Suspected Hypervitaminosis A in Lorikeets Maintained on Commercially Formulated Nectars – A Case Study.

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Abstract

Hepatic and serum vitamin A and E status were evaluated for captive colour mutation red collared (*Trichoglossus rubritorquis*) and rainbow lorikeets (*Trichoglossus haematodus*) housed in a private collection that had experienced health and productivity problems. Data were compared to samples collected from wild lorikeets in Western Australia over a 12 month period. Little variation was detected in hepatic vitamin A (19.6-56.2 mg/kg) or vitamin E (10.8-14.1 mg/kg) of wild birds, with the exception of one fledgling with symptoms of PBFD having low hepatic vitamin E (4.3 mg/kg). High hepatic vitamin A was detected in birds maintained on commercial nectars (up to 4093 mg/kg), while hepatic vitamin E was up to 108 mg/kg. While vitamin A content of hatchlings (46-61 mg/kg) compared favourably with that of wild birds (19.6-56.2 mg/kg), these levels rapidly declined by 4 weeks of age (<10 mg/kg) and may have contributed to poor hatchling survivorship. Similar declines were also noted for hepatic vitamin E concentrations for hatchlings. A single sample from a sun conure (*Aratinga solstitialis*) maintained on a seed-based diet in the same collection indicated that while vitamin E status of this bird was adequate (13.7 mg/kg), vitamin A status was possibly low (39.1 mg/kg) compared to other wild seed-eating psittacines (1.9-7 mg/kg vitamin E, 33-823 mg/kg vitamin A). Some improvements in health and productivity were observed when the collection was transferred to a diet devoid of vitamin A, with spirulina (high in carotenoids) and vitamin E added to the diet. Hepatic vitamin A has continued to decrease since the collection was transferred to the new diet but levels are still high compared to wild birds and it is possible that excess dietary vitamin A contributed to the problems but it is clear that further research is required.

Introduction

Hypervitamiosis A is not commonly diagnosed in companion birds in Australia as many birds are still maintained on seed-based diets that are deficient in provitamin A (carotenoids) and require vitamin supplementation. However, the incorporation of vitamin A in commercial products generally eliminates the need to provide further vitamin supplementation, with many of these products exceeding dietary requirements established for cockatiels (6,000 IU/kg; Koutsos and

Klasing, 2002). The higher vitamin A content of many commercial nectars can be exacerbated by the supplementation of vitamins.

Hypervitaminosis A results in severe skeletal lesions, osteodystrophy characterized by abnormal thickening of the proliferative maturation zone (broilers) or by a thin proliferative maturation zone (leghorns), metaphyseal sclerosis, hyperosteoidosis, decreased number of osteoclasts, parathyroid gland hyperplasia, relatively thickened hypertrophy zone, flattened spindle-shaped osteoblasts, and osteoporosis (Tang et al, 1985). Degenerative atrophy, fatty infiltration and reduced function of liver and kidney also result and increases in the activity of sucrase eliminate the ability to regulate the activity of this enzyme by the duodenum in the chicken small intestine (Shoshukova et al, 1991). Excess dietary vitamin A not only results in vitamin A toxicosis but can also lead to a deficiency of other fat-soluble vitamins, resulting in a decrease in growth or egg production. As the mechanisms of absorption and transport are similar for carotenoids, an excess of dietary vitamin A can also influence pigmentation and antioxidant status of birds. While there are differential responses to vitamin A toxicosis within breeds and across species (Veltmann and Hensen, 1986), higher dietary requirements correlated with high production rates may not be applicable to the comparatively small clutch sizes of lorikeets (approximately 2 eggs).

Case Study

A history of health and productivity problems was reported for a variety of mutation rainbow lorikeets (*Trichoglossus haematodus*) and red-collared lorikeets (*Trichoglossus rubritorquis*) by a private aviculturist in Australia. The birds had been maintained on a number of commercial nectars, providing varying levels of vitamin A (9,000-28,740 IU/kg). While egg production did not appear to be compromised, a high percentage of embryonic deaths at two weeks of age characterized by small embryos was recorded. Many hatchlings also died within a few hours with no evidence of bacterial infection at necropsy, although chicks that died post one day of age tended to have bacterial infections. While egg shell thickness was not perceived to be problematic, thin-shelled eggs that broke easily were reported for breeding birds in their first season. Egg fertility previously recorded at approximately 80% of eggs laid was reduced to less than 70% in the past year, with problems more significant in the first three months of each nine month breeding season. As sun conures (*Aratinga solstitialis*) were maintained in the same aviary complex (on different diets) with 79% fertility and 71% hatching and fledging success, flock infection was not considered to be an issue.

Pigmentation aberrations were noted in 10% of birds (n = 120) and were increasing in frequency. These included elongated oval marks in the centre of back feathers, general irregular spotting and loss of pigment on back feathers and underneath the legs, and marked deterioration of the ends of otherwise good feathers with black edges. Further deterioration of feathers (but not black edges) was associated with overpreening.

Birds were transferred to a home diet devoid of vitamin A, with provitamin A sourced from spirulina and vitamin E from wheat germ oil. To determine whether health and productivity problems in this collection were associated with excess dietary vitamin A, plasma and liver samples were obtained from a number of captive pied rainbow lorikeets (*Trichoglossus haematodus*) that had been maintained on various commercial nectars and compared to other unaffected species in the collection as well as samples from wild lorikeets in Western Australia over a 12 month period. Samples were analysed for vitamin A and E using HPLC by the Animal Health Laboratories, Western Australia. Samples of commercial foods were also chemically evaluated and compared to calculated values provided by manufacturers.

Results

Vitamin A and E content of various commercial nectar products are presented in Table 1. Vitamin A content of two products (Sheps wet and Shep dry) was originally at 12,500 IU/kg until the manufacturer removed the vitamins approximately 12 months prior to this study. Breeding histories of rainbow (*Trichoglossus haematodus*) and red-collared lorikeets (*Trichoglossus rubritorquis*) (n=71) are presented in Figure 1.

Summary data for vitamin A and E status are presented in Table 2, with details provided in appendices 1-5. Age related decline in both vitamin A and E was detected in chicks up to 4 weeks of age, Figure 2. While one day old chicks had hepatic vitamin A levels (46.1-61.6 mg/kg) in excess of most wild birds (19.6-56.2 mg/kg), it appears to be rapidly depleted up to at least four weeks of age. This is in contrast to captive adults that had hepatic vitamin A levels ranging from 97.5-4093 mg/kg, with only one individual at 14.7 mg/kg. There was little variation in hepatic vitamin E levels in wild birds from either 6 months of age (10.8-13.4 mg/kg) or adults (13.6-14.1 mg/kg), with the exception of one individual fledgling that was diagnosed with PBFD (4.3 mg/kg).

Data from three wild birds that were transferred to commercial diets in captivity indicate higher levels of hepatic vitamin A (89.9-588.4 mg/kg) and lower levels of vitamin E in two of the birds (7.7-6.9 mg/kg), when compared to wild birds (19.6-56.2 mg/kg and 4.3 14.1 mg/kg respectively). An adult purple crowned lorikeet (species name) also contained high hepatic vitamin A (823.9 mg/kg) but lower vitamin E (10.4 mg/kg). A sun conure (*Aratinga solstitialis*) in the same collection that was maintained on a seed-based diet contained only 39.1 mg/kg vitamin A and 13.7 mg/kg vitamin E.

Discussion

Hypervitaminosis A is rarely diagnosed in captive birds as birds that historically were maintained on seed-based diets tended to show symptoms of hypovitaminosis A and responded to treatment. However, symptoms of vitamin A toxicity can often resemble those of deficiency and the wider acceptance of commercially formulated diets has all but eliminated the need to supplement with vitamin A.

While there is considerable range in vitamin A content of commercial bird foods, the vitamin A contents of the commercial nectars used in this study ranged from 9,000 IU/kg to 28,740 IU/kg, exceeding dietary maintenance requirements of poultry of 1,500 IU/kg (NRC, 1994) and cockatiels (6,000 IU/kg; Koutsos and Klasing, 2002). Vitamin A competes with fat-soluble vitamins and carotenoids for sites of uptake so a dietary excess can result in a deficiency of other fat-soluble vitamins despite the appearance of adequate dietary intake. There was no evidence of vitamin A deficiency in any of the birds maintained on commercial nectars in this collection, with hepatic vitamin A levels exceeding that of many wild birds. Despite an extended period on a diet devoid of vitamin A with precursors provided predominantly from spirulina, hepatic vitamin A levels in this collection remain high. Studies of cockatiels indicate a period of up to 18 months is required on a diet devoid of vitamin A before symptoms of deficiency occur (Koutsos and Klasing, 2002) and it is possible that improvements will not be evident in this collection until the next breeding season. While fertility, hatchability and feather condition had increased in a number of individuals on the new diet, some health and productivity problems still persist in the collection. Wild lorikeets had higher hepatic vitamin A levels compared to other wild seed-eating psittacines (McDonald et al, *in prep*) but this may be attributed to a wild diet high in carotenoids compared to a seed-based diet that is low in carotenoids. Lorikeets may have adapted to a higher dietary provitamin A content but lack the ability to regulate uptake and storage of vitamin A from commercial diets.

While vitamin D status was not evaluated in this study, the high vitamin A levels may have interfered with uptake of this fat-soluble vitamin. Vitamin D deficiencies can result in the appearance of ruffled feathers and there were numerous problems with feather condition in this study. Vitamin D deficiency can also cause abnormal black pigmentation of some feathers of red or buff-coloured breeds of chickens, which is particularly pronounced in wing feathers (McDowell, 2000) and abnormal black pigmentation was observed primarily in green feathers of the lorikeets. Inadequate vitamin D can also result in the production of thin-shelled eggs and, while this did not appear to be widely problematic, 27% of eggs laid had broken shells, possibly indicating a dietary vitamin D deficiency. Hatchability is also reduced in birds suffering from vitamin D deficiency with embryos frequently dying at 18-19 days of age (McDowell, 2000). Hatchability was a significant problem in this study with most birds dying within the first few hours of hatching and embryonic deaths were recorded within two weeks of age, characterized by small embryos.

It is also possible that vitamin E uptake was compromised by the high vitamin A levels. Although hepatic and serum vitamin E levels of adults exceeded those of wild birds, the rapid decrease in hepatic vitamin E of hatchlings could suggest a compromised immune system and it is possible that young chicks require the vitamin E supplementation. Similar declines were observed in vitamin A content of captive hatchlings and it is possible that parents provide adequate provitamin A to chicks in the wild. As birds convert carotenoids to vitamin A on an 'as needs' basis, it is possible that the antioxidant system of chicks is compromised if hens are maintained on diets high in vitamin A.

Symptoms of vitamin E deficiency such as embryonic degeneration and immunodeficiency were evident in birds in this study. Chicks that failed to survive more than 2-6 days suffering from exudative diathesis may have been suffering from a deficiency of vitamin E and signs of encephalomalacia that generally affects chicks from two to six weeks of age were also evident in one chick that appeared healthy but died stretched out with clenched feet and erratic head movements. Degeneration of the Purkinje layer of cells in the cerebellum results in nervous signs typified as sudden prostration, with toes and legs outstretched, toes flexed and head outstretched (NRC, 1994).

Although there did not appear to be any vitamin E deficiencies in captive adults, if uptake of vitamin E was compromised by the high levels of vitamin A, fertility may have been impacted. Prolonged vitamin E deficiency can result in temporary or permanent sterility of males as there are high concentrations of vitamin E in spermatozoa (Surai et al, 1997,1998a/b, 2000, 2001). While approximately 27% infertility was recorded in an earlier season, this had increased to 36% in the following season. Hatchability of eggs from vitamin E deficient hens can be reduced (NRC 1994) with only 32.4% hatchability recorded in this study. Embryonic mortality may also be high especially during the first four days of incubation (McDowell, 2000) with circulatory failure impacting on later stages. 15.5% embryonic mortality was recorded in this study with most deaths recorded at two weeks and small embryonic sizes noted.

Conclusions

Despite the apparently adequate hepatic vitamin E levels in captive birds in this study it is possible that the excessively high hepatic vitamin A levels contributed to competitive uptake of vitamin E or carotenoids into other cells, compromising antioxidant status and immunocompentence of offspring. Studies of cockatiels indicate that up to 18 months may be required to deplete vitamin A stores and, although vitamin A content of birds in this study has decreased since commercial nectar mixes have been eliminated and carotenoids are provided from spirulina, levels remain high and numerous health and productivity problems still persist. It is clear that further studies of nutritional requirements of lorikeets are required and monitoring of birds in this private collection will continue.

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Product Name	Vitamin A IU kg^{-1}	Vitamin E mg kg^{-1}
Aristopet [1]	5,994	6
Aves Nectar[2]	24,150	22
Avione[1]	4,296	20
Elliots Dry[1]	666	2.8
Elliots Wild nectar[1]	666	1.3
HBD Adult Lifetime Fine (Maintenance)[3]	1,400	215
HBD High Potency Fine (Breeding)[3]	1,500	240
Lakes[6]	11,037	66
Lory Life Nectar[4]	52,900	54
Lory LifePowder[4]	10,130	11
Marion Lory[5]	8,500	250
Nekta Plus[4]	12,470	12
Nekton Lori[2]	60,550	34
Nekton Lori and Gelb[2]	244,820	136
Noah's Kingdom[4]	330	25
Passwells[1]	9,990	29
Quicko Nectar[4]	400	5
Rainbow Landing Lorikeet Nectar[4]	22,467	45
Roudybush 15 % Protein[4]	19,500	33
Roudybush 9 % Protein[4]	18,860	81
Sheps Lori dry[1]	167	1.7
Sheps Wet[6,(1)]	12,500 (333)	25 (1.8)
Wombaroo Nectar[5,(2)]	26,640 (28,740)	89 (27)

1. Independent laboratory analyses, Australia 2002
2. Author's laboratory analyses, 1999
3. Independent laboratory analyses, USA 2002
4. Laboratory analyses (Graffam, 1999)[23]
5. Manufacturer's data, 1999
6. Manufacturer's data, 2002

Table 1. Vitamin A and E content of commercial nectar mixes.

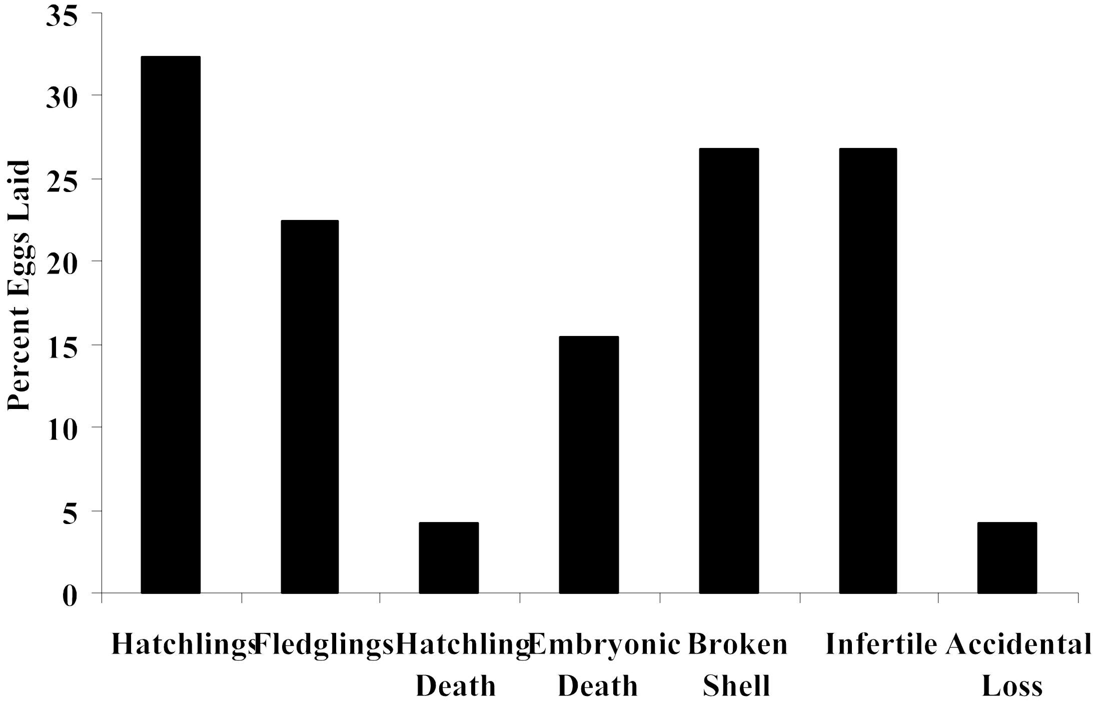

Figure 1. Productivity of Rainbow (*Trichoglossus haematodus*) and Red-collared Lorikeets (*Trichoglossus rubritorquis*) in captivity (n=71).

	Liver		Serum	
Species	**Vitamin A mg/kg**	**Vitamin E mg/kg**	**Vitamin A umol/L**	**Vitamin E umol/L**
Adult (wild) (n=2)	42.7 (29-56)	13.85 (14.1-13.6)		
Fledgling (wild) (PBFD)	26.4	4.3	0.49	11.8
Fledgling (wild)			0.1	17.6
6 months (wild) (n=3)	31.5 (19.6-37.8)	12.5 (10.8-13.4)	0	35.36
Adult (captive) (n=17)	1043.99 (14.7-1093)	44.81 (3.8-108.2)	0.44 (0.17-1.08)	52.83 (14.5-107.6)

Table 2. Vitamin A and E status of lorikeets

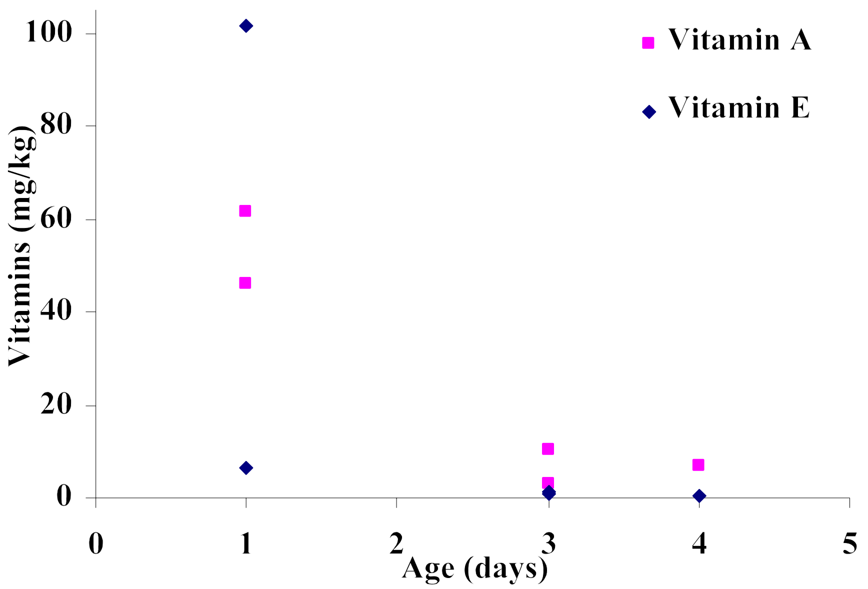

Figure 2. Decline of vitamin A and E in captive hatchlings.

		Liver		Serum	
Species	**Source**	**Vitamin A mg/kg**	**Vitamin E mg/kg**	**Vitamin A umol/L**	**Vitamin E umol/L**
adult	wild	29.2	14.1		
adult	wild	56.2	13.6		
fledgling (PBFD)	wild	26.4	4.3	0.49	11.8
fledgling	wild			0.1	17.6
6 months	wild	19.6	13.4	0	35.36
6 months	wild	37.8	13.4		
6 months	wild	37.2	10.8		

Appendix 1. Vitamin A and E status of wild lorikeets.

Species	**Source**	**Vitamin A mg/kg**	**Vitamin E mg/kg**
wild to captive diet (6 months)	wild	588.4	13.1
wild to captive diet (6 months)	wild	99.1	7.7
wild to captive diet (1 month)	wild	89.9	6.9

Appendix 2. Hepatic vitamin A status of wild birds transferred to captive diets.

		Liver		Serum	
Species	Source	Vitamin A mg/kg	Vitamin E mg/kg	Vitamin A umol/L	Vitamin E umol/L
1 day chick	captive	46.1	101.6		
1 day chick	captive	61.6	6.5		
3 week chick	captive	3.1	0.8		
3 week chick	captive	10.2	1.2		
4 week chick	captive	7	0.5		
4 month chick	captive			0.56	56.56
4 month chick	captive			0.56	42.33
4 month chick	captive			0.38	32.04

Appendix 3. Vitamin A and E status of captive chicks.

		Liver		Serum	
Species	Source	Vitamin A mg/kg	Vitamin E mg/kg	Vitamin A umol/L	Vitamin E umol/L
adult rainbow	captive	15	22		
adult rainbow	captive	98	4		
adult rainbow	captive	804	35		
adult rainbow	captive	813	34		
adult rainbow	captive	998	108		
adult rainbow	captive	1167	38		
adult rainbow	captive	1383	87		
adult rainbow	captive	4093	61		
adult rainbow	captive	26.6	16	1.08	15.86
adult rainbow	captive			0.17	29.16
adult rainbow	captive			0.21	46.95
adult rainbow	captive			0.21	14.54
adult rainbow	captive			0.31	73.58
adult rainbow	captive			0.35	76.23
adult rainbow	captive			0.45	39.22
adult rainbow	captive			0.59	107.62
adult rainbow	captive			0.63	72.35

Appendix 4. Vitamin A and E status of captive adult lorikeets.

		Liver		Serum	
Species	**Source**	**Vitamin A mg/kg**	**Vitamin E mg/kg**	**Vitamin A umol/L**	**Vitamin E umol/L**
Scaly-breasted (adult) *Trichoglossus chlorolepidotus*	captive			0.21	46.95
Scaly-breasted (adult) *Trichoglossus chlorolepidotus*	captive			0.28	10.89
Purple-crowned (adult) *Glossopsitta pusilla*	captive	823.9	10.4		
Lorikeet (15 day old yolk)	captive	29.9	382.8		
Sun Conure *Aratinga solstitialis*	captive	39.1	13.7		

Appendix 5. Vitamin A and E status of various species.