

Nutritional Considerations

Section II

Nutritional Disorders

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Nutritional disorders can result from malabsorption, a deficient diet, over-supplementation and/or overeating. Deficiencies and excesses of nutrients can both be harmful to birds.

Companion birds have been maintained for decades on diets that, while nutritionally inadequate, support limited breeding in a few species. While there are numerous publications regarding nutritional requirements of agricultural species, captive passerine energetics and feeding ecology, there are few controlled scientific studies on aviary and companion birds or their wild conspecifics. Variations in lifestyle and breeding ecology result in differing nutritional requirements. Clinically, many health problems are correlated with nutritional disorders. This chapter will provide an overview of these conditions observed in companion birds, with reference to anecdotal observations in a clinical context and summaries of nutrient implications that have been predominantly studied in agricultural species. Specific studies of companion and wild birds will be discussed. Parallels may exist between the following description of the improper diet cascade and the metabolic syndrome of humans and rats.^{90b}

The Improper Diet Cascade (IDC)

The 'improper diet cascade' (IDC) (Table 4.2.1) has been postulated by the author (GJH) from decades of clinical experience, reports from pathologists and nutritionists, as well as consultations with companies that produce commercially formulated diets. The IDC expresses itself in a highly individualistic fashion. The most common

thread is the history of a basic seed and table food diet. Generally, at presentation of a "sick" bird, the IDC patient exhibits pansystemic clinical signs that often include various behavioral problems. Typically though, the earliest clinical signs are reflected in the integument, followed closely by the digestive system. Often birds are not presented for evaluation until the reproductive or respiratory system is affected. Behavioral problems can be the proximal cause of veterinary presentation when other clinical signs have been missed or ignored.

The IDC can be initiated from a nutrient imbalanced diet as well as from influences, such as improper husbandry, diet handling and storage or over-supplementation of nutrients in formulated diets. Therefore, when evaluating nutritional disorders, consider the composition of the diet eaten, as well as the stability or availability of nutrients in that diet. Pathological influences such as parasite infestation, metal toxicoses, malabsorption syndromes, pancreatitis and gastroenteritis produce clinical signs similar to those seen in IDC, and therefore need to be ruled out (Table 4.2.2a).

The IDC is the result of improper nutrient utilization, usually from malnutrition that weakens the body immunologically and structurally. This can allow invasion of low level pathogens or commensals of viral, bacterial, or fungal origin.

Recent research by Dr. M. Beck, University of North Carolina¹⁵, showed that when the host is affected by a nutritional deficiency, the invading pathogen is affected as well. By sequencing the viral isolates recovered from selenium-deficient mice, she demonstrated mutations in the viral genome associated with increased pathogenesis of the virus affected by nutrient deficiency. Bhaskaram

Table 4.2.1 | Improper Diet Cascade (IDC)

Nutritional Imbalance						
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MULTISYSTEMIC ABNORMALITIES						
Cellular	Structural	Functional		Immunologic		
Impaired metabolism	Metaplasia of columnar epithelium	Goblet cells mucin production impaired		Commensal organisms normally bound to mucus are not excreted		
Altered cell wall permeability	Increased mucous viscosity	Loss of cleansing ability of mucous		Relationship with commensal organisms disrupted		
Cellular autointoxication Change in GI pH (less acidic)	Loss of normal collagen elasticity	Normal glandular production of various systems suppressed		Bone marrow suppression Decreased IgA, decreased lymphocytes		
Chronic: eg, • Hepatic lipidosis, fibrosis, cirrhosis • Iron storage disease • Irreversible degradation of retinal cones leading to blindness	Chronic: eg, • Abnormal cilia • Renal tubular nephrosis • Follicular atresia • Cataract formation • Bone/muscle abnormalities	Chronic: eg, • Diabetes mellitus • Deposits of high density lipids in vasculature • Exocrine pancreatic insufficiency • Infertility, decreased hatchability of chicks • Secondary hyperparathyroidism		Chronic: eg, • Secondary microbial infections • Increased susceptibility to neoplasia		
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ABNORMALITIES OF SPECIFIC SYSTEMS						
Integument	Gastrointestinal	Respiratory	Renal	Endocrine	Reproductive	Cardiovascular
• Skin • Feathers • Beak • Nails • Fat deposits	• Oropharyngeal • Pancreatic • Hepatic • Intestinal	• Nares • Infraorbital sinus • Syrinx • Air sacs	• Glomeruli • Renal tubules • Ureters • Urodeum	• Pancreatic • Thyroid • Parathyroids • Intestinal • Gonadal	• Ovarian • Uterovaginal • Testicular • Cloacal • Egg abnormalities	• Vasculature • Myocardium • Air capillaries • Pericardium
Biochemical		Hematological		Behavioral		
• AST, ALT • Bile acid • Glucose • HDL, LDL, Triglycerides • Cytokines		• Increased WBC • Altered total WBC		(see subsequent section)		

Table 4.2.2a | Commonly Encountered Etiologies of Improper Nutrient Intake or Utilization

Congenital/Developmental	Individual	Complicating Factors	Rule outs that impair digestion and/or absorption
Improper parental diet	Provision of improper diet	Little or no sunlight	Pancreatitis or organ failure
Improper handfeeding diet	Consumption of improper diet	Lack of bathing	Malabsorption syndromes
Weaned to improper diet	Improper diet supplementation	Lack of exercise	Viral, bacterial, fungal, or parasitic gastroenteritis
Diet constituents interfere with nutrient utilization	Improper food packaging/handling or storage		Metal toxicosis

expanded this theory by showing that several micronutrients such as vitamin A, β -carotene, folic acid, vitamin B₁₂, vitamin C, riboflavin, iron and selenium could be involved in such a scenario in humans.¹⁷ These micronutrient-compromised viruses can lead to the emergence of new infections.¹⁷ This hypothesis was further advanced by Lavender⁶¹, who showed that, at least for RNA viruses, host nutrient deficiencies and excesses can influence the genetic make-up of the pathogen. The majority of viruses are RNA viruses.⁶¹

The importation of wild caught psittacines has traditionally involved weeks to months of stress including severe nutrient imbalance. Such birds imported into the USA in the 1970s and 1980s were a part of a pandemic of new viral diseases. Psittacine beak and feather disease, proventricular dilatation disease and papillomatosis are three that still plague us. The research community has not adequately addressed the role of malnutrition in viral pathogenesis. It is interesting to ponder this hypothesis in light of the new expressions of these same viruses occurring in the European Union countries that still import wild-caught birds.

IMPROPER DIET FORMULATION

There is a general perception that 'fresh' is best. However, presenting a bird with an array of fresh produce, seeds and nuts does not necessarily provide a nutritionally balanced diet. Commonly fed seeds are deficient in a number of nutrients (**Table 4.2.2b**). Much of the produce is sold in its immature state of growth, and even when mature, it does not have the equivalent nutrient profiles of wild food items. Thus such produce is unable to improve the nutrient profile of the diet.

It is imperative that bird owners be informed of the nutritional inadequacies of such diets. In the wild, psittacines usually balance their diets by feeding on a variety of seeds and other plant parts. Primary issues of concern with captive diets are vitamin levels (vitamins A, D, E, and K and the water-soluble vitamins—biotin and B₁₂) and minerals. Seeds do not contain vitamin A and are generally low in the vitamin A precursor β -carotene. Hypovitaminosis A is particularly prevalent in birds on all-seed diets. Mineral levels of seeds can vary among plant species as well as geographically, depending on the composition of the parent soil. Calcium is deficient in most seeds and, while adequate phosphorous may appear to be present, up to 70% may exist in phytate form that is generally indigestible. Fatty acid composition will also vary among seed species and an imbalance can be an important cause of a number of health issues. Many seeds provide adequate total protein but do not contain the complete set of essential amino acids. A diet of predominantly millet seed will result in a lysine defi-

Table 4.2.2b | Nutrient Deficiencies of Seeds

The seeds most commonly fed birds, such as oats, corn, sunflower, safflower and millet, are generally missing 32 ingredients (from eight groups) needed to keep birds healthy. These include:

- **Vitamins** - choline, niacin, pantothenic acid, riboflavin (B₂), cyanocobalamin (B₁₂), biotin (H), D₃, E, K, and folic acid (M)
- **Minerals** - calcium, phosphorous (70% tied up as non-digestible phytates in plant products, such as grains), sodium
- **Trace minerals** - selenium, iron, copper, zinc, manganese, iodine, chromium, vanadium, bismuth, tin, boron
- **Pigments** - chlorophyll, canthaxanthin
- **Protein** - (amino acids) lysine, methionine
- **Fiber** - (mucopolysaccharide) both soluble and insoluble
- **Vitamin precursors** - β -carotene, converted to vitamin A in liver
- **Omega 3 Fatty Acids**

ciency not seen on other seed-based diets. The composition of commercially raised seeds differs dramatically from wild seeds (see Section I of this Chapter).

Birds do not exhibit nutritional wisdom when selecting dietary ingredients; they show a preference for high-energy, lipid-rich seeds, high carbohydrate seeds and fruits. The advent of formulated foods has diminished the incidence of nutritional disorders in the author's (GJH) practice. Yet not all formulated diets are created equal (**Tables 4.2.2c-e**). For example, products that offer the opportunity for selecting favored food items are poorly formulated and can be just as imbalanced as a seed-based diet in the end.

The Association of Avian Veterinarians (AAV) formed a committee of nutrition experts who developed a list of recommendations to assist veterinarians and owners in feeding pet birds (**Table 4.2.2f**).

While some essential nutrients are higher in organically certified plant products, a diet composed solely of organic seeds will present as many nutritional problems as a diet solely composed of non-organic seeds.

There are also the issues of diminished availability of some nutrients by interference from other nutrients and potential breakdown of key nutrients.

OVER-SUPPLEMENTATION

Vitamin toxicity is an aspect of dietary management that is frequently overlooked, but can be responsible for a number of clinical signs of a disease. Many commercially formulated products contain excessive levels of the fat-soluble vitamins A and D. The addition of vitamin supplements with high concentrations of these two vitamins compounds that excess. The generally low levels of

Table 4.2.2c | Provision of Improper Diet - Common Presentations

Excessive quantity of seeds or nuts provided (minimal vitamin A precursors, lysine deficient, decreased vitamin E absorption, inverted Ca:P ratio, excessive calories)	Excessive percentage of fruits and vegetables (deficient in essential amino acids and essential fatty acids, contain excessive sucrose) *Nutritional deficiencies vary widely between fruits and vegetables - see Figs 4.1.2, 4.1.3 and Tables 4.1.8, 4.1.9 in section 1)	Excessive quantity of "table foods" such as the carbohydrate rich pastas and breads (in addition to the aforementioned deficiencies, these provide a medium for yeast overgrowth in susceptible individuals)	Improper/excessive vitamin-mineral supplementation
			Potential toxicities eg, vitamin A,D ₃ , iron, selenium
			Competitive nutrient absorption, eg, excessive fatty acids, phytates, and fat soluble vitamins

Table 4.2.2d | Consumption of Improper Diet - Common Presentations

Formulated diet over-supplemented with vitamins (vitamin A) or minerals (iron). Deficiencies: lysine, L-carnitine	Diet provided requires bird to consume all components to achieve balance	Supplements needed to balance diet are provided as a coating on food that is not entirely consumed
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Table 4.2.2e | Preparation, Packaging and Storage Problems of Formulated Diets

Problems in Preparation	Packaging Concerns	Improper Storage
Inclusion of raw soybeans, oats or brown rice. Cooking soybeans improves the availability of methionine & cystine ^{14b} & destroys trypsin inhibitors. Oats & brown rice are high in lipase [break down fats to free fatty acids & lipoxygenase (oxidizes fatty acids to hydroperoxides)] ^{43b}	Use of oxygen-permeable packaging Oxidation → Rancidity	Continued mycotoxin production
Inclusion of mycotoxin producing agents	Exposure to light	
Poor quality control	Insect contamination	
Over cooking → degradation of nutrients and conversion of <i>cis</i> to <i>trans</i> fatty acids	Pesticide contamination	Insect infestation (eg, transmission of Sarcocystosis)
Addition of artificial coloring/dyes long term effects unknown	Soft plastics may act as phytoestrogens	Degradation of nutrients
Preservatives (such as ethoxyquin) may be toxic or teratogenic. However, in the absence of preservatives, proper packaging and storage are imperative to maintain quality and prevent rancidity.		

vitamin E in both commercial diets and vitamin supplements may exacerbate toxicity. Dietary supplementation should be undertaken only if there is an extensive knowledge of the nutrient composition of both the diet and the supplement. The common clinical practice of injecting vitamins into sick birds may not be defensible, especially if the bird has been on a formulated and/or supplemented diet. See Section 1, Nutrition and Dietary Supplementation for a more in-depth discussion.

RANCIDITY

Altering tissue structure mechanically (hulling, grinding, and crushing in the case of vegetable matter or maceration in the case of animal tissue) releases lipases.

Grains damaged at harvest also allow this lipase release to occur. Similarly, micro-organisms (fungal contaminants) contain lipases that cause hydrolysis of fats.^{43b} So

quality control of source products is essential. The exposure to oxygen, moisture and heat act with the catalysts naturally present in grains (iron, copper) to accelerate the deterioration process at all stages of grain handling and product manufacturing.

These lipolytic enzymes act on lipids to release free fatty acids and triglycerides. In the presence of oxygen, heat and moisture, these fatty acids and triglycerides are auto-oxidized or acted upon by enzymes (primarily stored in the germ) called lipoxygenases. Polyunsaturated fatty acids (oleic, linoleic, and linolenic) are the most likely to be oxidized, and they are usually the most abundant fatty acids in nuts and seeds.^{43b} This oxidation process produces free radicals in a dark environment. A similar but slightly different reaction occurs when exposed to light. Both reactions end with the production of lipid hydroperoxides which further break down, causing rancidity. This process is often self-perpetuating, starting

Table 4.2.2f | AAV Feeding Brochure

Association of Avian Veterinarians Feeding Recommendations

FEEDING COMPANION BIRDS

Feeding of companion birds has been one of the most challenging aspects of their care, primarily because of limited nutritional research on all species. However, based on studies of poultry and other animals, generalizations can be made on adequate feeding practices for companion birds.

FORMULATED DIETS

Formulated bird food products are available from the pet food industry as a convenience to the owner and to ensure a more nutritionally balanced diet than that offered by seeds alone. The current trend is toward specific formulations addressing age, activity, therapeutic, and stress-related needs of the bird. For example, birds have special nutritional needs during molting, egg laying, or raising young. However, improving a diet in the short term in anticipation of these life stages is not effective; the feeding practices must be optimal year round.

Commercial bird food products may be purchased as pellets, nuggets, crumbles, or hand feeding premixes. Converting a seed-eating bird to a formulated diet must be done with care because new items in the cage may not be immediately recognized as food. Your veterinarian can recommend a commercial formulated bird diet and help you with the conversion process.

ALTERNATIVE HOMEMADE DIETS

Where commercial diets are not available, attempts are made to produce a homemade diet. While not ideal for pet birds, these usually offer an improvement over an exclusive seed diet. Overall, however, homemade diets are often lacking in calcium, iodine, selenium, protein, fatty acid balance, fiber, pigments, and vitamins A, B complex, E, and D₃ while providing an excess of carbohydrates, and phosphorus. Additionally, homemade diets with moist ingredients tend to spoil easily and lose nutrients if not stored properly or if made too far in advance of feeding. The time and effort involved in preparing foods and the difficulty in balancing the nutrients make homemade diets impractical for the pet bird owner. Owners choosing a fresh food plan tend to offer too much variety and quantity of food each day, permitting birds to pick out what they like. Birds will not choose a balanced diet if given free choice. Consult your avian veterinarian for specific recommendations on items and quantities to feed.

FRESH WATER

Fresh water must be provided at all times. Some aviculturists and companion bird owners have had success using pet water bottles for birds, thereby limiting soiling of water.

FEEDING TIPS

- Carefully monitor TOTAL food consumption during any diet change.
- Introduce small amounts of a new food at a time.
- Gradually reduce the total volume of seeds as you increase the volume of more nutritional foods.
- Clean all food and water cups and remove old food from the cage daily.
- Do not provide supplemental vitamins unless recommended by your avian veterinarian.

BEHAVIORAL ENRICHMENT

A consistent daily feeding program contributes to physical and mental health as much as a varied diet. The availability of natural items such as branches, empty nutshells, leather pieces and coconut shells create a stimulating environment.

GRIT

Grit is small non-dissolvable rock. The necessity of grit in the diet is debatable. Some birds, such as pigeons, fowl, canaries and finches, appear to need the availability of grit. In psittacine species, an occasional grit particle is harmless but it is not necessary for healthy maintenance of pet parrots, macaws, parakeets and similar species.

SALT

Salt licks are not necessary for birds.

DEPRAVED EATING HABITS

Birds that routinely eat inappropriate materials (eg, feces, enclosure substrate) should be examined by a veterinarian. This behavior may be associated with disease or nutritionally deficient diets and is often prevented by the feeding of a more balanced formulated food product.

SPECIAL REQUIREMENTS

Lories and lorikeets require specialized diets in captivity. These nectar diets attract insects and result in liquid and messy feces. Your avian veterinarian can recommend a diet for these species. Soft-billed birds, waterfowl, backyard poultry and gamebirds Commercial foods are available for these birds. Some toucans and mynahs may have a special dietary requirement for a low-iron formula. Consult your avian veterinarian for recommendations.

slowly and increasing rapidly as reaction chemicals become available.

Expressing the oil from seeds increases the surface area being exposed to oxygen, which can increase the possibility of rancidity occurring.

The production of lipid hydroperoxides does not appear to alter flavor. Lipid hydroperoxides deteriorate to aldehydes in the presence of oxygen.^{43b} These do alter flavor and finally palatability. Alcohols and hydrocarbons are also produced. These latter products have been reported to be mutagenic.¹⁶ Rancid fats can lead to selenium and vitamin E deficiencies implicated in encephalomalacia, pancreatitis, myocardial necrosis, hepatic necrosis and general myopathy. Biochemical analysis of affected birds' blood may show anemia, elevated lactate dehydrogenase (LDH), aspartate aminotransferase (AST), creatinine phosphate (CK) and phosphorous levels. Many of these clinical conditions are not reversible.⁹⁸

Chickens fed diets with increased rancidity parameters (peroxide and aldehyde concentrations) experienced increased mortality from fatty liver syndrome (FLS). Total blood proteins of affected chickens were elevated, as were lipoproteins and total lipids.²⁸

HANDLING AND STORAGE

Wild birds naturally feed on an array of fresh foods while their captive counterparts are provided with foods that have been stored for extended periods. Nutritionally imbalanced food supplies are not uncommon in wild situations. Agriculture produces seeds and nuts only at the end of the growing season, usually in the fall. Storage increases the potential for nutrient degradation. Nitrogen flushing and storage under refrigeration are steps that discourage oxidation.

COLD DARK STORAGE HELPS PREVENT RANCIDITY

Storing walnuts in the light at 21° C resulted in profound oxidative changes.⁵¹ However, walnuts stored in the dark at 5° C for 25 weeks, even in 50% oxygen, were without a trace of rancid taste.⁵¹ However, it should be remembered that rancidity, as determined by chemical analysis, precedes taste detection.

Storing corn oil at room temperature for 48 months resulted in rancid oil, whereas storage in the refrigerator did not.¹⁶ A specific strain of mice fed the rancid corn oil showed significantly increased expression of oncogenes in all major organs. The results demonstrated that rancid oils, rich in *n*-6 polyunsaturated fatty acids, could initiate tumors and promote tumor growth.⁸⁶

COOKING

In the preparation of a formulated diet, cooking (roasting, pelletizing or extrusion) is designed to stabilize oils. However, depending on the condition of the products being mixed, some combinations may cause flash rancidity (D. Jones, personal communication 2000). This is due to the presence of enzymes in items like grains and peanuts that cause natural fermentation when exposed to warm moist air. The lipase concentration in some grains is very high. Oats and brown rice are examples.^{43b} Dehulling and milling these products causes rapid deterioration (rancidity) unless they are heat stabilized prior to storage or further processing. When these raw products are combined under the heat of processing, this flash rancidity can occur. For this reason, these ingredients need to be roasted or other wise partially cooked separately, then mixed with the other ingredients prior to final processing.

Raw soybeans contain trypsin inhibitors and can therefore be difficult to digest. This enzyme is a critical part of digestion in monogastric animals. Trypsin inhibitors are inactivated by heat.^{14b,70b} Cooking also improves the availability of methionine and cysteine.^{14b} Overcooking destroys or makes unavailable certain amino acids (lysine) and greatly reduces natural vitamin precursors such as tocopherols and carotenoids.

MOISTURE CONTENT

Lowering the moisture content of a product also acts as a stabilizer. Moisture plays a vital chemical role in most oxidation processes.^{43b} Levels below 5% are often required to deter degradation. The author (GIH) has shown that these low moisture levels cause minor proventricular irritation evidenced by excessive regurgitation and minor weight loss in some pet umbrella cockatoos. Even at these low moisture levels, over time non-free 'water' is all lipases need to act. Non-free water cannot be removed by drying.^{43b}

PACKAGING

Many bird foods are packaged in plastic, cellophane, coated paper or cardboard boxes. The latter two prevent exposure to light. Airtight containers (plastic, cellophane) prevent moisture from evaporating, but many do not stop oxygen from crossing into the food. The oxygen then breaks down essential nutrients or changes their biological activity. An advertised vitamin A content of 12,500 IU/kg may be reduced to as few as 1,500 IU/kg by inadequate packaging, with further deterioration once the package is opened. Even if one starts with a nutritionally sound, preservative-free formulated diet, the lack of proper packaging and resulting rancidity cancel its effectiveness.

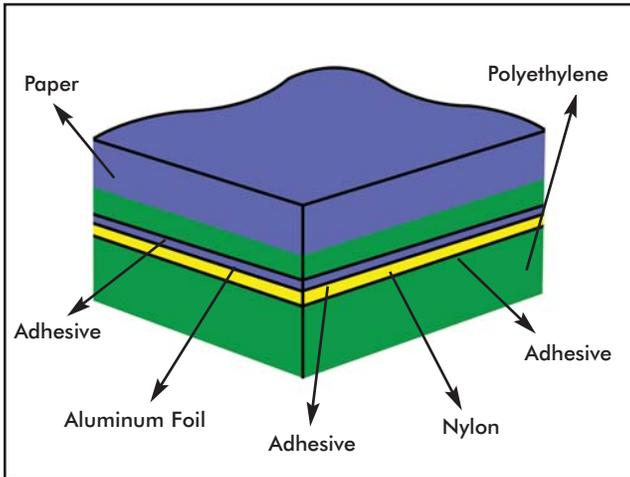


Fig 4.2.1 | Quadruple laminate packaging helps preserve the freshness of fomulate diets and prevents rancidity.



Fig 4.2.2 | A red-lored Amazon fed a seed and table food diet has an overgrown maxillary rhamphotheca that has been recently honed down to a more normal shape.



Fig 4.2.3a | A green-winged macaw fed a seed and table food diet. The red feathers are almost pink. Black pigment is co-mingled with green. The beak is hyperkeratotic. The breast and wing feathers are tattered and picked.



Fig 4.2.3b | A yellow-naped Amazon fed a seed and table food diet. The bird is obese, the maxillary rhamphotheca is overgrown and the feathers are abnormally pale green. The rectrices are tattered. Structural abnormalities make the coverts of the wing and body contour feathers lack the homogeneous interlocking appearance of a normal bird.

To avoid oxygen deterioration, chemical preservatives like ethoxyquin (originally used to soften rubber, later as a herbicide) and propylene glycol have been used for decades in dry animal foods. They have not been deemed safe for human foods. Recent public demand for more natural pet foods has led to a variety of newer techniques to avoid rancidity.

Lipid peroxidation can particularly affect products composed of organic ingredients that lack synthetic preservatives but is no less an issue for any products that are

inadequately packaged.

For these reasons, all foods need to be smelled when first opened. If they smell like old frying grease or linseed oil they are rancid. A taste test should be observed when first offering a new bag of food to the bird. If the bird acts hungry but rejects the food it might be rancid. Rancid foods should not be fed. Following the manufacturer's directions for handling the food and shelf life will usually prevent rancidity problems.

Table 4.2.3 | IDC and the Integument

Nutritional Imbalance			
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CLINICAL SIGNS			
Feathers	Skin	Beak and Nails	
<ul style="list-style-type: none"> • Frequent or incomplete molts • Retained feather sheaths • Abnormal coloration • Irritability due to feather discomfort • Deformities: <ul style="list-style-type: none"> - Cysts - Loss of elasticity - Barbules not interlocking • Chronic/Severe <ul style="list-style-type: none"> - Feather destructive behavior - Self-mutilation 	<ul style="list-style-type: none"> • Flaky • Dry • Pruritic • Hyperkeratotic • Loss of elasticity (tears readily) • Plantar surface - loss of pattern • Chronic/Severe <ul style="list-style-type: none"> - Pododermatitis 	<ul style="list-style-type: none"> • Excessive length • Exaggerated curvature • Friable texture • Splitting • Bruise readily • Chronic/Severe <ul style="list-style-type: none"> - Marked deformity - Secondary infections 	
↓			
INITIAL TREATMENT			
Husbandry	Medical	Procedural	Diet Conversion
<ul style="list-style-type: none"> • Increase UVB • Increase outdoor exposure: <ul style="list-style-type: none"> - Humidity - Ventilation - Sunlight - Psychological stimulation • Improve available perches, increased variety of sizes and textures • Increase exercise, both physical and mental • Verify or improve hygiene 	<ul style="list-style-type: none"> • Antipruritics: <ul style="list-style-type: none"> - Systemic - Topical • Treat secondary infection if present and significant • Psychotropic medications if self-mutilating • Parenteral vitamin supplementation for severe deficiencies • Blood work and other diagnostics if indicated 	<ul style="list-style-type: none"> • Trim overgrown beak • Trim overgrown nails • Remove damaged feathers • Trim rachis of feathers if irritating bird • Mechanical barrier to self-mutilation if needed • Medicated padding and or bandaging for pododermatitis when present 	<ul style="list-style-type: none"> • Evaluate for the following: <ul style="list-style-type: none"> - Essential amino acids - Balanced fat and CHO - Vitamins at physiologic levels (not excessive or deficient) - Free of dyes and preservatives - Need for supplemental essential fatty acids • Formulated diet is often most convenient and effective • Monitor weight during conversion
↓			
EVALUATION OF THERAPY			
<ul style="list-style-type: none"> • Dietary conversion is necessary for long-term treatment and control. • Anticipate exacerbation of clinical signs for 3-9 months (pruritus, flaking of skin and molting) with integumentary regeneration. • If clinical signs worsen, perform diagnostic work-up for systemic sequelae to IDC. • At 9-12 months, marked improvement should be noted in initial clinical signs. 			

There are few natural oxidative inhibitors. Tocopherols (vitamin E) and rosemary leaves have been tried. In the author's experience, preliminary studies of products containing rosemary had less than ideal acceptance, and the test subjects' had lower than desired body weights.

The natural antioxidants found in whole cereal grains have not been fully exploited.

The development of quadruple laminate bags (Fig 4.2.1), consisting of a layer of poly-coated extruded paper

(blocking light), a layer of nylon for puncture resistance, a metal alloy as a barrier to oxygen and a polyethylene layer to resist changes in moisture and retain oils, have increased shell life of non-synthetically preserved products by up to 14 months. However, once the seal is broken and exposure to oxygen and moisture increases, these products are only viable for up to six weeks before clinical signs produced in birds resemble those of birds maintained on diets depicted in Figs 4.2.2-4.2.3a,b. It is important that clients adhere to the manufacturers'



Fig 4.2.4 | A blue and gold macaw that was fed a diet of pasta, crackers, cookies, pellets and vegetables. The feathers are tattered and lack symmetry. The blue feathers contain a black pigment. Under the contour body feathers, the bird had an excessive number of pinfeathers.



Fig 4.2.5 | This blue and gold macaw hen died after laying a clutch of 5 infertile eggs. Note the pinfeathers after all the body and extremity feathers were removed. Also note the black pigment in the normally blue feathers. The bird had been fed a seed and table food diet.

Table 4.2.4 | Using a Formulated Diet

Shelf Life	Use within 4-6 weeks of opening*
Storage	Store in manufacturer's packaging only if adequate**
	Express air to minimize oxidation
Feeding Frequency	Offer fresh food 2-3 times daily dependent on species
Feeding Amount	Ensure bird eats all food offered, including crumbs. Amount fed should maintain normal body weight.
Selective Feeding	Don't allow bird to favor individual particles
Supplementation	Follow manufacturer's instructions as to types and amounts of supplementary foods
Water	Don't allow birds to dunk food in water as this degrades vitamins and pollutes water leading to bacterial and fungal overgrowth

*Diets composed of nonorganic ingredients may have a longer shelf life due to synthetic preservatives.

**Many products are packaged in inferior packaging resulting in breakdown of key nutrients before packaging is even opened. Quadruple laminate packaging preserves nutrients for extended periods of time.

storage directions, as even nutritionally adequate diets have a limited shelf life once opened (see Table 4.2.4).

The IDC from a Systemic Point of View

Although birds seldom present with only one system affected by improper diet cascade, diagnosis, treatment and prevention are best discussed by looking at a single system at a time.

Early recognition by the clinician of the effects of IDC on various systems allows diagnosis and implementation of dietary therapy. This is a key element in avian preventive health care.

INTEGUMENTARY SYSTEM

The integument is the site where clinical signs of dietary inadequacy often appear to be noticed first, but these early stages are so commonly encountered that they may not be perceived as abnormal (Table 4.2.3). The stratified squamous epithelial (SSE) cells characteristic of skin are involved in the production of integumentary components such as the nails, beak, feathers, and feather follicles. In addition to the integument, SSE cells are found in the rhinal cavity, mouth, salivary duct junctions, tear ducts, ear canal, syrinx, air sac junctions to the lungs, bile duct, pancreatic duct, cloaca, renal tubules and vagina. Nutritional imbalance can influence the structure and function of any of these sites. While nutritional inadequacies are most often manifested in the integument, the clinical presentation can be complicated by more serious underlying illnesses. The development of nutritionally balanced formulated diets has dramatically reduced the incidence of dermal disorders, but such diets are far from successful in totally eliminating these problems once they have developed.

The Physical Exam Form outlined in Chapter 6, Maximizing Information from the Physical Examination, is a useful tool for identifying signs and common clinical presentations listed in Table 4.2.3. Minor integumentary signs are often overlooked by the bird care industry. It is important to establish a program of wellness with regular checkups, especially for new birds, to identify problems with nutritional inadequacies at an early stage.

KERATINIZATION

Hyperkeratosis is characterized by failure of the new cells to differentiate beyond the squamous stage.

Table 4.2.5 | IDC and the Digestive System

Nutritional Imbalance → Pathology of gastrointestinal organs, liver, pancreas			
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CLINICAL SIGNS			
Body Condition	Behavior	Flora	Digestive
<ul style="list-style-type: none"> • Obesity • Loss of muscle mass • Bleeding • Chronic <ul style="list-style-type: none"> - Emaciation - Fatty liver - Cirrhosis - Hemochromatosis 	<ul style="list-style-type: none"> • Regurgitation • Vomiting • Loss of appetite • Listlessness • Aggression 	<ul style="list-style-type: none"> • Total # gram-positives decrease • Lower % gram-positive rods • Gram-negative bacteria increase • Yeast not budding • Chronic <ul style="list-style-type: none"> - Gram-negatives predominate - Budding yeast - Enterotoxemia (gram-positives explode) - Clostridial overgrowth 	<ul style="list-style-type: none"> • Bilestained urine, urates and stool • Occult blood in stool • Undigested food and fiber in stool = pancreatic failure • Liver shadow increases or decreases • Chronic <ul style="list-style-type: none"> - Ileus - Diarrhea
TREATMENT			
Diagnostics for Secondary Infections	Medical Treatment	Environmental Concerns	Dietary Conversion
<ul style="list-style-type: none"> • Endoscopy and organ biopsy • Culture and sensitivity • Radiology • Hematology • Biochemistry • Ultrasound 	<ul style="list-style-type: none"> • Fluids • Systemic treatment of secondary infections • GI stimulants • Bacteria, enzyme replacement • Lactulose • Milk thistle • SAMe • Apple cider vinegar • Chronic <ul style="list-style-type: none"> - Ultra clear^{®f} - Hepasan^{®e} 	<ul style="list-style-type: none"> • Heat • Proper humidity 	<ul style="list-style-type: none"> • Same as Table 4.2.3 • Formulated diet is often most convenient and effective

Dysfunctional, excessively keratinized cells replace normal cells. This can result in epithelial lesions and an increased susceptibility to infection. If the imbalance is severe and prolonged, columnar epithelium undergoes metaplasia to SSE. Keratinization can result in a loss of function of the tissues involved, including those of the alimentary, reproductive, respiratory and urinary tracts.

Clinical signs of hyperkeratosis involving the integumentary system can manifest as overgrowth of the beak and nails, which retain their outer covering due to a proliferation of basal cells. The keratinized outer coatings of pinfeathers are thicker, less flexible and retained much longer than normal. Retained coatings prevent pinfeathers from opening and such feathers appear to be painful to the birds if the unopened feathers are manipulated. Clients commonly report that birds with chronically retained pin feathers are irritable and vocalize as if in pain during preening (Figs 4.2.4 and 4.2.5).

While hyperkeratosis is generally associated with dietary deficiencies of vitamin A, excesses of vitamin A are also correlated with hyperkeratosis. The percent of squamous cells present in nasal flushes has been used as an indicator of vitamin A toxicosis.⁵⁸ It is important to obtain a full dietary history before prescribing vitamin A

supplementation to treat hyperkeratosis. In rodents, oral supplementation with vitamin A failed to raise serum vitamin A levels in the absence of adequate vitamin E.⁶ Therefore a mixture of both vitamin E and vitamin A may be required to treat hyperkeratosis due to a vitamin A deficiency. Deficiencies of zinc and biotin have been associated with hyperkeratosis. Biotin deficiencies, which can result from excess of salt, are correlated with hyperkeratosis on the footpad and the plantar surfaces of the toes.⁷ Thus the caveat to not treat all hyperkeratosis with vitamin A injections is valid.

GASTROINTESTINAL SYSTEM

Secondary to the dermal system (and some behavioral traits), the avian clinician is likely to observe gastrointestinal tract (GIT) dysfunction next in the unfolding of the IDC (Table 4.2.5). Vitamin A deficiency may interfere with normal growth, rate by influencing functionality of the small intestine by altering the proliferation and maturation of cells of the intestinal mucosa.¹⁰⁴ Hyperproliferation of enterocytes, decreased number of goblet cells, decreased alkaline phosphatase activity, and decreased expression of brush-border enzymes are all correlated with vitamin A deficiencies.¹⁰⁴

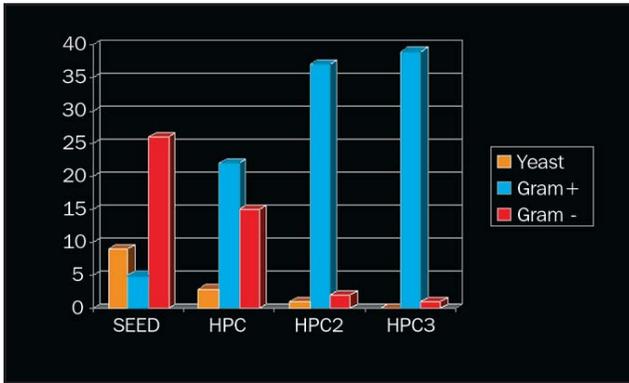


Fig 4.2.6 | Improvement of fecal Gram's stains over time with only the use of an organic formulated diet.¹⁰²

Key:

Seed = Initial assessment of 100 birds after 12 months of eating a seed-based diet
 HPC = Samples collected 4 weeks after changing 80 birds to Harrison's High Potency Coarse (HPC) pelleted formulation
 HPC2 = Samples collected from same birds after 8 weeks on HPC
 HPC3 = Samples collected from same birds after 12 weeks on HPC

The Fecal Gram's Stain in Psittacines

While early studies of captive birds indicate that they commonly have low levels of gram-negative bacteria in cultures of feces,^{5,9,18,21,54,99} other researchers maintain that autochthonous flora in healthy parrots are not gram-negative.^{37,38,45,46,47,102} Normal fecal flora of psittacines is comprised of 100% gram-positive, non-spore forming rods and cocci.^{37,38,45,46,47,102} One study of wild yellow-naped Amazon chicks showed 60% of cloacal cultures had Enterobacteriaceae.⁹⁹ The author (GJH) has hypothesized that this group of nesting birds were under undue stress from poachers, the presence of humans guarding the nests and a declining natural environment. For 20 years, this author (GJH) has used fecal Gram's stains to evaluate the normal flora of pet birds.⁴⁶ Studies of wild psittacines in a recent trial confirm the absence of gram-negative bacteria.^{45,47} Gram-negative bacteria were reduced to almost zero after conversion of African grey parrots from a typical seed-based diet to a nutritionally balanced one.¹⁰² (Fig 4.2.6) Glunder found (*E. coli* or *Klebsiella* spp.) it nearly impossible to colonize in the intestine of budgerigars on nutritionally balanced diets.³⁸ Joyner⁵⁴ whose MPVM thesis study looked at breeding pairs of aviary budgerigars on a seed-based diet, reported a 60% cloacal presence of gram-negative rods, and reported this as normal. This may be considered normal for a seed-eating budgerigar in this study, but it should not be considered normal for a healthy bird on a balanced diet.

Normal resident microflora maintains an acidic environment that inhibits the proliferation of gram-negative rods and yeast. An imbalance in the intestinal homeostasis results in alterations to the normal populations of microflora, and thus the distribution of bacteria in the

Table 4.2.6 | Techniques for Performing a Fecal Gram's stain

1. Fecal samples should ideally be collected at home and refrigerated until evaluated to prevent the proliferation of saprophytic gram-negative bacteria that may be interpreted as pathogenic.
2. A small amount of feces should be applied to a pre-cleaned glass slide using the wooden end of a cotton-tipped applicator. The sample should be spread into a uniform, thin, even film, using a single swath.
3. Heat fix
4. Place slide on staining tray:
 - Apply 3 drops of gentian violet to the sample and allow to stand for 30 seconds (stains all bacteria blue).
 - Rinse with water and drain excess water.
 - Apply 3 drops of Gram's iodine and allow to stand for 30 seconds (closes pores on gram-positive bacteria).
 - Apply 5 drops 95% ethyl alcohol to decolorize blue stain from gram-negative bacteria.
 - Rinse immediately with water and then add 5 drops saffron to stain gram-negative bacteria red.
 - Rinse immediately with water and blot dry with lens paper or tissue.
5. Scan slide under low microscope power for suitable evaluation site.
 - Using the oil immersion lens, scan several fields for a further idea of uniformity.
 - Choose a uniform field and begin to estimate the total number of bacteria, i.e., count 10 bacteria, assess the proportion of entire field occupied by those 10 bacteria and then estimate the total bacterial population per 1000x field.
6. Record results (see Table 4.2.7)
7. The presence of gram-negative cocci indicates improper staining technique.

With experience, the entire process should take less than 2-3 minutes to perform.

Table 4.2.7 | Recording Results of the Fecal Gram's Stain

Date _____

Species _____

Case ID _____

Results:

_____ total bacteria/1000x oil immersion field

_____ % G+ rods/field

_____ % G+ cocci/field

_____ % G- rods/field

_____ number yeast/field

_____ % budding yeast

_____ high fiber in feces

_____ undigested food

_____ parasites

_____ clostridial organisms

_____ hyperkeratotic cells

_____ normal intestinal cells

_____ RBC's

_____ WBC's

GIT. Normal intestinal flora of parrots, seen as gram-positive (blue) bacteria on a fecal Gram's stain, represent both aerobic and anaerobic bacteria such as *Bacillus*, *Corynebacterium*, *Streptomyces*, *Lactobacillus*, *Streptococcus* and *Enterococcus* spp.,³⁷ some of which are not able to be cultured using standard techniques.

Enterobacteriaceae are gram-negative (red) bacteria that include pathogens (eg, *Salmonella* spp., *E. coli*, *Acinetobacter* spp.) and non-pathogenic species. Enterobacteriaceae are not normal components of unstressed parrots' microflora³⁷ and are not detected in preliminary studies of wild parrots.^{45,47} However, normal flora bacteria can become secondary pathogens depending on the functional state of the host defense system.³⁷ Systemic disease, including septicemia and death, can occur when bacteria leave the mucosal surface and penetrate the intestinal wall, a situation that can be precipitated by an imbalanced diet influencing the integrity of mucosal surfaces. Parrot-specific *Lactobacillus*⁸ (currently only available in Europe) has been used successfully to treat chronic coliform infections, eliminating the incidence of *E. coli* on culture.³⁷ Techniques for performing and recording a fecal Gram's stain are outlined in [Tables 4.2.6](#) and [4.2.7](#).

The fecal Gram's stain is an important component of complete patient evaluation of psittacines. Although not definitive in making a diagnosis, it provides a visual screen of the proportions of bacteria present in the GIT at the time of sampling. When interpreted in conjunction with a complete physical exam and diet history, it can determine the next diagnostic step: whether to proceed to a culture and aggressive therapy or to treat conservatively with husbandry changes.

Interpreting a Fecal Gram's Stain

Ideally, one should use a fecal Gram's stain in conjunction with culture and antibiotic sensitivity testing and only then, antibiotic therapy. [Figs 4.2.7-4.2.22](#) represent a range of fecal Gram's stains commonly seen in clinical practice (1000x oil immersion field) from psittacines maintained predominantly on seed-based diets. [Figs 4.2.23-4.2.27](#) are representative of wild Australian psittacines taken from birds in the December breeding season when diets include a number of wild blossoms (D. Brennan, personal communication). A healthy psittacine should have a predominance of gram-positive rods and cocci, with an absence of gram-negative rods.

Malnutrition and liver disease are characterized by changes in the number and distribution of bacteria on the fecal Gram's stain. In the early stages, the change is reflected by:

- Decrease in total bacteria

- Decrease in percentage of gram-positive cocci
- Increase in % gram-positive rods

In the later stages of malnutrition and liver disease, the Gram's stain generally shows:

- Increase in presence of gram-negative rods (generally speaking, the more gram-negative rods, the more pathologic the situation)
- Presence of yeast which are judged as to their clinical significance by the number of budding yeast per field. The greater the percentage of budding yeast found, the more likely that the immune system is compromised.

The fecal Gram's stain of the stool from pet passerines should be free of bacteria, yeast and *Macrorhabdus* sp. organisms.

Clostridia (gram-positive, anaerobic organisms), are commonly associated with fetid stools in both cockatoos with cloacal prolapse and macaws with cloacal papillomas. Both the clostridial organism and the underlying cause require treatment¹⁰⁶ ([Figs 4.2.16](#) and [4.2.18](#)).

Hepatobiliary System

Fatty Liver Syndrome

The following discussion is offered because the author (GJH) believes fatty liver hemorrhagic syndrome (FLHS) of poultry is similar to a common clinical disease in psittacines, which is primarily a result of malnutrition.⁷¹ FLS, generally a consequence of an imbalance in energy metabolism, is associated with an accumulation of excessive abdominal and hepatic fat. Lipid infiltration weakens the hepatic cellular structure and results in hepatomegaly. Lipid deposits are also found in some skeletal muscles, alimentary tract, autonomic ganglia, CNS, pineal gland, kidney, heart and occasionally, small amounts are seen in the corneas, exocrine pancreas, adrenal medulla and epithelium of the thyroid follicles. Endogenous hypercholesterolemia and cessation of egg production are characteristic signs of a similar disorder in poultry, fatty liver hemorrhagic syndrome (FLHS).¹⁰⁰

The numerous blood vessels of an enlarged friable liver are easily ruptured during egg laying. The rupture of large blood vessels can result in death. This disease (FLHS) is most often seen in apparently healthy poultry in a high state of egg production. It also affects young birds, especially chicks from young parents, with 50% higher mortality in females than males. Fatty liver syndrome is especially evident in older, overweight pet birds that are fed a diet of seeds or nuts, but can be seen in handfed chicks as well. Trauma associated with adult birds falling from a perch or being held for a routine clinical examination has caused hepatic rupture in FLS

Figs 4.2.7-4.2.12 | Fecal Gram's Stains (FGS) Commonly Observed in Psittacines in Clinical Practice (oil immersion 1000x).

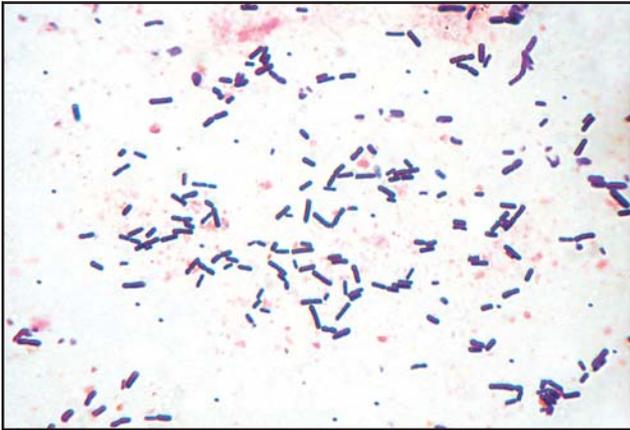


Fig 4.2.7 | Budgerigar, 4-year-old male: Hx = Apparently healthy bird, fed organic formulated diet.^b Normal flora. Clinical Signs (CS) = none. FGS = Normal distribution of organisms: 157 total bacteria per field, 70% gram-positive rods, 30% gram-positive cocci, 0 gram-negative bacteria, 0 yeast. Digestion of food is complete.

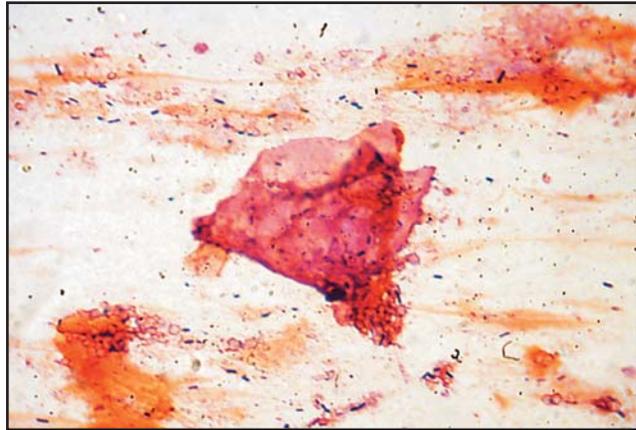


Fig 4.2.8 | Cockatiel, 14-year-old male: Hx = Bird presented for boarding, seed diet. CS = Dull feather color, retained pin feathers. FGS = 55 bacteria per field; 90% gram-positive rods, 10% gram-positive cocci. Hyperkeratotic cell with characteristic straight sides suggests intestinal microflora imbalance, probably due to malnutrition, early liver disease. Rx = Conservative, diet change.

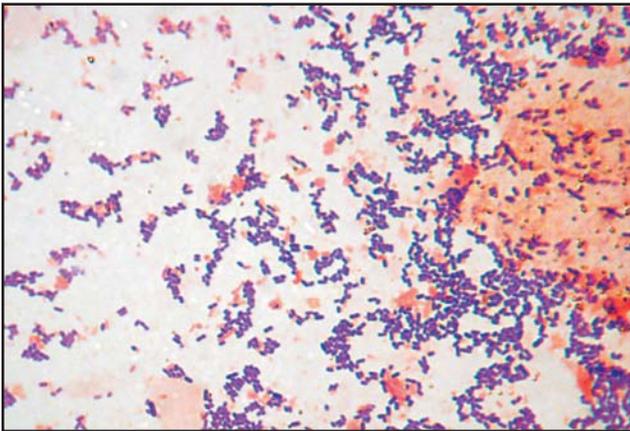


Fig 4.2.9 | African grey parrot, 4 years old, sex unknown: Hx = Intermittent vomiting or loose stool, not as playful. FGS = 400 bacteria per oil field, 95% gram-positive short rods, 5% gram-positive rods, 0 yeast. Overgrowth of intestinal bacteria, enterotoxemia, malnutrition. Rx = Aggressive, dietary change, antibiotics and supportive care.

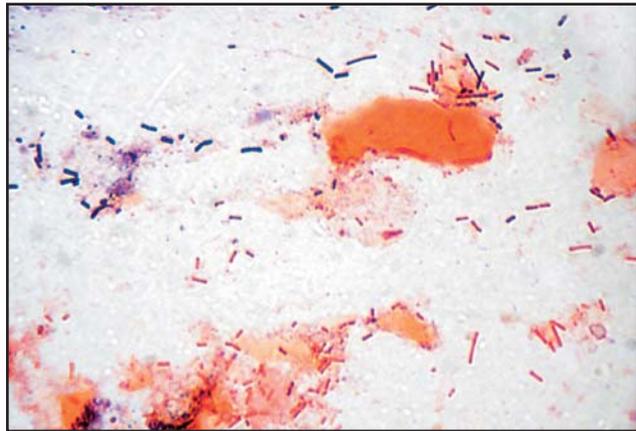


Fig 4.2.10 | Psittacine: Iatrogenic gram-negative rods due to staining error. An error is suspected when the demarcation of gram-positive and -negative is streaked and the groups are similar in shape and size, differing only in color. Note the presence of a normal intestinal epithelial cell, which is rounded and takes on a blue color. Compare this to the straight, pointed edges of the hyperkeratotic cell in Fig 4.2.8. Rx = None.

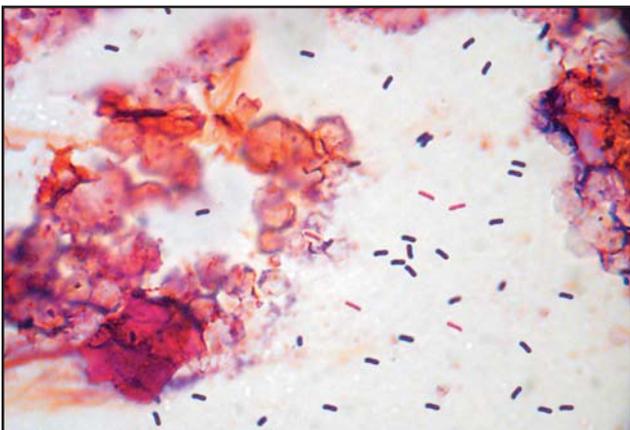


Fig 4.2.11 | Amazon parrot, 8-year-old, female: Hx = Finicky eater, occasionally grumpy. CS = Failure to molt correctly, balding of feet, obvious layering of beak, overgrowth of nails, minor feather-picking. FGS = 40 bacteria per field, 90% gram-positive rods, 0% gram-positive cocci, 10% gram-negative rods. (The normal binding of urates by protein is occasionally seen in fecal Gram's stains). Rx = Conservative diet change.

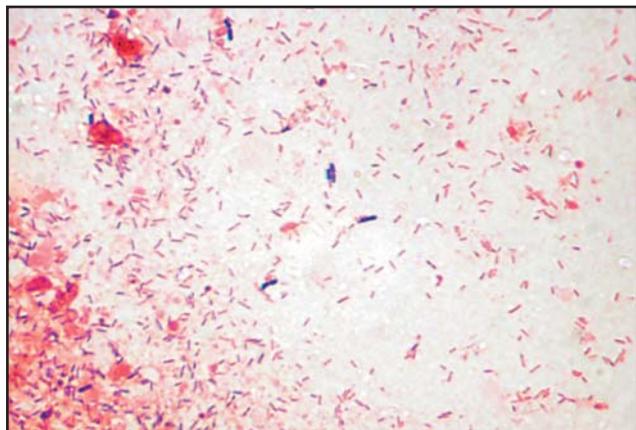


Fig 4.2.12 | Severe macaw, 7 years old, sex unknown: Hx = Depressed, not eating, weak. CS = Underweight, scant feces, dark yellow urine and urates, malcolored feathers. FGS = 200 bacteria per field, 1% gram-positive rods, 0% gram-positive cocci, 98% gram-negative rods. Rx = Aggressive for enteritis and septicemia.

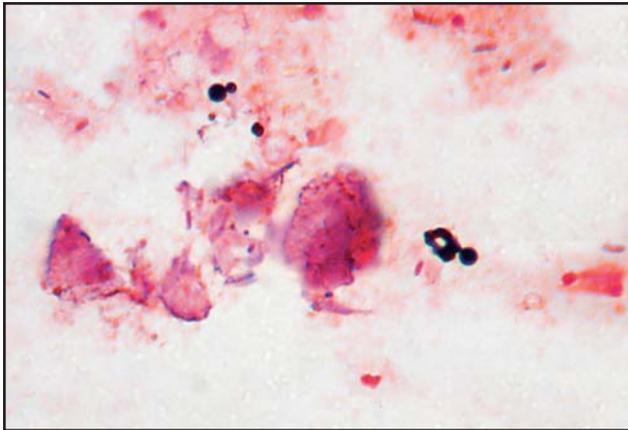
Figs 4.2.13-4.2.18 | Fecal Gram's Stains Commonly Observed in Psittacines in Clinical Practice (oil immersion 1000x).

Fig 4.2.13 | Meyer's parrot, 6 years old, sex unknown: Hx = Diet of seeds and supplements, treated previously for "bacteria." CS = Depressed, fluffed, poor appetite. FGS = Scant bacteria, two budding yeast organisms, suggesting early malnutrition. Rx = Aggressive, antimicrobials, dietary change and supportive care.



Fig 4.2.14 | Ring-necked parakeet, 9-year-old male: FGS = Scant gram-positive bacteria, occasional gram-negative, many apparent bacterial forms and colors; invasive filament of yeast budding bi-directionally. Rx = Aggressive, antimicrobials, supportive care, dietary correction.

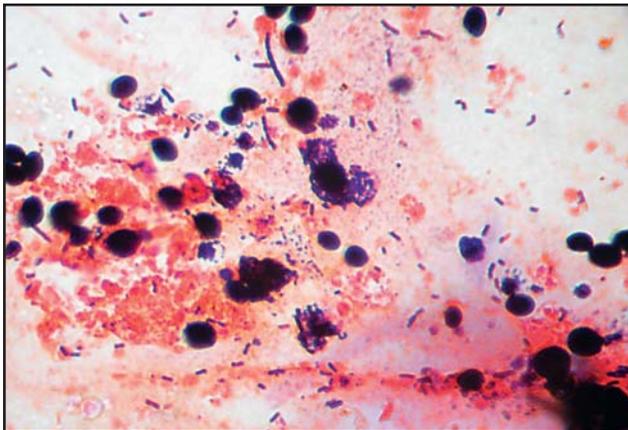


Fig 4.2.15 | Cockatiel, 8-year-old female: FGS = 80 bacteria per field, 80% gram-positive rods, 20% gram-positive cocci; 20 non-budding, yeast-like structures (possibly from bakery products in diet, not clinically significant). Rx = None.

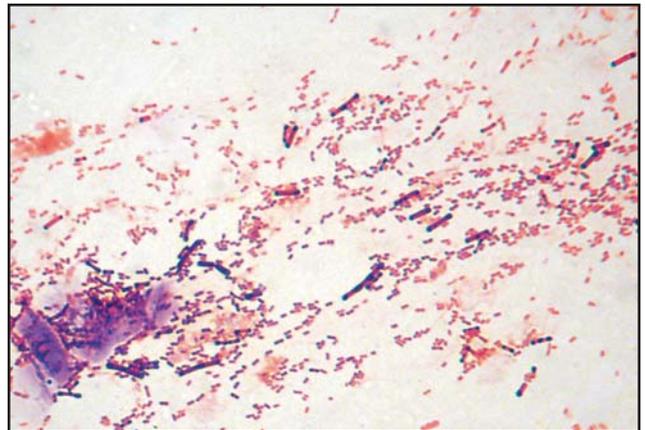


Fig 4.2.16 | Umbrella cockatoo, 6-year-old female: Hx = Exposure to carnivorous pets, seed only diet. CS = Fetid stool, weight loss, passing undigested food. FGS = 200 bacteria per field, 10% gram-positive rods of which 45% are *Clostridium* spp., 45% gram-negative rods. Rx = Aggressive antimicrobials, supportive care, dietary correction.

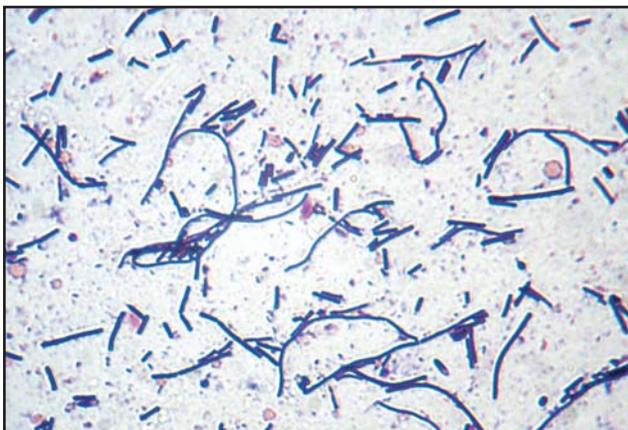


Fig 4.2.17 | Budgerigar, 4-year-old male: CS = Digestive upset. FGS = 200 bacteria per field, 5% gram-positive cocci, 95% gram-positive rods, of which half are large filamentous rods. Rx = Aggressive. See Chapter 30, Implications of *Macrorhabdus* in Clinical Disorders.

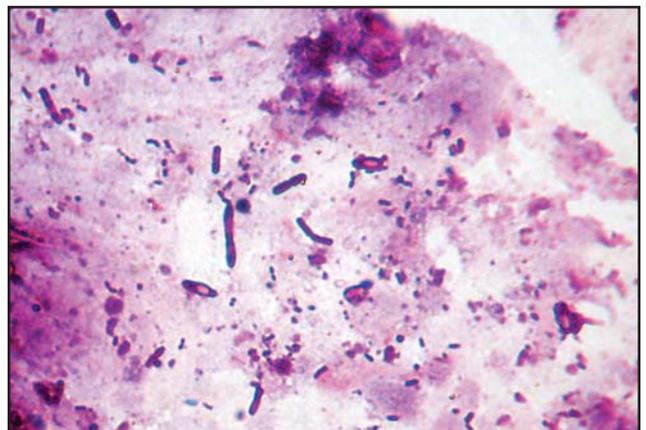


Fig 4.2.18 | Moluccan cockatoo, 7-year-old male: CS = smelly stool. FGS = 50 bacteria per field, 90% gram-positive rods, 10% gram-positive cocci, 30 *Clostridium* spp. organisms. Rx = Aggressive (see Fig 4.2.16).

Figs 4.2.19-4.2.22 | Fecal Gram's Stains Commonly Observed in Psittacines in Clinical Practice (oil immersion 1000X).

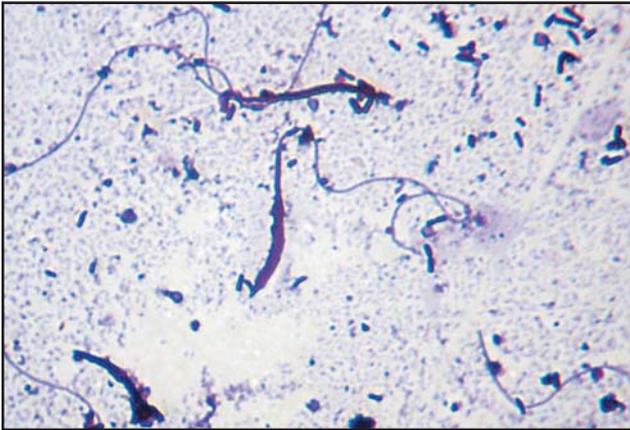


Fig 4.2.19 | Budgerigar, 3-year-old male: Hx = Frequent masturbation. FGS = Presence of sperm. Rx =None.



Fig 4.2.20 | Psittacine: Various forms of gastrointestinal diseases can be suspected if digestion of fiber or dietary ingredients is improper. Top slide = Normal fiber content of feces. Bottom slide = Undigested fiber.

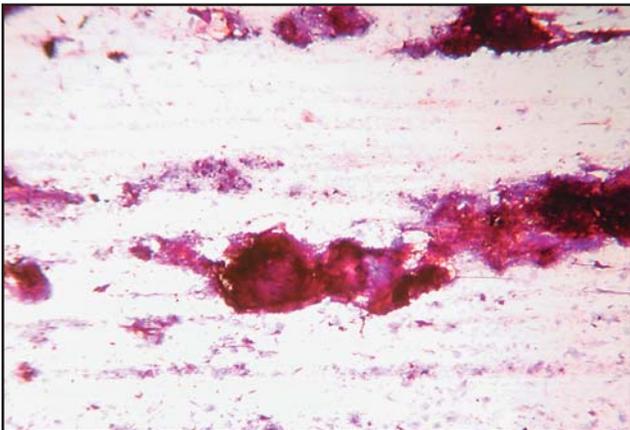


Fig 4.2.21 | Psittacine: FGS = Large amounts undigested fiber (low microscopic power).

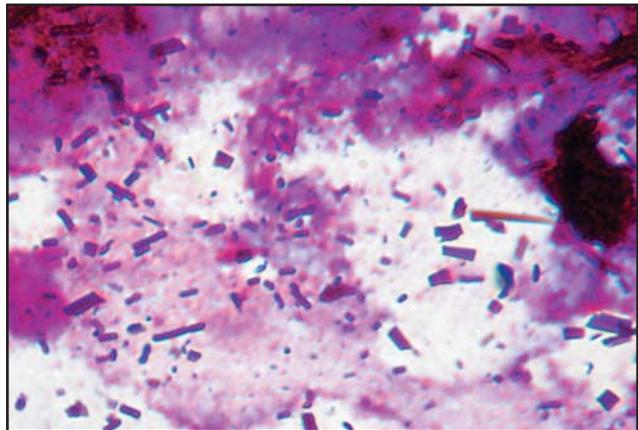


Fig 4.2.22 | Psittacine: FGS = 20 bacteria per field, 100% gram-positive rods, lots of undigested food particles cluttering field, suggesting some form of gastrointestinal disturbance.

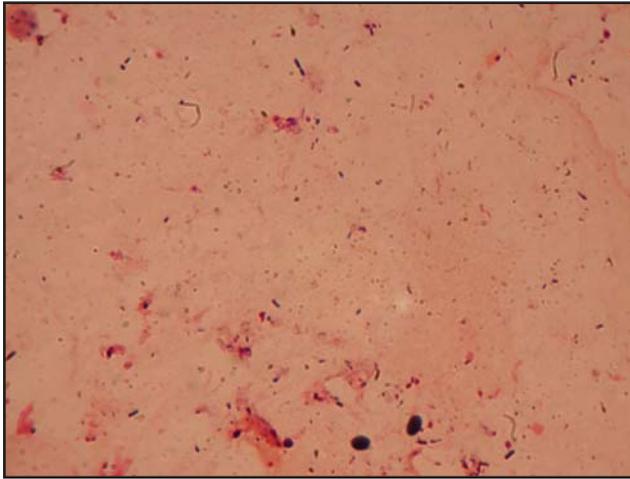
Figs 4.2.23-4.2.27 | Fecal Samples from Free-ranging Australian Psittacines (oil immersion 1000x).

Fig 4.2.23 | *Eolophus roseicapillus*: 30 bacteria/field, 50% small to medium gram-positive rods, 50% gram-positive cocci, no gram-negative rods, no yeast, slight debris, digested particles, two yeast-like forms.

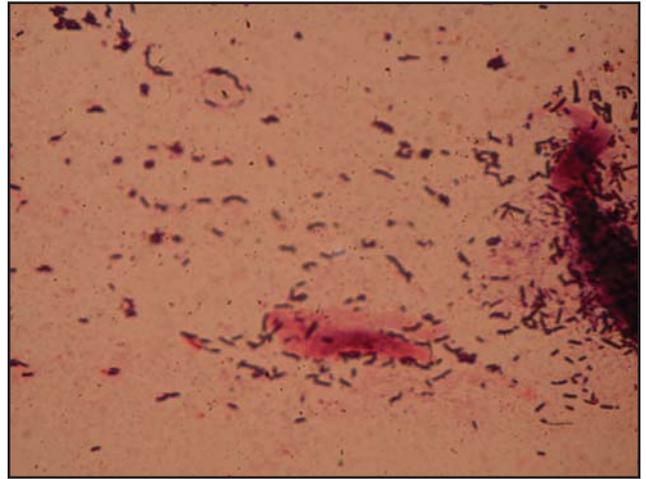


Fig 4.2.24 | *Eolophus roseicapillus*: 170 bacteria/field, 90% large gram-positive rods, 10% gram-positive cocci, no gram-negative rods, no yeast, moderate debris, digested particles.

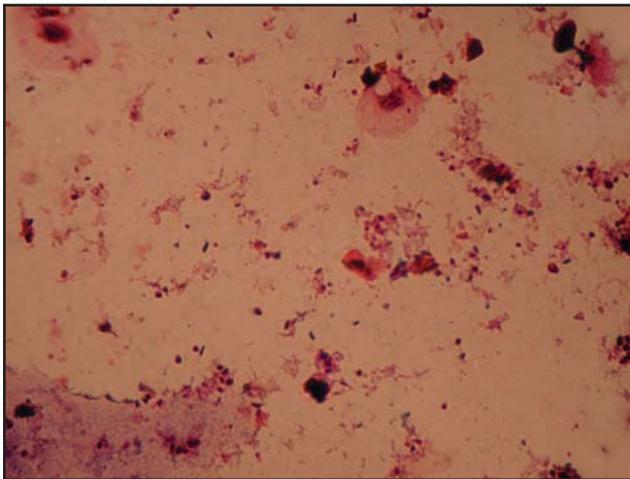


Fig 4.2.25 | *Cacatua tenuirostris*: 60 bacteria/field, 60% small to medium gram-positive rods, 40% gram-positive cocci, no gram-negative rods, no yeast, moderate amount debris, digested particles, two circular non-cornified cells with nucleus, one pollen-like form.

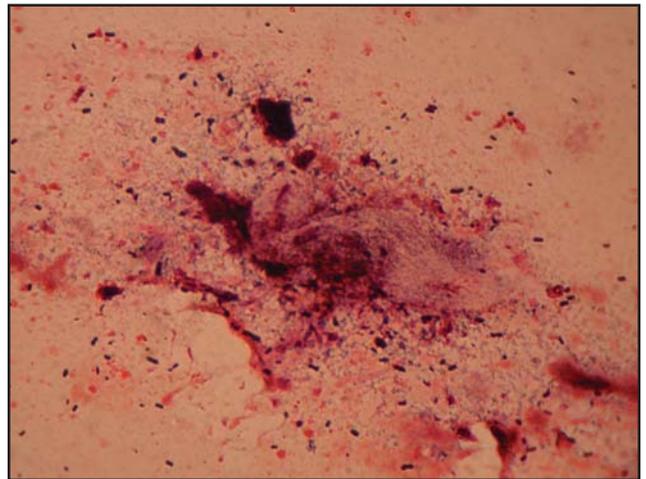


Fig 4.2.26 | *Cacatua tenuirostris*: 90 bacteria/field, 70% small to medium gram-positive rods, 30% gram-positive cocci, no gram-negative rods, no yeast, abundant debris (some not digested).

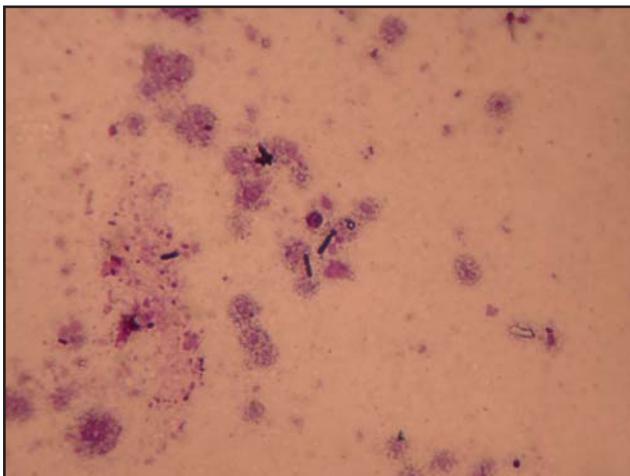


Fig 4.2.27 | *Cacatua tenuirostris*: 15 bacteria/field, 50% large gram-positive rods, 50% gram-positive cocci, no gram-negative rods, no yeast, moderate amount debris (cellular and digested particles).

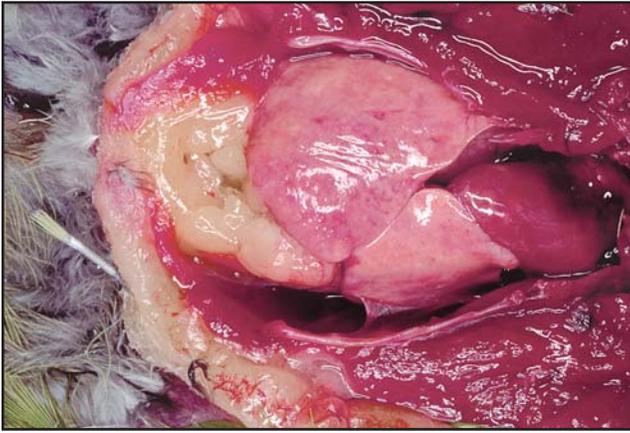


Fig 4.2.28a | Fatty liver in a Mexican red-headed Amazon. The bird died while being restrained for grooming. The serum was lipemic. The veterinarian never realized long nails and beak were a sign of a problem.



Fig 4.2.28b | The feces from an obese bird will on occasion have a red cream-colored urate after restraint. The renal vessel fragility is a strong prognostic indicator of fatty liver disease.

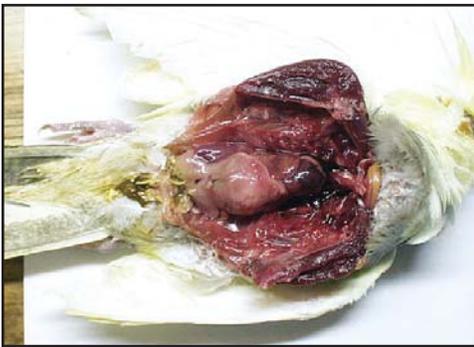


Fig 4.2.29 | Fatty liver in a baby cockatiel fed a commercial handrearing formula containing excessive fat.

birds. A fatty, swollen liver that compromises the abdominal and caudal thoracic air sacs can result in death from hypoxia (Figs 4.2.28a,b). Although there are some hereditary tendencies towards the disease, nutrition plays a major role in its development. There is little data on FLS in caged birds and a plethora of references on FLHS (Fig 4.2.29). Because FLHS is on the decline in poultry as a result of such data, we offer the following discussion for consideration.

Enzymatic Function and FLHS

A number of plasma enzymes increase with FLHS, such as AST, LDH and glutamate dehydrogenase (GDH). These can be used as indices of the syndrome in laying hens²⁷ (Table 4.2.8).

Signs and Symptoms

An overweight bird with a marked accumulation of fat is a likely candidate for hepatic lipidosis. Typically these birds may be considered behaviorally normal. Early signs include bile pigments in the urine, changes in the fecal Gram's stain and abnormal feather coloring. See Chapter 15, Evaluating and Treating the Liver for a further discussion.

Table 4.2.8 | Enzymatic Changes in Fatty Liver Affected Birds.^{10,11,105}

Enzyme	FLHS-affected birds
Acetylcholinesterase	↑ activity
Aspartate aminotransferase	↑ activity
Aspartate transaminase	↑ activity
Glucokinase	↓ activity
Gluconeogenesis	↓ activity
Lactate dehydrogenase	↑ activity
Phosphofructokinase	↓ activity
Sorbitol dehydrogenase	↓ activity

Nutritional Implications for Development of FLHS Dietary Fat

Liver fat and excess body weight (associated mainly with an accumulation of abdominal fat) are believed to be two predisposing factors contributing to the onset of FLHS in poultry.⁴² Unnecessarily force-feeding birds can increase liver fat and plasma estradiol, producing FLHS.^{11,42} A similar condition has also been observed in cockatoos and cockatiels fed improperly formulated diets (Fig 4.2.29). However, high liver lipid content alone may not be sufficient to cause FLS, as adequate dietary levels of lipid trigger a feed-back mechanism, enhanced by dietary starches, to prevent hepatic lipid accumulation.^{43a} Long chain fatty acids, especially those of the *n*-3 family are beneficial in the diet as a preventative measure.⁴⁴ Ground flaxseed (100 g/kg), flaxseed oil (40 g/kg) significantly decrease hepatic fat.⁹⁵ Safflower phospholipids decrease liver triglycerides (hepatic triglycerides increase with liver hemorrhage score⁸⁴), serum cholesterol and body weight.³ Palm kernel oil at 2% of dry matter weight of diet decreases FLS.⁷⁶ Palm oil is rich in vitamin E and carotenoids. The vitamin E fraction (400 mg/kg) is in an approximate ratio of 30:70 tocopherols:tocotrienols.

Tocotrienols have an unsaturated side chain rather than the saturated chain of the more common tocopherols. Tocotrienols more effectively lower cholesterol and show stronger antioxidant activity than tocopherols. See earlier discussion under Rancidity for oxidation's possible role in FLHS in poultry.

L-cysteine

Deficiencies in essential amino acids can increase mortality from FLHS and may be prevented with supplementation of L-cysteine at 6 g/kg of feed.²⁶ N-acetyl-L-cysteine (NAC) is the pre-crystallized form of the simple amino acid cysteine. It is a powerful antioxidant and immune support substance that neutralizes the free radicals produced by normal metabolic activity. While both cysteine and methionine are precursors of glutathione, NAC is more effective. During digestion, approximately 85% of the sulfur groups of L-cysteine are lost (these contribute to the active portions of glutathione), while only 15% are lost from NAC, resulting in up to six times more sulfur groups after digestion (for detoxification). NAC is also a better source of glutathione than supplementation with glutathione itself, because less than half the supplemental glutathione leaves the digestive system for other organs. This greater efficiency is important since cellular glutathione levels tend to drop 30 to 35% with age.

S-adenosylmethionine (SAME)

S-adenosylmethionine (SAME), a natural metabolite of the amino acid methionine, was discovered as a pharmaceutical in Italy in the 1970s and has been available in Europe for over 20 years. It is the most active of all methyl donors arising from the amino acid methionine. While healthy livers synthesize sufficient methionine, liver disease can impair SAME syntheses. On a cellular level SAME maintains mitochondrial function, prevents DNA mutations and, restores cellular membrane fluidity so that cell receptors become better able to bind hormones and other factors.

SAME's methyl groups make possible the production of the "fat burner" carnitine; the neuro nutrient acetyl L-carnitine; the primary ATP energy reservoir, creatine phosphate; the stress hormone and neurotransmitter, adrenaline; the neuro nutrient and chief membrane phospholipid, phosphatidyl choline; and the DNA bases methyl adenine and methylcytosine.

In addition to transmethylation, SAME is involved in transsulfuration, which begins with the by-products of the transmethylation of S-adenosylhomocysteine (SAH). SAH yields homocysteine, which can be converted to cysteine and then to a family of key sulphur biochemicals: glutathione, glutathione peroxidase, glutathione-S-transferase and taurine. As much as 80% of dietary cysteine, low in many foods, can lose its bioactive

sulfhydryl groups passing through the stomach. The glutathione compounds and taurine play important roles in liver detoxification.

SAME production decreases with age. Dietary supplementation may be required for older birds prone to fatty liver disease. Without SAME, the liver protective glutathione cannot be synthesized. While increasing glutathione levels through supplementation is desirable, glutathione alone is not a substitute for the combined actions of SAME and glutathione.

Betaine and SAME

Anhydrous betaine (trimethyl glycine, not to be confused with the digestive aid betaine hydrochloride), is a substance made from beet sugar that increases SAME levels. Impairment of SAME synthetase may result in SAME being manufactured through the betaine pathway, an alternative to the SAME synthetase-dependent methionine-plus-ATP route. Increasing levels of betaine reduce fatty infiltration and provide the precursors for the free radical scavenger glutathione.

It is recommended that SAME supplementation in humans for a diet comprising 16% protein range between 100 to 500 mg, with higher requirements in females. Mild stomach irritation may result if not using a product with an enteric coating. There have been no clinical trials on the effectiveness of SAME for birds with liver disease. Early empirical data is encouraging.

Vitamins and SAME

Once a SAME molecule loses its methyl group it breaks down to form homocysteine. On its own homocysteine can be extremely toxic, but the presence of vitamins B₆, B₁₂ and folic acid convert homocysteine into glutathione or re-methylate it into methionine. Deficiencies of any of the active coenzyme forms of vitamins B₂, B₆, B₁₂ or folic acid will disrupt SAME production. Reciprocally, diminished SAME production will impair conversion of folic acid and B₁₂ to their coenzyme forms.

In order to maximize the effectiveness of the interlocking SAME pathways, the addition of the water-soluble vitamins B₂ (20 to 200 mg), B₆ (40 to 400 mg), B₁₂ (0.5 to 5.0 mg) and folic acid (0.8 to 2.0 mg) is required. Vitamins B₆, B₁₂ and folic acid also convert the toxic homocysteine to glutathione or re-methylate it into methionine.

Biotin

While low dietary protein predisposes chicks to develop FLHS, high dietary protein can cause classical signs of biotin deficiency.¹¹ Biotin is an essential coenzyme involved in the conversion of protein to carbohydrate and the conversion of protein and carbohydrate to fat.

Biotin enzymes are important in protein synthesis, amino acid deamination, purine synthesis, and nucleic acid metabolism. Biotin itself is required for trans-carboxylation in the degradation of various amino acids; it also plays an important role in maintaining normal blood glucose levels when dietary intake of carbohydrate is low. Biotin deficiency is most severe in young chicks of heavier strain and greater rate of weight gain;⁸ promoting higher growth rates in psittacines may predispose birds to FLS.

Many of the problems associated with biotin deficiencies and FLHS result from biotin's role as a cofactor for many enzymes. These include: a decreased rate of lipogenesis; depressed gluconeogenesis from lactate and glycerol; an increase in the activities of fatty acid synthase (FAS), citrate cleavage enzyme (CCE) and phosphokinase¹¹; abnormal fatty acid composition of infiltrated lipid, with an increased proportion of monounsaturated fatty acids; severe hypoglycemia; and depleted hepatic glycogen.¹⁰⁵ Low fat or protein levels that increase the metabolic rate of biotin-dependent enzymes (pyruvate, acetyl CoA carboxylase) aggravate the condition. Biotin also serves as part of the prosthetic group, a transient carrier of CO₂, and is required for normal long-chain unsaturated fatty acid synthesis and is important for essential fatty acid metabolism.⁵⁹ FLHS is generally worsened by a high proportion of long chain saturated fatty acids.⁴⁸

Biotin deficiencies can result from a dietary deficiency of biotin or other factors that impact on the stability of biotin. The richest sources of biotin include: royal jelly, liver, kidney, yeast, blackstrap molasses, peanuts and eggs, while poor sources include: corn, wheat, other cereals, meat and fish. However, the chemical form of biotin (bound or unbound) as well as its overall content in feed is important, as less than one-half of the biotin in various feeds is biologically available. Starvation of birds can lower liver biotin levels, leading to an increase in liver weight and lipid content.¹⁰ The addition of raw egg white also decreases biotin availability as the proteinaceous avidin binds very tightly to biotin. While low mortality is seen in broilers fed freeze-dried egg white at 11.8 g/kg, mortality is high if dietary concentrations exceed 17.7 g/kg.

Not all forms of biotin are equivalent in their action; biotin contains three asymmetric carbonations, with eight different isomers. Only the isomer *d-biotin* contains vitamin activity; the stereoisomer *l-biotin* is inactive.⁵⁹ Biotin is inactivated by rancid fats and choline⁹⁷ and gradually destroyed by ultraviolet radiation. Structurally related analogues of biotin can vary in activity from anti-biotin activity, to no activity, to partial replacement.⁷ Oxybiotin has 1/3 biotin activity for chicks

whereas desthiobiotin and biotin sulfate are inhibitory to bacteria. Biotinidase, present in pancreatic juice and intestinal mucosa, releases biotin from biocytin during the luminal phase of proteolysis. Physiological concentrations of biotin are absorbed from the intestinal tract by a sodium-dependent active transport process, which is inhibited by desthiobiotin and biocytin.⁹²

Cecal microorganisms do not supply chickens with significant amounts of biotin; they compete with the host animal for dietary biotin, thereby increasing the requirement. In poultry, polyunsaturated fatty acids, ascorbic acid, and B vitamins may influence the demand for biotin. Biotin is rapidly destroyed as feeds become rancid, with 96% inactivation occurring in as little as 12 hours if linoleic acid of a high peroxide number is added to the diet. Supplementary choline in biotin-deficient diets decreases biotin status in chicks and increases mortality from FLHS.¹⁰⁵ The use of sulfa drugs can also induce a deficiency. Conversely, α -tocopherol decreases inactivation of biotin.

Minimum biotin requirements have been established for a number of commercial species, with higher requirements for turkeys compared to chickens (NRC, 1994). The minimum dietary requirements of 120 $\mu\text{g}/\text{kg}$ dietary dry matter determined for poultry increases to 160 $\mu\text{g}/\text{kg}$ in order to prevent fatty liver development and as high as 240 $\mu\text{g}/\text{kg}$ when sunflower seed meal is a dietary component.⁷⁵ Incorporation of 2% palm kernel oil can reduce the prophylactic dietary biotin requirements down to 120 $\mu\text{g}/\text{kg}$.⁷⁶

Choline

Choline plays an essential role in fat metabolism in the liver. Choline prevents abnormal accumulation of fat by promoting fat's transport as lecithin or by increasing the utilization of fatty acids in the liver itself. While conversion to betaine is required before choline can be a methyl donor, betaine itself fails to prevent FLHS. The addition of choline can decrease the amount of fat in the liver. Diets high in fat exacerbate choline deficiencies, thus increasing the dietary requirement. This is particularly important for chicks, as they are unable to synthesize choline until approximately 13 weeks of age.⁶⁸ However, mortality increases in chickens that are supplemented with B vitamins (other than biotin), with higher mortality if choline is also supplemented.¹⁰⁵ Only 57% of biotin in multivitamin premixes⁶⁸ is retained if the supplement contains choline.

Normal dietary choline requirements for poultry range from 800 to 2,000 mg/kg. Choline is largely absent in fruit and vegetables and is low in corn. Wheat, barley and oats have higher levels of choline. Peanuts are a good source of choline as are cereal germs, legumes and

Table 4.2.9 | IDC and Iron Storage Disease

Nutritional Imbalance → High iron content diet, presence of vitamin C and excess vitamin A	
↓	
CLINICAL SIGNS	
Insectivore/Frugivores	Parrots
Early <ul style="list-style-type: none"> Listless Respiratory wheeze or click Decreased exercise tolerance Sudden death Chronic <ul style="list-style-type: none"> Swollen coelomic cavity Dyspnea with forward leaning posture 	Early <ul style="list-style-type: none"> None - most common Found on necropsy Intestinal cellblock is saturated Chronic <ul style="list-style-type: none"> Sudden death
Diagnostics <ul style="list-style-type: none"> Radiology <ul style="list-style-type: none"> Pericardial effusion Hepatomegaly Ultrasound Endoscopy and biopsy (caution due to potential coagulation disorders) 	Diagnostics <ul style="list-style-type: none"> Liver biopsy
Treatment <ul style="list-style-type: none"> Low iron diet <80 mg/kg Avoid vitamin C and excess vitamin A Iron chelator Black tea Phlebotomy 	Treatment <ul style="list-style-type: none"> Low iron diet <80 mg/kg Avoid vitamin C and excess vitamin A

Table 4.2.10 | Avian Families Diagnosed with ISD^{20,25,35,36,57,70a,91,107*}

Order	Family	Common Name
Passeriformes	Sturnidae Paradisaeidae	Mynahs/starlings, tanagers Birds of Paradise
Galliformes	Cracidae	Guans, currasows
Ciconiformes	Ardeidae	Bitterns
Trogoniformes	Trogonidae	Quetzals
Piciformes	Rhamphastidae	Toucans, toucanettes, aracari
Ciconiformes	Phoenicopteridae	Roseate flamingos
Psittaciformes	Loridae Psittacidae	Lories Lorikeets Parrot/cockatoos

*See Chapter 4, Nutritional Considerations: Section I, Nutrition and Dietary Supplementation for further discussion

oilseed meals. Choline needs of poultry fed wheat-based diets are much lower than those fed on other grains. Wheat and sugar beets are high in betaine, which can spare choline for some reactions.

Vitamin E

Vitamin E powerfully combats the peroxidation of polyunsaturated fatty acids in the liver. Daily supplementation of 100 to 400 IU Vitamin E in conjunction with low dietary vitamin A is recommended for birds suffering from FLHS. See earlier discussion of rancidity for possible role of fat-soluble vitamin destruction and FLHS.

Silymarin (Milk Thistle)

Silymarin is a collective group of polyphenolic flavanolignans extracted from the seeds of the milk thistle (*Silybum marianum*). The flavanoids are powerful

antioxidants that increase levels of glutathione and protect the liver from oxidative damage. They may promote growth of new, healthy liver cells.^{32,85} While clinical trials in birds have not been undertaken to evaluate the effectiveness of silymarin, it has proven effective empirically when administered twice daily to birds with liver disorders. See Chapter 10, Integrative Therapies, Chapter 9, Therapeutic Agents and Chapter 15, Evaluating and Treating the Liver for further information.

Environmental Influences

While nutritional imbalances are the main factors contributing to both FLS and FLHS, stress alone can initiate FLHS. When birds are subjected to mild stress and/or short-term fasting, liver glycogen reserves become rapidly depleted and a progressive hypoglycemia develops that can prove fatal. Stress-associated lipogenesis increases cholesterol synthesis and converts excess glucose to fatty acids, which are stored as triglycerides.

Pesticides

While pesticide levels of individual ingredients may be deemed safe, a combination of a variety of pesticides or an accumulation of pesticides in tissues can result in pesticide toxicity. Polychlorinated biphenyls (PCBs) increase liver and body weights of birds associated with FLHS and can increase total cholesterol¹⁰³. Many pesticides have estrogenic actions; high estrogen levels are associated with FLHS.⁴² These estrogenic pesticides mimic the action of normal endogenous hormones and influence ovarian function. A combination of estrogen

and excess dietary energy create sufficient fat deposition in the liver for FLHS to occur.⁸⁹

Summary of Fatty Liver Disease

A variety of nutritional factors are implicated in the development of FLS and FLHS in chickens, but many are avoidable by providing a nutritionally balanced diet. Some factors are exacerbated by other dietary ingredients, environmental stimuli or infectious diseases.¹⁹ While the name of the disease, “fatty liver (hemorrhagic) syndrome,” implicates dietary fat levels as causative factors, there are many other dietary components that are important and warrant further consideration. We feel this model may serve the captive parrot industry well, as empirically the same corrections seem to apply.

IRON STORAGE DISEASE

Iron storage disease (ISD) is prevalent in many frugivorous and insectivorous birds maintained on commercially formulated foods (Table 4.2.9). Iron storage disease differs from one of its precursors⁵³, hemosiderosis, which is defined as the excessive accumulation of iron (hemosiderin) in hepatocytes or in free circulation in the blood, without alteration of normal tissue morphology or damage to any of the major organs. Various factors have been implicated in the development of this disease, including genetic predisposition, immunological stress, nutritional inadequacy and viruses; dietary iron content has been the main focus of nutritional investigations. The causative factors of ISD have been addressed by several authors.^{29,36,70a} Further studies on the interactions of dietary sugars, copper and iron metabolism have been proposed.⁸⁷ The discussion here will be confined to nutritional implications in the development of ISD. There is a high correlation with commercially formulated foods and ISD. Table 4.2.10 highlights a number of avian families in which the disease has been reported.

While iron is essential for fundamental cell functions, it is also a catalyst for chemical reactions involving free radical formation that can lead to oxidative stress and cell damage. Uptake of iron from the diet is regulated in the intestine, so acute intoxication is not observed under natural conditions. Cellular iron levels are generally regulated to maintain adequate substrate levels while minimizing the pool of potentially toxic ‘free iron.’ The main control of body iron homeostasis is in the duodenum where dietary iron is absorbed, but no controlled means of eliminating unwanted iron has evolved in animals. Consequently, chronic ingestion of large amounts of absorbable iron can lead to the storage of iron in the liver in many species.

Iron storage disease results from the accumulation of

iron in various tissues, with the liver most frequently involved. In severe cases, iron pigment is found in the liver, spleen, gut wall, kidney and heart; this leads to subsequent development of ascites, heart failure and multisystem pathology.²⁵ Iron may be found within the Kupffer cells in the liver³⁶ and the macrophage cells of the spleen, especially where concurrent diseases, such as hemolytic anemia, septicemia, neoplasia or starvation, are present.²⁵

The syndrome of excessive iron overload in mynahs shares most of the important histopathologic characteristics with idiopathic hemochromatosis in human beings. Iron storage disease has been correlated with immunological stress,^{25,36} as well as crowded conditions.⁵³ Reduced peristalsis or neuropathic gastric dilatation may increase iron absorption.³⁶ Stress increases lipid peroxidation and diminishes vitamin E levels, resulting in a lower level of antioxidant activity. Iron and vitamin E are involved in electron transfer in reduction/oxidation cycles; a dietary surplus of either iron or vitamin A decreases the α -tocopherol concentration. Therefore, any impact on vitamin E levels may reduce the protection of biological membranes against oxidation. In addition, diets high in saturated fats increase iron absorption.⁷⁹

Nutritional Implications for ISD

Highly frugivorous or highly insectivorous birds have adapted to foods low in iron (fruits and insects). The high vitamin C content of many fruits enhances iron uptake from iron deficient diets. Consequently, high dietary iron has been implicated in the development of the disease and it is generally recommended that iron content of commercial diets be maintained below 100 mg/kg⁵³, and in mynahs 19 to 25 mg/kg.^{29,94} However, birds have been maintained on commercial foods that reflect the high values of iron in some dietary components of wild toucans⁷⁸ (150 mg/kg) with no evidence of iron storage disease. Diet is not implicated in the development of ISD in the Rothschild mynah.

Vitamin A and Iron Uptake

Some commercially formulated products have high vitamin A content (see Chapter 4, Nutritional Considerations, Section I Nutrition and Dietary Supplementation). These high vitamin A levels are in contrast to the low vitamin A content of fruits and insects. Vitamin A from plants arises from conversion of carotenoids, a regulated process that avoids potential vitamin A toxicity. Productivity of psittacines increases when birds are transferred to formulated diets low in vitamin A but high in carotenoids.⁶⁷ Additional vitamin A is either supplied from plant based carotenoids in the diet or else it may

Table 4.2.11 | IDC and the Respiratory System

Nutritional Imbalance → Squamous Metaplasia of Respiratory Epithelium			
↓			
CLINICAL SIGNS			
Oral/Rhinal	Sinus	Trachea/Lungs	Air Sacs
<ul style="list-style-type: none"> • Blunting or loss of choanal papillae • Increased mucus viscosity • Serous rhinal discharge • Sneezing, scratching nares • Chronic - Rhinolith formation: Can be sterile, bacterial, or fungal. Dark discharge = hemocult+ 	<ul style="list-style-type: none"> • Infraorbital swelling • Respiratory wheeze or click • Lacrimal duct infection or impaction • Chronic - Secondary sinusitis: May be bacterial or fungal 	<ul style="list-style-type: none"> • Voice change or loss • Decreased exercise tolerance • Dyspnea with forward leaning posture • Chronic - Tracheal obstruction: Often <i>Aspergillus</i> granuloma 	<ul style="list-style-type: none"> • Increased panting • Decreased exercise tolerance • Mild, intermittent tail bob • Chronic - Secondary air sacculitis: <i>Aspergillus</i> is commonly isolated
TREATMENT			
Diagnostics	Medical Treatment	Environmental Concerns	
<ul style="list-style-type: none"> • Endoscopy (tracheal, coelomic for air sacs) • Cytology of respiratory exudate • Culture and sensitivity of exudate • Radiology • Hematology • Biochemistry • Serology (<i>aspergillosis</i>) 	<ul style="list-style-type: none"> • Debride rhinoliths • Nasal/sinus flushes as needed • Infraorbital sinus drainage as needed • Nebulization, this can be utilized for humidity, mucolytic agents, or antimicrobial therapy delivery • Systemic treatment of secondary infections - <i>Aspergillus</i> • Diet correction - (see Table 4.2.3: see diet conversion challenges at end of chapter) 	<ul style="list-style-type: none"> • Proper humidity • Adequate ventilation of enclosure • Filtration of ambient air (medical grade filters) • Avoidance of exacerbating aerosols: <ul style="list-style-type: none"> - cigarette smoke - perfumes - cockatoo dust - pollens 	

Recurrent or chronic respiratory infections are common with chronic IDC and marked changes in the respiratory epithelium.

be inferred that birds have an overall low requirement for vitamin A. The incidence of ISD is negligible in these birds.

Low serum retinol is associated with mild anemia in adult humans.³⁰ Retinol also plays a role in increasing levels of hemoglobin in children, especially those on iron supplementation.^{2,65} Dietary iron also influences conversion of β -carotene to retinol by enhancing β -carotene 15,15'-dioxygenase activity in the small intestinal mucosa of rats.³¹ High levels of dietary vitamin A may negatively influence availability of other fat-soluble vitamins such as vitamin E.

In contrast to vitamin A, the presence of carotenoids in microsomal membranes partially inhibits the loss of α -tocopherol, especially during the late phase of oxidative stress when β -carotene decreases phospholipid hydroperoxide production.⁷³ However, despite its beneficial antioxidant activity, β -carotene, like vitamin A, can increase the absorption of iron by preventing the inhibitory effect of phytate and tannins on iron absorption by forming complexes with iron that maintain solubility in the intestinal lumen.⁶²

The antioxidant activity of the different carotenoids is variable. The inhibitory effect of β -carotene on the production of lipid peroxidases is less than that of the extremely potent antioxidant astaxanthin from marine micro algae. Astaxanthin protects the mitochondria from

damage by Fe^{++} -catalyzed lipid peroxidation during vitamin E deficiencies.⁶² It is two-fold more effective than β -carotene in inhibiting production of lipid peroxidases.³⁹ Peridinin, another carotenoid of marine micro algae, limits oxidative damage on iron-liposomes, possibly by decreasing membrane permeability to initiators.¹² While the direct benefits of the blue-green algae *Spirulina platensis* have not been evaluated in birds, the phycobilins (phycocyanins and allophycocyanin) of *S. platensis* act as potent free-radical scavengers (hydroxyl and peroxy) and inhibit microsomal lipid peroxidation in humans.⁸⁸ Diets^b, low in vitamin A and containing micro algae, have demonstrated improvements in health and productivity of large psittacines.⁶⁷

Canthaxanthin is a carotenoid compound that is supplemented to promote feather pigmentation in flamingos and scarlet ibis. A recent study suggests that canthaxanthin can substantially alter the antioxidant status of murine liver tissue in vivo. In mice, canthaxanthin reduces both cellular content of lipophilic antioxidants and the activity of enzymatic antioxidants, as well as increasing iron concentrations in the liver by up to 27%. ISD has been diagnosed in flamingos.²⁰ The addition of canthaxanthin to the diets of these birds may alter the protective ability of tissues against oxidative stress in vivo and increase iron storage in the liver. The carotenoid canthaxanthin is associated with eye and liver damage in humans.



Fig 4.2.30 | An old female budgerigar with IDC, chronic rhinorrhea and secondary rhinal infection with yeast and/or bacteria.

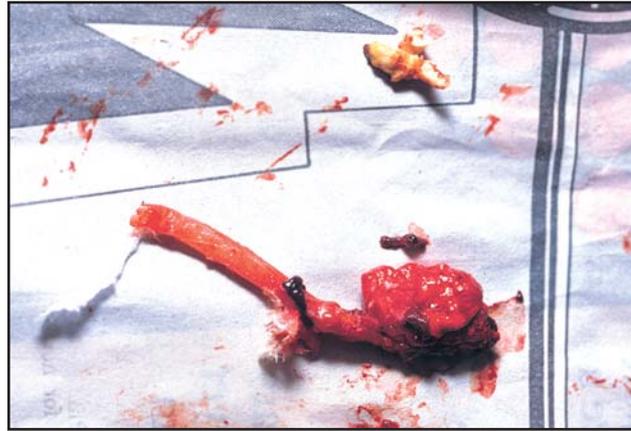


Fig 4.2.31 | Liths in the bronchial syrinx area or the air ostium to the lungs often become secondarily infected with yeast or fungal spores. This lesion was taken from a free-ranging Major Mitchell's cockatoo.

Table 4.2.12 | IDC in Geriatric Budgerigar with Goiter

History	Clinical Signs	Treatment
<ul style="list-style-type: none"> • Diet Seed based 	<ul style="list-style-type: none"> • Regurgitation • Crop mucus accumulation • Dyspnea • Swollen thyroid 	<ul style="list-style-type: none"> • Dexamethasone (if severe) • Iodine • Diet change

Summary of ISD

While diets low in iron are advocated for birds susceptible to ISD, the high vitamin A content of commercially formulated foods may also be implicated in the development of the disease. It is recommended that diets low in iron, vitamin A and saturated fats be presented to birds susceptible to this disease in addition to supplementation with vitamin E. Furthermore, high levels of dietary β -carotene may be detrimental. Vitamin A activity should be provided from other carotenoid sources, especially those present in blue-green algae, such as *Spirulina platensis*. Frugivorous species should be provided with fruits low in vitamin C to minimize uptake of iron from commercial diets. Recent recommendations on the use of tannins from tea to tie up iron have shown promise. It is evident that further research on this topic is required.

RESPIRATORY SYSTEM

Loss of function of epithelial tissue is a problem in the respiratory system because loss of cilia and mucus production decreases cleaning capacity (Table 4.2.11). In chronic cases, serum oozes from endothelial ulcerations and provides a culture media for bacteria, yeast or other fungi (Figs 4.2.30-4.2.31). The nares can reflect the internal condition.

Liths that roll up to form “balls” can develop from desquamated epithelial cells when proper cleaning function has not been maintained. These balls may randomly accumulate in such places as the connection between

the cul-de-sacs of the infraorbital sinus or the layering of the rhinal cavity under the operculum. In the case of the sinus, they may act as one-way valves causing the cervicocephalic air sac to hyperinflate. In the rhinal cavity, the accumulation leads to rhinitis or rhinorrhea (often containing blood). The liths in the bronchial syrinx area or the air sac junction to the lungs can become secondarily infected with yeast or other fungal spores (Fig 4.2.31). Recent reports from Europe indicate this is a common respiratory sign seen in African greys with circovirus.

Liths are not confined to the respiratory tract. They can accumulate in the bile duct. When flecks of liths partially obstruct the duct in cockatiels, the duct balloons to resemble a gall bladder. Liths in the kidney are often associated with gout. In the uterus, they can form egg-like structures of various sizes and shapes.

BUDGERIGAR GOITER

It has been common knowledge since the 1950s, that budgerigars (*Melopsittacus undulatus*) develop goiter on a seed diet low in iodine. Table 4.2.12 shows the typical scenario. The bird may only show obesity. Most common clinical presentations are “vomiting” (regurgitation) of a thick liquid (mucus) that accumulates on the feathers of the head. Respiratory sounds (squeaking) from impingement on the syrinx by the swollen thyroid are also a frequent complaint. While diagnostic tests are possible, they are seldom used due to their expense and the ease of diagnosis using response to treatment. Dexamethasone may speed the response to injectable

Table 4.2.13 | Influence of Prostaglandins on Renal Function

	PGE ₂	Prostacyclin	TBXA ₂
Vasodilation	↑	↑	
Vasoconstriction			↓
Renal blood flow	↑	↑	↓
GFR	↑	↑	↓
Platelet function		↑	↓

iodine by decreasing thyroid swelling and may be life saving in severely dyspneic birds. Complete recovery is insured by adding iodine to the water or seeds. Since other deficiencies will soon manifest on such a diet, only geriatric birds and birds that refuse to convert to formulated diets are treated by iodine supplementation alone.

INGLUVITIS

Baby psittacines from parents on seed-based diets commonly are plagued by ingluvitis. Seed-based malnutrition is the primary cause and can be prevented with formulated diets. Secondary invaders (yeast and Enterobacteriaceae) may require specific antimicrobials and resemble liver and gastrointestinal disorders caused by the same agents. In these latter conditions, crop atony followed by ileus is not unusual, especially in cockatiels. These birds are very difficult to cure and frequently suffer a prolonged wasting type malaise. See Chapter 7, Emergency and Critical Care and Chapter 14, Evaluating and Treating the Gastrointestinal System, for further discussion.

RENAL DISEASE

Excesses of dietary protein and the accumulation of waste products derived from the catabolism of protein result in uric acidemia and to a lesser degree, uremia, in birds. Excessive dietary protein is catabolized to uric acid and other nitrogenous compounds normally excreted by the kidneys. Decreased renal function leads to accumulation of these compounds. One goal of nutritional therapy is to achieve nitrogen balance by proportionally decreasing protein intake as renal function declines, except in cases of protein losing nephropathy.

Gout is associated with the deposition of a white chalky substance (urate) on body organs (visceral), in joints (articular) or in ureters (renal). Urates are the end products of protein metabolism in birds. Their accumulation impairs the function of key organs and can eventually be fatal. While excess dietary protein has been implicated in the increase of serum uric acid, birds are usually able to excrete excesses.⁵⁸ However, fasting, dehydration or a diet deficient in lysine may increase serum uric acid. Alterations to normal elimination of uric acid, which can

cause extensive renal damage, have also been associated with vitamin A deficiency leading to gout.⁵⁸

Dietary lipids have been implicated in the progression of chronic renal disease, especially in relation to a change in the balance of renal prostaglandins. A diet rich in arachidonic acid leads to a predominance of PGE₂, prostacyclin and thromboxane A₂ (TBXA₂), resulting in a shift toward greater vasodilatation and less platelet aggregation (Table 4.2.13). For further discussion on this topic, see Chapter 16, Evaluating and Treating the Kidneys. The effects of excess vitamin A and/or D are discussed in Section I of this chapter.

CARDIOVASCULAR DISEASE

Atherosclerosis

Atherosclerosis is problematic for psittacines in captivity⁵² that are provided with high-fat diets and little exercise compared to their free-ranging counterparts. Atherosclerosis involves the deposition of cholesterol within the innermost lining of the arteries and subsequent inflammatory response and fibrosis. While cholesterol-lowering agents and dietary changes are treatment mainstays in humans, the development of the disease may be prevented in birds with nutritionally balanced diets containing antioxidants.

Damage to the endothelial lining of the internal surfaces of the heart increases permeability to lipoproteins and macrophages. Increased endothelial permeability leads to the accumulation of lipoproteins within the subendothelial space, which initiates the formation of atherosclerotic plaques. The oxidation of lipoproteins retained within the subendothelial space is responsible for the inflammatory response seen in atherosclerosis.

Lipoprotein oxidation is a necessary step in the development of atherosclerotic plaques; the oxidation of low-density lipoprotein (LDL) is implicated in lesion formation in the aorta. Vitamin A can attenuate the oxidation of LDL and consequently minimize or even reverse aortic plaque development and endothelial dysfunction.

Oxidants are produced by the normal metabolic functions of the endothelium, macrophages, and smooth muscle of the arterial wall. Lipoproteins that accumulate within the subendothelial space are exposed to these cellular oxidants. Although a small degree of lipoprotein oxidation may occur during circulation, most lipoprotein oxidation occurs within the arterial wall. It is therefore noteworthy that lipoproteins are themselves enriched with antioxidants such as vitamin E, coenzyme Q₁₀ (ubiquinone) and carotenoids. Water-soluble antioxidants found in the plasma, such as vitamin C, may also be important in preventing oxidation of lipoproteins in the circulation.

In a recent abbreviated study, two high fat and two low fat diets were fed for periods of 24, 28 and 32 days. Results showed that palm kernel oil (derived from the seed) produced significantly higher levels of plasma cholesterol and phospholipid concentrations than did sunflower oil.¹³ No conclusions on the effect on atherosclerosis could be drawn. The authors had previously shown 84% sudanophilic staining levels in aortas of parrots presented for necropsy.^{14a} The authors recommended diets of up to 10% fat on a dry matter basis.

Nutritional Supplementation for the Treatment of Atherosclerosis

In studies on humans, palm oil (derived from the fruity coating outside the seed), which is distinct from palm kernel oil, has proven beneficial in the treatment of atherogenesis. Palm oil is very rich in carotenes, vitamin E and coenzyme Q₁₀. The cholesterol lowering action of palm oil is attributed to its high vitamin E content, especially the tocotrienol fraction. A typical palm oil vitamin E concentrate contains up to 30% α -tocopherol and 70% mixture of different tocotrienol isomers (20% α -tocotrienol, 20% γ -tocotrienol, 40% α -tocotrienol, 10% δ -tocotrienol and 10% others).⁴⁹

Cardiomyopathy

Cardiomyopathy has been associated with a number of disease entities including malnutrition, as the pathogenesis in birds is similar to mammals, it is possible that similar nutritional inadequacies are implicated. While supplementation levels have not been established for birds, studies of mammals indicate that supplementation with taurine, vitamin B₆, coenzyme Q₁₀ and carnitine are helpful.

Taurine

Taurine is an essential amino acid in cats as they have a limited ability to synthesize taurine from cysteine and methionine. Because there are many physiological similarities between carnivorous birds and felines, it is possible that taurine deficiency is implicated in the development of cardiomyopathy in carnivorous birds, especially those provided with commercial dog foods that are typically low in taurine. Plasma taurine concentrations can be influenced by food intake and food deprivation. Whole blood concentration is a more reliable index of taurine status, as this only declines after prolonged periods of depletion. While taurine levels for cats (plasma <20 to 30 nmol/ml; whole blood <150 nmol/ml) are indicative of dietary deficiencies, similar data is not available for carnivorous birds.

Vitamin B₆

Mild deficiencies in vitamin B₆ can interfere with taurine conversion. The heat treatment associated with the pro-

duction of commercial pet foods degrades B₆.

Coenzyme Q₁₀ (CoQ₁₀)

Coenzyme Q₁₀ is an antioxidant that plays a role in mitochondrial function. A deficiency of CoQ₁₀ is correlated with deterioration in heart function. CoQ₁₀ is similar in structure to vitamin K and can interfere with the blood-clotting mechanism. Studies with humans indicate that supplementation at 2 mg/kg body weight reduces symptoms associated with cardiomyopathy and heart failure. β -blockers and cholesterol-lowering drugs from the statin family can interfere with the body's production of CoQ₁₀.

Carnitine

Carnitine is a small, water-soluble, vitamin-like quaternary amine found in high concentrations in mammalian heart and skeletal myocytes. L-carnitine is synthesized primarily in the liver from the amino acids lysine and methionine. Long-chain fatty acids are important for maintaining a constant energy supply to the heart. Carnitine is a critical component of the mitochondrial membrane enzymes that transport activated fatty acids. In addition to its role in fatty acid transport, free carnitine serves as a mitochondrial-detoxifying agent. Because of the high-energy requirements of the heart muscle, it is particularly vulnerable to carnitine deficiencies. A recent report shows the potential for L-carnitine to reduce the percent body weight and lipoma size in budgerigars.^{14c}

OPHTHALMIC DISORDERS

Cataracts

While causative factors have not been identified clearly, cataracts have been described in aging macaws.²⁴ Nuclear cataracts associated with aging occur in the center of the lens. The nucleus of the lens is particularly sensitive to nutrient deficiencies. Nuclear cataracts are associated with deficiencies in the fat-soluble vitamins A and α -tocopherol and the water-soluble vitamins B₂ (riboflavin) and B₃ (niacin). Carotenoids have potent antioxidant activity with marginal inverse associations between the carotenoids lutein and cryptoxanthine and the development of nuclear cataracts. Riboflavin is important in the production of glutathione peroxidase; deficiencies in glutathione peroxidase have been correlated with cataracts. Selenium and vitamins C and E are also helpful in preserving glutathione levels. However, supplementation with selenium is not recommended as cataracts have been correlated with both deficiencies and excesses of this trace mineral. Taurine deficiency (particularly in animals fed heat-processed diets) has also been correlated with cataracts.

Table 4.2.14 | The IDC and the Reproductive System

History	Signalment	Diagnosis	Treatment
Diet <ul style="list-style-type: none"> • Seeds, nuts, produce, supplemental vitamins, table foods 	Eggs <ul style="list-style-type: none"> • No eggs, small clutches, soft/rough-shelled eggs, infertile eggs Hatchlings <ul style="list-style-type: none"> • Failure to hatch, (need assistance), early neonatal death, bent legs/beaks, crop/digestive disorders, require hand raising (from day 1) Adults <ul style="list-style-type: none"> • Egg binding, masturbation, regurgitation, nest building, mate mutilation 	Blood <ul style="list-style-type: none"> • Increased cholesterol, hyperlipidemia Diet Change <ul style="list-style-type: none"> • Response takes up to a year Reproductive Tissue <ul style="list-style-type: none"> • Culture uterus, biopsy uterus/testis, endoscopy (cystic ovaries) Eggs <ul style="list-style-type: none"> • Necropsy, culture 	<ul style="list-style-type: none"> • Treat specific disorder Diet <ul style="list-style-type: none"> • Nutritionally balanced formulated organic foods, limit supplements: low sugar produce - Breeding Birds: 5-10% high fat items (sunflowers seeds, nuts) sugary fruits & vegetable (necessary for breeding stimulation) Genetics Cockatiels <ul style="list-style-type: none"> • Choose birds that are not chronic egg layers - Deplete body stores of nutrients <ul style="list-style-type: none"> • Hormone therapy Salpingohysterectomy

The cortical cataract occurs in the cortex of the lens and a subcapsular cataract starts as opacity under the capsule, usually at the back of the lens. The prevalence of cortical cataracts is reduced in the presence of polyunsaturated fatty acids. Insufficient *n-3* fatty acids or excess saturated or 'trans' (hydrogenated) fats may impact on the progression of eye disease. While high levels of α -tocopherol reduce the risk of nuclear opacity, medium levels are associated with a reduced risk of cortical opacities.

Macular Degeneration

While macular degeneration has not been reported in birds, there are different kinds of macular problems. In other animals the most common is age-related macular degeneration. Zinc deficiencies can exist in older birds from poor absorption from food. Zinc is highly concentrated in the eye, particularly in the retina and tissues surrounding the macula. Zinc is necessary for the action of over 100 enzymes, including chemical reactions in the retina. While zinc supplementation may be beneficial if there is a dietary deficiency or malabsorption problem, excess zinc may also interfere with other trace minerals such as copper. The xanthophylls lutein and zeaxanthin selectively accumulate in the macula, providing what is known as the macular pigment. Singlet oxygen production in the eye can be increased by UV light exposure, but this is yet to be established in birds. It is assumed this increased oxidative damage is due to increased free radicals in the retina. Lutein and zeaxanthin are scavengers of these free radicals. Anti-oxidants (vitamin A, C and E) may also help slow down macular degeneration and other aging factors associated with activated oxygen from exposure to light, but this has yet to be established.

REPRODUCTIVE SYSTEM

Various reproductive problems can be caused by a nutritionally imbalanced diet (Table 4.2.14). A high fat and sugar based diet is strongly suspected to be involved in the overly stimulated breeding bird. See Chapter 3, Concepts in Behavior, Section III Pubescent and Adult

Psittacine Behavior for a table of foods hypothesized to be stimulatory.

Protein and Amino Acids

Amino acid requirements increase at least one week prior to the first oviposition for growth of the oviduct and accretion of egg proteins. While overall protein requirements for birds laying small clutches may be little more than maintenance requirements, a deficiency in essential amino acids may increase overall protein requirements. Budgerigars maintained on seed-based diets that provided only half the lysine, methionine and cysteine required, produced fewer hatchlings, fledglings, fertile eggs, and total eggs.⁴

Vitamins

Fat-soluble vitamins can influence breeding potential; a reduction in vitamin A and E reduces antioxidant function and increases exposure of lipid-rich tissues to peroxidation. Both deficiencies and excesses of Vitamin A influence epithelial integrity. The resulting hyperkeratosis can influence mucin production as well as proper tone and elasticity of reproductive tissue. Vitamin A deficiencies can result in failure of spermatogenesis, decreased size of testes and decreased sexual activity in males.

Dietary excesses of vitamin A may negatively influence reproductive output of birds. Improved productivity has been recorded in a variety of larger psittacines maintained on diets low in vitamin A and high in vitamin E.⁶⁷ A study of blue and gold macaws (*Ara ararauna*) highlights the importance of maintaining breeding birds year round on nutritionally balanced diets. Productivity significantly decreased when birds were transferred to seed-based diets during the nonbreeding season.⁶⁷ Maternal diets can influence nutrient transfer to embryos, as well as the antioxidant status of the developing embryo and hatchlings.

Table 4.2.15 | Action of Pesticides at a Cellular Level

Action	Result	Pesticide
Altered membrane integrity	Interferes with fluid and electrolyte movement	DDT Pyrethrins
Altered cell volume regulation	Alters energy metabolism, reducing energy availability to drive active transport systems, synthesis of macromolecules and maintenance of osmotic balance	Dinitrophenol Chlorophenol fungicides Arsenates Tin fungicides
Results from metabolic defects	Abnormal accumulation of lipids and pigments	industrial estrogenic wastes
Alteration of protein synthesis	Denaturation or inactivation of enzymes	Oxalic acid Fluoroacetate Organophosphates Carbamates
Disturbance of growth regulation	DNA damage that is not properly repaired or exceeds homeostatic control	Damage documented but not the cause

Lipids

While obesity can be detrimental to breeding birds, an imbalance of essential fatty acids is also problematic. Linoleic acid (*n*-6) typically reduces liver fat and improves egg production.^{42,43a} It is preferentially retained by laying hens. Conversely, high levels of linolenic acid reduce egg production in laying hens.¹

Typical fatty acid profiles of chicken spermatozoa display considerable resistance to manipulation by dietary means.⁵⁶ Dietary supplementation of docosahexaenoic acid (DHA) may inhibit synthesis of *n*-6 fatty acids in the testes²³ resulting in an accumulation of DHA, a concurrent decrease in vitamin E concentrations, and increased susceptibility to lipid peroxidation. While supplementation with *n*-3 fatty acids may be beneficial for the treatment of inflammatory diseases (see Chapter 16, Evaluating and Treating the Kidney), it may influence fertility by impacting the integrity of the component fatty acids in the spermatozoa's phospholipid membranes.

Sperm output generally decreases with age, but this decrease can be prevented by supplementing diets with oils rich in either arachidonic acid or DHA, in conjunction with vitamin E (200 mg/kg). Testes mass can also be increased up to 1.5 times in aging birds when supplemented with essential fatty acids. However, supplementation with linoleic acid in the absence of vitamin E can result in 50% reduction in spermatozoa per ejaculate in aging birds.

The cloaca can transmit microbes retrograde into the reproductive tract. The hypothesis that there is a passage of beneficial microbes at the time of copulation is intriguing. It has been hypothesized that females copulate with multiple males to gain microfloral advantage for the offspring from flora transmitted at copulation.⁶⁴ The female is thought to benefit by protection from future as well as present infections that become over-

whelmed by the inoculum.⁶⁴ This is another area that, if proven, shows the value of the microflora to the body.

ADVERSE FOOD REACTIONS

Food Allergies

The bird should undergo a complete physical examination including appropriate diagnostics to look for any underlying pathology, which may be exacerbated by or in addition to the suspected food allergy. Not all dermal problems are related to nutritionally imbalanced diets as some birds, like cats and dogs, have allergic reactions to certain dietary ingredients. Advanced stages often result in feather picking and self-mutilation. While early signs of these food allergies are yet to be described, failure of nutritional therapy may warrant skin allergy testing and/or a simplified organic diet^a where ingredients such as corn and sunflower seeds are eliminated. While these ingredients have been incriminated in the development of dermal disorders⁶⁵, they are regularly included in both formulated and homemade diets as primary ingredients with no reports of digestive disorders. Organic formulated diets are free from pesticide residues and preservatives that could be potentially allergenic. Common allergens for mammals (wheat, gluten, egg and dairy products) should also be eliminated from sensitive birds, although no proof exists they are a problem. A commercial organic mash diet^a is composed of the following ingredients: buckwheat, hulled gray millet, hulled white (proso) millet, spirulina, chia, alfalfa, clay, sea kelp, anise, natural sources of vitamins, minerals and trace minerals. (Can be wrapped in thin slices of banana for feeding). This mash has been clinically correlated with the abatement of pruritis in several birds suspected to be suffering from food allergies. Once the mash has been accepted, the proportion of banana can be gradually decreased until eliminated. However, a bird could potentially be allergic to any dietary ingredient.

DIETARY ANTIFEEDANTS AND XENOBIOTICS

As the practice of organic farming diminishes the detrimental impact of pesticides on wildlife and their habitats, the consumption of organic products minimizes the need for animals in captivity to modify and detoxify antifeedants in their diet. Evaluating the potential burden that residual pesticides in non-organic ingredients place on a bird requires understanding the biochemical complexities of detoxification mechanisms that enable animals to process these potentially harmful chemicals. This can apply equally to foodstuffs with high concentrations of antifeedants, such as alkaloids, cardiac glycosides and phenolic compounds, that require detoxification. However, pesticide tolerance has not been evaluated in

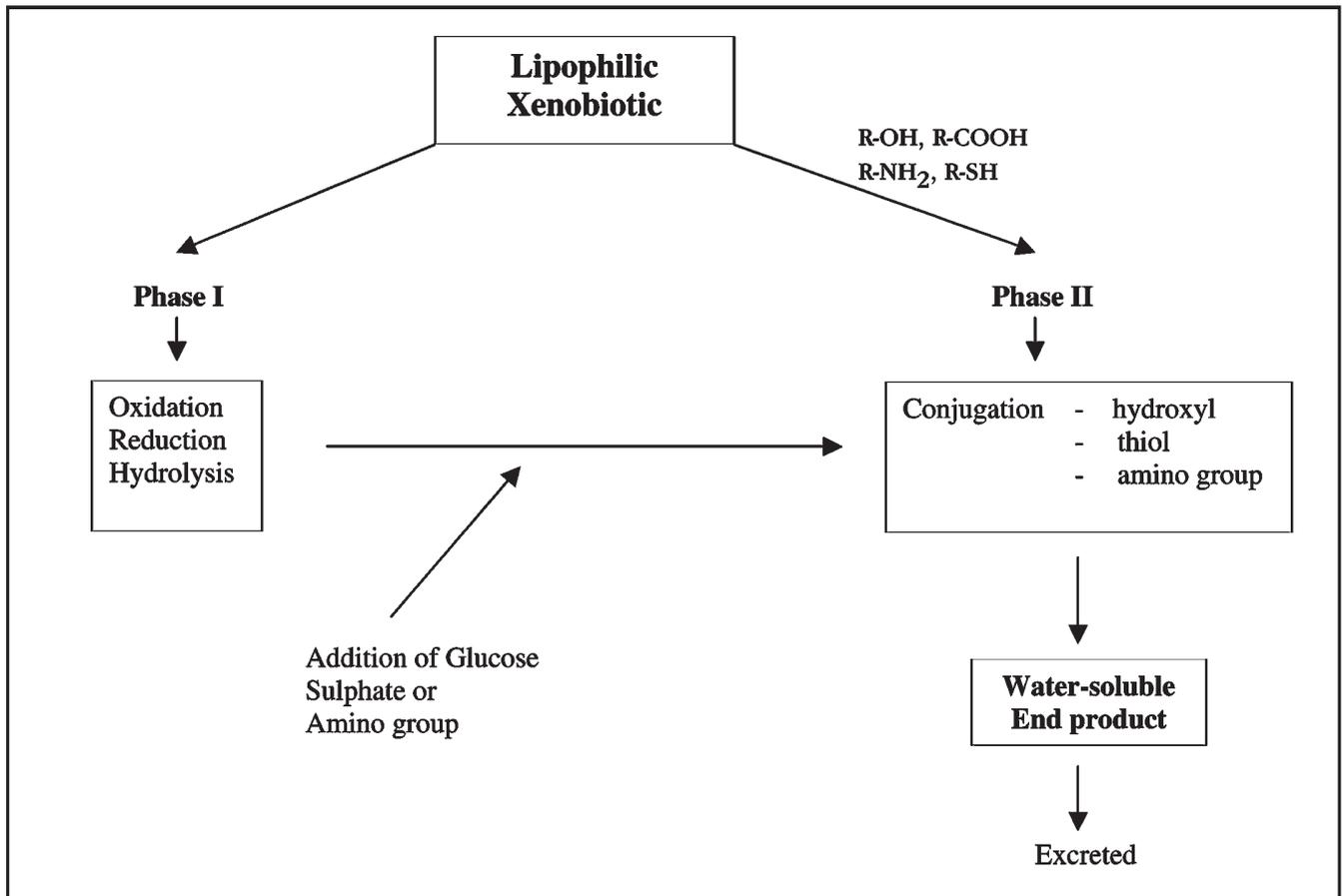


Fig 4.2.32 | Phase I and II detoxification of xenobiotics.

companion birds. Clinically significant improvements in health and productivity have been reported for birds maintained on organic diets.⁶⁷

Actions of Chemical Contaminants

Most toxicological injury results in cellular damage. The toxic response to any specific chemical is the result of dysfunction of relatively few basic biologic processes. Normal processes can be suppressed or stopped completely, or they can be enhanced beyond normal physiologic limits and, in turn, affect other systems dependent on their controlled functions. Cellular responses to chemical toxins occur through both structural and metabolic mechanisms in the cell. A single response or a number of actions can be elicited from an individual pesticide (Table 4.2.15).

Detoxification of Xenobiotics

Xenobiotics are pharmacologically, endocrinologically, or toxicologically active substances not endogenously produced and therefore foreign to an organism. Pesticides acting as estrogens (xenoestrogens) are a common subject of discussion at wild bird disease seminars^{33,34,40,41,103} (Fig 4.2.32).

The detoxification of xenobiotics is a biphasic process

carried out by a suite of non-specific microsomal enzymes referred to as mixed function oxidases (MFOs) that act primarily in the endoplasmic reticulum. The detoxification process converts a lipophilic compound into a highly water-soluble product that is suitable for excretion in urine or feces. The two phases of detoxification place different demands on nutrient stores in the body. Therefore, the ability to digest and process foreign chemicals may vary from one individual to another, depending on species, feeding ecology, gender and developmental stage. If liver function is diminished from any other disease process, detoxification capability may also be affected.

Nutritional Requirements for Detoxification

Dietary nutritional deficiencies, such as minerals (calcium, copper, iron, magnesium, zinc), vitamins (E, C and the B complex) and proteins, can limit the chemicals necessary for the synthesis of enzymes or conjugating agents. Energy deficits induced by fasting decrease the activity of xenobiotic metabolizing enzymes. Protein malnutrition may also impair enzyme synthesis, MFO activity and hepatic glutathione concentration.

Detoxification may also have an impact on ascorbic acid (vitamin C) levels, as glucuronate is a precursor in the

Table 4.2.16 | Behavioral Traits Attributed to Nutritional Inadequacy

History	Signalment Individualistic	Treatment
Overly permissive owners Behavior <ul style="list-style-type: none"> • Craves sweets/fats Diet <ul style="list-style-type: none"> • Poorly balanced 30-60% warm table foods and numerous treats • Seed based Husbandry <ul style="list-style-type: none"> • Poor 	Distrustful Obsessive/Compulsive Depressed, Separation Anxiety Reproductive <ul style="list-style-type: none"> • Masturbation, hiding, nest building, egg laying Feather Picking, Mutilation Biting, Screaming Sweet/Lovable Clinging Aggression	Formulated Diet <ul style="list-style-type: none"> • Nutritionally balanced Environment <ul style="list-style-type: none"> • Adjust, move/redecorate cage Medication <ul style="list-style-type: none"> • Human chorionic gonadotropin • Lupron, dexamethasone Behavior <ul style="list-style-type: none"> • Modification, tolerance training, exercise

biosynthesis of ascorbate in most animals. Deficiencies of ascorbate reduce the ability to detoxify foreign chemicals. Stress may also increase the rate of ascorbate metabolism. Any changes in environmental attributes (structural or nutritional) may impact on levels of ascorbate and thus a species' ability to detoxify antifeedants in its diet.

By consuming several different plant foods, and so using various rate-limiting detoxification pathways, an animal can ingest larger amounts of food per unit time enabling them to obtain more energy and nutrients. While varying the diet may be beneficial to some animals, there is the risk of exposure to a greater variety of biologically active compounds. If more than one xenobiotic is absorbed there can be an additive, synergistic or antagonistic interaction between them. Therefore, the ability to utilize a single food substrate or to incorporate various plant species into the diet will be based on the individual.

Implications for Pesticide Residues in Avian Diets

Birds maintained in captivity are confronted with a different set of stresses than their wild counterparts. In addition to adjusting to the stress of being maintained in a confined environment, they are generally supplied with foods that have been domesticated for the human palate or formulated foods composed of foreign ingredients. There are various natural antifeedants in foods that the avian system must detoxify, and the addition of residual pesticides in non-organic foods adds to this burden. Tannins differ in their actions. Condensed tannins have the potential to bind to proteins, rendering them insoluble and unavailable for assimilation; hydrolysable tannins do not bind to proteins, but require detoxification, placing a drain on nutrients associated with the detoxification system. A nutritionally balanced diet is still not optimal if levels of feed contaminants require extensive detoxification.

The toxicity of certain pesticides may be underestimated, as cumulative effects or delayed toxicity can occur long after exposure. This confuses the interpretation of signs credited to pesticide exposure. Synergistic effects may also be observed, if one chemical affects the solubility, binding, metabolism or excretion of another. Therefore,

while studies on the effects of individual pesticides may not indicate any detrimental actions, a combination of two or more pesticides may enhance toxic actions. Potentiation occurs when one chemical enhances the toxicity of another, even though the toxicity of the potentiator is minor or nonexistent. See Chapter 11, Low-risk Pest Management for actions one might consider in avoiding pesticides.

Pesticides and Behavioral Abnormalities

Levels of pesticide contamination in bird foods composed of non-organic ingredients have yet to be evaluated. Behavioral abnormalities associated with pesticide exposure include: reduction in courtship behavior^{33,34,74}, reluctance of females to take food from males, changes of activity patterns in males⁴⁰, reduced levels of nest defense^{33,34,41}, alterations of incubation behavior, decreased parental attentiveness resulting in increased embryonic mortality^{33,41,82}, decreased time feeding young, fewer sorties to feed young and increased time away from nests.^{33,34,82}

Eggshell thinning induced by dichlorodiphenyldichloroethylene (DDE) is species specific. Organochlorines reduce levels of androgens in males and estrogen and progesterone in females. Levels of thyroxine in both sexes decrease in a dose-related fashion. There are also links between hyperthyroidism, PCB's and dichlorodiphenyltrichloroethane (DDT).⁴¹ Chlorinated hydrocarbons induce changes in the metabolism of steroid hormones by mixed function oxidases (MFO).⁸²

BIRD BEHAVIOR

A range of behavioral traits can be attributed to nutritionally imbalanced diets, inappropriate dietary ingredients, and dietary exposure to pesticides (Table 4.2.16). These include changes to vocalization patterns and breeding behavior. Behavioral traits of birds also need to be evaluated when converting birds to formulated diets (see Chapter 3, Concepts in Behavior, Section III Pubescent and Adult Psittacine Behavior). The subjects of high-fat and high-sugar diets along with elevated sodium are presented.

Dietary Influences on Vocalization Patterns

Changes in vocalization patterns have been reported in cockatiels maintained on diets with deficient or excessive levels of vitamin A.⁵⁸ Excessively high levels of vitamin A (100,000 IU/kg) increase the number of vocalizations and reduce the peak frequency of vocalizations, while moderately excessive levels of vitamin A (10,000 IU/kg) result in a reduction in peak amplitude and total power. These changes may influence breeding behavior, social interactions and responses of adults to begging behavior of chicks.

Diet and Behavioral Changes

Reproductive behavior, normally regulated by photoperiod, can be influenced by dietary components. Additional seeds stimulate breeding behavior in African grey parrots.⁶⁹ Endocrine malfunction has been implicated in anecdotal studies of male budgerigars on all-seed diets that display continuous feeding behavior towards a mirror. Reducing the synthetic vitamin A content of food fed to lorikeets reduced defecation in nest boxes (Unpublished data, D. McDonald). See Chapter 3, Concepts in Behavior, Section III Pubescent and Adult Psittacine Behavior.

DIET CONVERSION CHALLENGES

Modifying a bird's diet is one of the biggest behavioral challenges. Most issues can be overcome with patience and perseverance. Educating the owner about the benefits of a formulated diet versus a seed-based diet is the first challenge. Feeding the new dietary items early in the morning when the birds are most hungry is beneficial, but dietary changes should be undertaken gradually. If birds continue to have problems with acceptance of new dietary items, placement near birds that are already feeding on similar foods is beneficial as birds usually mimic feeding habits of other birds. Mixing the new diet with a favorite fruit can be helpful; mushy fruit works best as it sticks to the formulated food. Remove the fruit after 4 to 6 hours to avoid consumption of spoiled food.

An important concern is the bird's refusal to eat or significant weight loss. Although weighing the bird in grams on a daily basis is the best method of monitoring adequate food consumption, monitoring droppings can also indicate if the bird is eating enough. Prior to the diet change, note the number and character of the droppings (color, amount, liquid, form, shape, lack of odor, staining). See examples in Chapter 6, Maximizing Information from the Physical Examination. Any change in volume and number of droppings, usually a dramatic decrease in amount, indicates insufficient consumption. Character of droppings will change as the bird consumes more formulated diet. Weight fluctuations greater than 10% are considered

problematic. Even if provided with surplus food, birds can starve to death if they don't consume the food offered. Therefore, it is imperative to immediately return a bird to its original diet if it refuses to eat the new diet. The following guidelines should be given to owners when a diet conversion is being initiated.

Tips for Converting Birds to a Formulated Diet

- *Visit the avian veterinarian for a general health exam to decide if the bird is healthy enough to undergo a diet change at this time.*
- *Discuss which formulated product is best for the bird.*
- *Determine the goal body weight that is appropriate for the bird.*
- *Purchase a gram scale and learn how to use it correctly.*
- *Weigh the bird at the same time every morning for a week to establish normal fluctuations in weight. Report any serious fluctuations (10% or more) to the avian veterinarian.*
- *Mix half formulated diet and half the old diet. Expect the bird to exhibit negative behavior by throwing the pellets/nuggets at owner, screaming, yelling, and having tantrums. Talking to the bird may soothe it. As the bird starts to eat the formulated diet, gradually reduce the amount of the old diet and increase the proportion of the new diet.*
- *Remove all perches from cage so the bird is forced to sit on the food dish or put the bird in a plastic or glass box (aquarium) and sprinkle food on the floor. Provide a small water container—no toys.*
- *Place formulated food on a mirror located on the floor (Figs 4.2.33a,b).*
- *Place the bird in a cage with another bird that is already consuming a formulated diet. Do not provide any seed and separate birds at night. If the bird has not consumed any of the formulated food by evening, provide it with seed.*
- *Once it starts to eat, place a bowl of formulated food near the highest perch.*
- *Feed the old diet for 30 minutes in the morning, remove and replace with formulated food for remainder of the day. Feed the old diet for 30 minutes at night if formulated diet is not eaten.*
- *Grind formulated food in a blender (or purchase a mash product) and mix crushed millet in with the mash.*
- *Using a less palatable seed (hulled white millet), break the seeds up in a blender with the pellets. After a few days use less ground millet.*
- *Thoroughly mix bird's favorite fruit into formulated diet so bird gets a mouthful of new diet with fruit.*
- *Under the guidance of an avian veterinarian, try returning to hand feeding a juvenile formula with a*



Fig 4.2.33a | Budgerigar that has become infatuated with its image in a mirror.

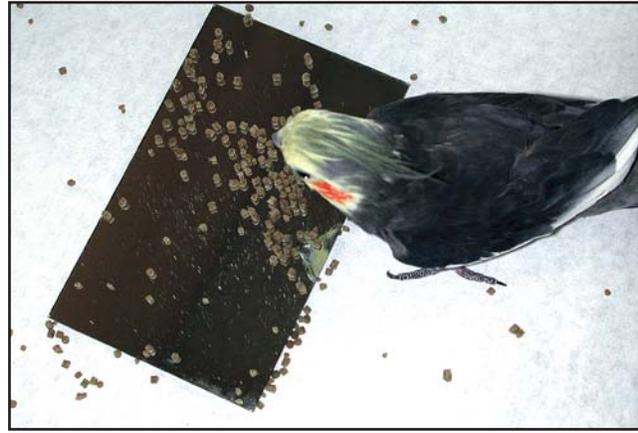


Fig 4.2.33b | Cockatiel in a container with only a mirror. Food is placed on the mirror. Return the bird to its own cage at night for regular diet and water. Replace in mirror box next day after having at least 30 minutes to eat regular diet and drink. Diminish the time with seeds daily until weaned.

syringe and then wean to formulated food.

- *Very small particles of formulated foods seem more acceptable to small birds*

If patience and persistence doesn't pay off, it is best to board the bird under clinical supervision. The veterinarian can convert the bird to the new diet while carefully monitoring weight and health of the bird. Most birds switch diets very quickly when removed from the "comfort" of the home environment. Avian veterinarians generally have more experience with dietary changes. Birds should be left for a sufficient period of time to ensure conversion is complete ie, held for 2 to 10 days. Wait until the bird maintains body weight for at least two days after normal diet has been totally removed. It is best that the bird is not returned to the same routine at home (ie, move cage, redecorate cage, don't place in kitchen at mealtime).

Some of the IDC clinical signs are idiosyncratic in nature. A 20-year old cockatiel maintained solely on sunflower seeds or a 50-year-old Amazon provided with chicken bones, seeds, nuts and table foods may never present with the advanced signs of a clinical nutritional disorder. However, this should be seen as the exception and not the rule. Additionally, individual birds may develop clinical signs of the IDC at different rates or to varying degrees.

While problems associated with the integument can precede breakdown of other systems, they are seldom recognized in the early stages of the IDC. Therefore, digestive upsets associated with gastroenteritis and ileus are more likely to instigate an investigation into yeast or coliform bacterial infection rather than dietary history. A proliferation of meetings, publications, investigative consultancies, analytical laboratories and therapies aimed at the presenting problems over the past 30 years have all contributed to a better understanding of illnesses in pet birds. While secondary problems associated with any one of dozens of common, yet serious, secondary pathogens still warrant attention, preventive medicine remains the most effective therapy.

Products in Text

- Adult Lifetime Mash. HBD International, Inc., Brentwood, TN, 1-800-346-026
- Harrison's Bird Foods. HBD International, Inc., Brentwood, TN, 1-800-346-0269
- Avian Enzyme- HBD International, Inc., Brentwood, TN, 1-800-346-0269.
- Prozyme- www.prozymeproducts.com
- Hepasan- www.vetpharm.unizhoch/tpp/0000
- Ultraclear. Metagenics, 1152 Ensell Rd. Lake Zurich, IL. www.metagenics.com
- Parrot Specific Lactobacillus (Munich)- www.janeczek.de
- Bird Builder, AV1x - www.exoticdvm.com

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