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Verh.ber. Erkrg. Zootiere (1995) 37.

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(Director: M.H. ROBINSON)

NUTRITION AND DISEASE IN ZOO ANIMALS

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Introduction

Quantitative nutritional requirements for domestic and laboratory animals have been established for most nutrients (NATIONAL RESEARCH COUNCIL, 1994, 1993, 1989a, 1989b, 1988, 1986, 1985a, 1985b, 1984, 1982, 1981). The determination of a requirement for a specific nutrient is a lengthy process and involves feeding diets with different concentrations of a particular nutrient. Animal growth, reproductive effort, and organ and tissue responses are then assessed for treatment effects. The need for animal nutrition research stems largely from the need for cost-effective production of milk, meat and eggs. While economic factors have driven research in domestic animal nutrition, no such factors exist to encourage the studies of nutritional requirements of zoo animals.

Over 46 nutrients have been identified as essential to the health of humans and other vertebrates. When these nutrients are not supplied in appropriate amounts and forms, decreased disease resistance and a variety of nutrient-specific pathological conditions may result (see ULLREY, 1993). Specific signs associated with the inadequate or inappropriate intake of individual nutri ents have been well studied in most domestic and laboratory animals (NATIONAL RESEARCH COUNCIL, 1987, 1980; see MACHLIN, 1991; see MERTZ, 1987), whereas this is not the case for zoo animals. Few reports exist in the literature which describe experimentally produced deficiencies or toxicities in non-domestic animals. Nutritionists, veterinarians and veterinary pathologists must use information gathered from domestic animal nutritional research to aid in the diagnosis of nutritional disorders in zoo animals. Reliance on clinical chemistry measures alone to diagnose nutritional problems in zoo animals is not without risk (ULLREY and ALLEN, 1993; RUBEN and BENNETT, 1981). In addition, many nutrient interrelationships exist which may complicate diagnoses of specific deficiencies or toxicities (WISE, 1980). Nutritional disorders of zoo animals are often reported as case or retrospective studies after animals present with signs. In this paper we present a review of the more common and well-documented nutritional problems in zoo animals.

Calcium, Phosphorus and Vitamin D

Many zoo animal diets contain feeds that are low in calcium or have poor calcium to phosphorus ratios. Muscle meats, organ meats, seeds, nuts, fruits and insects are all common foods used in the diets of mammals, birds and reptiles. Rickets, osteomalacia and nutritional secondary hyperparathyroidism can and do occur in animals that are offered cafeteria-style diets containing calcium and/or vitamin D-poor foods (DUNCAN et al., 1995; ULLREY et al., 1991; FOWLER, 1986; NICHOLS et al., 1983). Primates tend to consume the more palatable fruits and seeds to the exclusion of a well-balanced complete biscuit or canned diet. Attempts to fortify such mixed diets with calcium and vitamin D supplements are not always successful. TOMSON et al. (1978) have reported that lemurs (*Lemur catta* and *L. variegatus*) developed nutritional secondary hyperparathyroidism even though a calculated analysis of the diet revealed appropriate levels of calcium, phosphorus and vitamin D. Hyperphosphatemia, hypocalcemia, soft tissue mineralization, increased activity of alkaline phosphatase and poorly mineralized bone were all thought to result from dietary imbalances. One explanation was that the supplement, in powder form, adhered poorly to the foods. A second hypothesis was that some individuals may have made poor choices with respect to dietary items. Actual nutrient intakes are impossible to document in group feeding situations, particularly when mixed diets are offered.

Other accounts of nutrition-related bone diseases of primates have been reported (SNYDER et al., 1980). Although accurate dietary histories and feed consumption data are difficult to establish, these conditions usually result when voluntary selection of foods poor in calcium and/or vitamin D is allowed or when intake of an appropriate complete feed is not encouraged.

Laboratory investigations of calcium requirements of non-human primates have been reviewed (NATIONAL RESEARCH COUNCIL, 1978). It is suggested that the calcium requirement for Old World and New World primates is 0.5% of diet dry matter. However, most of the investigations of dietary requirements have been done with few genera, and some families are very poorly studied.

The issue of dietary vitamin D and exposure to ultraviolet light of the appropriate wavelengths is an area in need of much research. Birds, reptiles and amphibians are thought to require vitamin D_3, being unable to use vitamin D_2 effectively (HAY and WATSON, 1977). In addition, it has been reported that, in those species studied, New World primates use vitamin D_3 much more effectively than vitamin D_2 and appear to circulate levels of 25-OH-D_3 and 1,25$(OH)_2D_3$ that differ from those in other primates (GACAD et al., 1992; PORTMAN, 1970; HUNT et al., 1967; LEHNER et al., 1966). In the wild, since these primates consume primarily plant material, their requirement for vitamin D_3 is likely satisfied by exposure to sunlight. Many zoo primates in northern climates are housed entirely indoors thus receive little or no natural sunlight. Sunlight contains ultraviolet light (UVB; 280-320 nm) which is essential for the synthesis of vitamin D in the skin (WEBB and HOLICK, 1988). Most skylights and window materials will not allow transmission of UVB, unless specifically fabricated for this purpose (ULLREY et al., 1986). New World primates housed in zoos are usually fed diets of fruits and vegetables but also receive canned or dry commercial products, fortified with vitamin D_3, and manufactured especially for these species.

A number of cases of rachitic, juvenile primates (*Alouatta seniculus*, *Colobus guereza*, *Gorilla gorilla* and *Presbytis francoisi*) have been reported (MORRISEY et al., 1994; MEEHAN, 1993; ULLREY, 1986). It is suspected that there is insufficient transfer of mothers' dietary vitamin D into milk. When housed without access to UVB, juvenile primates seem particularly at risk, presumably since their intake of vitamin D-fortified foods is insufficient and since there is likely little vitamin D in maternal milk. Radiographically the long bones of the juvenile primates appeared bowed, and cortical thinning and widened epiphyseal plates were observed. In euthanized animals histological findings supported the diagnosis of rickets. It is suggested that husbandry practices for suckling primates should include direct dietary supplementation with vitamin D_3 or irradiation with natural sunlight.

Some species of lizards may require not only adequate dietary calcium and an appropriate calcium to phosphorus ratio, but may require exposure to UVB to maintain health and normal skeletal development (BERNARD et al., 1991; ALLEN, 1989; TOWNSEND and COLE, 1985). Exposure to artificial lamps which emit UVB radiation may be effective, but very little is presently known about the efficacy and safety of commercially available ultraviolet light sources. Studies with green iguana (*Iguana iguana*) have demonstrated that after consuming diets containing 2,000 IU vitamin D_3 and receiving no UVB for over one year, serum 25-OH-D_3 concentrations were extremely low. Animals presented with signs of muscle weakness and tetany, and radiographs revealed fractures and cortical thinning, yet necropsy results revealed extensive metastatic mineralization of soft tissues. In addition, serum chemistries were abnormal in most affected animals (R. MONTALI, pers. obs.). After exposure to UVB emitting lamps, these animals appeared to recover and had circulating levels of 25-OH-D_3 of over 400 ng 25-OH-D_3 per ml serum (BERNARD et al., 1991; O. OFTEDAL, R. MONTALI and M. ALLEN, unpublished data).
We have recent evidence that young varanid lizards may also benefit from a UVB source (ALLEN et al., 1994). Radiographs of Komodo dragon (*Varanus komodoensis*) hatchlings at two months of age revealed femur fractures in 9 of 13 animals and circulating 25-OH-D_3 concentrations of 13.8 ng/ml. After six hatchlings were exposed to UVB emitting bulbs for one month, 25-OH-D_3 concentra tions were 45.8 ng/ml compared to 17.1 ng/ml in the 7 hatchlings not exposed to UVB. While measurement of circulating vitamin D compounds may be helpful diagnostically, there is evidence that reptile species differ with respect to cutaneous precursor compounds (HOLICK et al., 1995; TIAN et al., 1994). A better understanding of the mechanisms whereby lizards and other animals synthesize vitamin D will help in our efforts to house and feed them appropriately.

ANDERSON and CAPEN (1976a, 1976b, 1976c) produced nutritional osteodystrophy in *Iguana iguana* by feeding diets low in calcium and phosphorus or low in calcium and adequate in phosphorus. The animals developed tetany, hypocalcemia, osteoporosis, osteomalacia and pathologic fractures. Histological and fine

structural changes in bone, parathyroid and ultimobranchial tissues were reported. Given our present knowledge of the green iguana and its responses when exposed, or not exposed, to UVB, some of the pathological changes reported by ANDERSON and CAPEN (1976b) may have been caused, in part, by the lack of exposure of the iguanas to UVB.

Insectivorous lizards will also develop signs of rickets and bone demineralization when fed unsupplemented insects. The calcium:phosphorus ratio in feed crickets and mealworm larvae, is approximately 1 : 10 (ALLEN and OFTEDAL, 1994; ALLEN, 1989). In studies with the nocturnal leopard gecko, *Eublepharis macularius*, softened bones, folding fractures in the long bones and significant demineralization occurred in the bones of animals receiving crickets unsupplemented with calcium. Bone ash, as a percent of dry, fat-free bone was 27.7%. By feeding crickets a diet containing 8% calcium, on a dry matter basis (DMB), the calcium level in the cricket could be increased from approximately 0.09% to 1.2% (DMB). Geckos receiving crickets supplemented in this way grew significantly better, had well-mineralized skeletons and bone ash values of 61.0% (ALLEN, 1989).

Some frogs and lizards possess what appear to be unique calcium storage organs. Calcium carbonate-filled sacs (chalk sacs), either in the neck region (endolymphatic) or running caudally along the vertebral column (paravertebral) can be seen radiographically and/or grossly. The function of these extra-cranial stores of calcium carbonate is thought to relate to acid-base regulation, especially in frogs. In lizards the calcium is believed to play a role in calcification of eggshell (ALLEN et al., 1993; SIMKISS, 1967; PILKINGTON and SIMKISS, 1966). The exact nature of the regulation of these structures needs further study. The pathological enlargement of these structures has also been reported in the gecko, *Phelsuma dubia dubia* (REICHENBACH-KLINKE, 1963).

Carnivorous species such as wolves, tigers and hawks, succumb to nutritional bone disease when all-meat rations are consumed (FOWLER, 1986; GRAHAM, 1976; VAN PELT, 1974; DIETRICH and VAN PELT, 1972; GORHAM et al., 1970). As with domestic animals, pathological bone disease in exotic animals, resulting from dietary imbal ances of calcium, phosphorus and vitamin D, manifests itself most readily in growing or lactating animals. The calcium : phosphorus ratio of muscle and organ meats range from 1 : 16 to 1 : 44 (NATIONAL RESEARCH COUNCIL, 1986; PENNINGTON and CHURCH, 1980). Historically, it was not unusual to find that carnivorous species kept either as pets or in zoological collections had been fed all-meat diets, although awareness of the problems associated with such diets is now common knowledge, at least among most zoo staff.

Sodium

Marine mammals maintained in fresh water may develop hyponatremia, apparently as a result of insufficient dietary sodium (GERACI, 1986; 1972). Animals may present with signs of a general central nervous system disorder and may appear listless and uncoordinated. Tremors, convulsions and death can result although early intervention, peritoneal or oral treatment with sodium chloride, may restore sodium levels to normal. GERACI (1972) suggests that dietary levels of 3 grams of sodium chloride per kilogram of fish be used for pinnipeds in fresh water pools although it has been suggested that this level may be excessive (BERNARD and ULLREY, 1989). Sodium concentrations in fish are reported to contain between 0.23 to 1.11% on a dry matter basis (BERNARD and ULLREY, 1989). The common practice of thawing fish in fresh water may result in considerable loss of sodium.

Iron

Excessive accumulation of iron in the livers of captive birds has been frequently reported (KINCAID and STOSKOPF, 1987; TAYLOR, 1984; WADSWORTH et al., 1983; GOSSELIN and KRAMER, 1983; RANDELL et al., 1981; LOWENSTINE and PETRAK, 1980). From a study of hepatic hemosiderosis in birds at the London Zoo (WADSWORTH et al., 1983) in which the livers of 531 birds were examined histologically, significant amounts of iron pigment were found in 37 birds, primarily in Ciconiiformes, Cuculiformes, Coraciiformes, and Passeriformes. The livers of all Psittaciformes examined appeared histologically normal. Iron accumulation and subsequent liver degeneration is believed to be the primary cause of death in birds of paradise (*Paradisaeidae*) at the Blijdorp Zoo (ASSINK and FRANKENHUIS, 1981) and a common cause of death in lesser Indian hill mynahs, *Gracula religiosa* (LOWENSTINE and PETRAK, 1980). GOSSELIN and KRAMER (1983) found that

detectable iron was present in hepatocytes of young Rothschild's mynahs, *Leucospar rothschildi*, as early as 10 days post hatch. Iron accumulation in hepatocytes appeared to increase with age when tissues from 20 birds, were compared. Major differences in pathogenicity of iron storage occur among species. In the hill mynahs the disease is usually progressive and fatal, whereas the Rothchilds appear to tolerate heavy accumulation of iron. While liver iron stores of free-ranging birds have not been widely investigated, ENSLEY and OSBORN (1993) reported hemosiderosis in a King Bird of Paradise *Cicinnurus reguis* and in other species captured in New Guinea.

The etiology of this condition is unclear. Faulty mechanisms of iron absorption, transport, storage or excretion may be responsible. Since only certain species of birds appear to be susceptible, genetic predisposition, as in human idiopathic hemochromatosis, has been suggested. Dietary factors such as excessive iron or low copper intakes have also been implicated as has the presence of tannins which may prevent or reduce absorption. In a retrospective study of 19 tanagers (7 species) it was reported that iron accumulation in liver, spleen and other organs was excessive and was believed to be the result of extremely high iron concentrations (1,230) in the commercial mynah diet (KINCAID and STOSKOPF, 1987). In an experiment with European starlings *Sturnus vulgaris* birds were fed, for 18 weeks, diets containing either 3,000 mg/kg or 148 mg/kg iron on a dry matter basis (DMB). Liver iron was significantly greater (5,929 mg/kg, DMB) in the high-iron group than in the low iron group (CRISSEY et al., 1993). DORRESTEIN et al. (1992) suggest that doves and pigeons be used as avian model species in testing various theories of iron storage disease in more rare species, although they found that vitamin C did not influence iron accumulation in pigeons, *Columba livia*.

Since absorption of iron from the intestinal tract depends on the form of iron, care should be exercised in evaluating dietary iron concentrations in animal feeds. Iron oxide, which is an unavailable form, may be added to animal feeds to impart a dark color, yet analytical results will reflect its presence. In the U.S., zoos commonly feed commercially produced diets with iron concentrations of less than 100 mg/kg dry matter for those birds considered at risk.

Iron storage disease in other species has also been reported. Iron-containing pigment was present in the tissues of 9 out of 10 rock hyraxes, *Procavia capensis*. Although death in most cases was attributed to infectious disease, in livers from older animals there was focal hepatocellular necrosis, portal fibrosis and cirrhosis. (REHG et al., 1980). In captive lemurs, Lemur variegatus variegatus, a species in which abnormal accumulation of iron in visceral organs is said to occur, GONZALES et al. (1984) were not able to demonstrate differences in iron retention between lemurs and rhesus monkeys, *Macaca mulatta*, a species in which the condition is not seen. Hemosiderosis is also seen in captive black rhinoceros and may be a sequela to hemolytic anemia syndrome, while wild populations do not appear affected (KOCH et al., 1992). The etiology of idiopathic hemolytic anemia in black rhinoceros remains unclear.

Iodine

BLACKMORE and COOPER (1982) reported iodine deficiency in certain seed-eating birds, such as parakeets (budgerigars) and suggest that the condition could be corrected by supplemental iodine added to the food or water. In tortoises goiter with hypothyroidism has been attributed to the feeding of vegetable material of the genus Brassica which contain thioglycoside goitrogens (FRYE, 1981; FRYE and DUTRA, 1974). Such cases are usually treated by the addition of iodine to the diet and the removal of the goitrogenic feeds. Adult-onset goiter, possibly caused by a familial defect in thyroglobulin, has been reported in bongo antelope, *Tragelaphus eurycerus* (SCHILLER et al., 1995; DOI et al., 1990).

Selenium and Vitamin E

Myopathies associated with low dietary vitamin E and / or low selenium have been reported in a number of zoo species (LIU et al., 1985; RUEDI et al., 1980; HELDSTAB and RUEDI, 1980; DECKER and McDERMID, 1977; GANDAL, 1968; FINLAYSON et al., 1971). Wallabies appear particularly susceptible to nutritional muscular dystrophy (HUME, 1986). KAKULAS (l961, 1963a) found that vitamin E administration reversed myopathy in the Rottnest quokka, *Setonix brachyurus*, but that selenium had no effect. He also reported that stress increased the requirement for vitamin E in the quokka (KAKULAS, 1963b). MacKENZIE and FLETCHER (1980) noted that myopathy in Goodfellow's tree kangaroos, *Dendrolagus goodfellow*, could be reversed with supplemental vitamin E. Grossly, cardiac and skeletal muscle is typically pale or streaked. Atrophic fibers, hyaline

degeneration, and dystrophic calcification may be seen histologically. It is felt that neonates or animals not well acclimated to confinement may be particularly susceptible (BRADY and ULLREY, 1976; HELDSTAB and RUEDI, 1980).

The specific dietary requirements of vitamin E and selenium for zoo ungulates have not been well-studied, although the white-tailed deer, *Odocoileus virginianus*, may represent a suitable model. In studies with does and fawns vitamin E requirements were found to be approximately 85 IU/kg when dietary selenium is 0.25 ppm. This level of selenium did not prevent myopathy when dietary vitamin E was 5 IU/kg (ULLREY et al., 1983). In the U.S. pelleted feeds for ungulates are formulated to contain 250 to 300 IU vitamin E per kg, DMB, with selenium concentrations of at least 0.3 mg/kg, DMB. Since selenium concentrations in forages depend on soil conditions, it is best to formulate pelleted feeds with enough selenium to offset hays that contain insufficient amounts.

A number of recent studies have focused on possible differences among species in their abilities to absorb various forms of vitamin E (SADLER et al., 1994; WALLACE et al., 1992; PAPAS et al., 1991; PAPAS et al., 1990; HIDIROGLOU et al., 1988a; 1988b). Most manufactured feeds include tocopheryl acetate as a source of vitamin E, since it is more stable than the alcohol form. Black rhinoceros, *Diceros bicornis*, and elephants, *Elephas maximus* and *Loxodonta africana*, appear to absorb fat-soluble and water-dispersible forms (D- or D,L-alpha tocopheryl acetate and D-alpha-tocopherol) of vitamin E poorly as compared to a water-soluble form of vitamin E, D,L-alpha-tocopheryl polyethylene glycol 1,000 succinate (TPGS) (PAPAS et al., 1991). TPGS is apparently well absorbed by horses (PAPAS et al., 1990) whereas in green iguanas, Iguana iguana, deer, *Odocoileus virginianus*, tapirs, *Tapirus sp.*, and pigs, TPGS is poorly absorbed (HOWARD et al., 1990; PAPAS et al., 1990). WALLACE et al. (1992) reported that a micellized form of vitamin E (D-alpha-tocopherol) produced increases in circulating alpha tocopherol levels in Asian and African elephants after 8 months of administration. Differences in the efficacy of oral vs. injected vitamin E have been noted in Swainson's hawks, *Buteo swainsonii* (MAINKA et al., 1994).

Captive fish-eating animals are also reported to suffer from nutritional myopathy which is thought to be related to the high levels of unsaturated fats found in many vertebrate fish (GERACI, 1980). Normally fish are good sources of the fat soluble vitamins. However improper packaging, transport, storage, and thawing of fish may result in rapid oxidation of the fish oils and destruction of vitamin E. Brown pelicans, *Pelecanus occidentalis*, were found to have extensive degeneration of cardiac and skeletal muscle and in Eastern white pelicans, *P. onocrotalus*, the pectoral muscles appeared primarily affected (CAMPBELL and MONTALI, 1980; MONTALI and WALLACE, 1986). In both of these cases the birds had been fed unsupplemented fish. Similar findings of muscle pathology were evident in a young California sea lion, *Zalophus californianus*, fed unsupplemented fish (CITINO et al., 1985). Steatitis and myodegeneration of cardiac and skeletal muscle have been reported in many species of fish-eating birds, both wild and captive (see LOWENSTINE, 1986; NICHOLS et al., 1986). Van FLEET and FERRANS (1977) have described ultrastructural changes in skeletal muscles of ducks maintained on vitamin E and selenium deficient diets. The effects of other elements on the induction of vitamin E and selenium deficiency signs in ducks have also been experimentally studied (VAN FLEET, 1982).

In studies of the effects of different forms of vitamin E, ENGLEHARDT (1977) reported that harp seal, *Phoca groenlandica*, plasma alpha-tocopherol was 30 times higher after oral administration of alpha-tocopherol than after dosing with the same amount of gamma-tocopherol. ENGLEHARDT and GERACI (1978), in experiments on the effects of vitamin E deficiency in harp seals maintained in fresh water, found that sodium requirements in the deficient seals were twice as high as those of the control animals. Vitamin E overdoses are generally considered rare although NICHOLS (1989) reported a vitamin K responsive coagulopathy in pink-backed pelicans, *Pelecanus rufescens*, that were apparently receiving excessive vitamin E supplementation.

In reptiles, steatitis and necrotic adipose tissue has been reported in animals consuming fish unsupplemented with vitamin E. In crocodilians accumulations of yellow and brown staining ceroid in body fat stores were thought to be due to a diet high in unsaturated fats (FRYE and SCHELLING, 1973). Snakes fed obese laboratory rats may also develop steatitis (FRYE, 1981). Steatitis has also been described in mink, cats and pigs (MASON and HARTSOUGH, 1951; GASKELL et al., 1975; GORHAM et al., 1951). In farmed American alligators, *Alligator mississippiensis*, LARSEN et al. (1983) reported a high prevalence of granulomatous steatitis and fat necrosis. Although the animals appeared healthy, tissues examined at slaughter revealed yellow-brown lesions in the fat tissues throughout the body, with accumulations in the tail being particularly pronounced. The dietary history revealed that the alligators had been fed fish and fish scraps for a period of over ten years.

Primarily due to the economic importance of alligator farming, the effects of diet on vitamin E and selenium levels in alligator plasma have been investigated (LANCE et al., 1983). Animals fed whole fish, Atlantic croaker, *Micropogon undulatus*, had higher plasma selenium and lower plasma vitamin E levels than did alli gatorsfed ground nutria, *Myocastor coypus*, or than did wild alligators. There is increasing interest in the role that vitamin E might play in the successful reproduction of commercially raised alligators.

Vitamin A

Vitamin A deficiencies may result from the feeding of unsupplemented muscle meat to zoo carnivores. Exotic felines are believed to be similar to domestic cats in that conversion from beta-carotene to vitamin A cannot occur. Alopecia, anorexia, follicular hyperkeratosis and general unthriftiness has been observed in cats fed insufficient vitamin A (SCOTT, 1968). Neurological signs have been associated with vitamin A deficiency in lions, *Panthera leo* (O'SULLIVAN et al., 1977).
RODAHL and MOORE (1943) found extremely high vitamin A levels (13,000-18,000 IU/g) in polar bear (*Thalarctos maritimus*) livers. Hypervitaminosis A has been reported in captive mink fed whale liver (FRIEND and CRAMPTON, 1961). Signs included impaired reproduction, hemorrhage, weight loss, and bone fractures. It has been suggested that a higher requirement for vitamin A may exist in certain carnivores (HEYWOOD, 1967). FOSTER (1981) has used vitamin A therapeutically (200,000 IU vitamin A per kilogram of food) in the diets of polar bears for the treatment of a persistent dermatitis that was unresponsive to other measures. It is not known whether such massive doses will have adverse, long-term effects. Excessive vitamin A is reported to result in testicular degeneration in domestic cats (LADDS, 1985). Excessive dietary vitamin A has been suggested as one of a number of possible causes of veno-occlusive disease in snow leopards, *Panthera uncia*, and cheetahs, *Acinonyx jubatus* (MUNSON and WORLEY, 1991; GOSSELIN et al., 1989).

Vitamin A deficiency has been diagnosed in a number of different bird species (ALTMAN, 1976; LOWENSTINE, 1982; ZWART et al., 1979). Overall loss of integrity to epithelial tissues, including epithelial hyperkeratosis and epithelial squamous metaplasia, especially in respiratory and urogenital tracts, characterizes the deficiency signs in birds (LOWENSTINE, 1986). The condition is most likely to be seen in seed-eating birds since seeds, many grains, and nuts are particularly poor sources of the vitamin (ULLREY et al., 1991; PENNINGTON and CHURCH, 1980; PAUL and SOUTHGATE, 1978).

In reptiles, problems related to epithelial tissues characterize vitamin A deficiency signs (JACOBSON, 1984). In aquatic turtles the adnexal structures of the eye appear abnormal and horny mouth parts are typically overgrown. ELKAN and ZWART (1967) have described histopathological changes in the eye of turtles that are typical of vitamin A deficiency in other species, including hyperkeratosis and squamous metaplasia in lacrimal glands.

B Vitamins

Deficiencies of certain of the B vitamins have occurred in captive exotic animals. The need for supplemental thiamin when feeding fish has been well-documented. Deficiencies have been documented or suspected in pinnipeds (GERACI, 1972; RIGDON and DRAGER, 1955), cetaceans (WHITE, 1970), raptors (WARD, 1971) and gulls (GILMAN, 1978). Thiaminases present in the viscera and trimmings from many fish species will destroy thiamin present in the fish (HARRIS, 1951; GREEN et al., 1942). In zoos it is common to use thiamin supplementation when feeding whole fish, especially when fish represent the entire diet for an animal. SCOTT et al. (1976) state that thiaminases are also found in the heart and spleen of warm-blooded vertebrates. Thiamin deficiency in a peregrine falcon, *Falco peregrinus*, was produced from a diet mainly of domestic chicks but supplemented with muscle, gizzard and heart (WARD, 1971). Deficiency signs included vertigo, seizures and opisthotonos which gradually disappeared after intramuscular and oral administration of thiamin was initiated.

A case of suspected riboflavin deficiency was reported in a debilitated, wild golden eagle, *Aquila chrysaetos* (STAUBER, 1973). The animal was emaciated, listless, and appeared paralyzed. Because it also exhibited curled-toe paralysis, riboflavin deficiency was suspected. The bird responded rapidly to oral administration of vitamin B complex which included 40 mg of riboflavin. KEYMER (1972) also reports the occurrence of B vitamin deficiencies in other raptors.

It had been suggested that lesions in snakes, resembling necrotic enterohepatitis in swine, responded to supplemental niacin (SAUER et al., 1970). BARTKIEWICZ et al. (1982) conducted a study on niacin requirements of the bull snake, *Pituophis melanoleucus sayi*, and found that snakes receiving a niacin-free diet exhibited weight gains equal to those of snakes fed diets with 80 milligrams niacin per kilogram. No signs of deficiency or gastroin testinal lesions were noted in any of the snakes at necropsy. It was concluded that either 132 days was an insufficient length of time to produce deficiency signs or that the snakes had the ability to convert tryptophan to niacin, as is the case in all other species studied, with the exception of the cat.

Vitamin C

The ability of certain species to synthesize vitamin C has been reviewed by ROY and GUHA (1958), ELLIOT, YESS and HEGSTED (1966), CHAUDURI and CHATTERJEE (1969) and CHATTERJEE (1973). It is believed that synthesis of ascorbic acid in the kidney occurs in amphibians, reptiles and most birds. Certain species of birds must rely on dietary sources since they lack the ability to synthesize ascorbic acid either in the kidney or liver. The mammals that have the capability apparently synthesize ascorbic acid in the liver. Species differences become important when formulating diets for zoo animals. The ascorbic acid requirement of common marmosets, *Callithrix jacchus*, was found to be higher than that of humans (FLURER et al., 1987). Primates fed only commercially produced biscuits must rely on the vitamin C present in the product which is usually applied on the outside of the biscuit after manufacture. Since vitamin C is labile, rapid turnover of the stored product must occur. Usually zoo diets for primates and most birds include fruits and vegetables, many of which provide ample supplies of the vitamin.

HANSSEN et al. (1979) has described vitamin C deficiency in willow ptarmigan, *Lagopus lagopus lagopus*, chicks fed diets containing 265 mg per kilogram of diet, even though the adults have the ability to synthesize the vitamin in the kidney. Adult willow ptarmigan in captivity do not show signs of deficiency when fed vitamin C-free diets. The chick requirement, probably elevated due to the stresses of growth and development, is felt to be 125-150 mg per kilogram of body weight and reported to be greater than the amount the bird can synthesize. It has been suggested that the ulcerative stomatitis seen in snakes and lizards may be associated with a vitamin C deficiency (FRYE, 1981), although studies with garter snakes, *Thamnophis sp.*, given supplemental vitamin C, revealed that tissue levels and body stores remained stable while synthesis of vitamin C was reduced (VOSBURGH et al., 1982). Vitamin C synthetic capability has been reported in a number of reptiles (BIRNEY et al., 1980; CHATTERJEE et al., 1975).

Additional Problems

Gout

There are other conditions that have been observed in captive exotic animals that are poorly understood. Gout, both visceral and articular, has been described in reptiles and birds and is commonly reported as the cause of death (LOWENSTINE, 1986; JACOBSON, 1984; KNOX, 1980; APPLEBY and SILLER, 1960). Consumption of excess protein, water deprivation, vitamin A deficiency and impaired renal function have all been suggested as causative factors. In birds it is commonly believed that a diet high in protein will result in gout. Nitrogen consumed in excess of requirements will obviously result in the need for excretion, and in birds and some reptiles, the excretory product is uric acid. When poor quality protein (unbalanced amino acids) is fed or when tissue is catabolized for energy, uric acid excretion will increase. In some bird species, excessive feeding of diets with unbalanced amino acid profiles (seeds) may predispose to gout (see ULLREY et al., 1991). In reptiles gout is associated with increased circulating uric acid levels although pre- and post-feeding levels of serum uric acid are distinctly different within a species with post-feeding levels significantly greater than pre-feeding levels (MAIXNER et al., 1987). These authors caution that time of bleeding relative to feeding and extrapolation among species as to normal circulating uric acid levels may confound a diagnosis. Visceral gout has been produced in captive alligators as a result of intraperitoneal injections of D-serine (COULSON and HERNANDEZ, 1964).

Cystinuria

Maned wolves, *Chrysocyon brachyurus*, are particularly susceptible to urinary cystine stone accumulation. The extent to which cystine excretion can be moderated in maned wolves by dietary factors, such as reduced dietary methionine and cystine loads, remains to be demonstrated. We are currently conducting long-term

feeding experiments with maned wolves to test the effect of low protein (cystine/methionine) diets on urinary cystine excretion. Elevated urinary cystine levels are also seen in wild maned wolves. Excessive urinary cystine levels were found In 34 out of 42 captive and wild maned wolves in South America (BOVEE et al., 1981). Four of the wolves had cystine stones and three died due to the obstructions. In renal clearance studies per formed on five of the wolves there were abnormalities (dibasic aminoaciduria) in all animals. The condition is attributable to a renal defect for resorption of cystine and dibasic amino acids. The maned wolf is uncommon in the wild and infrequently exhibited in zoos. The exceptionally high incidence of this disease coupled with its debilitating and fatal effects, and the possibility that it is of genetic origin, may seriously impair efforts to successfully propagate the species.

Urolithiasis

Urolithiasis is a persistent problem among some zoo carnivores, notably otters (*Mustelidae*) and its occurrence has been also been noted in mink and foxes (CALLE and ROBINSON, 1985; LONG, 1985; KARESH, 1983; KEYMER et al., 1981; GORHAM et al., 1972). Uroliths and calculi may be comprised of calcium oxalates or urates or magnesium ammonium phosphates. In a retrospective study of Asian small clawed otters, *Aonyx cinerea*, held in 16 North American zoos, renal and cystic calculi were found in 66% and 23% of the radiographed or necropsied animals (CALLE, 1988). Although the condition is fairly well studied in domestic felids, the etiology is still poorly understood (LEWIS et al., 1978; NATIONAL RESEARCH COUNCIL, 1986) and dietary factors have been suggested as causative (CALLE, 1988; KEYMER et al., 1981)

Herbivorous Primates

Some herbivorous primates have gastrointestinal tracts more similar to a ruminant than to a monkey (BAUCHOP and MARTUCI, 1968). Some species are considered to be entirely folivorous and have adapted to natural diets relatively high in fiber (MILTON, 1984; see OFTEDAL, 1991). Some species with rumen-like foreguts (*Presbytis* spp., *Pygathrix* spp., *Nasalis* spp.) are considered particularly difficult to maintain in zoos. The excessive use of foods with rapidly fermentable carbohydrates (e.g., monkey biscuits, cultivated fruits and vegetables) has resulted in cases of bloat and other serious gastrointestinal disturbances (HILL, 1964). Many zoos that exhibit these specialized primates attempt to feed natural plant material as a regular part of the diet. While this practice is generally considered effective in promoting foregut fermentation, there is evidence that, unless particular care is paid to the type and maturity of the browse material, digestive disturbances can result. ENSLEY et al. (1982) have reported that certain types of acacia (*Acacia saligna, A. longifolia*) may be inappropriate as food for hanuman langurs, *Presbytis entellus*, and douc langurs, *Pygathrix namaeus*. They found that phytobezoars, largely comprised of lignin, formed and occluded the digestive tracts of animals offered acacia as a dietary supplement. The lignin content of the browse was found to be 25% on a dry matter basis. Dietary fiber levels appropriate for leaf-eating primates are not known. Work by CRISSEY and EDWARDS (1989) and M. EDWARDS (San Diego Zoo, unpublished observations) suggest that the use of a high fiber manufactured primate biscuit may be useful in the captive management of leaf-eating primates.

Taurine Requirement in Cats

Signs associated with taurine deficiency in cats include central retinal degeneration and dilated cardiomyopathy (MORRIS et al., 1990; PION et al., 1987). Studies with domestic cats have demonstrated that dietary taurine is an essential nutrient. A level of 400 mg/kg dry matter sustains normal growth in kittens (NATIONAL RESEARCH COUNCIL, 1986). However, a recent report of retinopathy in leopard cats, *Felis bengalensis*, suggested that the use of a canned feline diet resulted in low circulating taurine levels and retinal degeneration, despite the high concentration of taurine in the diet (1,500 mg/kg dry matter) (HOWARD et al., 1987). In subsequent studies with domestic cats HICKMAN et al. (1990) showed that a heat-processed diet results in lower circulating taurine while the same diet, preserved by freezing, resulted in normal circulating levels of taurine. Heat-processed diets were later shown to result in a greater intestinal losses of taurine although the exact mechanisms have not been defined (HICKMAN et al., 1992).

Additional Problems Related to Feeding

Bacterial hazards have been associated with the exclusive feeding of raw, meat-based diets. Septicemia and deaths in carnivorous small mammals have been linked to *Streptococcus zooepidemicus* cultured from horse-meat based diets (SCHILLER et al., 1989; NICHOLS, 1986; SHAW et al., 1984). Callitrichid hepatitis in tamarins and marmosets has been linked to the feeding of new-born mice that were apparently infected with

lymphocytic choriomeningitis virus (MONTALI et al., 1993). In Black-footed ferrets *Clostridium perfringens* Type A gastroenteritis was associated with abdominal distension syndrome due to overfeeding (SCHULMAN et al., 1993).
Soft diets which require little chewing or tearing may contribute to excessive plaque and calculus formation. Gingivitis, loose teeth, abscesses in the oral cavity and bacteremia are likely sequellae (FAGAN, 1980). It has been suggested that recurrent sepsis resulting from chronic dental infections may contribute to or cause compromised renal function, liver abscesses or endocarditis (ROBINSON, 1986). Lumpy jaw, or necrobacillosis, is noted to be the most prominent oral disease in wallabies and kangaroos and is characterized by facial and mandibular swelling, periodontal infection and osteomyelitis (ROBINSON, 1986). Coarse, sharp feed items may cause initial trauma to the oral tissues allowing bacteria (*Corynebacterium pyogenes* and / or *Fusobacterium necrophorum*) to invade (HUME, 1986). In Australia, the feeding of dry grasses and fibrous tree bark seems to result in a lower incidence of the disorder (HUME, 1986). Because of the high incidence of lumpy jaw in macropods, the use of dried forages for kangaroos or wallabys in U.S. zoos is not common. However, since macropods rely on fermentation of plant fiber by microbes in the foregut, they should recieve sufficient dietary fiber to promote normal gut function. Fiber can be provided by offering second/third cutting grass hay, which will have a much greater leaf:stem ratio than first cut grass hay, high fiber pellets or appropriate browse.
Bones with muscle attached are often used to supplement the diets of lions, tigers and other carnivores. Cats spend considerable time and effort in tearing the meat and tendons from bone. It is felt that these practices may help abrade the teeth. Some zoo canids may receive dry, extruded dog food which can provide some stimulation to the teeth and gums. The effects of diet consistency have been quantitatively measured in wolves (*Canis lupus*) fed a dry, extruded dog food or soft meat-based diets. Wolves fed the dry food had approximately half the plaque than did wolves fed a soft diet (VOSBURGH et al., 1982). In a study with Amur tigers (*Panthera tigris altaica*) the effects of using a dietary supplement of beef femur bones, with some muscle still attached, were evaluated. Tigers were maintained on a soft, meat-based diet and received bones either once or twice per week. When bones were offered only one day a week, the tooth surface areas covered by plaque and calculus were not significantly different from those of the control group which received the soft diet only. Tigers receiving bones twice weekly had significantly less maxillary tooth area covered by plaque and calculus than did those cats in the control group. Gingival sulcus depths adjacent to maxillary teeth were also significantly decreased in the group receiving bones as compared to controls (HABERSTROH et al., 1984).

Summary

Nutrition and disease in zoo animals

A review of some of the commonly seen, nutritional disorders of zoo animals is reported. Many clinical signs and lesions associated with specific nutrient deficiencies or imbalances in domestic species are paralleled in zoo animals, although diagnoses must often be based on post-mortem examinations. Since there are only rare opportunities to study experimentally induced nutritional problems with large sample sizes, zoo animal health professionals frequently must rely on blood parameters, radiographic or necropsy evidence and/or tissue biopsies. For these reasons, the diagnosis and resultant treatment for a suspected nutritional problem should be made not only on the basis of these common diagnostic tools, but should include an assessment of dietary history, nutrient intake measurements with a computer analysis of the diet, and husbandry conditions.

Zusammenfassung

Ernährung und Erkrankung von Zootieren

Die Arbeit gibt einen Überblick über einige häufig auftretende Emährungsstörungen von Zootieren. Viele klinische Anzeichen und krankhafte Veränderungen, die mit speziellen Nährstoffdefiziten oder -unausgewogenheiten bei Haustierarten in Verbindung gebracht werden, treten bei Zootieren ebenfalls auf, obwohl die Diagnose sich oft auf post mortem Untersuchungen stützen muß. Da es nur wenige Gelegenheiten gibt, experimentell induzierte Emährungsprobleme mit großen Probenmengen zu untersuchen, müssen sich Zootierexperten ständig auf Blutparameter, radiologische oder Sektionsnachweise und / oder Gewebebiopsien verlassen. Aus diesen Gründen sollten Diagnose und anschließende Behandlung eines vermuteten Emährungsproblems sich nicht nur auf diese üblichen diagnostischen Mittel stützen, sondern eine Anamnese der Emährung, Messung der Futteraufnahmemengen mittels Computeranalyse und Haltungsbedingungen einschließen.

Résumé

L'alimentation et les maladies des animaux gardés

L'exposé a établi toute une liste de troubles végétatifs alimentaires souvent observés chez des animaux gardés au zoo. Un nombre considérable de signes cliniques et d'altérations liés à des déficits nutritionnels ou à un déséquilibre alimentaire chez des animaux domestiques sont également observés chez des animaux gardés quoique le diagnostic doit se baser très souvent sur des résultats obtenus post mortem. Puisqu'il n'y a pas beaucoup d'occasions pour étudier expérimentellement des problèmes nutritionnels sur la base de grandes quantités de prélèvements, les experts en la matière des jardins zoologiques sont obligés de se fier en permanence aux paramètres sanguins, aux résultats des examens radiologiques et des nécropsies et/ou des biopsies de tissus. Voilà pourquoi aussi bien la diagnose que le traitement consécutif d'un problème alimentaire supposé ne devrait pas se fonder exclusivement sur ces méthodes diagnostiques connues mais devrait comprendre entre autres, une anamnèse de l'alimentation, une analyse par ordinateur des quantités avalées et une étude des conditions dans lesquelles sont gardés les animaux.

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