(d) measurement of urinary PdG appears to provide a useful method for detecting mid-late pregnancy in rhinos. Further work is needed to establish tests for early pregnancy.

3. Monitoring of estrus cycles and ovulation.
   In contrast to the Indian rhino, attempts to monitor the estrus cycle in black and white rhinos by measurement of urinary estrogen metaboiltes and pregnanediol-3x-glucuronide have so far proved unsuccessful. Other methods need to be investigated.
4. New assay methodology.
   A new, simple microtitre plate ELISA (enzyme assay) for urinary pregnanediol-3x-glucuronide has been developed and validated for all three species of rhino.

UPDATE ON DEVELOPMENT AND APPLICATION OF REPRODUCTIVE TECHNOLOGY TO RHINOS

As head of the research team of the Cincinnati Wildlife Research Federation (CWRF) which is a combined effort of the Cincinnati Zoo, Kings island Wild Animal Habitat and the University of Cincinnati College of Medicine, Dr. Betsy Dresser reported on the development and application of reproductive technologies to rhinos. At the Cincinnati Zoo , there are two breeding pairs of black rhinos and they have produced 13 offspring to date. At Kings Island, there is a group of white rhinos, and four nonpregnant cows that are being worked with now in some areas of embryo transfer technology.

It is the hope of the CWRF to eventually be able to do embryo transfer within the white and black rhino species and also at some point attempt interspecies embryo transfer between the black and white rhinos. There is a lot of talk about embryo transfer but until actual manipulation of these animals is tried, working with them is a little more difficult than is first thought. So, first there is a need to determine if catheters can be physically inserted into the cervix and uterus manipulated before superovulation by hormones can be attempted.

As has been mentioned by other investigators, there is a need to be able to determine the estrus cycle in rhinos, It will be important to know when we can artificially inseminate on when to breed these animals before embryo transfer can be attempted. And then, after that, we have to know when embryos can be recovered. Also, embryo recipients will need to be hormonally prepared in order to establish a pregnancy. At Kings Island in Ohio, in an ongoing effort to develop embryo transfer technology for white rhinos, animals were first immobilized, placed in sternal recumbancy and rectally palpated to evaluate the reproductive tract. To date, it has been determined that uterus and ovaries could be palpated. but it is often very difficult. Ultrasound equipment is now being used to aid these efforts.

Specula are being developed in order to visualize the cervix for catheter insertion. A lengthy catheter has been developed for this procedure and attempts to flush the uterus with fluids are underway. Once superovulation techniques are pursued. embryo recovery techniques will be correlated.

Another technique that the CWRF team have been trying with rhinos came out of work that is being done with domestic cattle. It involves a small radio transmitter that sends out pulses. It has been used successfully in cattle to determine internal body temperature. It is inserted into the vagina and is similar to the method used to measure internal body temperature in women when they ovulate and there is a measurable increase in body temperature. When a cow's internal body temperature increases, these pulses increase and are received through a radio receiver. Dr. David Zartman, of Ohio State University. has inserted many of these into cattle. He custom-made the transmitter for the rhinos (larger that that used in a horse). The rhino cows have been monitored for at least six months and a trend does appear to be emerging.

Dr. Terry Blasdel. research coordinator for the Houston Zoo, has organized a program to produce offspring from white rhinos at the Houston Zoo by artificial Insemination. This project involves at least eight other zoos in North America, but had not yet begun at the time of the meeting.

Session Chairman ERIC MILLER

HEMOLYTIC ANEMIA IN THE BLACK RHINO

Summary of presentation by R. Eric Miller (St. Louis Zoological Park), co-authored by Hugh Chap//n (Washington University School of Medicine), Donald E. Paglia (University of Cal/torn/a at Los Angeles) and Will/am J. Boever (St. Louis Zoological Park).

Hemolytic anemia in the black rhino *(Diceros bicornis)* is a frequent occurrence and cause of death in the captive population of this species. Twenty-eight episodes of hemolytic anemia have been identified in 21 animals in zoos in North America, Europe and Japan. Eighty percent of the affected rhinoceroses have died during their initial or a recurrent episode of the anemia.

In man and in domestic animals, hemolytic anemia may result from a variety of factors that lead to a decrease in the survival time of the red blood cells (RBC's) and their early intra- or extra-vascular destruction within the body. Intravascular destruction of the RBC's leads to the release of their hemoglobin into the serum (hemoglobinemia) and may result in its passage into the urine (hemoglobinuria). The latter results in a clear, dark red coloration in the urine that is often the first sign that a black rhinoceros is developing a hemolytic crisis.

The case that occurred in St. Louis in 1981 (studbook 183/STL 6) was typical of the majority of the cases (8). A nine-year-old nulliparious female was noted to be weak and passing red urine. She was anesthetized for further evaluation, and blood values reflected a marked anemia — a haemocrit of 14.5% (normally 45-50%) (6). in other cases this value has ranged from 4.5% to 36**%** on initial presentation. Nucleated red blood cells — cells that in the horse are indicative of intensive efforts to replace the RBC loss—— were noted Similar findings, including regenerative bone marrow, have been found in two subsequent cases. The St. Louis animal died during attempts to reverse the anesthetic, no doubt complicated by the severe anemia present. Necropsy find-

ings were unremarkable except for massive deposition of iron in the liver (3 000 ppm) and the digestive tract. Similar iron deposition had been noted in previous cases, and in one animal without any signs of hemolysis. Further evaluation is warranted to determine if this reflects a subacute or chronic stage to the peracute form of cell destruction that is the hallmark of the syndrome described here.

A common cause to link the majority of the cases of hemolytic anemia has not been identified. Leptospirosis is strongly suggested in several cases (1,4), including one recent case (Osaka 209/LAX 5). Two cases were noted in Frankfurt that temporarily responded to steroids (7). Fatal hemic parasitism has been noted in newly captured wild black rhinoceroses (9), but its relationship to hemolytic anemia is unclear (2). No evidence for similar parasitism in captive animals outside of Africa has been noted to date, and titers for *Ehrlichia* sp. and *Babesia* sp. using reagents for domestic animals have been negative. Attempts to identify the agents of equine infectious anemia, copper toxicity, equine arteritis and clostridial infection have not identified any of these as possible causes of anemia in the black rhinoceros.

In a previous survey (8), respondents reported that they had kept 98 black rhinoceroses in captivity from 1972 to 1982. Twenty-five deaths occurred in animals greater than one year of age, and 11 of these deaths were associated with hemolytic anemia. Additional animals were located, bringing the total number of episodes to 28 ln 21 individuals. No sex ratio or seasonality is apparent. The greatest difference in age at death was noted between wild-caught (average 13.6 years) and captive-bred animals (average 7.0 years). Familial groupings were evident in one vertical grouping (mother-daughter-grandaughter) at the Frankfurt Zoo and multiple siblings from pairs at St Louis (three of four) and Denver (two of three). However, these three families appear unrelated to each other and only account for eight of the 21 affected individuals. At Toronto and Memphis Zoos, two and three cases occurred at one- and ten-day time intervals, perhaps suggesting a common agent or exposure. (indeed leptospirosis was strongly suggested at Memphis). However, at the majority of institutions single deaths occurred with apparently normal black rhinoceroses in the same enclosure or nearby. Despite the pre-mortem exposure of one animal to isoniazid and two others with an inadvertent exposure to the rodenticide diaphacinon, no common environmental exposure could be found.

Further efforts to identify a cause for the syndrome were directed at finding a “common denominator”, a basic defect that could lead a number of factors, e.g. leptospirosis or a toxin exposure, to trigger a massive hemolytlc event. A”two–fold approach was chosen: (i) evaluate basic RBC parameters of stability and a possible immune basis for the anemia, and (ii) an evaluation of the function of the RBC’s via a study of their enzymes and metabolites. The former approach was designed to evaluate the stability of the black rhinoceros RBC and the apparent response of several European animals to steroids. The latter study was designed to evaluate the RBC enzymes of the black rhinoceros due to several similar hemolytic syndromes in some human populations that are due to specific enzyme defects in their RBC’s.

For the first study, specific Coomb’s reagents for the black rhinoceros were developed (3). Using black rhinoceros sera inoculated into rabbits, both anti-black rhinoceros whole sera and a more specific anti-IgG were developed. Reactions with these reagents have been negative in all presumed normal animals, and one of the two anemic black rhinoceroses studied to date. in the second individual in a hemolytic crisis, the test indicated a possible coating of the RBC’s with the C3 component of the complement system. in man, this may occur in a number of chronic conditions and does not necessarily indicate an immune, basis to the disease. The reagents continue to be available for use in any future cases of hemolytic anemia. To facilitate their use, they will most likely be disseminated to centers in North America, Europe and Africa.

Additional studies (3) also indicated an increase in osmotic fragility of the black rhinoceros RBC in saline solutions in comparison to man. The haemaglobin electrophoretic pattern of the black rhinoceros indicated two bands at a pH of 8.6, the majority (80%) of the hemoglobin migrating slightly distal to the region of the unstable human hemoglobin H. The significance of both findings remains uncertain at this time. Electrophoretic patterns of RBC membranes of affected and unaffected individuals found no discernible differences between the two.

A separate study (10) evaluated the red blood cell enzymes and metaboiltes of aerobic glycolysis, glutathione cycling, and nucleotide metabolism. Ten animals were tested’— seven of East African origin, including two during hemolytic episodes, and one who was the dam of three affected individuals; and three apparently normal animals from southern Africa. Though the values found differed markedly from human normals, no differences were noted between apparently normal and affected rhinoceroses. Values were comparable to those found in a previous study of two rhinos (5). Further tests on an anemic Individual found no differences between the time of the hemolytic crisis and the convalescent period, nor was evidence of a heterozygous carrier state evident in the dam of the affected animals.

An interesting notation to this study was the variation between the animals of the eastern and southern origin in two of the enzymes studied. The seven samples from the eastern animals had only one third of the 2,3-diphosphoglycerate activity, and twice as much reduced glutathione in their RBC’s as did the southern animals (10).

Another area of possible importance to the etiology of hemolytic anemia is the overall nutritional status of the captive black rhinoceros population. Nearly an exclusive browser in the wild, captive diets for this species often predominate in feeds more closely approximating those of a grazer. Four captive black rhinoceroses were assayed for alpha-tocopherol levels. Levels were undetectable in two, and levels 0.2 ug/ml and 0.23 ug/ml were found in two additional animals. Selenium levels were 0.122 ug/ml to 0.170 ug/ml in the four animals. Further vitamin and mineral evaluation of the captive and wild animals is planned. Assays from wild animals are needed to supply standard values for animals on natural feeds.

Suggested treatment for the syndrome at the present time remains empirical: (I) high doses of penicillin or tetracycline if the case is acute leptospirosis or other infectious agent; (ii) vitamin E and selenium supplementation due to their role in the stability of red blood cell membranes; and (iii) possible use of short-acting steroids due to the apparent response of several European cases.

In all future cases, major emphasis must be given to the re-evaluation of each of the possibilities discussed —leptospirosis equine infectious anemia, copper toxicity, clostridial infect ion, hemic parasitism, undetected infectious agent or exposure to a toxin. Wherever possible, frozen tissue and serum

should be saved and stored at -75 degrees C for future reference.

Possible future avenues of research identified at the meeting include: (I) repetition of many of the previous tests on additional black rhinoceroses and also on white and Indian rhinoceroses, (ii) further evaluation of the immunological status of these animals in addition to the continued use of the Coombs reagent, (iii) further evaluation of the stability of the black rhinoceros RBC and its hemoglobin, (iv) evaluation of the iron metabolism of this species and attempts to identify a possible chronic stage of the anemia process, and (v) an overall evaluation of the nutritional status of this species In captivity. One emphasis of the latter study should be the determination of vitamin E and selenium levels in both captive and wild populations. The importance of a multi–faceted diagnostic approach was emphasized in a species in which so little is known. in man, with a much broader data base available, the cause of less than 50% of nonspherocytic hemolytic anemia is identified.

Since the syndrome has not been reported in white and Indian rhinoceroses, results from these species may help to establish a comparative data base for the black rhinoceros. A blood collection protocol for diagnostic and genetic studies in black, white and Indian rhinoceroses has been distributed to North American and European institutions holding these species (copies are available on request from the senior author).

Finding the specific etiology for the hemolytic crisis so frequent in the captive black rhinoceros population rests on further research in the areas enumerated above and perhaps others yet to be identified. The authors welcome suggestions of additional tests and approaches to this perplexing problem in the successful maintenance of this species in captivity.

Authors' notes

(i) Collecting large volumes of blood from the black rhinoceros can be difficult if the ear vein is used as the primary venipuncture site. Animals at St. Louis have been routinely bled from a large vein that passes over the medial carpus and ante-brachium. Though it is not always visible under the thick skin, a tourniquet applied proximally on the leg allows it to be palpated and cannulated. Up to one litre of blood has been collected rapidly from this site.

(ii) Since the Cincinnati meeting, an additional three adult (14.16 and 24 years of age) black rhinoceroses have died of hemolytic anemia in North America. The deaths occurred from November 2 to December 17, 1986. Preliminary laboratory data from these cases parallels that from previous hemolytic events. Two of the cases were tested with the autoimmune reagents described in this paper, and both were negative. Further tests are pending. No common factors could be identified to link the cases.

HAEMATOLOGICAL STUDIES OF BLACK RHINOS IN ZIMBABWE

Summary of presentation by Raoul du ToIt (IUCN African Elephant and Rhino Specialist Group), co-authored by Beverley Paul (University of Zimbabwe)

Various haematological studies were carried out with blood samples from 31 black rhinos that were translocated from the Zambezi Valley, Zimbabwe, in mid-1986.

In a field laboratory, within 3 hours of the collection of each sample, the following procedures were carried out haematocrit, white blood count, red blood count measurement of haemoglobin, plasma protein, erythrocyte sedimentation rate, and osmotic fragility; preparation of slides for differential cell counts, reticulocyte counts and parasite screening Additional blood samples from each animal were transported too Harare on wet ice, where standard blood analyses were performed on a Coulter counter (within at most 48 hours, and generally within 24 hours, of collection) in Harare, additional tests were carried out to investigation haemoglobin stability: isopropanol precipitation, heat test acidified glycerol lysis time test, and staining for Heinz bodies with methyl violet. Human blood specimens stored for similar periods were used as controls. Haemoglobin electrophoresis was performed on cellulose acetate Giucose-6-phosphate-dehydrogenase was assayed using a commercial kit (Sigma), which had been supplied by St. Louis Zoo.

The findings of these investigations are to be published (Journal of Zoology, in press). Consistent results were obtained from the standard haematological tests, an measurements of haemoglobin, haematocrit, and cell count conform closely with those obtained by veterinarians at Whipanade Park, using blood from a few captive black rhinos Thus it is felt that these data constitute reliable baseline information on the haomatology of the species Reticulocytes, not generally seen in rhino blood smeared occurred in some of the samples. The osmotic fragility of the red cells was somewhat greater than that of human red cells with 50% lysis occurring at a salt concentration of about 4.9 g/l. A significant observation was that all samples showed rapid precipitation of haemoglobin when inclubated will isopropanol. Heinz bodies could be demonstrated by methyl violet staining in up to 10% of fresh red cells. Very high levels of G-6-P-D activity were found in the red cells.

These results, Indicating an inherent tendency towards collapse of haemoglobin under oxidant stress, are obvious highly relevant to the problem of intravascular haemolysis. It seems unlikely that there is any single agent responsible for triggering haemolysis episodes; these are probably the end result of a variety of oxidant stresses.

There are indications that some die-offs of black rhinos in the wild could be related to haemolytic anaemia (e.g. about 30 rhinos died in Tsavo National Park in 1960-61, due to what was tentatively described as "'nutritional anaemia"). With wild animals, it would be worth investigating if parasitaemia aggravated by Inadequate nutrition, capture stress and other debilitating factors, is associated with haemolytic anaemia-abnormal haemoglobin and red cell enzyme systems may have developed in rhino as an evolutionary response to parasitaemia (as with sickle cell anaemia and possibly G-6-P D deficiency in humans), but under extra physiological stresses the balance could tip towards excessive haemolysis The Zambezi rhinos, from which blood samples were taken. were translocated to another reserve in Zimbabwe, where at least 20% of them died some weeks after translocation. in the pathological examinations that were carried out on a couple of sick and dead rhinos in this group, an unidentified piroplasm parasite was found In blood smears to a greater extent than in biood smears taken at the time that the animals were first captured, and large amounts of haemosiderin were found in spleen and liver tissue. This indicates a possibility of the mortality being due to stress-induced parasitaemia and a degree of haemolytic anaemia (although it has also been suggested that the deaths were due to the use of the drug ivermectin, for controlling skin and gut parasites). Further

investigation of rhino blood parasites and haematology is Intended In the hope of clarifying these health problems before more animals are lost In translocation operations, which will become an increasingly important part of rhino conservation In Africa.

POPULATION AND VETERINARY STATUS OF BLACK RHINOS IN THE UNITED KINGDOM

Summary of presentation by Richard Kock
(Zoological Society of London)

Introduction

The black rhino population in the British isles numbers 12 at present: five wild-caught and seven captive-bred individuals. The latter derive from two genetic lines. One pair came direct from East Africa to the United Kingdom In 1950. The other genetic line is derived from a pair at Hannover which were wild-caught in 1955 and 1957, and two Whipsnade animals and one London animal which were also wild-caught. Fifteen animals In total were caught from the wild. Twenty-four individuals have been born in captivity since 1958. From 1969-1986, 24 deaths occurred including both captive-bred and wild-caught individuals. The major reason for this poor record includes a high mortality in both sexually Immature and mature individuals. A relatively short reproductive period over the life span and a long calving internal are also problematic. Deaths In this species, when compared to the white rhinoceros in captivity, are premature.

Of the 24 deaths recorded, 21 died between October and May, there was one still-birth in July and two deaths between May and October, but both of these had been Ill during the previous winter. In general the clinical syndromes recorded are associated with winter management, i.e. indoor housing, fluctuating climatic conditions, dry fodder nutrition and inactivity. There appears to be no sex or age susceptibility to Illness.

Nine collections have exhibited the black rhino, including 5 currently.

From 1969-1986, 20 deaths occurred in collections as follows:

Chester (5), Marwell (2), Bristol (6), Dublin (3), Paignton (1), Manchester (2), Whipsnade (2), London (1) and Howletts (2). The most "successful" records are from London/ Whipsnade and Howletts/Port Lympne. Only three post-natal deaths (two juveniles and one adult which was Ill on arrival from Bristol) occurred in these collections. Five offspring from these two zoos are at present alive In Great Britain. The two animals at Howletts are in their sixteenth year of captivity and include a captive born animal. The animals at London/Whipsnade are over 20 years old. A major difference between these two collections and the rest is in feeding management, with more browse and green foods being provided during the summer due to a rural location.

Clinical histories 1969-1986

From the case records available only a few individuals died without clinical signs prior to death. The clinical signs have included nasal discharges; with muco-purulent material, serosanguinous fluid and frequently whole blood clots. Skin ulcers, diffuse and punctate in appearance and with a remarkably regular patterning over the skin surface, were common. A few cases presented with diarrhoea, laminitis or haemoglobinurea. During periods of illness animals were in general lethargic and on occasions inappetant. Many of the animals have shown respiratory distress In the last two or three days of illness particularly where recumbency was evident.

Due to the difficulty of clinical examination without anaesthesia in this species clinicians rarely performed extensive diagnostics.

Haematology

The information available is primarily from the Zoological Society's collections (Table 13). It appears that the red cell numbers, haemoglobin concentrations, packed cell volumes and mean cell volumes are extremely variable in the individuals examined when compared with the white rhino. There appears to be no correlation between the time of year and the red cell/mean cell volume values or the presence of heinz bodies. The heinz body findings are unlikely to be significant as they are a common occurrence in white rhinos. High mean cell volumes have been recorded in several animals and this was suggested to be due to a vitamin B or folate deficiency. it may have been an indication of a response to red cell loss by haemolysis. In general, the black rhinoceros has lower red cell haemoglobin values, packed cell volumes and higher mean cell volumes than the while rhinoceros. The only comparative data to hand are from an individual case in another collection which showed dramatically lower red cell haemoglobin and packed cell volume values to those in the collection. it is worth noting here that none of-the deaths in the Society's collections have been during a haemolytic crisis, as has occurred in other collections in this country and abroad.

Table 13. Haematological data from African rhinos, obtained by the Zoological Society of London.

	Black rhino (n = 7)			White rhino (n = 16)		
	Lowest	Mean	Highest	Lowest	Mean	Highest
Red cell count (x 1012/1)	2.69	4.80	6.90	5.48	6.82	8.16
White cell count (x10^9/1)	3.0	8.5	14.0	4.7	8.6	12.5
HaemoglobIn (g/l)	9.76	14.70	19.64	13.94	17.03	20.13
Packed cell volume (%)	30.7	41.6	52.6	37.9	46.4	54.9
Mean cell volume (f1)	76.1	86.8	114.2	67.3	68.0	69.1
Mean cell HaemoglobIn (pg)	28.5	30.6	36.3	24.7	25.0	25.4
Erythrocyte Sedimentation rate (mm/hr)	2.00	22.50	54.00	5.00	16.98	33.00
Platelets (x 10^9/I)	14.3	314.2	614.1	2.28	5.34	8.41
Reticulocytes						
Neutrophils (x10^9/I)	0	0	0	0	0	0
Lymphocytes (x 10^9/I)	2.38	5.09	7.79	2.28	5.43	8.41
Monocytes (x 10^9/I)	0.00	0.24	0.95	0.00	0.32	0.83
Eosinophils (x10^9/I)	0.00	0.22	0.72	0.16	0.41	1.00

Biochemistry

The biochemical parameters did not show any consistent abnormality except for very low plasma vitamin E levels of less than 0.1 mu/ml. Low values are seen frequently in white rhinos and elephants so this is difficult to interpret. Plasma vitamin A levels varied between 15-140 iu/litre. Very little

biochemistry is available from animals terminally; one case showed raised creatine kinase and urea, a very high calcium/phosphorus ratio and hyperglobulinaemia.

Bacteriology, etc.

The bacteriology of oral and skin ulcers was inconsistent and bacteriology of post-mortem materials was inconclusive. In general virological investigations have not been performed. No evidence of fungal infections of any significance was recorded.

Pathology

Relatively few cases were investigated thoroughly at post-mortem. For example, only viscera was examined from certain individuals. Full histological series were rarely obtained with tissues taken according to the gross post-mortem.

Fortunately a few cases were thoroughly examined and provide valuable information. General comments on the table of pathological findings (Table 14) follow.

There appears to be a pattern of pathological change with similarities between individuals. The suggestion is that a number of animals suffered from a similar condition. This was not recognized hitherto due to the scattered nature of the cases and inconsistent examination through the involvement of many individual clinicians and pathologists.

A significant number of animals were found on histology, to have heavy haemosiderin deposition in a variety of tissues which, although not an uncommon finding in normal horses, is rarely seen to the extent in evidence in the rhinoceroses. This suggests haemolysis during life. These changes have also been noted in zoo equids to a lesser extent.

An interesting finding was glomerulopathy which requires further investigation. Fortunately a number of tissues are available for this purpose from previous cases. The possibility of immune complex deposition in the kidney is under investigation. The ulcerative dermatoses were frequently encountered but rarely investigated histologically. The range of findings in the skin suggest low grade dermatitis unlikely to be infectious in origin with hyperkeratosis, vesication, arterial changes (including endarteritis obliterans) and deposition of pigments amongst other pathological findings. Ulcerative and inflammatory changes of the alimentary tract were common. One case of a typical myopathy was reported, one liver hepatosis and two cases with pathology in the respiratory system other than emphysema. Two cases which died acutely with apparently no preliminary signs showed evidence of acute haemolysis, one with extensive alimentary tract ulceration and the other with apparent acidosis in the colon.

In summary, the pathogenesis of the ‘condition’ (if it is one condition) involves the development of ulcers in the skin and alimentary tract with mild inflammatory changes suggesting ischiemia rather than infectious agents and glomerulopathy, the cause of which is undetermined at present. in addition the deposition of pigments (predominantly haemosiderin) suggests haemolysis may be an important component of the syndrome.

Conclusion

In the opinion of the author there is sufficient evidence of a syndrome affecting black rhinos in captivity in the British Isles and leading to abnormal mortality in the species in captivity. The most likely predisposing factors or causes are winter nutrition and possibly stresses at this time of the year including enclosed housing, inactivity and fluctuating environmental temperature. The black rhino is almost exclusively a browser and the rations in captivity during the winter have been based on dry matter (primarily lucerne hays) and concentrated foods based on cereals. These diets are not consistent with the natural dietary intake and might lead to malnutrition in the species. The clinical response in a few cases to vitamin supplementation, particularly A and E, is

Table 14. Summary of pathological findings ——black rhinos———British Isles ——1969-1986

Local ID	Birthdate	Sex	Location	Skin Ulcers	Ulcers/ inflammation Alimentary Tract	Pigment Deposition Haemosiderin/ Lipofuschin or unidentified	Kidney Pathology (Giomerulopathy)
Willie	01.07.50	M	Bristol	*	*	*	*
Stephanie	01.07.50	F	Bristol	*	*	*	No histo
Rebecca	17.05.70	F	Bristol	*	*		
Rupert	28.06.65	M	Whipsnade	*	*	*	*
Kes		20.09.78	M	Marwell	*	*	* *
Joanna	15.11.72	F	Paignton	+	+		
Susie	01.07.56	M	Chester	*	*	*	*
Paul	01.07.63	F	London	*	*	*	+
Laura	01.07.60	M	Dublin?	+	+*	*	*
Johnny	01.07.65	F	Dublin	No histo			
M’kuzl	31.08.73	M	Whipsnade	No histo	+*	No histo	No histo
Linda	20.11.73	F	Chester	+			+ (myoglobin deposits)
Kijana	25.11. 70	M	DublinNo histology. Gross evidence of acute haemolysis and ulcers (skin & gut)				
Katie	16.09.79	F	MarwellAcute haemolysIs and colon acidosis. No histology.				

• Histology reported — positive findings
+ Gross findings
No histo: Histology not completed or reported

suggestive of a deficiency. These elements are liable to degradation in dry forage during the winter and may be a component of the syndrome. It is notable that the collections which have been most successful have provided large amounts of green food in the form of cut grass or browse during spring, summer and autumn.

Two facts are clear from this review. There needs to be an improved coordination between collections to ensure optimal investigation of cases and a more detailed examination of available tissues is necessary.

In the Society we have initiated a change in the nutritional management of the rhinos which includes an attempt at a browser ration for the black rhino and an improved supplementation of elements considered to be deficient in winter diets. The browser concentrate is fed at approximately 6 kg per day and the analysis is shown in Table 15. Lucerne hay is also provided plus vitamin E cubes to a level ensuring an intake of 6 000 international units per day per animal.

Table 15. Analyses of diets of black rhinos at London/ Whipsnade. All values are calculated to nominal 10% moisture content; all values are total calculated values; 1 mcg retinol = 3.3 i.u. vitamin A activity; total retinol content includes the retinol equivalent of carotene; 1 mcg B-carotene = 1.6 i.u. vitamin A activity: 1 mcg cholecalciferol = 40.0 i.u. vitamin D3 activity; 1 mg tocopherol = 1.1 i.u. vitamin E activity; 1 MJ = 239.23 calorIes.

General Purpose Diets

1. Bovine (Browser) Breeder Pellets
2. Bovine (Browser) Maintenance Pallets

		1	2			1	2
Crude Oil	%	4.5	2.8	Glycine	%	1.07	0.65
Crude Protein	%	16.4	12.9	Aspartic Acid	%	1.03	0.71
Crude Fibre	%	10.6	15.2	Glutamic Acid	%	2.98	2.36
Ash	%	10.9	10.7	Proline	%	1.04	0.88
N.F.E	%	47.6	48.4	Serine	%	0.66	0.47
				Hydroxyproline	%	–	—
Dig. Crude Oil	%	4.2	2.5	Hydroxylysine	%	—	—
Dig. Crude Protein	%	14.4	10.5	Alanine	%	0.12	0.12
Tot. Dietary Fibre	%	28.5	35.6				
Pectin	%	2.7	2.7	Calcium	%	1.20	1.24
Hemicellulose	%	13.2	14.2	Phosphorous	%	1.14	1.05
Cellulose	%	10.0	11.7	Phytate Phosphorous	%	0.33	0.30
Lignin	%	2.6	7.0	Sodium	%	0.73	0.72
Starches	%	16.8	16.4	Chlorine	%	1.11	1.09
Sugars	%	12.9	11.6	Magnesium	%	0.65	0.55
				Potassium	%	1.41	1.26
Gross Energy	MJ/kg	14.6	14.1				
Dig.. Energy	MJ/kg	9.4	7.7	Iron	mg/kg	194	153
Met. Energy	MJ/kg	8.5	6.9	Copper	mg/kg	18	16
				Manganese	mg/kg	117	116
Myristoleic Acid	%	–	—	Zinc	mg/kg	114	108
Palmitoleic Acid	%	0.05	0.02	Cobalt	mcg/kg	1104	1107
Oleic Acid	%	1.02	0.71	Iodine	mcg/kg	1431	1419
Linoleic Acid	%	1.19	0.58	Selenium	mcg/kg	356	314
Linolenic Acid	%	0.71	0.40	Fluorine	mg/kg	69	69
Arachidonic Acid	%	0.35	0.13				
Clupenodonic Acid	%	–	—	Retinol	mcg/kg	27889	26667
				Cholecalciferol	mcg/kg	103	52
Lauric Acid	%	0.06	0.03	dl %Tocopherol	mg/kg	116	69
Myristic Acid	%	0.20	0.15				
Palmitic Acid	%	0.44	0.33	Vitamin B1	mg/kg	13.8	9.5
Stearic Acid	%	0.13	0.07	Vitamin B2	mg/kg	10.3	6.3
				Vitamin B6	mg/kg	9.1	5.5
Arginine	%	1.10	0.76	Vitamin B12	mcg/kg	81.0	40.7
Lysine	%	0.80	0.54	Vitamin C	mg/kg	160.0	111.0
Methionine	%	0.22	0.16	Menadione	mg/kg	41.4	36.7
Cystine	%	0.24	0.18	Folic Acid	mg/kg	72.5	61.0
Tryptophan	%	0.24	0.18	Nicotinic Acid	mg/kg	45.1	31.6
Histdine	%	0.39	0.28	Pantothenic Acid	mg/kg	35.5	24.6
Threonine	%	0.60	0.42	Choline	mg/kg	1248.0	902.0
Isoleucine	%	0.66	0.46	Inositol	mg/kg	995.0	879.0
Leucine	%	1.13	0.79	Biotin	mcg/kg	519.0	378.0
Phenylalanine	%	0.71	0.50	p Aminobenzoic Acid	mg/kg	—	—
Valine	%	0.78	0.57				
Tyrosine	%	0.55	0.38	6 Carotene	mg/kg	49.6	50.2
Taurine	%	—	—	Carophyll Red	mg/kg	—	—

Vitamin E Supplementary Foods

1. Vitamin E Cubes
2. High Potency Vitamin E Pellets

		1	2			1	2
Crude Oil	%	6.4	16.8	Glycine	%	1.26	0.37
Crude Protein	%	17.4	9.3	Aspartic Acid	%	1.40	0.45
Crude Fibre	%	7.1	8.2	Glutamic Acid	%	2.76	1.50
Ash	%	9.1	15.6	Proline	%	0.91	0.61
N.F.E	%	50.0	40.1	Serine	%	0.72	0.29
				Hydroxyproline	%	0.02	—
Dig. Crude Oil	%	5.6	16.3	Hydroxylysine	%	—	—
Dig. Crude Protein	%	15.7	7.3	Alanine	%	—	0.09
Tot. Dietary Fibre	%	19.5	24.0				
Pectin	%	3.4	2.2	Calcium	%	0.85	0.34
Hemicellulose	%	8.5	12.6	Phosphorous	%	0.47	0.34
Cellulose	%	6.5	7.8	Phytate Phosphorous	%	0.23	0.20
Lignin	%	1.1	1.4	Sodium	%	0.08	0.14
Starches	%	31.0	9.2	Chlorine	%	0.15	0.24
Sugars	%	6.6	15.1	Magnesium	%	0.27	0.35
Gross Energy	MJ/kg	15.4	16.3	Potassium	%	1.03	0.96
Dig.. Energy	MJ/kg	11.5	11.8	Iron	mg/kg	145	61
Met. Energy	MJ/kg	10.4	10.6	Copper	mg/kg	10	7
Myristoleic Acid	%	0.04	—	Manganese	mg/kg	31	43
				Zinc	mg/kg	25	7
Palmitoleic Acid	%	0.22	0.08	Cobalt	mcg/kg	48	36
Oleic Acid	%	0.48	1.34	Iodine	mcg/kg	241	85
Linoleic Acid	%	0.66	2.18	Selenium	mcg/kg	120	163
Linolenic Acid	%	0.71	0.40	Fluorine	mg/kg	17	6
Arachidonic Acid	%	0.06	0.09				
Clupenodonic Acid	%	0.01	—	Retinol	mcg/kg	24799	24501
				Cholecalciferol	mcg/kg	10	—
Lauric Acid	%	0.01	0.09	dl %Tocopherol	mg/kg	23342	92651
Myristic Acid	%	0.07	0.25				
Palmitic Acid	%	0.25	0.49	Vitamin B1	mg/kg	4.0	4.0
Stearic Acid	%	0.10	0.20	Vitamin B2	mg/kg	4.1	2.0
				Vitamin B6	mg/kg	3.1	1.2
Arginine	%	1.25	0.47	Vitamin B12	mcg/kg	4.9	0.4
Lysine	%	0.71	0.34	Vitamin C	mg/kg	57.0	58.0
Methionine	%	0.32	0.12	Menadione	mg/kg	31.1	30.7
Cystine	%	0.29	0.12	Folic Acid	mg/kg	1.2	0.5
Tryptophan	%	0.25	0.13	Nicotinic Acid	mg/kg	45.1	31.6
Histdine	%	0.36	0.19	Pantothenic Acid	mg/kg	12.0	9.7
Threonine	%	0.62	0.27	Choline	mg/kg	1149.0	459.0
Isoleucine	%	0.67	0.28	Inositol	mg/kg	2349.0	509.0
Leucine	%	1.09	0.51	Biotin	mcg/kg	270.0	183.0
Phenylalanine	%	0.77	0.34	p Aminobenzoic Acid	mg/kg	—	—
Valine	%	0.84	0.38				
Tyrosine	%	0.51	0.25	6 Carotene	mg/kg	49.5	49.0
Taurine	%	—	—	Carophyll Red	mg/kg	—	—

Supplementary Foods

1. Rhino Cubes
2. Zebra Cubes

		1	2			1	2
Crude Oil	%	7.3	5.8	Glycine	%	0.41	1.48
Crude Protein	%	9.4	17.5	Aspartic Acid	%	0.64	1.25
Crude Fibre	%	18.7	11.8	Glutamic Acid	%	1.57	3.09
Ash	%	9.9	7.8	Proline	%	0.62	1.01
N.F.E	%	44.7	47.1	Serine	%	0.35	0.74
				Hydroxyproline	%	–	—
Dig. Crude Oil	%	6.9	5.4	Hydroxylysine	%	—	—
Dig. Crude Protein	%	7.5	15.6	Alanine	%	0.09	0.07
Tot. Dietary Fibre	%	43.9	30.2				
Pectin	%	2.4	3.2	Calcium	%	1.06	1.56
Hemicellulose	%	18.0	13.5	Phosphorous	%	0.53	0.66
Cellulose	%	18.1	11.0	Phytate Phosphorous	%	0.26	0.28
Lignin	%	5.4	2.5	Sodium	%	0.67	0.48
Starches	%	10.1	21.7	Chlorine	%	1.05	0.70
Sugars	%	9.4	7.0	Magnesium	%	1.20	0.47
				Potassium	%	1.97	1.21
Gross Energy	MJ/kg	15.2	15.5				
Dig.. Energy	MJ/kg	7.4	10.0	Iron	mg/kg	233	230
Met. Energy	MJ/kg	6.7	9.0	Copper	mg/kg	23	32
				Manganese	mg/kg	197	342
Myristoleic Acid	%	0.01	0.03	Zinc	mg/kg	153	437
Palmitoleic Acid	%	0.12	0.15	Cobalt	mcg/kg	10128	5207
Oleic Acid	%	1.50	0.82	Iodine	mcg/kg	15777	19236
Linoleic Acid	%	2.35	0.53	Selenium	mcg/kg	196	362
Linolenic Acid	%	0.47	0.36	Fluorine	mg/kg	40	30
Arachidonic Acid	%	0.10	0.08				
Clupenodonic Acid	%	–	—	Retinol	mcg/kg	80065	30951
				Cholecalciferol	mcg/kg	377	51
Lauric Acid	%	0.12	0.11	dl %Tocopherol	mg/kg	123	258
Myristic Acid	%	0.29	0.09				
Palmitic Acid	%	0.57	0.33	Vitamin B1	mg/kg	44.6	26.3
Stearic Acid	%	0.21	0.06	Vitamin B2	mg/kg	84.3	23.4
				Vitamin B6	mg/kg	21.7	17.3
Arginine	%	1.55	1.31	Vitamin B12	mcg/kg	1916.8	251.6
Lysine	%	0.41	0.84	Vitamin C	mg/kg	108.0	310.0
Methionine	%	0.15	0.29	Menadione	mg/kg	62.1	51.6
Cystine	%	0.14	0.25	Folic Acid	mg/kg	15.7	11.7
Tryptophan	%	0.14	0.25	Nicotinic Acid	mg/kg	237.4	62.1
Histdine	%	0.21	0.42	Pantothenic Acid	mg/kg	87.9	39.2
Threonine	%	0.37	0.65	Choline	mg/kg	1282.0	1330.0
Isoleucine	%	0.34	0.73	Inositol	mg/kg	722.0	1643.0
Leucine	%	0.60	1.18	Biotin	mcg/kg	217.0	483.0
Phenylalanine	%	0.40	0.83	p Aminobenzoic Acid	mg/kg	—	—
Valine	%	0.45	0.87				
Tyrosine	%	0.27	0.59	6 Carotene	mg/kg	99.2	49.5
Taurine	%	—	—	Carophyll Red	mg/kg	—	—

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