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VITAMIN E DEFICIENCY IN CAPTIVE ELEPHANTS & RHINOCEROSES

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Vitamin E deficiency syndrome has been well established in domestic animals since the early 1900s and this vitamin has been recognised as a health problem in captive exotic species for more than 50 years (Dierenfeld, 1989). Little information is available concerning the levels of vitamin E in wild animals in captivity, their requirements or its degree of physiological importance. In domestic animals, deficiency of this vitamin has been associated with myodegenerative disorder and cardiomyopathy (Blaxter,1962; Vanvleet & Ruth, 1977), as well as impaired reproductive capacity (Trinder et al.,1986) and nervous system disorders (Muller et al; 1983, Nelson 1980). Vitamin E may also play a role in the immune response to infection and in Prostaglandin synthesis (Lipinski and Machlin, 1981,Stuart,1982).

But the pathologies in wild animals are various and there is great species difference in utilisation of dietary vitamin E, its effects and pathology. There are few reports of vit E deficiency manifestation of wild species which include cardiomyopathy in Camels (Finlayson et al.,1971; and Nyla Liu et al., 1982) and myodegenerative disorders in ruminants (Wallach, 1970) Pathology consistent with Vit E deficiency has aslo been reported in cases of capture myopathy in elephant (Dierenfeld 1989). Actual cause of Vit E deficiency in captive wild animals are various and there is great variation in physiology species individuality of vit E absorption as well as diet supplied to them. It has been found that even prolonged vitamin E supplement to animals can show low level of plasma tocopherol. There are several forms of vitamin E available as feed supplements for animals. But it is very important to be sure which forms of vitamin E available as feed supplementation with D,L α tocopheryl acetate in good quality has been proved to be unsuccessful to raise the tocopherol level of plasma in elephants and black rhinoceroses (Dierenfeld & Dolensek, 1988; Lewis and Kirkwood 1990).

In this context, the etiology and pathophysiology of Vit E deficiency in elephants and rhinoceroses are discussed. A trial has also been made towards supplementation of vitamin E to Asian elephant.

MATERIALS & METHODS FOR VITAMIN E ESTIMATION

Vitamin E has become synonymous with the most active form α tocopherol and its esters in most cases. But in natural foods and feeds, addition to alpha, beta, gamma and delta tocopherol, are found to have some Vitamin E activity.

The vitamin E is usually available in the form of D,L α tocopheryl acetate (Iμ/g), D α tocopheryl polyethylene glycol succinate TPGS, 20% solution in water, 77.4 Iμ/ml, Dα tocopheryl oil (Iμ/g) D α tocopheryl acetate in oil or water dispersible solid. The D,L forms of vitamin E are mixture of eight stereoisomer and are produced synthetically and Dα tocopheryl is a single stereoisomer derived from natural sources and modified chemically to produce its esters. The TPGS is prepared by esterification of polyethylene glycol 1000 to the acid group to the Dα tocopheryl succinate This waxy solid can be dissolved in water to form up to 20% solution (Weight basis).

Blood samples are taken usually by catheterizing an accessible ear vein and aspirating in a disposable syringe. The samples are then transferrerd into a tube and sent to veterinary investigation service of the Ministry of Agriculture, Fisheries & Food for plasma tocopheryl estimation. The blood samples are collected for plasma α tocopheryl because circulatory blood concentration reflect the amount absorbed in the gut (Machlin, 1980). But tocopherol level in the liver is a more accurate indicator of vit E status than in the plasma though it is very difficult to get sample in case of live and especially large animals (Blood et al, 1983). Also Vit E levels in tissues other than blood have not been studied in most zoo species. It has also been established that no changes occur on long storage of plasma at temperature below -70°C.

FACTORS CAUSING VITAMIN E DEFICIENCY

Primary vitamin E deficiency occurs most commonly when animals are fed on inferior quality hay or straw and on root crops. Cereal grains, green pasture and well cured fresh hay contain adequate amounts of vitamin. Higher serum tocopherol has been seen in new born calves born from an individual fed on grass silage than in those born from cows fed the same grass as hay and also from animals fed on high moisture grain which was treated with propionic acid as a method of inexpensive storage and for protection from fungal infection (Allen 1975). Marked decrease of the vitamin E concentration has also been observed in case of acid treated grains with an increase in the levels of peroxides of fat which is consistent with a loss of naturally occuring antioxidant like tocopherol.

Though vit E deficiency of farm animals is due to vit E deficient diet, the cause of the deficiency in captive wild animals differs to a great extent. It has been found that vit E supplement in the form of α tocopheryl acetate fails to meet the vit E demand in some animals like rhinoceroses and elephants. The form in which vit E supplement is to be given differs in different species. Hidiroglou *et al*, (1988) compared the natural and synthetic form of α tocopherol in cattle. While all experimental cattle received 1000 Iμ/day and similar circulating levels would be expected but the circulating levels were higher with natural form. In addition the α tocopherl form produced higher increase over acetate form.

In practical feeding, the non alpha tocopherols are assigned zero Iμ value and are not included in feed supplement because of their low activity in rat fetal resorption and other bioassay in comparision to α tocopherol. In vitro gamma tocopherol is found to be more active than alpha and there is evidence that

the gamma is absorbed that may have a sparing effect on the alpha (Machlin,1980; Traber and kayden, 1989).

Many physiological factors are also responsible for vit E absorption and that is why Iμ value does not predict the absorption of various forms by different species. It is important to note that in general, vit E absorption is poor ranging from 20-40% (Papas *et al.*, 1989). A variety of factors such as billary and pancreatic secretions influence the vit E absorption (Sokol *et al.*,1987). This may be a factor in elephants and rhinoceroses especially as they have no gall bladder (Reuther, 1977) and absence of bile acid which may result in incomplete emulsification.

The dietary items which are rich in unsaturated fatty acids such as cod liver oil, other fish oil, fishmeal, used as a protein concentrate, linseed, soyabean, corn oil have been found to cause destruction of vitamin and thus increasing the dietary requirement of vitamin E.

Micro-organisms also play an important role in vitamin E absorption. These micro-organisms in the gut of animals may cause degradation of this vitamin before absorption. Alderson *et al* .(1971) reported a high degree of vit E in the rumen increasing with large concentrate feeding.

Inadequate dietary fat may limit vitamin E absorption, thereby increasing requirements.

NORMAL α TOCOPHEROL LEVEL IN BLOOD

Circulation level of vitamin E in wild black rhinoceros has been found to be 0.77+/- 0.05 μg/ml, much higher than in captive rhinoceroses which was found to be 0.18+/- 0.03 μg/ml. However there is no information available about vitamin E status of blood in Indian Rhino. The black and Indian rhinoceroses are more prone to this deficiency. The level of α tocopherol observed however for captive elephants and black rhinoceroses were significantly lower than those reported for other species in captivity (Brush & Anderson, 1986, Dierenfeld 1989).

In these species, the deficiency seemed to be caused by physiological factors in fat metabolism. Mean plasma level of α tocopherol in healthy animals are highly dependent upon blood lipid levels. It has been seen that shrubs preferred by black rhino usually contain 25% crude fat as well as higher tocopherol content than grasses or dried forages and pellets commonly fed captive animals. Vitamin E concentration in tissues also may have an important role in the deficiency of this vitamin. The circulating level of vit E in African elephant has been found to be .80 μg per ml. The mean serum or plasma level of α tocopherol and cholesterol are presented in Table-1. There are few reports available in those, where vitamin E deficiency can cause death (Papas *et al.*, 1991) .

CLINICAL SIGNS AND PATHOLOGY

Though neurologic abnormalities i.e. degenerative myoencephalopathy are very common clinical manifestation of vit E deficiency in horse, it is not commonly observed in elephant or rhinoceroses. Clinical and pathological findings have been reported in captive zebras (Montable *et al.* 1974) and Przewalski horse (Liu *et al.* 1983). Skeletal muscle degeneration due to vitamin E deficiency has been reported in elephant due to vitamin E deficiency but muscle tissue α tocopherol level were not reported (Papas *et al.*,1989, 1991). The changes noted included acute and chronic severe rhabdomyolysis of skeletal muscle in the hind leg and mild acute degeneration of heart muscles.

Cardiomyopathy resembling "Mulbery heart disease" of swine has been reported in Asian elephant by Dierenfeld & Dolensek (1988). The vitamin E concentration in blood was found 0.26 μg/ml and it was 0.64 μg/g in the liver. The liver concentration was considered below normal (10 μ g/g % as compared to equine liver). The pathology included micro-angiopathy, edema, liver necrosis and steatitis.

The microscopic and histological appearance of the muscle lesion is quite constant but their distribution varies widely in different animals. Affected group of skeletal muscles are always bilaterally symmetrical. In skeletal muscle the diaphragm, there are localised white or graysh areas of degeneration which have an appearance of fish flesh. In cases of myocardial involvement, white areas of degenaration are usually visible under endocardium.

TRIALS ON VITAMIN E

The etiology of vitamin E deficiency as discussed earlier are varied and several efforts have been made to supplement an adequate quantity of tocopherol to raise the normal blood levels in elephant and rhino especially. Black and Indian rhinoceroses are browsers and grazers respectively. For this reason, vitamin E deficiency is common in these animals in captivity, though elephant are also equally affected.

In London zoo, a trial was made to supplement vit E in the form of α tocopherol acetate with an amount of 12500 IU/day, apart from normal diet consisting of vegetables, concentrate and forage. After eight months supplementation, no significant increase in plasma vit E was observed. (Lewis & Kirkwood, 1990).

In the case of elephant (both Asian and African) the same results were obtained after supplementation with D L α tocopherol acetate at 1.5-2.0 IU/Kg bodyweight for 9 months. The D α tocopherol acetate in oil form at a high dose also failed to increase α tocopherol level in blood (Papas et al., 1991)

It has been shown that instead of α tocopherol acetate, D α tocopheryl poly ethylene glycol 1000 succinate (TPGS) is to be given then α tocopherol level of plasma can easily be raised.

In a study of TPGS supplementation to black rhinoceroses it was observed that plasma α tocopherol was eleveated after 13 days of supplementation to 3.9. mg/lit (Kirkwood *et al.*, 1991). In another study it has been seen that TPGS supplementation elevated the α tocopherol level in plasma within a very short time in black rhino and captive elephant. In elephant TPGS increased the plasma α tocopherol level to 0.4 μg/ml immediately after supplementation and did not return to base line after withdrawal of the supplementation. From research

work in Whipsnade Wild Animal Park, two Asian elephant were provided with 13500 IU TPGS which showed very good reflection of α tocopherol level in blood 3.75 µg/lit. The supplementation was continued for six months. The initial elevation of α tocopherol level was followed by decrement and maintained a standard level 2-3 µg/lit after 60 days. In contrast to fat soluble and water dispersible forms, TPGS does not require biliary secretion, ie bile acid for solubilisation and absorption (Sokol *et al.*, 1987, Traber *et.al.*, 1986).

CONCLUSION

1. There are great differences in the absorption and utilisation of vitamin E in different species. So selection of vit E supplementation should be done on the basis of bioavailability to animal species.

2. Elephants and black rhinoceroses absorb fat soluble forms of D L α tocopherol acetate, D α tocopherol poorly. In contrast TPGS is readily absorbed.

3. These findings explain the persistently low circulating blood α tocopherol in captive elephant and black rhino despite of dietary supplementation of D L α tocopherol acetate.

4. Some evidence exists that hypovitaminosis E in elephants may result in myodegeneration of both skeletal and heart muscles and is suspected in hemolytic anemia in black rhino.

5. Research is needed to determine if similar peculiarities of vit E metabolism exist in the Indian rhino.

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Table -1

Mean serum or palsma tocopherol (E) and cholesterol in elephant rhinoceros.

A	Elephant	Tocopherol µg/ml	Cholesterol mg/ml
1	Captive\ (Asian&African)	0.4	0.62
2	Free ranging(African)	0.8	0.69
B	Rhinoceros		
1	Captive (3 species)	0.4	0.79
2	Free ranging (black rhino)	0.6	