The Impact of Vitamin A Deficiency on the Health & Reproductive Success of Exhibition Budgerigars

PRIMARY VERSUS SECONDARY VITAMIN A DEFICIENCY

Vitamin A deficiency can be categorised as either primary or secondary deficiency. Understanding the differences is key to managing the many problems associated with vitamin A deficiency in exhibition budgerigars. Vitamin A is not synthesized in the body and must be provided in the diet to ensure adequate stores are maintained in the liver (the primary vitamin A storage organ in birds). Signs of vitamin A deficiency occur when liver stores drop below 10% homeostatic functional levels.

Primary vitamin A deficiency occurs when the dietary intake of vitamin A does not meet the amount required for maintenance. This form of vitamin A is uncommon in exhibition studs because carotenoid-rich foods such as carrots and leafy greens are usually regularly fed throughout the year and provide sufficient vitamin A to maintain those organs and body systems that depend upon vitamin A for their normal functions.

On the other hand, secondary vitamin A deficiency is a common problem in exhibition budgerigars and occurs when any increased demand for vitamin A above maintenance requirements outweighs the dietary intake of vitamin A and reduces liver stores below 10%. This means there is not sufficient circulating vitamin A to fulfill the many vital functions of vitamin A in the body. When vitamin A liver stores drop below the critical 10% threshold level, the body prioritizes vitamin A to those organs essential for survival. Simultaneously it restricts vitamin A delivery to those organs and systems that are not essential for immediate survival - notably the testes, ovary, immune system and thermoregulation system amongst others. This biological mechanism helps to restore functional levels of circulating vitamin A when vitamin A supplementation is given. This recovery system fails to protect against the consequences of vitamin A deficiency when digestive disorders associated with vitamin D deficiency prevent the absorption of vitamin A. This is a complex concept but important to understand the connections between vitamin A and vitamin D deficiencies and how best to manage them.

Secondary vitamin A deficiency is a common problem in exhibition budgerigar studs because without regular supplementation, the vitamin A needs of reproduction far exceed the amount of vitamin A (beta carotene) provided by vegetables which are only 3-9% absorbed. Beetroot is often included in the diet of exhibition budgerigars as a source of iron, magnesium, zinc and B vitamins. However, it is an inferior source of vitamin A compared to carrots, which contain 300% more bioavailable vitamin A.

Vitamin A supplementation is best achieved through ZADE liquid or NVA power which are 90-98% absorbed. Prior to the breeding season, these supplements are given (see accompanying brochures) in addition to fresh carotenoid foods (e.g. carrots, green leafy vegetables) to lift the liver reserves of vitamin A to optimal levels (75-100%). Supplementation continues throughout the breeding season, but leafy greens should be restricted to prevent gizzard disease. This condition is a common problem in studs housed indoors with insufficient exposure to direct sunlight and are therefore often vitamin D deficient. Vitamin D deficiency limits calcium absorption and predisposes to gizzard disease which impacts digestive function and the absorption of vitamin A and other nutrients. This complication of vitamin D deficiency culminates in a vicious cycle of nutritional deficiency that leads to reproductive failures, disease and deaths.

VITAMIN A DEFICIENCY AND REPRODUCTION

The greatest demands for vitamin A occur during spermatogenesis (the mating phase of reproduction) and folliculogenesis (the development of egg yolk) prior to ovulation. This means studs with marginal levels of liver stores will be impacted by secondary vitamin A deficiency during the breeding season when the demands for vitamin A outweigh the intake of vitamin A. Budgerigar studs are particularly prone to the widespread consequences of secondary vitamin A deficiency because they are opportunistic breeders and produce multiple eggs per clutch. Males in the flights can engage in prolonged periods of courtship and mating behaviours during repeat 7-week cycles. This unregulated sexual activity drains vitamin A liver stores leading to a state of perpetual marginal secondary vitamin A deficiency. Females are most prone to the consequences of secondary vitamin A deficiency when they start to lay second round eggs. We have designed a vitamin A booster plan to prevent the consequences of secondary vitamin A deficiency during the breeding season (see accompanying brochure).

VITAMIN A DEFICIENCY AND THERMOREGULATION

Budgerigars with vitamin A deficiency have impaired thermoregulation and are more prone to the harmful effects of heat stress during heat waves, or cold exposure during cold snaps. Altered glucose metabolism and vitamin B requirements associated with these temperature extremes leads to increased susceptibility to illness. The immune function of birds with secondary vitamin A deficiency is also impaired. The combination of vitamin A deficiency, heat or cold stress predisposes budgerigars to life-threatening opportunistic infections following periods of extreme or fluctuating weather. ZADE Liquid combined with Quikgel given for 2 consecutive days prior to forecast cold or hot spells is an effective prevention measure. Follow up treatments of ZADE for 2 days each week are continued for 6-8 weeks to restore optimal vitamin A stores.

VITAMIN A DEFICIENCY AND IMMUNE FUNCTION

Birds with vitamin A deficiency have impaired immune function and are susceptible to opportunistic bacterial, fungal and viral infections. These include *Macrorhabdus ornithogaster* (**Megabacteria**, AGY) infection, Polyomavirus (**French Moult**) infection, *Staphylococcus* infection (**"Red Eye Disease"** and **"Yellow Belly"** in babies), *Chlamydia psittaci* (**Psittacosis**), *Aspergillus* and *Candida* fungal infections.

TELL TALE SIGNS OF SECONDARY VITAMIN A DEFICIENCY

- Any type of breeding failure (e.g. infertile eggs, soft belly, sudden death of incubating hens, stunted chicks, fledgling disease, second round mortality etc.)
- Opportunistic infections (e.g. red eye (staph. Aureus), dirty nostrils (streptococcus / enterococcus), french moult (polyomavirus), scaley face mite (cnemidocoptes mite)
- Pasted vents (antibiotic resistant gut infections)
- Obesity (dyslipidemias, pectoral muscle atony causing weakened flight)

TELL-TALE SIGNS OF COMBINED VITAMIN A AND VITAMIN D Deficiency

The most severe consequences of vitamin A deficiency occur in flocks with co-existing vitamin D deficiency because of the cumulative impact of vitamin A and vitamin D on immune function.

- Signs of vitamin D deficiency (egg binding, delayed moult, dry feathers, splay legs, soft shelled eggs etc.)
- Preen gland abscesses
- Infections with poor response or resistance to medications (E. coli, staphylococcus, trichomoniasis, coccidiosis, candidiasis, Macrorhabdus etc.)