

# Nutrition and practical feeding of aviary and caged birds

Helen Rooney<sup>1</sup>

---

## Summary

Nutrient deficiencies and excesses play a major role in many diseases of caged and aviary birds. Current knowledge of nutrient requirements, natural sources of these nutrients, and malnutrition syndromes, along with treatment protocols, is presented. When formulating a diet, it is essential that the species of bird, as well as its age, weight and physiological state are considered, so that specific nutrient requirements are fulfilled. Although the complete nutrient requirements for aviary and caged birds have not yet been determined, it is unquestionable that unsupplemented seed - only diets are unsuitable. Processed diets show promise for the future, and as research reveals more information about avian nutrition, these diets should continue to improve.

## Introduction

Nutrient deficiencies and excesses play a major role in many diseases of caged and aviary birds. Despite this, relatively little is known about their nutritional requirements. Extrapolations have been made from studies of poultry, however due to the great biodiversity among birds, this method is less than ideal. With respect to aviary and caged birds, the majority of research has focused on pittacine birds, which will be evident in this essay. It must be remembered, though, that although nutrient deficiencies may not necessarily produce specific clinical signs, they may produce subclinical effects, particularly on growth and reproduction.

## Energy Requirements

Birds eat primarily to satisfy their energy requirements, thus other dietary components should ideally be considered in relation to the energy density of the diet. If the energy density is too high, as may occur in seed - based diets high in fat, intake is decreased in response, and nutrient deficiencies are more likely to occur. Conversely, if the energy density is too low, intake is increased and nutrient excesses may result. Furthermore, gastrointestinal capacity may become a limiting factor, and the bird may not be able to meet its nutrient requirements, particularly for growth, recuperation from illness or reproduction.

Dietary energy may be supplied by fat, carbohydrate or protein (Table 1). Due to incomplete oxidation of protein and excretion as uric acid, the available energy content of protein is decreased, compared to mammals.

*Table 1. Physiological fuel values of foods (Whittow, 1986)*

Protein	4.29 kCal/g
Carbohydrate	4.09 kCal/g
Fat	9.29 kCal/g

A bird's energy requirement may be numerically expressed in terms of the basal metabolic rate (BMR). This is equivalent to the amount of heat that is produced by a bird that is resting, awake, post - absorptive, and in a thermoneutral environment. Using allometric scaling, BMR may be determined with the following formula :

---

<sup>1</sup> Veterinary Science V student, University of Sydney, 1997

$$\text{BMR} = K (W^{0.67}),$$

where K = theoretical constant for kcal used during a 24 hour period

and K = 129 for passerines

K = 78 for non-passerines

W = bodyweight in kg

(Bennett and Harvey, 1987, Sedgwick, 1988, cited in Quesenberry *et al*, 1989)

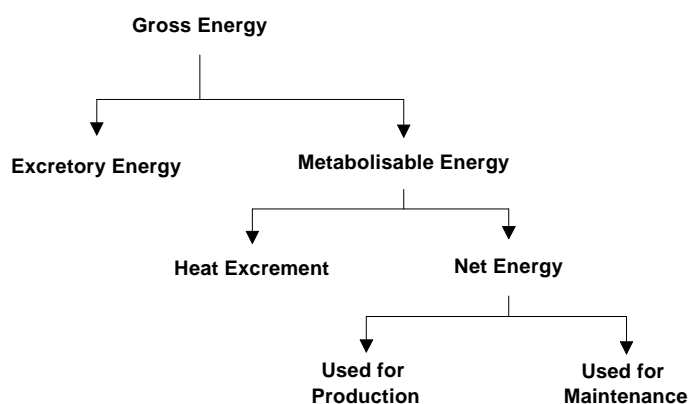
To be practically useful, BMR must be converted to maintenance energy, (M). This refers to the energy expenditure of a caged bird that maintains a constant body mass over a period of days when it is not undergoing reproduction, moulting, migratory unrest, growth or fat deposition. M varies between 1.3 and 7.2 times the BMR in passerines, the variation due to seasonal factors (Whittow, 1986). Adjustments for different physiological states are detailed in Table 2

**Table 2. Adjustments to maintenance, expressed as multiples of maintenance energy (Donoghue, 1989).**

Physical inactivity	0.7-0.9
Starvation	0.5-0.7
Hypometabolism	0.5-0.9
Elective surgery	1.0-1.2
Mild trauma	1.0-1.2
Severe trauma	1.1-2.0
Growth	1.5-3.0
Sepsis	1.2-1.5
Burns	1.2-2.0
Head injuries	1.0-2.0

The energy requirements detailed above must be met by the energy content of the diet, however the gross energy of a diet is not totally available. In the bird, faeces and urine are voided together, hence faecal and urinary energy are combined and termed excretory energy. (Figure 1.)

**Figure 1. Partition of food energy in the bird (Adapted from McDonald *et al*, 1992)**



## Major Nutrient Requirements

### Fat Requirement

The main functions of dietary fat are to provide essential fatty acids (EFA), to serve as a concentrated energy source, and to deliver fat-soluble vitamins. Fat also plays an important role in palatability, as illustrated by the preferential selection of high fat seeds such as sunflower by many psittacine birds. Table 2 illustrates the variation in crude fat content of various seeds.

Linoleic acid is considered to be the essential fatty acid in birds because it will eliminate all of the signs of EFA deficiency in poultry. It is postulated however, that *alpha*-linoleic acid (Watkins, 1991), and arachidonic acid (Scott, 1996), may also be essential. Signs of EFA deficiency are related to the metabolic role of EFAs in cell membranes and prostaglandin synthesis. Deficiency is characterised by retarded growth in chicks, rough, flaky skin, poor feathering, increased thirst, and decreased resistance to disease (Watkins, 1991, Nott and Taylor, 1993). EFA deficiency is uncommon in adult birds because of body reserves, however it is recommended from studies of poultry that linoleic acid should comprise approximately 1 % w/w (NRC, 1994). Oil seeds such as linseed and sunflower are good sources of linoleic acid (Table 2).

Excessive dietary fat may lead to obesity, particularly in those species with a breed predilection such as budgies, cockatiels, cockatoos, amazons and canaries (LaBonde, 1992). Furthermore, high fat diets may lead to lipomas, diarrhoea, atherosclerosis, fatty liver syndrome and secondary mineral and vitamin deficiencies, particularly of calcium and vitamin E (Fowler, 1986, Harrison and Harrison, 1986, Murphy, 1992, Johnson *et al*, 1992). Sudden death following exertion is the most common presenting sign of atherosclerosis in psittacine birds (Johnson *et al*, 1992), however it is often an incidental lesion at necropsy. Although the exact cause of psittacine fatty liver syndrome has not yet been elucidated, malnutrition from a seed or other high fat diet, which is inadequate in lipotropic factors, is suspected. Total elimination of dietary fat however, is integral to treatment (Murphy, 1992). Diets high in fat also carry the risk of rancidity, which increases requirements for the antioxidants, vitamins C and E.

Table 2. Major nutrient composition of seeds fed to psittacine birds (g/kg DM)  
(Ullrey, Allen and Baer, 1991)

<i>nutrient</i>	<i>corn</i>	<i>proso millet</i>	<i>hulled oats</i>	<i>peanuts</i>	<i>pump-kin</i>	<i>saf-flower</i>	<i>sun-flower</i>	<i>wheat</i>
<i>dry matter</i>	890	919	870	934	931	944	946	880
<i>crude protein</i>	97	150	182	275	264	171	241	144
<i>crude fat</i>	43	40	70	527	492	407	524	18
<i>crude fibre</i>	26	185	30	52	24	26		28
<i>Arg</i>	5.6	3.8	10.2	37.0	58.2	18.5	25.4	6.7
<i>Ilu</i>	4.2	5.4	5.9	10.7	18.2	7.6	12.0	6.7
<i>Lys</i>	2.7	1.4	6.1	10.6	26.5	5.7	9.9	4.6
<i>Met</i>	2.2	1.0	2.4	2.8	7.9	2.7	5.2	2.2
<i>Thr</i>	4.4	3.8	5.1	8.0	13.0	5.5	9.8	4.3
<i>Trp</i>	1.0	2.1	2.2	3.3	6.2	1.7	3.7	2.1
<i>linoleic acid</i>	24.9	7.8	-	166.3	204.3	297.5	344.9	6.8

### Carbohydrate Requirement

Carbohydrates are the primary energy source for granivores, nectivores and frugivores (Raidal, 1989). Seeds are generally high in starch, so although an excess of carbohydrate is possible, a deficiency in granivores such as psittacine birds is unlikely (Table 3).

*Table 3. Analysis of carbohydrate content of seed kernels commonly fed to budgerigars (g / kg kernel, fresh weight) (Adapted from Earle and Clarke, 1991)*

<i>seed kernel</i>	<i>nitrogen free extract (carbohydrate)</i>
<i>white millet</i>	712 +/- 25.0
<i>red millet</i>	699 +/- 10.0
<i>canary</i>	615 +/- 58.0
<i>hulled oats</i>	707 +/- 45.0

### **Protein Requirement**

A bird's protein requirement comprises the obligate need for essential amino acids, and the requirement for additional protein or non-protein nitrogen for synthesis of the non - essential amino acids. Furthermore, amino acids should be correctly balanced, without individual excesses or deficiencies. Birds require a dietary supply of ten essential amino acids ; growing birds have an additional essential requirement for glycine and proline (*Table 4*).

*Table 4. Avian amino acid requirements*

<i>Essential amino acids</i>	<i>non - essential amino acids</i>
lysine	alanine
arginine	aspartate
histidine	proline-----glutamate*
leucine	glycine -----serine*
methionine-----	cystine
threonine	tyrosine
tryptophan	glutamate
isoleucine	serine
phenylalanine	
valine	

Note : ----indicates a requirement for either the amino acids referred to

\* indicates that these amino acids are essential for growth

Protein requirements for maintenance, growth and reproduction have been determined for various species, and are illustrated in *Table 5*; they vary markedly with breed and physiological state of the bird. This variation with breed may be true, or may be contributed to by differences in approach by researchers to the term 'requirement', and the difficulty of determining maintenance requirements.

Table 5. Protein Requirements for Maintenance, Growth and Reproduction

<i>Species</i>	<i>Protein Requirement for Maintenance (% DM)</i>	<i>Protein Requirement for Growth / Reproduction (% DM)</i>	<i>Reference</i>
<i>cockatiel</i>	10 - 15	20	Roudybush and Grau, 1991
<i>budgerigar</i>	17 - 22	25	Earle and Clarke, 1991 Nott and Taylor, 1993
<i>passerines</i>	8 - 9	16.5 - 22	Nott and Taylor, 1993, Kamphues and Meyer, 1991
<i>pigeon</i>	13	18	Nott and Taylor, 1993

Although, as stated earlier, birds eat primarily to satisfy their energy requirements, this is modified by the energy : protein ratio of the diet. Insufficient (or unbalanced) dietary protein with respect to energy, results in increased food intake to compensate, which may lead to obesity (Underwood *et al*, 1991). In the majority of psittacine diets, lysine is the limiting amino acid, which may also lead to poor feathering (Scott, 1996). Excess dietary protein with respect to energy is energetically wasteful in terms of protein metabolism and excretion, dry matter digestibility and fat utilisation. Furthermore, excess dietary protein has resulted in weight loss and death (Underwood *et al*, 1991), depression of growth and behavioural abnormalities (Roudybush and Grau, 1991) and may contribute to the development of gout in susceptible birds.

#### Water Requirement

Water requirement varies greatly with environmental temperature, diet composition, species, size and age of the bird. Caged and aviary birds should be supplied with fresh water daily. This is critical for those on a dry seed based diet, and for canaries in particular. If water intake is inadequate, droppings become infrequent and small, and appear dark green in colour due to proportionally increased faeces (Nott and Taylor, 1993).

#### Requirement for Vitamins and Major and Trace Elements

Despite obvious limitations, most of the nutritional requirements of aviary and caged birds have been extrapolated from known requirements of poultry. Thus, they have involved assumptions that the maintenance requirements for major and trace elements, and the utilisation rates for these dietary components are the same as for poultry (Kamphues and Meyer, 1991). For numerical requirements of major and trace elements for poultry, please refer to the NRC (1994). The natural sources, function, signs of deficiency and excess of the major and trace elements required by birds are summarised in Table 6. They are predominantly derived from studies of poultry.

**Table 6. Major and trace elements required by birds : natural sources, function, signs of deficiency and excess (McDonald *et al*, 1992, Murphy, 1992, Nott and Taylor, 1993, Doneley, 1996, Scott, 1996)**

<i>Element</i>	<i>Natural Source</i>	<i>Function</i>	<i>Signs of Deficiency</i>	<i>Signs of Excess</i>
<i>calcium</i>	cuttlefish, lucerne, nuts, dairy products	bone, nerve impulses, muscle contraction, blood coagulation	nutritional secondary hyper-parathyroidism	nutritional secondary hyper-parathyroidism
<i>phosphorus</i>	brewer's yeast, peanuts, egg	component of cell membranes, bone, energy metabolism	nutritional secondary hyper-parathyroidism	nutritional secondary hyper-parathyroidism
<i>potassium</i>	lucerne, soy bean, brewer's yeast, dried fruit, vegetables	maintenance of osmolality of fluids, acid - base balance	depressed growth, death	excess rare
<i>sodium</i>	salt, eggs, dairy products, lucerne	as above	as above	excess rare
<i>chlorine</i>	salt, dairy products, carrots, greens	as above	as above	excess rare
<i>sulfur</i>	proteins containing methionine, cystine, cysteine	as for these proteins; feather growth, protein metabolism	as for deficiency of these proteins; poor feathering	as for excess of these proteins; excess rare
<i>magnesium</i>	sunflower seeds, nuts, lucerne, brewer's yeast	bone structure, enzyme activator	deficiency rare	excess rare
<i>zinc</i>	soy bean, wheat, nuts, cereal grains	enzyme activator	retarded growth, bone and feather abnormalities, reproductive failure, scaly feet and skin	poor growth, anaemia, death, induced copper deficiency
<i>manganese</i>	soy beans, seeds other than corn, level higher in acidic soils	enzyme activator	perosis, depressed growth, ataxia, repro. failure	excess rare
<i>iodine</i>	egg, cheese, water (variable)	component of thyroid hormones	goitre	goitre
<i>copper</i>	brewer's yeast, sunflower seeds, nuts, peas, lucerne	enzyme systems, component of blood proteins	feather hypo-pigmentation, anaemia	gastroenteritis, renal and hepatic degeneration
<i>cobalt</i>	present in most foods	microbial vitamin B12 synthesis	signs of vitamin B12 deficiency	excess rare
<i>iron</i>	green leaves, brewer's yeast, most cereal grains	incorporation into haemoglobin for oxygen transport	anaemia, feather hypo-pigmentation	possibly related to haemosiderosis, haemochromatosis
<i>selenium</i>	brewer's yeast, lucerne, rice, cheese, egg	component of glutathione peroxidase	reproductive failure, myopathies	excess rare
<i>molybdenum, nickel, silicon, vanadium</i>	widely distributed in feeds	metabolic roles	deficiency rare	excess rare

The mineral content of many seeds and other plant products is dependent on both the species of plant, and environmental factors such as mineral concentration in the soil and soil acidity. The availability of these minerals to the bird is further affected by the presence of other minerals or chelating agents such as phytate which interact to impede absorption. For details of major and trace element and vitamin levels in seeds commonly fed to psittacine birds, please refer to *Table 7*. Vitamins may be divided into the fat - soluble vitamins, namely A,D,E, and K, and the water - soluble vitamins, B - complex and C. The water - soluble vitamins in general are not stored in the body in appreciable quantities, and thus a regular dietary supply is required (*Table 11*).

Table 7. Vitamin and mineral composition of seeds commonly fed to psittacine birds (mg / kg DM).  
Adapted from Ullrey, Allen and Baer (1991)

<i>nutrient</i>	<i>corn</i>	<i>proso millet</i>	<i>hulled oats</i>	<i>peanuts</i>	<i>pump-kin</i>	<i>saf-flower</i>	<i>sun-flower</i>	<i>wheat</i>
<i>calcium</i>	200	100	800	600	500	800	1200	600
<i>total P</i>	3100	4000	4800	4100	12600	6800	7500	4300
<i>Mg</i>	1300	1800	1300	1900	5700	1700	3700	2000
<i>K</i>	3400	4800	3900	7700	8700	4600	7300	5200
<i>Na</i>	300	28	575	17.1	22	40	32	460
<i>Mn</i>	6	14	32	12	31	34	21	37
<i>Zn</i>	15	22	-	35	88	65	53	36
<i>Fe</i>	30	82	84	35	54	89	72	57
<i>Cu</i>	4	12	7	11	19	19	19	1
<i>Se</i>	0.04	0.08	-	0.34	0.14	0.84	0.77	0.23
<i>β-carotene</i>	1.4	-	0	0	2.3	0	0.3	0
<i>α l p h a - tocopherol</i>	9	-	17	98	-	-	-	15
<i>riboflavin</i>	1.1	4.2	1.4	14.0	3.4	4.4	2.6	1.6
<i>pantothen-ic acid</i>	4.5	12.1	15.7	30.0	-	-	-	11.4
<i>niacin</i>	27.0	25.3	11.5	151.5	18.7	24.2	47.7	55.2
<i>B12</i>	0	0	0	0	0	0	0	0
<i>choline</i>	697	484	1300	-	-	-	-	1253
<i>biotin</i>	0.07	-	-	-	-	-	-	0.13
<i>folacin</i>	0.45	-	0.57	1.08	-	-	-	0.46
<i>thiamin</i>	3.9	8.0	7.5	7.1	2.3	12.3	24.3	5.2
<i>pyridoxine</i>	7.9	-	1.1	3.2	-	-	-	3.9

### Syndromes of Nutrient Deficiency and Excess in Caged and Aviary Birds

#### i) Metabolic Bone Disease

As discussed by Nott and Taylor (1993), aviary and caged birds have high calcium and phosphorus requirements, as well as a high Ca : P ratio (2 : 1) compared to the requirements of most mammals. This is due in part to the rapid growth rate of many birds, as well as to the drain placed on the hen for egg shell formation. The plasma calcium level is very tightly regulated by parathyroid hormone, calcitonin and vitamin D (1,25-dihydroxycholecalciferol). Bird seed is commonly low in calcium, has a poorly balanced Ca : P ratio, or in the case of oil seeds, and may form insoluble calcium soaps in the intestine. In legumes, the Ca : P ratio is markedly imbalanced ranging from 6:1 to 10:1 (Hays and Swenson, 1993). The calcium content of seed kernels commonly fed to psittacine birds ranges from 0.01 % (0.1 g /kg DM) to 0.12 % (Table 8.). These values are well below the minimum dietary requirements reported in the literature (Table 9.).

**Table 8. Calcium and Phosphorus Concentration of Seed Kernels Commonly Fed to Budgerigars (g / kg kernel fresh weight) (Earle and Clarke, 1991)**

	<i>Ca</i>	<i>Total P</i>	<i>Phytate</i>	<i>Available P</i>	<i>Ca : Avail P</i>
<i>white millet</i>	0.15	2.6	2.6	0.1	1.5
<i>red millet</i>	0.13	2.5	2.1	0.4	0.33
<i>canary</i>	0.41	4.1	3.0	1.1	0.37
<i>hulled oats</i>	0.44	3.4	2.3	1.1	0.4

**Table 9. Minimum Dietary Calcium Requirements for Psittacine birds**

<i>Calcium Level</i>	<i>Physiological State</i>	<i>Species</i>	<i>Reference</i>
<b>1 %</b>	reproduction	large psittacine birds	Ullrey <i>et al</i> (1991)
<b>0.35 %</b>	reproduction	cockatiels	Roudybush and Grau, (1991)
<b>0.04 %</b>	reproduction (depressed)	cockatiels	Roudybush and Grau, (1991)
<b>0.8 %</b>	reproduction / growth	budgerigars	Nott and Taylor (1993)

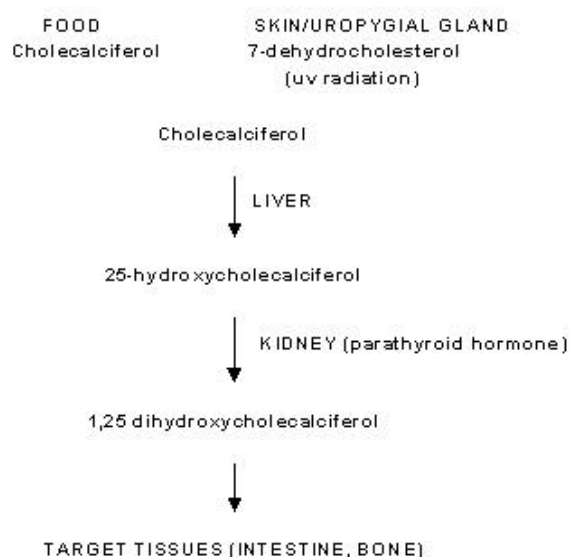
Although cereal grains contain high levels of phosphorus, much of it is in the form of phytate, and is thus unavailable. *Table 8* shows the concentration of available phosphorus in the kernels of seeds commonly fed to budgies. Cellular membranes and energy metabolism have priority over bone when available phosphorus is low. (Nott and Taylor, 1993).

Vitamin D plays a vital role in calcium and phosphorus homeostasis, by stimulating absorption of calcium and phosphorus from the intestinal lumen, renal tubules, and bone. Birds require a supply of cholecalciferol (vitamin D3), which may be derived from a dietary source, or may be synthesised in the skin and uropygial gland, and then either absorbed or ingested while preening. Synthesis of cholecalciferol is stimulated by the effect of ultra violet light on the precursor molecule, 7-dehydrocholesterol.

According to Raidal (1989), the uropygial gland and feathers contain low quantities of 7-dehydrocholesterol compared to the skin of the head and neck. The metabolic pathway for production of hormonally active vitamin D (1,25-dihydroxycholecalciferol,) is detailed in *Figure 2*.



**Figure 2. Metabolic Pathway for Production of the Hormonally Active Form of Vitamin D (from McDonald et al, 1992)**



Most seeds, particularly those which are sun-ripened, contain high levels of vitamin D (type unspecified,) according to Nott and Taylor, (1993), however this is disputed by Fowler (1986), who states that most seeds are low in vitamin D3. Signs of deficiency however, are most likely to occur when young birds fed a diet that is low in vitamin D3 are given limited exposure to ultra violet light.

Not surprisingly, deficiencies in calcium, vitamin D3 and phosphorus, or an improper calcium : phosphorus ratio are common in pet psittacine birds, and can lead to nutritional secondary hyperparathyroidism, rickets or osteomalacia. Rickets and osteomalacia refer to cases when the calcium - phosphorus abnormalities are caused by a vitamin D3 deficiency in young and older birds respectively. The term nutritional secondary hyperparathyroidism is used when there is a primary abnormality of calcium and / or phosphorus levels.

Metabolic bone disease is most commonly diagnosed in nestlings and juvenile parrots of the larger species. Insufficient deposition of calcium and phosphorus in the beak and bones results in a soft beak weak, curved bones, and multiple pathological fractures, particularly of the wing bones. In the adult bird, demineralisation of bones occurs. Reported clinical signs in psittacine birds include appetite loss, weakness, lethargy, feather chewing with abnormal blackening of feathers, eggs with thin shells, decreased hatching, embryos with malformed or partially formed beaks (Nott and Taylor, 1993), as well as rapid bone thinning, curved bones and fractures (Roudybush, 1993). Abnormalities of calcium - phosphorus homeostasis are also related to the multifactorial problems of egg-binding in laying hens, and hypocalcaemic syndrome in African Grey and Timneh Grey Parrots (McDonald, 1988, Scott, 1996).

#### **Recommended treatment :** (Doneley, 1996)

Supplement diet with calcium syrup to restore the 2 : 1 (Ca : P) ratio (Calcium Sandoz syrup).  
Ensure adequate exposure to sunlight or provide a vitamin D3 supplement (Calcivet - Vetafarm).  
Avoid high fat seeds such as sunflower, which may interfere with calcium absorption.

#### **Recommended prophylaxis :**

Provide a well - balanced diet, with adequate Vitamin D and sunshine (Doneley, 1996). Calcium supplements in the form of dry cuttlefish, limestone or oyster shell grit, however these are inaccurate methods of supplementation, particularly since many of the larger psittacine birds will not eat cuttlefish. Furthermore, high concentrations of calcium carbonates or phosphates in the diet may make it unpalatable (Nott and Taylor, 1993). Cod liver oil is a valuable source of vitamins A and D as well as iodine, however oversupplementation may result in vitamin A toxicity (Nott and Taylor, 1993), induced vitamin E deficiency due to excessive

polyunsaturated fatty acids (McDonald et al, 1992), or vitamin D toxicity. Susceptibility to the latter varies between species ; macaws appear to be particularly susceptible (Scott, 1996).

## **ii) Goitre (Iodine Deficiency)**

Iodine plays a vital role in the metabolism of birds as with mammals, by its incorporation into the thyroid hormones, triiodothyronine and tetraiodothyronine (thyroxine). Goitre may result from:

- a) ingestion of insufficient iodine in the diet,
- b) ingestion of excessive goitrogens such as found in ground nuts, soya beans and members of the Brassica family such as rape and cabbage, or
- c) ingestion of other elements such as calcium, which interact with the dietary iodine, thereby interfering with its absorption.

Iodine deficiency has been documented in budgerigars, passerines (Blackmore and Cooper, 1982), cockatiels (Fowler, 1986) and pigeons (Hollander and Riddle, 1946). It results in decreased production of the aforementioned thyroid hormones, which leads to an increase in the secretion of thyroid stimulating hormone (TSH). This in turn stimulates enlargement of the thyroid gland, resulting in what is commonly referred to as goitre. Clinical signs most commonly observed in birds are respiratory difficulties and regurgitation due to pressure by the enlarged thyroid on the trachea and oesophagus respectively. **Recommended treatment :**

0.3 % Lugol's iodine, 1 drop / 20 mL water, daily for one week, three times weekly for the second week, then once weekly (Doneley, 1996)

### **Recommended prophylaxis :**

1 drop of dilute Lugol's iodine in 30 mL water once a week, or 1% cod liver oil (Blackmore and Cooper, 1982). Alternatively, a supplemented commercial diet may be fed, such as Trill ®.

## **iii) Iron Storage Disease**

Iron storage disease is a poorly understood and most likely multifactorial disease that affects caged and aviary birds. It is characterised by accumulation of excess iron, and is classified as either haemosiderosis (with no functional or morphological alterations to the tissues involved), or as haemochromatosis (where there is a functional or morphological alteration). It may occur as a primary or secondary condition. Currently, opinion varies as to the importance of nutrition in the aetiology of this disease. Fowler (1986) considers an inappropriate diet as the causative factor (with respect to birds of paradise, *Parasidae spp.*), while Roudybush (1993) is of the opinion that nutrition is only of minor importance.

### **Recommended treatment:** (Doneley, 1996)

Weekly phlebotomy to remove a blood volume equal to 1 % of the bird's bodyweight, combined with a low iron diet (< 150 mg / kg). Recommended prophylaxis : (Doneley, 1996) Feed a low iron diet

## **iv) Selenium - Vitamin E Deficiency**

Vitamin E and selenium play a vital role in preventing damage to cellular membranes by peroxide radicals. In addition to its role as a non - specific antioxidant, vitamin E acts within the cellular membranes to prevent formation of peroxide radicals. Selenium, a component of the enzyme glutathione peroxidase, acts within the cellular cytoplasm to hydrolyse radicals that have already formed. Hence, by the nature of their complementary roles, it is not surprising that vitamin E and selenium share many signs of deficiency in common, and to a certain extent, a deficiency of one can be overcome by supplementation with the other.

The level of selenium in crops is highly variable, and is dependent on the soil conditions in the region. Ullrey, Allen and Baer (1991) support this, noting that three samples of safflower seed (with hulls) were recorded to have selenium concentrations ranging from 0.11 to 1.76 mg / kg DM, and that selenium concentrations in corn alone ranged from 0.01 to 2.03 mg / kg DM. Cereal grains contain reasonable levels of vitamin E, however requirements are increased by diets high in unsaturated fatty acids, such as those high in peanuts, safflower,

sunflower and pumpkin seeds, particularly when these are stored for too long, and the fats turn rancid (Griffiths, 1961). Furthermore, vitamin E activity declines with storage. Thus, deficiency is common, particularly in cockatiels (Scott, 1996). Vitamin E (*alpha* - tocopherol) and selenium levels of a selection of seeds are indicated in *Table 7*.

Vitamin E and selenium deficiencies have resulted in myopathies affecting skeletal, cardiac or the smooth muscle of the gizzard in cockatiels, waterfowl and birds of prey (Nott and Taylor, 1993, Fowler, 1986).

**Recommended treatment :** (Doneley, 1996)

Vitamin E (ADEC - Vetafarm)

**Recommended prophylaxis :** (Doneley, 1996)

Avoid rancid fats, and ensure reliable sources of vitamin E are present in diet

#### v) Hypovitaminosis A

Vitamin A plays two major roles in the body. Firstly it is involved in the differentiation of epithelial cells, and hence in the formation and maintenance of mucous membranes and other epithelial tissues. Its second role is as a component of rhodopsin, the photo - receptor enabling vision at low light intensity.

In plants, vitamin A exists as precursor compounds named carotenoids, which are converted into vitamin A in the intestinal wall and liver. As illustrated in *Table 7*, most seeds are deficient in carotenes. Consequently, hypovitaminosis A is commonly seen in pet psittacine birds fed all seed diets, particularly in African Grey, *Amazona* and *Eclectus* parrots, Gang - Gang cockatoos, and Budgerigars (Earle and Clarke, 1991, Raidal, 1989, Nott and Taylor, 1993), as well as in Columbiforme birds (Tudor, 1982). Clinical signs of vitamin A deficiency are numerous, due to the wide distribution of epithelial cells in the body, degree of deficiency, as well as species variations in susceptibility to deficiency. Reported manifestations of hypovitaminosis A include :

- S** pustular lesions in the larynx and soft palate of pigeons, and in the mucous glands and their ducts of the nasal passages, mouth, oesophagus and crop of chickens (Tudor, 1982)
- S** illthrift in young birds (Fowler, 1986)
- S** sinus infections (Roudybush, 1993)
- fading of pigmentation of skin and feathers (Ingram, 1978)
- night-blindness, retarded growth, weakness, ruffled feathers, staggering gait, decreased egg production and hatchability, as well as mortalities in chickens (McDonald, 1992)
- S** squamous metaplasia of epithelium in the lacrimal and salivary glands as well as of the respiratory and urogenital tracts, the latter resulting in renal or systemic gout (Fowler, 1986)

**Recommended treatment :** (Doneley, 1996)

Parenteral vitamin A (ADEC - Vetafarm) followed by dietary changes to ensure adequate vitamin A or precursors

**Recommended prophylaxis :** (Doneley, 1996)

Balanced diet, with adequate vitamin A or precursors

Foodstuffs containing high levels of vitamin A or precursors are detailed in *Table 10*. Vitamin A toxicity occurs commonly due to over - supplementation with foodstuffs rich in vitamin A, such as cod liver oil, and may prove lethal in severe cases (Nott and Taylor, 1993, Earle and Clarke, 1991). Hypervitaminosis A will not occur in birds fed a diet high in carotenes, however, because the maximal rate of conversion to vitamin A is insufficient (Olson, 1989).

*Table 10. Foodstuffs Containing High Levels of Vitamin A or Precursors (Nott and Taylor, 1993)*

<i>Foodstuff</i>	<i>Vitamin A (IU / 100g as fed)</i>
<i>beet greens</i>	6000
<i>carrot</i>	11000
<i>dandelion greens</i>	14000
<i>spinach</i>	8000
<i>sweet potato</i>	9000
<i>turnip greens</i>	7500
<i>dried red pepper</i>	77000
<i>beef liver</i>	45000
<i>egg yolk</i>	3000

#### **vi) Vitamin C Requirement**

The majority of birds do not have a dietary requirement for vitamin C, however it has been suggested that during times of physiological stress, such as reproduction, moulting, growth or climatic stress, supplementary vitamin C may be beneficial (Nott and Taylor, 1993, McDonald *et al*, 1992).

#### **vii) Vitamin K Requirement**

Intestinal microbial synthesis also occurs, however whether absorption occurs directly (Nott and Taylor, 1993, Fowler, 1986), or requires coprophagy (McDonald *et al*, 1992) is debated. Although rare, vitamin K deficiency may occur following disruption to intestinal flora by prolonged antibiotic therapy (Nott and Taylor, 1993, Fowler, 1986).

#### **viii) Manganese Deficiency**

Signs of deficiency as reported in poultry have not been reported in psittacine or passerine birds. It is postulated by Nott and Taylor (1993) that this is due to the shorter limbs and lighter bodyweight of psittacine and passerine birds compared with ground-living species, rather than due to a lower requirement by the former.

#### **ix) B - Complex Vitamins**

The requirements for B-complex vitamins by caged and aviary birds are derived from poultry research. Deficiency syndromes are commonly characterised by poor growth and feather abnormalities. In addition to this, single deficiencies may produce classic clinical signs. (*Table 11.*)

Table 11. Natural Sources of B - Complex Vitamins and Signs of Deficiency in Birds (Scott, 1996, Nott and Taylor, 1993, McDonald *et al*, 1992, Murphy, 1992)

<i>Vitamin</i>	<i>Natural Source</i>	<i>Clinical Signs of Deficiency</i>
<b>Thiamin, B1</b>	cereal grains, green leaves, egg yolk	anorexia, weakness, seizures, polyneuritis
<b>Riboflavin, B2</b>	green, leafy plants	curled toe paralysis, embryonic mortality mid - incubation
<b>Pyridoxine, B6</b>	cereal grains	perosis, reproductive failure
<b>Biotin</b>	grains, vegetables	dermatitis of foot pads, fatty liver and kidney syndrome
<b>Vitamin B12</b>	intestinal bacterial synthesis	anaemia, gizzard erosion
<b>Nicotinamide</b>	groundnuts, sunflower	inflammation of tongue and oropharynx, bone disorders
<b>Pantothenic Acid</b>	groundnuts, cereal grains	dermatitis, ataxia
<b>Choline</b>	cereals, green leaves, synthesis from methionine	leg abnormalities, fatty liver, perosis
<b>Folic Acid</b>	grains, oil seeds, green leaves	anaemia, nervousness, beak deformity

### Diet Formulation and Feeding

As discussed above, seeds which form the basis of the diet fed to most caged and aviary birds, are deficient in many of the major nutrients, and contain excessive fat.

Furthermore, birds often become fixated on one particular dietary component, commonly sunflower seeds. Contrary to popular belief, birds, with the exception of the domestic fowl, are not able to balance their own intake if offered a mixture of seeds. Thus it is highly important to offer a diet that is balanced to the requirements of the specific bird, according to its species, size, age and physiological state. This may be achieved by providing either a processed diet for example in pellet form, or by combining varied feedstuffs and supplements as a prepared diet.

#### Prepared diets

Doneley (1996) has suggested a prepared parrot diet comprising :

seed and grain products (60 % w / w of diet) : seed mix (20 % sunflower, 30 % millets, 30 % canary seed, 20 % hulled oats), toast, biscuits, rice, pasta, vegetables (10 % w / w of diet) : broccoli, cabbage, dandelion, carrot, corn on the cob, fruit (5 % w / w of diet) : apple, pear, orange, banana, protein (25 % w / w of diet) : meal worms, dog / cat biscuits, peas, soy beans calcium supplementation : cuttlefish, oyster shell grit or calcium syrup

If a diet containing seeds is fed, allowing the seeds to sprout or germinate prior to feeding may increase digestibility and vitamin (particularly vitamin C) concentration. If the water used for these processes is not changed regularly however, bacterial growth may be encouraged (Scott, 1996). Storage conditions for seeds should be such that their moisture content does not exceed 14 %, to ensure that they do not provide a substrate for fungal growth with the subsequent production of mycotoxins.

Considering the great variation in nutrient content of seeds and other foodstuffs, prepared diets commonly require addition of supplements in order to make them complete. Supplements may be delivered to the bird in either its feed or water, however both methods have their shortcomings. Powdered supplements are prone to separate from the food that is eaten, and hence are not consumed in the desired quantity ; coating seeds with suspensions or solutions is ineffective, because the supplement is lost when the bird removes the hull to ingest the kernel ; and when added to water, vitamin and mineral supplements are subject to interactions with dissolved minerals and the irregular nature of drinking of many birds.

One must bear in mind that even if a seed mixture were provided, which when completely consumed meets all

of the nutritional needs of a particular species of bird, preferential selection of more favoured components of the diet by the bird may yet result in an unbalanced diet. Hence, processed diets have been developed.

### **Processed Diets**

Processed pelleted diets are becoming more popular. Arguments for and against the use of such diets are presented below.

#### **Advantages of Processed Diets**

- \* convenience
- \* apparently balanced for various physiological states
- \* nutritionally consistent
- \* increased digestibility of starches by depolymerisation
- \* wholly edible (little waste)
- \* destruction of pathogenic micro-organisms during production

#### **Disadvantages of Processed Diets**

- \* expensive
- \* often not balanced
- \* poor acceptance by some birds
- \* should be fed as sole energy intake
- \* do not stimulate the bird's inquisitive nature
- \* perception that birds do not perform as well as when on a 'natural diet'

### **Conversion to a Processed Pelleted Diet**

Offering a combination of pellets and seeds is an ineffective way of meeting nutrient demands (Ullrey *et al*, 1991). Thus, complete conversion to a pelleted diet is recommended, either gradually or abruptly. With either method it is advisable to provide the new feed in the same feed bowls, in the same position as the previous diet ; to consider meal feeding ; and most importantly, to weigh the bird daily, at a regular time, noting any weight loss. Conversion to pellets is most likely to be successful in a colony situation, where birds are able to mimic the eating habits of others (Underwood *et al*, 1991).

### **Handrearing Altricial Birds**

Energy and nutrient requirements for growth vary with the rate of bodyweight gain and with its composition. Kamphues and Meyer (1991) found that the differential nature of growth of components such as feathers and the skeleton, markedly influenced the composition of bodyweight gain and hence nutrient requirements of growing canaries. Furthermore, the energy density requirement of the diet varies with species and age. For the first three days after hatch, handfed cockatiels require less than 10 % solids in their diet (Roudybush and Grau, 1991), whereas this is insufficient for Blue and Gold macaw chicks (McFadden, 1993). In contrast, from three days of age until weaning, 70 % solids are desirable for cockatiels (Roudybush and Grau, 1991). One must note however, that feeding a diet that is excessively energy dense, particularly if high in fat, can lead to rapid adipocyte hyperplasia in the young bird, and may predispose to obesity later in life (LaBonde, 1992). Furthermore, weak hatchlings should not be fed a high fat diet, as this may result in slowing of gastrointestinal function.

Handfeeding diets must be made freshly for each meal, using cooled, boiled water to minimise risk of bacterial and fungal contamination. Meals should be spread out evenly during the day, and the crop filled at each meal. As a general rule, each meal should consist of a volume equal to 10 - 15 % of the chick's bodyweight (Vetafarm pamphlet, 1994). Chicks should be weighed daily, at a regular time, and should gain weight every day until weaning.

### **Conclusion**

When considering the nutrition and practical feeding of aviary and caged birds, it is essential to consider in concert the energy density, nutrient content, digestibility, and palatability of the diet. Furthermore, these variables should be related to the species of bird, as well as its age, weight and physiological state. Although the complete nutrient requirements for aviary and caged birds have not yet been determined, it is unquestionable that seed - only diets are unsuitable, and may lead to numerous syndromes of deficiency and excess. Processed diets show promise for the future, and as research reveals more information about avian

nutrition, these diets should continue to improve.

### Reference List

- BASKI, S.N. and KENNY, A.D. (1978) Vitamin D Metabolism in Japanese Quail : Gonadal Hormones and Dietary Calcium Effects, *Am. J. Physiol.* **234** (6) : E622 - E628
- BENNETT, P.M. and HARVEY, P.H. (1987) Active and Resting Metabolism in Birds : Allometry, Phylogeny and Ecology, *J.Zool.* **213** : 327 - 363
- BLACKMORE, D.K. and COOPER, J.E. (1982) Diseases of the Endocrine System, pp 478 - 490 in Diseases of Cage and Aviary Birds, Petrak, M.L. (ed.), 2nd ed., Lea and Febiger, Philadelphia
- DONELEY, B. (1996) Control and Therapy of Diseases of Birds, Series A, No 21, University of Sydney Post Graduate Foundation in Veterinary Science
- DONOGHUE, S. (1989) Nutritional Support of Hospitalised Patients, in Clinical Nutrition, Kaffelz, F. A. (ed.), *Vet Clinics of North America* **19** (3) : 475 - 495
- EARLE, K.E. and CLARKE, N.R. (1991) Nutrition of the Budgerigar (*Melopsittacus undulatus*), *J.Nutr.* **121** : S186 - S192
- FRIEDMAN, A., MEIDOVSKY, A., LEITNER, G. and SKLAN, D. (1991) Decreased Resistance and Immune Response to E. coli infection in Chicks, *J. Nutr.* **121** : 395 - 400
- FOWLER, M.E. and LOWENSTINE, L.J. (1986) Nutritional Disorders of Birds, pp202 - 212 in Zoo and Wild Animal Medicine, 2nd ed., , Fowler, M.E. (ed.), W.B. Saunders, Philadelphia
- GINN, H.B. and MELVILLE, D.S. (1983) Moults in Birds, The British Trust for Ornithology, Hertfordshire, England
- GRIFFITHS, T.W. (1961) Studies on the Requirement of the Young Chick for Vitamin E, *Br. J. Nutr.* **15** : 271 - 279
- HARRISON, G.J. and HARRISON, L.R. (1986) Clinical Avian Medicine and Surgery, W.B. Saunders, Philadelphia
- HOLLANDER, W.F. and RIDDLE, D. (1946) Goiter in Domestic Pigeons, *Poultry Science* **25** : 20
- INGRAM, K. (1986) Humingbirds and Miscellaneous other Birds, pp 335 - 346 in Zoo and Wild Animal Medicine, 2nd ed., Fowler, M. E. (ed.), W.B. Saunders, Philadelphia
- JOHNSON, J.H., PHALEN, D.N., KONDIK, V.H., TIPPIT, T. and GRAHAM, D.L. (1992) Atherosclerosis in Psittacine Birds, *Proceedings Association of Avian Veterinarians*, pp 87 - 92
- KAMPHUES, J. and MEYER, H. (1991) Basic Data for Factorial Derivation of Energy and Nutrient Requirements of Growing Canaries, expanded abstract, *J.Nutr.* **121** : S207 - S208
- LABONDE, J. (1992) Obesity In Pet Birds, The Medical Problems and Management of the Avian Patient, *Proceedings Association of Avian Veterinarians*, pp 72 - 77
- MCDONALD, L.J. (1988) Hypocalcaemic Seizures in an African Grey Parrot, abstract, *Can. Vet. J.* **29** : 928 - 930
- MCDONALD, P., EDWARDS, R.A. and GREENHALGH, J.F.D. (1992) Animal Nutrition, 4th ed., Longman Scientific and Technical, Essex, England

- MCFADDEN, C.B. (1993) Anecdotal Report : Caloric Density Requirements in Neonatal Blue and Gold Macaws, *Proceedings Association of Avian Veterinarians*, pp 240 - 243
- MINSKY, L. and PETRAK, M.L. (1982) Metabolic and Miscellaneous Conditions, pp 638 - 645 in *Diseases of Cage and Aviary Birds*, Petrak, M.L. (ed.), 2nd ed., Lea and Febiger, Philadelphia
- MURPHY, J. (1992) Psittacine Fatty Liver Syndrome, *Proceedings Association of Avian Veterinarians*, pp 78 - 82
- NATIONAL RESEARCH COUNCIL (1994) Nutrient Requirements of Poultry, 8th revised edition, National Academy Press, Washington
- NOTT, H.R. and TAYLOR, E.J. (1993) The Energy Requirements of Pet Birds, *Proceedings Association of Avian Veterinarians*, pp 233 - 239
- NOTT, H.R. and TAYLOR, E.J. (1993) Nutrition of Pet Birds, pp 69 - 84 in *The Waltham Book of Companion Animal Nutrition*, Burger, I.,(ed.), Pergamon Press, Oxford
- OLSON, J.A. (1989) provitamin A Function of Carotenoids : the Conversion of beta - carotene to Vitamin A, *J.Nutr.* **119** : 94 - 95, 105 - 108
- PERRY, R.A. (1983) Diseases of Birds, Vade Mecum No. 2, Sydney University Post Graduate Committee in Veterinary Science
- QUESENBERRY, K.E., MAULDIN, G and HILLYER, E (1989) Nutritional Support of the Avian Patient, *Proceedings Association of Avian Veterinarians*, pp 11 - 19
- RAIDAL, S. (1989) Nutrition of Companion and Aviary Birds, Veterinary Science V essay
- RANDELL, M.G. (1981) Nutritionally Induced Hypocalcaemic Tetany in an Amazon Parrot, *J.A.V.M.A.*, **179** (11) : 1277
- REECE, R.L., DICKSON, D.B. and BURROWES, P.J. (1986) Zinc Toxicity in Aviary Birds, *A.V.J.* **63** (6) : 199
- ROUDYBUSH, T.E. (1993) Nutrition, Chicago Seminar
- ROUDYBUSH, T.E. and GRAU, C.R. (1991) Cockatiel (*Nymphicus Hollandicus*) Nutrition, expanded extract, *J.Nutr.* **121** : S206
- SCOTT, P.W. (1996) Nutrition, pp 17 - 26 in *Manual of Psittacine Birds*, Beynon, P.H., Forbes, N.A. and Lawton, M.P.C. (ed.), British Small Animal Veterinary Association, Gloucestershire. U.K.
- SCOTT, M.L., AUSTIC, R.E. and GRIES, C.L. (1978) Nutritional Deficiency Diseases, pp 49 - 78 in *Diseases of Poultry*, Hofstad, M.S. (ed.), 7th ed., Iowa State University Press
- TUDOR, D.C. (1982) Vitamin A Deficiency in Pigeons, *Veterinary Medicine / Small Animal Clinician*, Veterinary Medicine Publishing Company, Kansas
- T-W-FIENNES, R.N. (1982) Diseases of the Cardiovascular System, Blood and Lymphatic System, pp 422 - 431 in *Diseases of Cage and Aviary Birds*, Petrak, M.L. (ed.), 2nd ed., Lea and Febiger, Philadelphia
- ULLREY, D.E., ALLEN, M.E., BAER, D.J. (1991) Formulated Diets Versus Seed Mixtures for Psittacines, *J.Nutr.* **121** : S193 - S205
- UNDERWOOD, M.S., POLIN, D., O'HANDLEY, P. and WIGGERS, P. (1991) Short Term Energy and Protein Utilisation by Budgerigars (*Melopsittacus undulatus*) Fed Isocaloric Diets of Varying Protein Concentrations, *Proceedings Association of Avian Veterinarians*, pp 227 - 237



WALLACH, J.D. (1969) Nutritional Secondary Hyperparathyroidism in Captive Psittacine Birds, *J.A.V.M.A.* **155** (7) : 1046 - 1051

WALLACH, J.D. (1970) Nutritional Diseases of Exotic Animals, *J.A.V.M.A.* **157** (5) : 583 - 599

WATKINS, B.A. (1991) Importance of Essential Fatty Acids and Their Derivatives in Poultry, *J. Nutr.* **121** : 1475 - 1485

WHITTOW, G.C. (1986) Energy Metabolism, pp 253 - 268 in *Avian Physiology*, 4th ed., Sturkie, P.D. (ed.), Springer - Verlag, New York