the intestinal wall, decreasing the absorption of vitamin A.[69] Vitamin A deficiency in chicks is characterized by poor feathering on the head, neck and breast regions, as well as facial dermatitis.

Reproduction

Defects in reproduction, including increased time between clutches, reduced hatchability, increased embryonic mortality, decreased survival time of progeny, decreased testes size, failure of spermatogenesis and a decline in sexual activity in males, are correlated with deficiencies of vitamin A. These may be associated with failure to maintain healthy epithelium.[17]

Immune Function

Both deficiency and excess of dietary vitamin A suppress immune function. Vitamin A deficiency in chicks leads to a rapid loss of lymphocytes. Diarrhea, pneumonia and blunted immune response are characteristic of vitamin A deficiency in cockatiels.[33] Deficiencies lead to phagocytic activity in macrophages and neutrophils, and impair intestinal IgA response.[33]

Vitamin A Toxicity

In the wild, noncarnivorous birds are rarely exposed to dietary excess of vitamin A. These birds probably depend on the conversion of carotenoids to biologically active vitamin A. Toxicities are avoided because the efficiency of conversion of vitamin A from ß-carotene decreases with higher levels of intake. Conversion efficiency in the chicken drops from a ratio of 2:1 to 5:1.[46] Studies of Japanese quail also indicate a saturation of the retinol-transporting system, as birds supplemented with ß-carotene do not develop increased levels of retinyl palmitate.[60] Cockatiels at maintenance are more susceptible to vitamin A toxicity than deficiency.[30] Perhaps ß-carotene would be a superior source of vitamin A in some psittacine diets; however, ß-carotene may not be appropriate for hand-rearing mixes because chicks may not efficiently convert ß-carotene to vitamin A. Toxicities have been reported in cockatiels maintained at 10,000 IU/kg of vitamin A. Many commercial diets exceed this level.[30] Some hand-rearing diets have levels in excess of 47,000 IU/kg.[85] In lorikeets, commercial diets high in vitamin A and deficient in vitamin E are correlated with high rates of infertility, decreased hatching and survivability of chicks.[42] The author hypothesizes similar levels may contribute to the increased incidence of iron storage disease in these birds. Vitamin A toxicity causes epithelial damage and keratinization of squamous cells.[33] Epithelial damage results from penetration of retinol into the lipid portion of the membrane, causing it to expand. Weakening of the membrane results from the inelastic protein portion of the membrane resisting expansion and increases access to pathogens and infection. Clinical signs of Vitamin A toxicity include:

Vocalization Patterns

Cockatiels maintained on excess dietary vitamin A exhibit frequent stress calls of greater intensity and duration.[30] Vitamin A toxicities may contribute to behavioral problems in companion birds. Vocalization changes were observed in psittacines maintained on diets that contained recommended levels of vitamin A.[47,75]

Iron Storage Disease

See Section II Nutritional Disorders for the potential contribution of excess vitamin A to iron storage disease. Splenic hemosiderosis has been correlated with excess vitamin A in cockatiels.[30]

Pancreatitis

Pancreatitis was diagnosed in cockatiels fed excessively high levels of vitamin A.[33] Hypervitaminosis A increases the activity of sucrase and eliminates the duodenum's ability to regulate this enzyme in the small intestine[71] which may lead to diabetes and digestive difficulties.

Table 4.1.11 | Ultraviolet Light

Type of light	Distance (ft)	UVA (microwatts/cm^2)	UVB* (microwatts/cm^2)
Reptisun 5.0	1	23	10
Vitalite	1	6	1.6
Blacklight	1	153	2.6
Active UV Heat			
100 watt flood	1	400	50
100 watt flood	2	110	12.5
160 watt flood	1	640	85
160 watt flood	1.5	480	55
160 watt flood	2	320	25
160 watt flood	3	142	11
275 watt flood	2	720	66
275 watt flood	2.5	520	48
275 watt flood	3	320	30
275 watt flood	4	180	20
100 watt spot	2.5	1130	70
100 watt spot	3	562	40
100 watt spot	4	400	30
160 watt spot	3	1500	137
160 watt spot	4	1200	100
Location	**Time**	**Direct/ Shade**	**UVB** (microwatts/cm^3)
Natural Sunlight			
Equator	noon	direct sunlight	265
Germany	noon	direct sunlight	175
Yucatan	noon	direct sunlight	250
Illinois	noon	direct sunlight	260
Illinois	7:00 am	direct sunlight	12
Illinois	7:00 pm	direct sunlight	17
Illinois	1:00 pm	shade	54
Illinois	5:00 pm	shade	22

*UVB is the biologically active ultraviolet light

Reproduction

Excesses of vitamin A may interfere with uptake of vitamin E, compromising fertility, hatchability and survivability of chicks.

Antioxidant Status

Vitamin A supplementation of laying hens increases liver, egg yolk and embryonic liver concentrations at the expense of vitamin E, compromising the antioxidant status of progeny. High levels of vitamin A reduce uptake of astaxanthin, a powerful carotenoid antioxidant that protects mitochondria from damage by Fe^{+2} catalysed lipid peroxidation.

Vitamin D

Vitamin D is a group of closely related compounds that possess antirachitic activity. They are obtained directly from the diet or from irradiation of the body. The two major natural sources (provitamins) are cholecalciferol (D_3 in animals) and ergocalciferol (D_2, predominantly in plants). Both D_2 and D_3 forms also can be ingested and further metabolized to 25-hydroxyvitamin D_3 through hydroxylation first by the liver, and then again to 1,25-dihydroxyvitamin D_3 in the kidneys. Cholecalciferol can be produced in the skin of most mammals from provitamin 7-dehydrocholesterol via activation with ultraviolet light in as little as 11-15 minutes daily. Vitamin D enhances intestinal absorption and mobilization of calcium and phosphorus through the hormone 1,25-dihydroxyvitamin D_3. Circulating levels of 25-hydroxyvitamin D_3 are indicative of vitamin D status. One IU of vitamin D activity is equivalent to the activity of 0.025 μg vitamin D_3. As vitamin D_2 has only 1/10 the activity of vitamin D_3 in chicks the International Chick Unit (ICU) is used with reference to vitamin D_3 in poultry. Plasma half-lives vary with the form of the vitamin ranging from 5 to 7 days (vitamin D) to 20 to 30 days (25-$(OH)D_3$) (see Chapter 5, Calcium Metabolism). In mammals and reptiles, activation depends on UVB radiation (290-320 nm) **(Table 4.1.11)**.

Table 4.1.12 | Vitamin D and Calcium Content of Various Commercial Foods*

Product	Manufacturer	Vitamin D_3 (IU/kg)	Calcium (%)
Avipels	Blue Seal	4170	1.11
Bird of Paradise	Zeigler	3970	1.34
Bird of Prey (frozen)	Animal Spectrum	3470	1.05
Chick Starter	Blue Seal	4000	1.19
Crane	Mazuri	10830	2.62
Exotic Game Bird	Mazuri	2500	0.89
Flamingo	Mazuri	6670	1.72
HPC	HBD International	150	0.69
Nutribird Parrot	Nutribird	1200	0.9
Palm Cockatoo	SSP	1900	1.1
Psittacine Breeder	Roudybush	1560	1.0
Psittacine Handfeeder	Roudybush	1560	1.0
Psittacine Maintenance	Roudybush	890	0.44
Scenic Bird	Marion	1600	1.2
Poultry	NRC	200	0.99
Turkey	NRC	900	0.5

*From manufacturer's published data 1999-2002.

Dietary Requirement

The dietary requirement for vitamin D in poultry is 200 IU/kg. While higher dietary requirements are evident for the turkey (900 IU/kg) and Japanese quail (1200 IU/kg), optimum levels for companion birds have yet to be established. It has been suggested that dietary levels for poultry are adequate for breeding African grey parrots[76] **(Table 4.1.12)**, but many formulated foods exceed these levels.

Vitamin D Deficiency

Vitamin D synthesis can be affected by liver malfunction; intestinal disorders can reduce absorption of the vitamin and kidney failure can prevent synthesis of 1,25-$(OH)_2D_3$. Inadequate exposure to UVB radiation prevents production of vitamin D in the skin. Glass windows block the penetration of UVB rays. The first signs of vitamin D deficiency include decreased egg production, thinning or absence of eggshells, and an increased incidence of embryonic death. Inadequate maternal transfer of vitamin D_3 and 25-(OH)D_3 results in the failure of development of the upper mandible and failure to pip.

Vitamin D Toxicity

Vitamin D toxicity may arise from an excess of dietary vitamin D or a deficiency in the other fat-soluble vitamins. Toxicity leads to widespread calcification of soft tissue. Toxic levels can be transferred maternally to the embryo, leading to abnormalities in chick development. Safe upper limits in chicks less than 60 days old are 40,000 IU/kg and 2800 IU/kg in birds older than 60 days.[69]

Vitamin E

Vitamin E consists of tocopherols and tocotrienols in four isomeric forms, α, ß, δ, and γ. α-tocopherol has the highest vitamin E activity followed by ß, δ and γ, respectively. Dietary requirements of vitamin E are dynamic, with increased requirements with diets high in PUFA, oxidizing agents, vitamin A, carotenoids, trace minerals and decreased requirements in diets high in other fat-soluble antioxidants, sulphur-containing amino acids and selenium. Vitamin E is one of the least toxic vitamins, however, high doses decrease absorption of vitamins A, D and K, resulting in reduced hepatic and egg yolk storage of vitamin A,[77] impaired bone mineralization[20] and coagulopathies.[53] Studies of pelicans indicate that 500-10,500 IU vitamin E/kg result in decreased growth and coagulopathy.[53] Japanese quail under heat stress (34° C) require 250 mg/kg vitamin E and 0.2 mg/kg Se.[66] Various researchers have recommended up to 60 mg α-tocopherol per gram of PUFA. Many formulated foods have less than 200 mg/kg of vitamin E. Vitamin E status can be evaluated from single blood samples, but the magnitude of body stores may not be reflected in α-tocopherol concentrations.[45,81] High dietary concentrations of vitamin E elevate vitamin E levels in the blood. Deficiencies affect the neuromuscular, vascular and reproductive systems.

Vitamin K

Vitamin K plays a major role in blood clotting factors and is involved in the synthesis of osteocalcin, with deficiencies resulting in increased bleeding times and toxicities resulting in kidney tubule degeneration. Vitamin K is available as phylloquinone (K_1) from plants, menaquinone (K_2) from bacteria and menadione (K_3) which is synthetic. Vitamin K_1 is present as the fat-soluble portion of plant chlorophyll (Table 4.1.13). An energy-dependent process absorbs vitamin K_1 from the intestine, whereas vitamins K_2 and K_3 are passively absorbed. Estrogens stimulate the absorption of vitamin K_1. Vitamin K_3 has twice the potency of natural vitamin K_1 on a weight-to-weight basis. Vitamin K_3 is the most common form in commercial bird foods. Choline can impact the activity of water-soluble K_3, destroying up to 80% within 3 months. γ-irradiation of foods to increase storage life also inactivates vitamin K, while heat treatment can increase its bioavailability (Table 4.1.14).

Table 4.1.13 | Vitamin K Content of Fruit on a Dry Matter Basis

Fruit	Vitamin K (mg/100 g)
Apple peel, green	60.0
Apple peel, red	20.0
Apples, no skin	0.4
Avocado, raw	40.0
Banana, raw	0.5
Grapefruit, raw	0.02
Grapes, raw	3.0
Kiwifruit, raw	25.0
Melon, raw	1.0
Orange, raw	0.1
Peach, raw	3.0
Pineapple, raw	0.1
Plum	12.0

Table 4.1.14 | Influence of Heat Treatment on Vitamin K Content on a Dry Matter Basis

Vegetables	Vitamin K (mg/100 g) raw	Vitamin K (mg/100 g) cooked
Broccoli	205	270
Carrot	5	18
Cauliflower	5	10
Coriander leaf	310	1510
Mint leaf	230	860
Parsley	540	900

Table 4.1.15 | Conversion of Carotenoids to Vitamin A Relative Rat-biopotency[46]

Carotenoid	Biopotency
α-carotene	25
β-carotene	100
γ-carotene	14
Cryptoxanthine	29

It has been suggested that mortality from cerebral hemorrhage in some species of fig parrots (*Opopsitta* spp.) is the result of dietary deficiency of vitamin K.[11] If fig parrots have developed a dependency on vitamin K_2 produced by the gut microbes of termites, they may be unable to process sufficient vitamin K_1 from plant sources. Daily supplementation of 300 μg vitamin K_1 per fig parrot appears to alleviate clinical signs.[11]

Nutritional Influence on Feather Pigmentation

The dietary pigments utilized by passerines for their coloration are referred to as carotenoids. Psittacines do not use carotenoids for feather pigmentation. Each carotenoid produces a specific color. Carotenoids are subdivided into carotenes and xanthophylls (Table 4.1.15). Carotenoids may be used directly in the plumage or modified to other forms prior to incorporation into the feathers or skin (Fig 4.1.8, Tables 4.1.16-4.1.18). Each carotenoid appears to have its own individual pattern of absorption, plasma transport and metabolism. There are considerable species differences in the types of carotenoids that are preferentially absorbed and metabolized. Many carotenoids act as potent antioxidants and stimulate the immune system.

The yellow pigmentation of the helmeted honeyeater

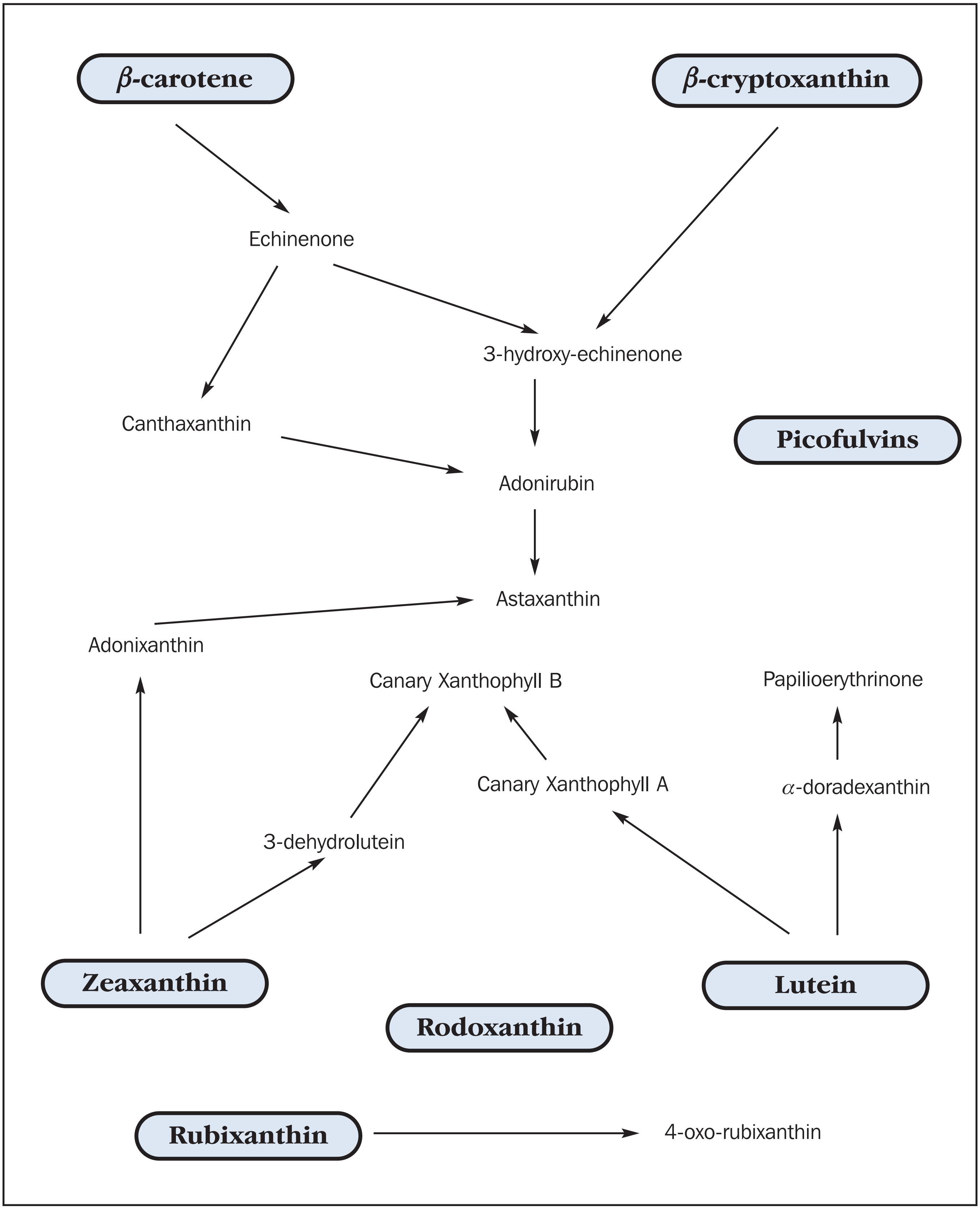

Fig 4.1.8 | Metabolic pathways for various dietary carotenoids. Rodoxanthin and picofulvins are deposited directly from the food into the feathers and are not metabolically transformed.

Table 4.1.16 | Metabolic Pathways of Various Carotenoids Responsible for Pink-colored Feathers of Passerines

Species Name	Common Name	Feather Pigment	Metabolism	Original Pigment	Feather Pigment
Aegithalos caudatus	Long-tailed tit	3-hydroxy-echinenone	oxid	ß-crypto/ß-carot	pink
Carduelis cannabina	Common redpoll	3-hydroxy-echinenone	oxid/hydrox	ß-crypto/ß-carot	carmine red
		4-oxo-rubixanthin	oxid	rubix	
		4-oxo-gazaniaxanthin	oxid	rubix	
Carduelis flammea	Linnet	3-hydroxy-echinenone	oxid/hydrox	ß-crypto/ß-carot	carmine red
		4-oxo-rubixanthin	oxid	rubix	
		4-oxo-gazaniaxanthin	oxid	rubix	
Carpodacus roseus	Pallas's rosefinch	3-hydroxy-echinenone	oxid/hydrox	ß-crypto/ß-carot	bright pink
		4-oxo-rubixanthin	oxid	rubix	
		4-oxo-gazaniaxanthin	oxid	rubix	
Fringilla coelebs	Chaffinches	3-hydroxy-echinenone	oxid	carots	copper-pink
		4-oxo-rubixanthin			
		dehydrolutein			
		astaxanthin			
Pyrrhula pyrrhula	European bullfinch	α-doradexanthin	oxid	ß-carot	pinkish-red
		astaxanthin			
		adonirubin			
Rhodospiza obsoleta	Desert finch	canthaxanthin	oxid	ß-carot	pink

Oxid=oxidation, hydrox=hydroxylation, carots=carotenoids, ß-crypto=ß-cryptoxanthine, ß-carot=ß-carotene, rubix=rubixanthin

Table 4.1.17 | Metabolic Pathways of Various Carotenoids Responsible for Red-colored Feathers of Passerines

Species Name	Common Name	Feather Pigment	Metabolism	Original Pigment	Feather Pigment
Bombycilla cedrorum	American waxwing	rodoxanthin	direct	rodox	red
Carduelis carduelis	Gold finch (face mask)	canary xanthophylls B/C/D	dehydrog	lut/keratin	red
Carduelis cucullata	Red siskin	α-doradexanthin	oxid	lut	red
		canthaxanthin			red
Colaptes auratus	Northern flicker	astaxanthin	oxid	carots	red
		lutein/zeaxanthin	direct	lut/zea	yellow
		ß-cryptoxanthin	direct	ß-crypto	yellow
Dendrocopos major	Great spotted woodpecker	astaxanthin	oxid	lut/zea/carots	red
		α-doradexanthin			
		adonirubin			
Hirundo rustica	Swallow	lutein	direct	lut	red
		zeaxanthin	direct	zea	
		3-hydroxy-echinenone	oxid	ß-crypto/ß-carot	
Loxia curvisrostra	Common crossbill (m)	3-hydroxy-echinenone	oxid	ß-crypto	red
Loxia leucoptera	White-winged crossbill	4-oxo-rubixanthin	oxid	rubix	red
		4-oxo-gazaniaxanthin		gazan	
Luscinia calliope	Siberian rubythroat	astaxanthin	oxid	carots	ruby red
		α-doradexanthin			
		adonirubin			
Pericrocotus flammeus	Scarlet minivet (m)	astaxanthin	oxid	carots	red
		α-doradexanthin			
		adonirubin			
		canthaxanthin			
Picus viridis	Green woodpecker	α-doradexanthin	oxid	lut	red
		picofulvins		lut/zea/ß-crypt	green/yellow
Pinicola enucleator	Pine grosbeaks (m)	3-hydroxy-echinenone	oxid	ß-crypt	red
		4-oxo-rubixanthin		rubix	
Pyrrhula erythaca	Grey-headed bullfinch	canary xanthophylls A/B	dehydrog	lut	orange-red
Serinus pusillus	Red-fronted serin	canthaxanthin	oxid	ß-carot	red
Trichodroma muraria	Wallcreeper	astaxanthin	oxid	zea	red
Uragus sibiricus	Long-tailed rosefinch	3-hydroxy-echinenone	oxid	ß-crypt/ß-carot	red

Rodox=rodoxanthin, lut=lutein, zea=zeaxanthin, gazan=gazaniaxanthin

Table 4.1.18 | Metabolic Pathways of Various Carotenoids Responsible for Yellow-colored Feathers of Passerines

Species Name	Common Name	Feather Pigment	Metabolism	Original Pigment	Feather Pigment
Bombycilla garrulus	Bohemian waxwing	canary xanthophylls (tail)	dehydrog	lut/zea	yellow
		astaxanthin (wing patches)		ß-caro	
C. sinica/C. spinoides	Asiatic finches	canary xanthophylls	direct	lut	yellow
Carduelis atrata	Black siskin	canary xanthophylls A/B	dehydrog	lut	yellow
Carduelis carduelis	Gold finch (wing bars)	canary xanthophylls B/C/D	dehydrog	lut	yellow
Carduelis chloris	Greenfinch	canary xanthophylls	dehydrog	lut	yellow
Carduelis citrinella	Citril finch	canary xanthophylls A/B	dehydrog	lut	yellow
Carduelis spinus	Siskin finch	canary xanthophylls A/B	dehydrog	lut	yellow
Emberiza citrinella	Yellowhammer	lutein/zeaxanthin	direct	lut/zea	yellow
E. melanocephala	Black-headed bunting	lutein/zeaxanthin	direct	lut/zea	yellow
Fringilla coelebs	Chaffinches (secondaries)	lutein	direct	lut	yellow tinge
Fringilla coelebs	Chaffinches (rump)	lutein	direct	lut	
Leiothrix lutea	Peking robin	dehydrolutein	dehydrog	lut/zea	yellow
		α-doradexanthin	oxid	carots	
		astaxanthin	oxid	carots	
Loxia curvisrostra	Common crossbill (f)	canary xanthophylls A/B	dehydrog	lut	yellow
Motacilla flava	Yellow wagtail	lutein	direct	lut	yellow
		zeaxanthin		zea	
Oriolus oriolus	Golden oriole	lutein	direct	lut	yellow
		zeaxanthin	direct	zea	yellow
		oxo-carotenoids	oxid	lut/zea	bright yellow
Parus ater	Coal tit	lutein	direct	lut	yellow
		zeaxanthin		zea	
Parus ceruleus	Blue tit	lutein	direct	lut	yellow
		zeaxanthin		zea	
Parus major	Great tit	lutein	direct	lut	yellow
		zeaxanthin		zea	
Pericrocotus flammeus	Scarlet minivet (f)	lutein/zeaxanthin	direct	lut/zea	yellow
Pinicola enucleator	Pine grosbeaks (f)	lutein	direct	lut	yellow
		dehydrolutein	dehydrog	zea	
Regulus regulus	Goldcrest	lutein	direct	lut	yellow/green
		zeaxanthin	direct	zea	orange
Serinus mozambicus	Yellow-fronted canary	canary xanthophylls A/B	dehydrog	lut	yellow
Serinus serinus	Serin	canary xanthophylls	dehydrog	lut	yellow

(*Lichenostomus melanops cassidix*) (Fig 4.1.9) is replaced with near white pigmentation (Fig 4.1.10) when maintained on commercial nectar mixes high in vitamin A. Feather structure and light refraction can influence feather color. Carotenoids also can act synergistically with melanin pigments. Dark colors (black, brown, gray and related tints) produced by melanin and porphyrin pigments complexed with trace minerals are influenced by amino acid nutrition. Stress can influence feather quality and color.

Minerals

CALCIUM AND PHOSPHORUS

Adequate dietary calcium can be negated by high phosphorus content. The dietary ratio of Ca:P should range from 1:1 to 2:1.[61] Calcium availability can be influenced by solubility and particle size. Foods high in oxalic acid form insoluble calcium oxalates, while phytates bind phosphorus and decrease its availability. Additional oxalic acid can be produced from excesses of vitamin C. Fats can form insoluble calcium soaps.

The calcium content of nuts and seeds is variable (Fig 4.1.11). Some green leafy vegetables and tubers that are high in calcium also are high in oxalic acid (Figs 4.1.12, 4.1.13), which decreases the calcium availability.

Invertebrates generally have poor Ca:P ratios (Table 4.1.19). Crickets should be maintained on 80% poultry mash and 20% calcium carbonate. Water should be provided *ad lib* from produce (such as a slice of apple) or free water. If die-off of crickets occurs from constipation, restrict gut loading to 48 hours prior to feeding. When

Fig 4.1.9 | Helemeted honeyeater, full pigmentation (in-house nectar mix).

Fig 4.1.10 | Helemeted honeyeater, light pigmentation (commercial nectar mixes).

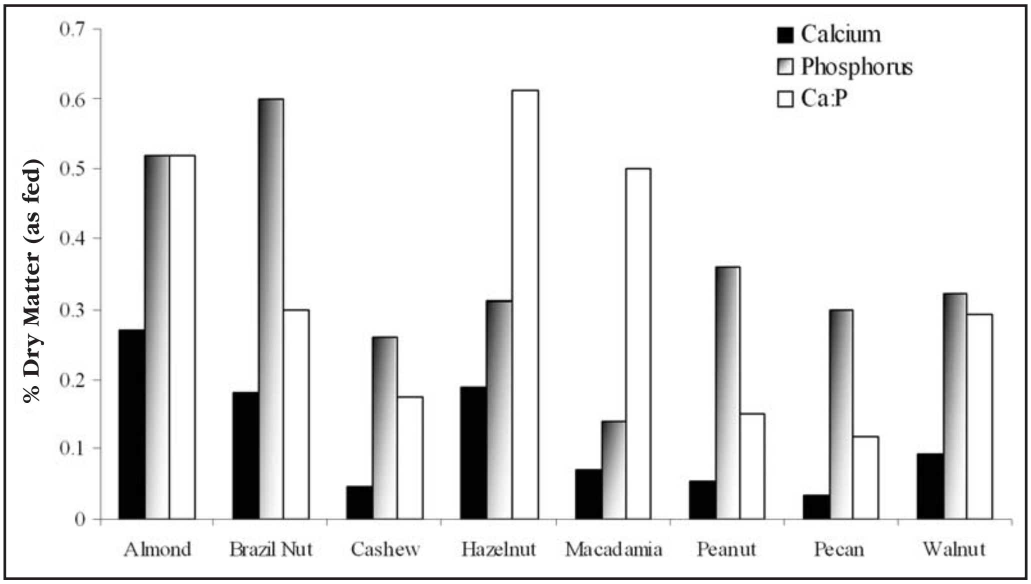

Fig 4.1.11 | Calcium and phosphorus content of nuts and seeds.

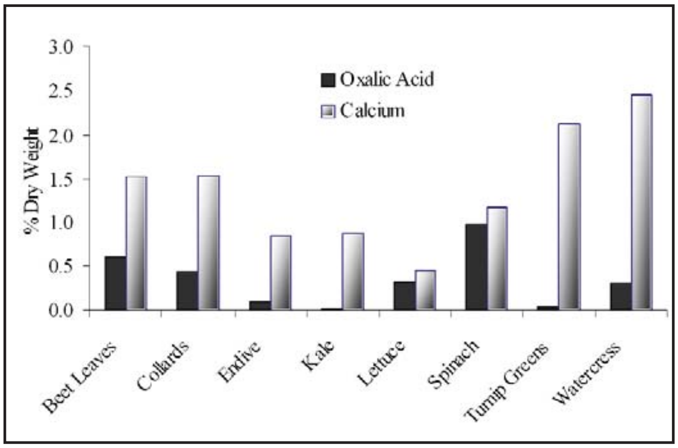

Fig 4.1.12 | Oxalic acid and calcium content of greens.

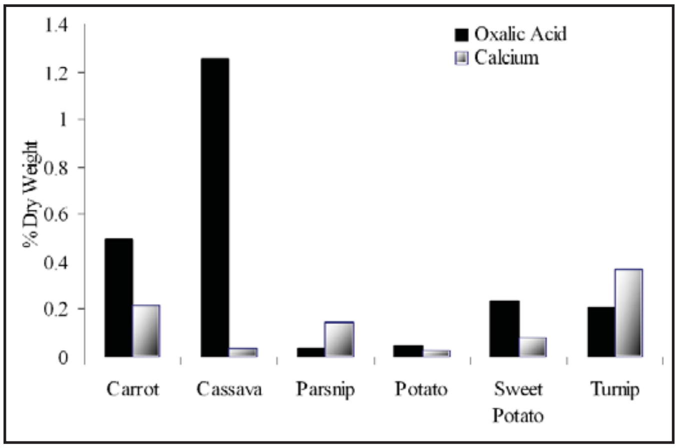

Fig 4.1.13 | Oxalic acid and calcium content of tubers.

Table 4.1.19 | Calcium and Phosphorus Content of Invertebrates

Invertebrate	Part	Calcium (% DM)	Phosphorus (% DM)	Ca:P
Bogong Moth (*Agrotis infusa*)	Abdomen	0.64		
	Wings	0.17		
	Whole	0.25		
Cricket (*Acheta domesticus*)	Adult	0.21	0.78	0.27
	Pinhead	1.29	0.79	1.63
Fruit fly (*Drosophila melanogaster*)	Pupae	0.77	2.73	0.28
	Larvae	0.59	2.3	0.26
	Adult	0.1	1.05	0.10
Mealworm (*Tenebrio molitor*)	Larvae	0.11	0.77	0.14
	Beetle	0.07	0.78	0.09
	Pupae	0.08	0.83	0.10

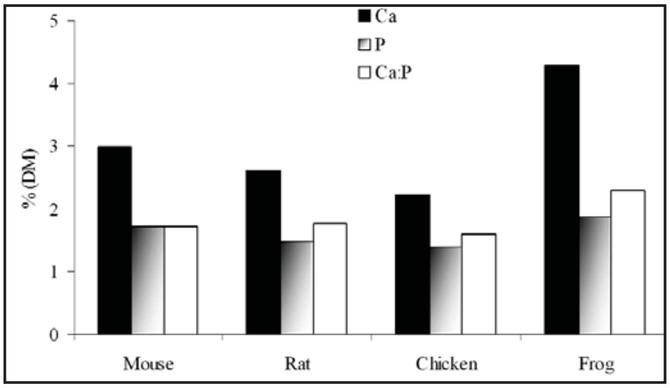

Fig 4.1.14 | Calcium and phophorus content of whole vertebrate prey.

feeding whole vertebrate prey, it is not necessary to supplement with calcium (Fig 4.1.14).

It has been suggested that optimum levels of calcium in feed are between 0.3 to 0.7%.[58] Many wild seeds provide 0.1 to 0.3% calcium (Table 4.1.20). Companies continue to provide formulated feeds in excess of 0.7%. It is possible that the high calcium content of formulated foods contributes to the development of renal disease that is observed in color mutation cockatiels.

IRON

Body iron is either hemal or non-hemal (Table 4.1.21). Hemal iron forms part of the porphyrin group and comprises 70 to 75% of total iron. Iron from animal sources is generally more available than that from plant sources. Iron in soy isolates may be unavailable. Pectin, vitamins A and C, and amino acids such as cysteine, histidine and lysine enhance iron uptake. High levels of calcium and/or phosphate decrease iron absorption in chicks. Cellulose and oxalate, as well as the heat and pressure of food processing, increase the bioavailability of iron. (Tables 4.1.22-4.1.25).

Table 4.1.20 | Calcium Content of Wild Food Resources of the Orange-bellied Parrot[44]

Species	Common Name	Calcium (%)	Seed Weight (mg)
Mainland Indigenous			
Halosarcia pergranulata	Black-seed glasswort	0.1	0.3
Samolus repens	Creeping brookweed	0.68	0.017
Sarcocornia quinqueflora	Beaded glasswort	0.28	0.3
Suaeda australis	Austral seablite	0.08	0.4
Introduced Species			
Atriplex prostrata	Hastate orache	0.08	1-3
Cakile maritima	Beach rocket	0.16	7.4-9.6
Chenopodium glaucum	Goosefoot	0.07	0.4
Tasmanian Species			
Baumea tetragona	Square-twig rush	0.04	0.3
Gahnia grandis	Brickmaker's sedge	0.3	7.7
Restio complanatus	Flat cord rush	0.2	0.5

SELENIUM

Selenium (Se) and vitamin E function synergistically as antioxidants; the actions of vitamin E cannot be replaced with selenium. Selenium toxicity decreases hatchability, growth and reproductive success and results in deformed embryos, diminished immune function, abnormal feather loss, emaciation and liver lesions.[23,74] Dietary selenium affects whole blood levels.[56] Mallards fed more than 10 mg/kg Se developed bilaterally symmetrical alopecia of the scalp and dorsal cervical midline, broken nails and necrosis of the tip of the beak.[56] Chicks raised on diets depleted of both vitamin E and Se show signs of exudative diathesis on the superficial pectoral muscles. Deficiencies of selenium are characterized by increases in heterophils and decreases in lymphocytes, basophils and hemoglobin. Selenium deficiency depresses plasma T_3 concentrations.[25] Dietary selenium up to 0.4 mg/kg appears to be adequate for large psittacines maintained on extruded diets with 200 mg/kg vitamin E.[43]

ZINC

Zinc is involved in cell replication and in the development of cartilage and bone. Normal serum zinc concentrations reported for parrots range between 0.5 and 5.8 ppm (7.65-84 μmol/L).[82,45] However, plasma and serum concentrations above 2 ppm (30 μmol/L) have been considered diagnostic for zinc toxicity in most species.[5,15,55,59,72,82] A lack of correlation between hepatic zinc levels and clinical diagnosis of zinc toxicity also has been reported.[12]

Zinc toxicosis usually arises from ingestion of zinc-coated aviary wire or metallic foreign bodies.[62] Clinical signs of zinc intoxication include anorexia, acute gastroenteritis, ataxia, lethargy, yellow-colored feces, vomiting, extreme loss of plumage and hepatomegaly.[72,85] It can cause pancreatic cell necrosis.[72] Excess dietary zinc negatively impacts tissue concentrations of α-tocopherol.[36] Zinc is more toxic in iron-deficient chicks than in properly iron-supplemented birds.[6,55] Liver biopsy is

Table 4.1.21 | Forms of Organic Iron

Hemal Iron	Non-hemal iron
Hemaglobin	Transferrin
Myoglobin	Ferritin
Cytochromes	Hemosiderin
Cytochome oxidase	Iron Proteinates
Catalase	—
Peroxidase	—

Table 4.1.22 | Iron and Vitamin C Content of Native Australian Fruits

Fruits	Iron mg/kg	Vitamin C mg/kg
Lillypilly, *Acmena smithii*	15.15	303
Wild ginger, *Alpinia caerulea*	5.7	199
Davidson plum, *Davidsonia pruriens*	127.66	BDL
Quandong, *Santalum acuminatum*	108.61	BDL
Wild fig, *Ficus platypoda*	80.12	59.35
Native gooseberry, *Physalis minima*	265.82	63.29

Data reprinted with permission from Elsevier Science[41]

Table 4.1.23 | Iron and Vitamin C Content of wild Australian Insects (expressed as dry matter)

Insect	Iron mg/kg	Vitamin C mg/kg
Honeypot ant, *Melophorus spp*	35	15
Lerp scale, *Psylla eucalypti*	78	100
Witchety grub, *Cossidae spp*	102	127
Bogong moth, *Argrotis infusa*	159	20
Green tree ant, *Oecophylla smaragdina*	400	58

Data reprinted with permission from Elsevier Science[41]

Table 4.1.24 | Iron Content of Invertebrates

Invertebrate	Diet	Iron mg/kg
Mealworm	wheat, grain, carrots	40
Mighty Mealy	wheat, brain, supplements	26
Super Mealworm	wheat, grain, carrots	50
Cricket, adult	cornmeal, wheat, soybean hulls, meat meal molasses, fish meal	110
Cricket, juvenile	shipped with raw potato	200
Wax Worm	none	80
Fruit Fly	commercial feed	450
Earth Worm, wild		11,100
Earth Worm, commercial	peat humus soil	5,800

Table 4.1.25 | Iron and Vitamin C Content of Fruits Commonly Fed to Birds

Fruit	Iron mg/kg	Vitamin C mg/kg
Apple, apricot, banana, fig, grape, raisin	—	Low <1,000
Watermelon	20	1130
Canteloupe	21	4130
Orange	9	4344
Papaya	9	5530
Strawberry	45	6725

Data reprinted with permission from Elsevier Science[41]

Table 4.1.26 | Concentrations of Hepatic Zinc in Birds

Species	**Zn** (mg/kg) Physiological Status	**Zn** (mg/kg) Intoxication	Reference
Budgerigar (*Melopsittacus undulatus*)	50.5 ± 12.7 (37.6-70.5) n=10	153 250	7
Budgerigar (aviary bred) (*Melopsittacus undulatus*)	64.7 ± 37 (29-126) n=8	No clinical signs of toxicity	12
Monk Parakeet (*Myiopsitta monachus*)	57.9 ± 34.5 (28.1-156) n=14	179 ± 73.7 (n=7)	13
Lovebird (*Agapornis roseicolli*)	42.5 ± 8.9 (37.5-50.2) n=5	75 156	62
Macaws (*Ara chloroptera, A. macao*)	38.9 ± 22 (12.0-115) n=77	150 ± 37.0 (n=3)	13
Rosellas and Lorikeets	74 ± 63 (27-166) n=4	Wild-caught	12
Galah (*Eolophus roseicapilla*)	31.6 ± 5.4 (24-45) n=16	Wild-caught	45
Sulphur-crested Cockatoo (*Cacatua galerita*)	37.5 ± 7.8 (25-59) n=21	Wild-caught	45
Long-billed Corella (*Cacatua tenuirostris*)	37.3 ± 9.8 (29-64) n=13	Wild-caught	45

not definitive as a diagnostic tool (Table 4.1.26). Signs of zinc deficiency include reduction in immune response, alterations to cell division, early embryonic death, fetal abnormalities, weak chicks at hatching, retarded growth, alopecia, dermatitis, delayed sexual development, abnormal skeletal formation and feathering.

Specific Diets

FRUIT AND POLLEN

Nectarivorous birds feed on a variety of pollens, plants, insects and their exudates.[9,19,57,89] Pollen is high in protein (Table 4.1.27), while its digestibility is relatively low in hummingbirds and lorikeets (4.5-6.6%).[8] As the sugar content of fruits increases, the volumetric intake and passage rate decrease.[84] The protein content of fig species is variable (4-25%).[26]

WHOLE PREY

Whole prey fed to birds in captivity can differ from that available in the wild (Tables 4.1.28, 4.1.29; Figs 4.1.15, 4.1.16). and may require supplementation. Feeding individual pieces of prey or eviscerated meat can contribute to nutrient imbalances. Feeding high proportions of liver can result in hypervitaminosis A. Supplement meat-based diets with $CaCO_3$, which has

Table 4.1.27 | Protein and Amino Acid Content of Pollen Sources in Australia[73]

Pollen source	Poultry requirements	Eucalypts	Banksias	She-oak	Hakea	Wattles	Almond	Black /Spear thistle	Lavender	Onion weed	Saffron thistle
Amino acids (% protein)											
Threonine	3.77	3.66 (3.38-4.11)	4.06 (3.81-4.3)	3.67	4.26	3.97 (3.01-4.63)	4.58 (4.47-4.7)	3.25 (1.7-3.6)	4.17	3.27	4.05
Valine	3.47	4.94 (4.38-5.83)	4.92 (4.7-5.4)	4.07	4.78	4.70 (3.95-5.49)	5.21 (4.83-5.4)	4.33 (3.4-5.1	4.54	11.08	5.69
Methionine	1.68	2.14 (1.0-2.69)	2.24 (2-2.273)	2.44	2.04	2.58 (2.21-2.84)	1.77 (0.7-2.57)	1.78 (1.2-2.1)	2.21	1.30	2.55
Leucine	5.51	6.60 (5.97-7.63)	6.49 (5.6-7.6)	6.03	6.59	6.54 (5.35-7.28)	6.81 (6.41-7.4)	5.98 (4.6-6.4)	6.04	6.91	6.94
Isoleucine	3.35	3.97 (3.36-5.47)	3.89 (3.5-4.5)	3.34	3.93	3.89 (2.94-4.64)	4.3 (4-4.7)	3.98 (3.2-4.5)	3.59	4.56	5.03
Phenylalanine	2.99	3.94 (3.48-5.37	4.43 (3.71-5.4)	3.29	3.81	3.76 (3.21-4.24)	3.57 (2.3-4.9)	3.55 (2.6-4.1)	4.11	3.38	4.18
Lysine	4.01	5.65 (5.17-6.34)	5.74 (5.1-6.5)	4.37	4.66	5.3 (4.66-6.19)	4.97 (3.1-6.48)	3.93 (1-6.8)	6.38	3.77	6.77
Histidine	1.44	2.31 (1.8-3.84)	2.58 (2.37-2.98)	1.73	2.4	2.05 (1.73-2.36)	1.95 (1.82-2.1)	2.7 (1.4-3.1)	3.67	1.64	4.43
Arginine	5.51	6.2 (4.13-7.18)	7.36 (6.7-8.6)	6.44	6.41	5.92 (4.66-7.2)	5.05 (4.6-5.48)	4.5 (3.7-6.5)	4.31	7.40	4.48
Crude protein (%)	16.7	24.87 (20.5-29.4)	33.06 (31.2-36.9)	12.50	18.4	23.75 (21.7-24.9)	25.94 (23.3-30.7)	20.94 (16.1-31.8)	19.4	18.25	18.1
Fat (%)		2.01 (0.48-3.9)	2.18 (1.9-2.45)	1.93	2.82	1.52 (0.9-2.52)	2.32 (1.89-2.74)	2.42 (2.25-2.59)	2.9	4.50	3.86

Note: Data in parentheses indicate ranges.

Table 4.1.28 | Mineral Content of Whole Vertebrate Prey

	Ca	P	Ca:P	Mg
Mouse	3.0	1.7	1.7	0.16
Rat	2.6	1.5	1.8	0.08
Chicken	2.2	1.4	1.6	0.5
Frog	4.3	1.9	2.3	2.47

Table 4.1.29 | Fat-soluble Vitamin Content of Whole Prey

	Vitamin A (IU/g)	Vitamin E (mg/kg)
Mouse (12weeks)	657	74
Rat (adult)	335.3	152
Chicken (6 weeks)	35.59	61
Frog, green	25.11	82.2

Table 4.1.30 | Calcium Content of Various Supplements

Supplemental source	Ca (%)	P (%)
Calcium borogluconate	8.32	0
Calcium carbonate (ground limestone, oyster shell, cuttlebone)	40.04	0
Calcium gluconate	9.31	0
Calcium glucobionate (4.6% Ca)	23mg/ml	0
Calcium lactate	18.31	0
Calcium phosphate (monobasic)	17.12	24.47
Calcium phosphate (dibasic)	29.46	22.77
Calcium phosphate (tribasic)	38.76	19.97
Bone meal, steamed	31.74	15

Table 4.1.31 | Iron and Vitamin A Content of Invertebrates

Invertebrate	Diet	Iron (mg/kg)	Vitamin A (IU/kg)
Mealworm	Wheat, grain, carrots	40	810
Mighty mealy	Wheat, grain, supplements	26	160
Super mealworm	Wheat, grain, carrots	50	970
Cricket, adult	Cornmeal, wheat midds, soybean hulls, meat meal, molasses, fish meal	110	810
Cricket, juvenile	Shipped with raw potato	200	470
Waxworm	None	80	150
Fruit fly	Commercial feed	450	not detected
Earthworm, wild		11,100	2400
Earthworm, commercial	Peat humus soil	5800	330

Table 4.1.32 | Calcium Content of Insects

	Supplement	Ca:P
Mealworm	nil	1:9
Cricket	nil	1:16
Cricket	Gut loaded	1:5
Cricket	Gut loaded/dusted	1:3

the highest calcium content (Table 4.1.30).

INSECTS

There are limited varieties of invertebrates for captive birds, with mealworms, earthworms and crickets forming the bulk of the available diet. Hard-bodied insects that contain up to 50% of their body weight as chitin may be important sources of dietary fiber, as chitin is chemically similar to cellulose. Chitinase activity has been identified in starlings, raptors and a variety of seabirds. Vitamin E content of many insects is adequate, but vitamin A content is relatively low or undetectable (Table 4.1.31).[4] Insects (especially from colder climates) contain high levels of polyunsaturated fatty acids. Insects generally concentrate a number of carotenoids that may be important for pigmentation or antioxidant activity. Insects generally have poor Ca:P ratios (Table

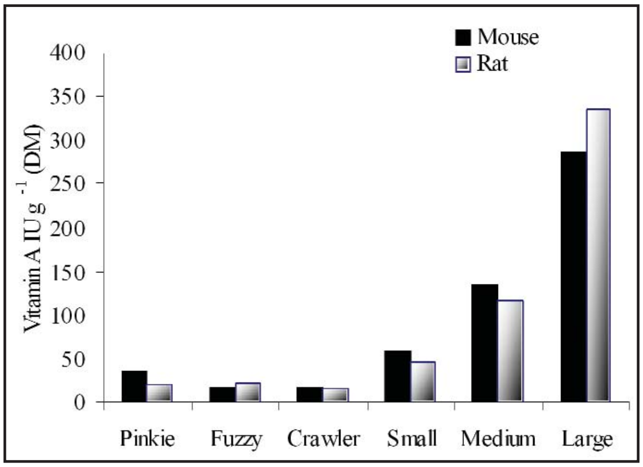

Fig 4.1.15 | Vitamin A content of rodents at various stages of development.

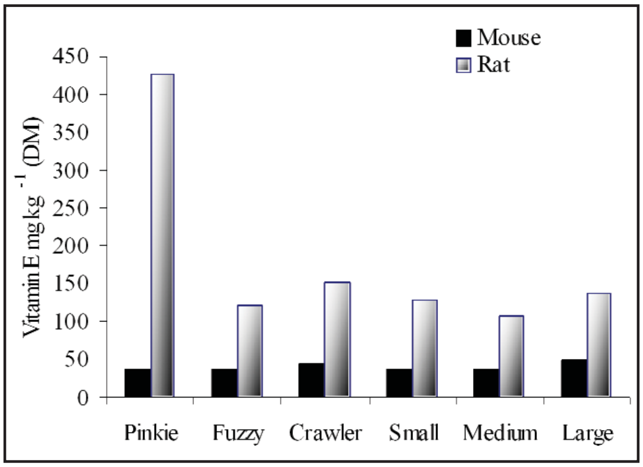

Fig 4.1.16 | Vitamin E content of rodents at various stages of development.

4.1.32). Calcium content of termites is low, but the content of their nest material (1.7%) provides a valuable source of calcium to birds, such as fig parrots, that nest in termitaria.[70]

FISH

Fish must be stored below -18° C to maintain nutritive value. Fish should be dry-thawed at <4° C up to 48 hours before use, and emergency thaws should be undertaken in plastic bags under cold running water to prevent the loss of nutrients. Supplementation of piscivorous diets is required for some key nutrients, depending on the species of fish fed and the method of preparation. Feeding whole fish is imperative to maintain proper Ca:P ratio. Iodine content of marine fish is considered adequate (0.9 mg/kg), while that of freshwater fish may be as low as 0.03 mg/kg. Sodium levels of marine fish are adequate if fish are not thawed in fresh water. Heat stress may increase a bird's sodium requirement. Vitamin A content of fish commonly fed to birds is adequate (Fig 4.1.17). Supplementation may be required if eviscerated fish are fed. Vitamin E levels of frozen fish are generally inadequate (Fig 4.1.18). Thawing of fish in water will deplete water-soluble vitamins. However, there is no data to support the supplementation of fish with water-soluble vitamins other than thiamine (B_1) if they are dry-thawed. Many fish contain thiaminase; these require 25 to 30 mg of thiamine per kg of fish fed.

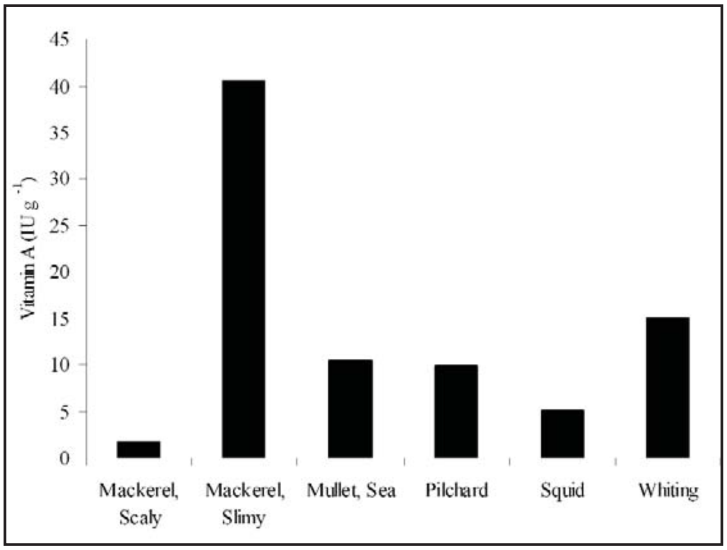

Fig 4.1.17 | Vitamin A content of fish stored in a frozen state (-10°C).

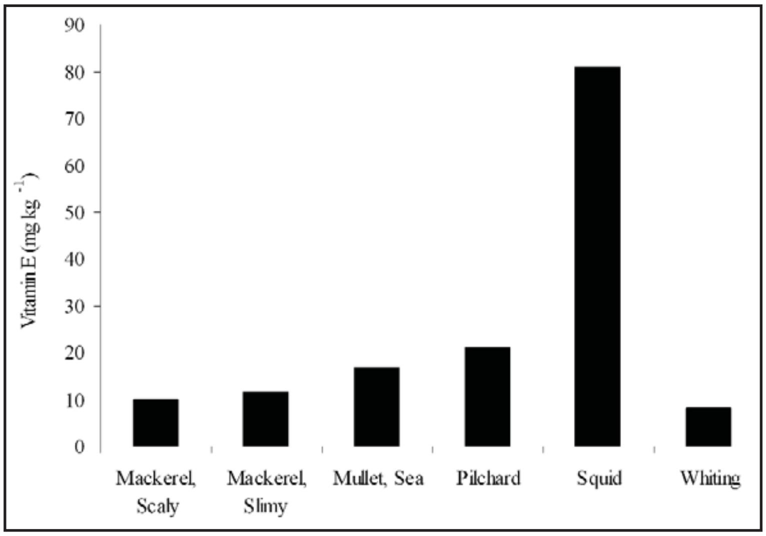

Fig 4.1.18 | Vitamin E content of fish stored in a frozen state (-10°C).

Labeling

Most products will display information regarding only a "guaranteed analysis." This provides an indication of maximum or minimum levels of crude protein, fat and fiber. However, there is no legal requirement for every batch to be chemically evaluated, rather these values often are derived from calculated values and thus may not be accurate. Furthermore, a value for "crude protein" provides no information about the digestibility of the protein or the proportions of various essential amino acids. A crude protein value for products containing invertebrates does not account for the proportion of nitrogen bound up in chitin. A crude fat content indicates neither whether the fats are saturated or unsaturated, nor the proportions of essential fatty acids. A crude fiber value does not delineate the proportion of soluble or digestible fiber, and provides no information about the lignin content.

SUMMARY

Nutrition is the single most important aspect of bird husbandry. Nutrition impacts the health, longevity, appearance and behavior of birds in captivity. The complex biochemistry and interactions between levels of nutrients coupled with the paucity of research in companion birds make choosing an appropriate diet very difficult.

Species differences between various psittacines makes dietary recommendations even more complex. Table 4.1.33 offers current estimated nutritional requirements for psittacines. A summation of critical factors in the selection of a diet should include:

1. Consideration of any species — specific dietary requirements or sensitivities.
2. Avoidance of excessive amounts of nutrients, as well as assuring adequate mineral levels.
3. Analysis of the components and availability of the listed manufacturers, "crude" protein, fat, calcium, etc., and knowledge of what method is used to measure these levels.
4. Awareness, as in human, dog and cat nutrition of the potential dangers inherent in preservatives and additives.
5. Recognition of the discrepancy between wild natural food sources and substitutions made by many commercial manufacturers.

Table 4.1.33 | Estimated Nutritional Requirements for Psittacines

Nutrient	Unit	Maintenance	Breeder
Protein			
Crude protein	%	10-15	15-22
Lysine	%	0.8-1.5	
Lipid			
Crude fat	%	5	10-15
Macrominerals			
Calcium	%	0.3-0.7[a]	0.7-1.2
Magnesium	%	0.15	
Phosphorus	%	0.3-0.7	0.5-0.8
Potassium	%	0.7	
Sodium	%	0.2	
Microminerals			
Copper	mg/kg	4-12	
Iron	mg/kg	100[b]	100
Manganese	mg/kg	65	
Selenium	mg/kg	0.30	0.4-0.5
Zinc	mg/kg	40-50	50-80
Vitamins			
Vit A	IU/kg	4000[c]	6000
Vit D_3	IU/kg	200-1200	2000
Vit E	mg/kg	200-250	250-350
Vit K_1	mg/kg	0.5[d]	0.5

[a] Calcium requirements established for budgerigars, some species may have higher requirements.
[b] Species susceptible to ISD may require less than 80 mg/kg.
[c] Beta carotene 22.4 mg/kg.
[d] Supplementation of 300 μg daily for fig parrots susceptible to vitamin K deficiency. Expressed on a dry matter basis. These values are estimates only and may not apply to all species.

References and Suggested Reading

1. Angel R, Ballam G: Dietary protein effect on parakeet reproduction, growth and plasma uric acid. Proc First Annu Conf Nutrition Advisory Group, Toronto, p 91, 1995.
2. Aye PP, et al: Induction of vitamin A deficiency in turkeys. Avian Dis 44(4):809-817, 2000.
3. Barker D: Preliminary observations on nutrient composition differences between adult and pinhead crickets, *Acheta domestica*. Bull Assoc Reptile Amphib 7(1):10-13, 1997.
4. Barker D, Fitzpatrick MP, Dierenfeld ES: Nutrient composition of selected whole invertebrates. Zoo Biol 17:123-134, 1998.
5. Bauck L, LaBonde J: Toxic diseases. *In* Altman RB, et al: Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 604-316.
6. Blalock TL, Hill CH: Studies on the role of iron in zinc toxicity in chicks. Biol Trace Element Res 17:17-29, 1988.
7. Bourke AM: Vitamin A toxicity in conures. AAV Newsletter 3-5, 1996.
8. Brice AT: Protein nutrition of captive hummingbirds. Proc 9th Dr Scholl Conf Nutr of Captive Wild Animals, 1991, pp 10-24.
9. Brice AT: The essentiality of arthropods in the diet of hummingbirds. Comp Biochem Physiol 101A(1):151-155, 1992.
10. Buttemer WA, et al: Time-budget estimates of avian energy expenditure: physiological and meteorological considerations. Physiol Zool 59:131-149, 1986.
11. Crosta, personal communication.
12. Doneley R: Zinc toxicity in caged and aviary birds - "new wire disease." Aust Vet Pract 22:6-11, 1992.
13. Dorrestein GM, et al: Zinc intoxication in two parrot flocks. Proc Deutsche Veterinärmedizinsche Gesellschaft e.V.125-133;2002
14. Drepper K, et al: Unteruschunen zum protein- aund energiebedarf adulter welesittche (*Melopsittacus undulatus*). Kleintierpraxis. 33, 1988.
15. Dumonceaux G, Harrison GJ: Toxins. *In* Ritchie BW, Harrison G J, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 1034-1052.
16. Frankel TL, Avram DS: Protein requirements of rainbow lorikeets, *Trichoglossus haematodus*. Aust J Zool 49:435-443, 2001.
17. Fu Z, et al: Retinoic acid accelerates the development of reproductive organs and egg production in Japanese quail (*Coturnix coturnix japonica*). Biol Reprod 63:1795-1800, 2000.
18. Grajal A: The nutritional ecology and digestive physiology of the hoatzin, *Opisthocomus hoazin*, a folivorous bird with foregut fermentation. PhD, Uni of Florida, 1991.
19. Grant B: Pollen digestion by Darwin's finches and its importance for early breeding. Ecology 77:489-499, 1996.
20. Gross KL, et al: Nutrients. *In* Hand MS, et al: Small Animal Clinical Nutrition. Kansas, Mark Morris Institute, 2000, pp 21-110.
21. Hanssen I, et al: Vitamin C deficiency in growing willow ptarmigan. J Nutr 109:2260-2278, 1979.
22. Harrison G, personal communication.

23. Hoffman DJ: Role of selenium toxicity and oxidative stress in aquatic birds. Aquat Toxicol 57:11-26, 2002.
24. Jenni L, Winkler R: Moult and Ageing of European Passerines. London, Academic Press, 1994.
25. Jianhua H, Ohtsuka A, Hayashi K: Selenium influences growth via thyroid status in broiler chickens. Br J Nutr 84:727-732, 2000.
26. Kalina J: Ecology and behavior of the black-and-white-casqued hornbill (*Bycanistes subcylkindricus subquadratus*) in Kibale Forest, Uganda. PhD. Michigan State Uni, 1988.
27. Kamphues J, Otte W, Wolf P: Effects of increasing protein intake on various parameters of nitrogen metabolism in grey parrots (*Psittacus erithacus erithacus*). *In* Kamphues J, Wolf P, Rabehl N: First Int Symp of Pet Bird Nutrition, 1997, pp 118.
28. King JR: Energetics of avian moult. Acta 17th Congr Int Orn Berlin, 1980, Vol. 1978, pp 312-317.
29. Klasing KC: Comparative Avian Nutrition. UK, CAB International, 1998.
30. Koutsos EA, Klasing KC: Vitamin A nutrition of cockatiels. Proc Joint Nutr Symp, Antwerp, Belgium, 2002, p 141.
31. Koutsos EA, Matson KD, Klasing KC: Nutrition of birds in the order Psittaciformes: A review. J Avian Med Surg 15:257-275, 2001.
32. Koutsos EA, et al: Adult cockatiels (*Nymphicus hollandicus*) metabolically adapt to high protein diets. J Nutr 131:2014-20, 2001.
33. Koutsos EA, et al: Adult cockatiels (*Nymphicus hollandicus*) at maintenance are more sensitive to diets containing excess vitamin A than to vitamin A deficient diets. J Nutr 2003 Jun; 133(6): 1898-1992.
34. Lindström Å, Visser GH, Daan S: The energetic cost of feather synthesis is proportional to basal metabolic rate. Physiol Zool 66:490-510, 1993.
35. Lowenstine LS: Nutritional disorders of birds. *In* Fowler MF (ed): Zoo Animal Medicine, Philadelphia, WB Saunders Co, 1986, pp 202-212.
36. Lü J, Combs GFJ: Excess dietary zinc decreases tissue α-tocopherol in chicks. J Nutrition 118:1349-1359, 1988.
37. MacMillen RE, Baudinette RV: Water economy of granivorous birds: Australian parrots. Funct Ecol 7:704-712, 1993.
38. MacWhirter P: Malnutrition. *In* Ritchie BW, Harrison GJ and Harrison LR (eds): Avian Medicine: Principles and Application, Lake Worth, Florida, Wingers Publishing, 1994, pp 842-861.
39. Massey DM, Selwood EHB: The amino-acid composition of budgerigar diet, tissues and carcass. Vet Rec 72:283-287, 1960.
40. Maynard LA, et al: Animal Nutrition 7th ed. New York, McGraw-Hill, 1979.
41. McDonald DL: Feeding Ecology and Nutrition of Australia Lorikeets. *Seminars in Avion and Exotic Pet Medicine; 12(4): 195-204; 2003*.
42. McDonald DL: Dietary considerations for iron storage disease in birds and implications for high vitamin A contents of formulated bird foods. Proc Joint Nutr Symp, Antwerp, Belgium, 2002, p 162.
43. McDonald DL: Evaluation of the use of organic formulated bird foods for large psittacines. Proc Joint Nutr Symp, Antwerp, Belgium, 2002, p 110.
44. McDonald DL: Failure of the wild population of the endangered Orange-bellied Parrot (*Neophema chrysogaster*) and implications for nutritional differences in indigenous and introduced food resources. Proc Aust Assoc Avian Vet, 2004.
45. McDonald DL, Nutritional status of the Australian long-billed corella (*Cacatua tenuirostris*) and sulphur-crested cockatoo (*Cacatua galerita*): an evaluation of hepatic and serum vitamin and mineral concentrations. Proc Aust Assoc Avian Vet, 2004.
46. McDowell LR: Vitamins in Animal and Human Nutrition. Ames, Iowa State University Press, 2000.
47. Meade J: Amazons. Proc Smokey Mountain Cage Bird Society Meeting, 1998.
48. Mezes M, et al: Nutritional metabolic diseases of poultry and disorders of the biological antioxidant defense system. Acta Vet Hung 45:349-360, 1997.
49. Murphy ME: Amino acid composition of avian eggs and tissues: Nutritional implications. J Avian Biol 25:27-38, 1994.
50. Murphy ME, King JR: Diurnal constancy of feather growth rates in white-crowned sparrows exposed to various photoperiods and feeding schedules during the postnuptial molt. Can J Zool 66:1403-1413, 1986.
51. Murphy ME, King JR: Nutritional aspects of avian molt. Acta 20th Congr Int Orn, Christchurch, pp 2186-2193, 1991.
52. Murphy ME, King JR: Energy and nutrient use during moult by white-crowned sparrows, *Zonotrichia leucophrys gambelii*. Ornis Scand 23:304-313, 1992.
53. Nichols DK, et al: Coagulopathy in pink-backed pelicans (*Pelecanus rufescens*) associated with hypervitaminosis E. J Zoo Wildl Med 20(1)57-61,1989.
54. NRC: Nutrient Requirements of Poultry. Washington, National Academy Press, 1994.
55. Osofsky A, et al: Determination of normal blood concentrations of lead, zinc, copper and iron in Hispaniolan Amazon parrots (*Amazona ventralis*). Avian Med Surg 15:31-36, 2001.
56. O'Toole D, Raisbeck MF: Experimentally induced selenosis of adult mallard ducks: Clinical signs, lesions, and toxicology. Vet Pathol 34(4):330-340, 1997.
57. Paton DC: The diet of the New Holland honeyeater, *Phylidonyris novaehollandiae*. Aust J Ecol 7:279-298, 1982.
58. Phalen D: Study determines safe diets for companion birds. Animal News Vol III, 2001.
59. Puschner B, St Leger J, Galey FD: Normal and toxic zinc concentrations in serum/plasma and liver of psittacines with respect to genus differences. J Vet Diagn Investig 11:522-527, 1999.
60. Pusztai A, Agota G, Bardos L: Effect of large dietary doses of beta-carotene on plasma retinoid and beta-carotene levels and on progesterone production in the granulosa cells of Japanese quail. Acta Vet Hung 48: 81-7, 2000.
61. Rabehl N: Untersuchungen zur Körperzusammensetzung und deren Entwicklung bei vershiedenen Ziervogelarten (Kanarien, Wellensittiche, Agaporniden, Nymphensittiche, Amazonen und Graupapagein). Hannover, Institut für Tierernährung der Tierärzlichen Hochschule, 1995.
62. Reece RL, Dickson DB, Burrowes PJ: Zinc toxicity (new wire disease) in aviary birds. Aust Vet J 63:199, 1986
63. Remington TE: Why do grouse have ceca? A test of the fiber digestion theory. J Exp Zool Suppl 3:87-94, 1989.
64. Roudybush TE, Grau CR: Lysine requirements of cockatiel chicks. Proc 34th West Poult Dis Conf, 1985, vol. 34, pp 113-115.
65. Roudybush TE, Grau CR: Food and water interrelations and the protein requirement for growth of an altricial bird, the cockatiel (*Nymphicus hollandicus*). J Nutr 116:552-559, 1986.
66. Sahin K, et al: Protective role of supplemental vitamin E and selenium on lipid peroxidation, vitamin E, vitamin A, and some mineral concentrations of Japanese quails reared under heat stress. Bio Trace Elem Res 85:59-70, 2002.
67. Schoemaker NJ, Lumeij JT, Beynen AC: Polyuria and polydipsia due to vitamin and mineral oversupplementation of the diet of a salmon crested cockatoo (*Cacatua moluccensis*) and a blue and gold macaw (*Ara ararauna*). Avian Pathol 26:201-209, 1997.
68. Scott ML: Environmental influences on ascorbic acid requirements in animals. Annals NY Acad Sci 258:151-155, 1975.
69. Scott ML, Nesheim MC, Young RJ: Nutrition of the Chicken. Ithaca, NY, 1982.
70. Sergio E, et al: Nutrition of the tamandua: I. Nutrient composition of termites (*Nasutitermes* spp.) and stomach contents from wild tamanduas (*Tamandua tetradactyla*). Proc 1st Ann Conf NAG, Toronto, 1995, pp 170-171.
71. Sheshukova T, et al: The adaptation of the digestive system to carbohydrates in experimental A hypervitaminosis. Fiziol Zh SSSR Im I M Sechenova 77:70-76, 1991.
72. Smith A: Toxicosis in a flock of Hispaniolan Amazons. Proc Conf Assoc Avian Vet, 1995, pp 447-453.
73. Somerville DC: Nutritional Value of Bee Collected Pollens. RIRDC, Victoria, 2001.
74. Spallholz JE, Hoffman DJ: Selenium toxicity: Cause and effects in aquatic birds. Aquat Toxicol 57:27-37, 2002.
75. Stobart, personal communication.
76. Stanford M: Calcium metabolism in grey parrots: The effects of husbandry. Proc Assoc Avian Vet, 2002.
77. Sunder A, Flachowsky G: Influence of high vitamin E dosages on retinol and carotenoid concentration in body tissues and eggs of laying hens. Arch Tierernahr 55:43-52, 2001.
78. Takeshita K, Graham DL, Silverman S: Hypervitaminosis D in baby macaws. Proc Assoc Avian Vet: 1986, pp 341-345.
79. Taylor EJ, Nott HMR, Earle KE: Dietary glycine: Its importance in growth and development of the budgerigar (*Melopsittacus undulatus*). J Nutr 124:2555S-2558S, 1994.
80. Tsiage VK, et al: Identification of volatile sulfur derivatives released from feathers of chicks fed diets with various levels of sulfur-containing amino acids. J Nutr 117:1859-1865, 1987.
81. Ullrey DE, et al: Limitations of plasma or serum analysis in assessing vitamin E status of domestic and wild animals. First Annu Conf Nutrition Advisory Group (NAG) of the Amer Zoo and Aquarium Assoc (AZA), 1995, pp 195-202.
82. Van Sant F: Zinc and parrots: More than you ever wanted to know. Proc Assoc Avian Vet, 1998, p 305.
83. Voitkevich AA: The Feathers and Plumage of Birds. London, Sidgwick and Jackson, 1966.
84. Witmer MC: Ecological and evolutionary implications of energy and protein requirements of avian frugivores eating sugary diets. Physiol Zool 71:599-610, 1998.
85. Wolf P: Handfeeding of young parrots. Techniques, diets and recommendations. Proc Joint Nutrition Symposium. Antwerp, Belgium, 2002, pp 113-136.
86. Wolf P, Kamphues J: Die futter- und wasseraufnahme bei kanarien - Einflußfaktoren und abhängigkeiten-. Kleintierpraxis 37:545-552, 1992.
87. Wolf P, Wendler C, Kamphues J: Untersuchungen zur Zusammensetzung handelsüblicher Mauserpräparate für Ziervögel. Kleintierpraxis 41:669-670, 1996.
88. Wolf P, et al: Vitamin A metabolism in recessive white canaries. Animal Welfare 9(2):153-165, 2000.
89. Wooller R, Richardson K, Pagendham C: The digestion of pollen by some Australian birds. Aust J Zool 36:357-362, 1988.