which had been constipated for some time were usually very weak and were treated systemically with antibiotics, corticoids (cortisone acetate) and vitamin B_{12}.

Elephants in the Kruger National Park are usually infested with parasites of the gastro-intestinal tract and liver. We attempted to control intestinal nematodes by the oral administration of thibenzole (MSD) at a dosage rate of 15 mg/kg. We found this anthelmintic very safe and quite effective for controlling intestinal nematodes.

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Mortalities associated with the capture, translocation, trade and exhibition of Black rhinoceroses

Diceros bicornis

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INTRODUCTION

The Black rhinoceros *Diceros bicornis* will not usually retreat before human encroachment and, as a result, it is killed by incoming settlers or by the Game Division in defence of human life and property. Even in the game controlled areas, where human rights are limited to settlement only, and in conservation areas where human rights are denied, rhinoceroses are poached for 'rhino horn', which is regarded as having anti-toxic and aphrodisiac properties.

In the early 1960s such poaching of the rhinoceros was occurring in and around the Grumeti and Lamai game controlled areas. As a result, in 1962, the Tanzania Game Division in conjunction with Mr Carr-Hartley, a professional game trapper, began to catch and remove rhinoceroses from these areas. In 1964, the Veterinary Division undertook to co-operate in this work, and to investigate causes of mortality. Rubondo Island and Saanane Island, both in Lake Victoria, were designated as a game reserve and a game reserve zoo respectively. It was planned to translocate rhinoceroses to Rubondo, exhibit two animals on Saanane and to send some rhinoceroses to Arusha, and to Kenya, for overseas markets.

Under this programme from 1964, the Government required that an investigation be made into the trade in captured rhinoceroses, with specific reference to mortality caused by disease or management practice. As professional trappers hold their animals in captivity for some time before overseas shipment, we simulated these conditions to some extent, and kept the rhinoceroses in field captivity for varying periods before allocating them to their final destinations.

Mr Carr-Hartley and his sons were responsible for the capture of the rhinoceroses and a team

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from the Game and Veterinary Divisions was responsible for care, management and feeding. Many of the animals which fell sick received professional examination and treatment. It was not possible, however, to arrange for professional attendance all the time and, therefore, some animals which died were not subjected to post-mortem examination.

METHODS

Rhinoceroses were captured in daylight. They were chased, usually one at a time, and noosed from one of two pursuing vehicles. Once caught, the rhinoceroses were cast, hobbled, loaded on to a lorry, taken to camp and then released in individual holding pens. At some stages of the work blood slides were prepared and prophylactic drugs were administered at capture. Most, but not all, of the animals were allocated numbers.

The holding pens were made of hardwood poles which were wired closely together with heavy gauge fencing wire and sunk in trenches 0·75 m deep. At the Grumeti, a line of contiguous pens was constructed. Each pen was approximately 5·5 m square. At the Lamai, separate and circular pens of some 4·5 m diameter were built over a larger area.

RESULTS

Grumeti, 1962. Two rhinoceroses were caught and released in the Serengeti.

Grumeti, 1963. Sixteen rhinoceroses were caught of which four died at, or shortly after, capture. Three were translocated to Rubondo and five to Arusha.

The remaining four were held in pens at the Grumeti after heavy rains had made travelling conditions impossible. Of these one escaped, two died and one was taken to Arusha.

No post-mortem examinations were carried out on the six rhinoceroses which died and the cause of death remains unknown.

Grumeti, 1964. Eight rhinoceroses were caught between February and August. As a prophylactic treatment against trypanosomiasis, 1·5 to 3·0 gm of diminazine aceturate were administered subcutaneously to all of them. No deaths were directly attributable to the method of capture. One death occurred in captivity when a mature male rhinoceros broke into a pen of a young male (3434), and in the ensuing fight, the young animal sustained injuries from which it later died in Arusha.

Lamai, 1964. Seventeen rhinoceroses were captured between the beginning of October and the end of November. They were treated with a prophylatic dose of 1·5 to 3·0 gm of diminazine aceturate. There was one death directly attributable to capture. A rhinoceros (3474) charged the catching truck at full force, staggered back dazed and died almost immediately. No post-mortem examination was carried out. One rhinoceros (5788) died suddenly after 14 days in captivity. No post-mortem examination was carried out, but many large forms of *Babesia* piroplasms were seen in blood smears from this animal. Two rhinoceroses (5789 and 5645) died after 17 and 13 days in captivity. These animals were less aggressive and more lethargic than usual. Although there was only some slight loss of appetite, loss of weight was rapid and a marked deterioration was followed by death. Post-mortem examinations were carried out and showed marked pulmonary oedema, with froth exuding from the cut bronchioles. Stomachs and intestines contained normal ingesta but the small intestines showed catarrhal inflammation. The carcasses were emaciated, with depletion of the cardiac, omental, perirenal, intermuscular and subcutaneous fat deposits. Post-mortem examinations suggested that active trypanosome infections were involved. Blood smears from 5645 showed trypanosomes of the brucei subgroup. No haematozoa were seen in slides from 5789.

Two deaths were thought to be due to mismanagement. A heavily pregnant cow (3480) died after 31 days in captivity. She had a tear in the vagina from which she had lost a lot of blood. Post-mortem examination showed marked emaciation and, although blood smears were negative, trypanosomiasis may have contributed to her state of cachexia and weakness, and may have precipitated her death. Rhinoceros 5786 was roped in the holding pen and pulled struggling into a travelling crate, where it died shortly after the ropes had been removed.

One death was later reported amongst the rhinoceroses which were taken to Kenya. Post-mortem examination of this animal (5646) was carried out at the Faculty of Veterinary Science, University College, Nairobi. Salient features

reported were pulmonary congestion, pulmonary oedema and haemorrhagic gastro-enteritis.

Lamai, 1965. Between the beginning of August and the end of October 1965, 34 rhinoceroses were caught. Trypanosome and piroplasm control methods were reviewed and the rhinoceroses subsequently fell into 5 groups.

In the first group (5 rhinoceroses: diminazine aceturate 3·0 to 10·0 g) all died. One animal (4675) died on the day of capture. 4674 died after 15 days in captivity and post-mortem examination indicated trypanosome infection. Blood smears were positive for brucei subgroup and *Trypanosoma vivax*. 3406 and 3464 died after 12 and 9 days in captivity. No post-mortem examinations were carried out and no blood or tissue smears were prepared. Clinical symptoms suggested that active trypanosomiasis was concerned in both these deaths. 3510 died suddenly after 26 days in captivity. No post-mortem examination was carried out, but very many large forms of *Babesia* were seen in blood smears. A count of 100 contiguous red cells showed 44 cells with one form; 11 cells with two forms; four cells with three forms; and three cells with four forms. Nearly every red cell showed anulocytosis, and anisocytosis was marked. There were signs of poikilocytosis. A similar count in a slide from 5788 showed five cells with one form, and four cells with two forms. There was also some evidence of anulocytosis and anisocytosis.

In the second group (7 rhinoceroses: diminazine aceturate 6·0 g; pyrithidium bromide 3·0 g) there were three deaths in late captivity. 3470 died 50 days after capture. Post-mortem examination showed a massive load of gut nematodes, including *Strongylus tremletti* and *Kiluluma* spp. This worm load was regarded as responsible for death. The carcass was emaciated. The cardiac, omental, perirenal, intermuscular and subcutaneous fat deposits were completely depleted, but the stomach and bowels were full and contained normal ingesta. 3469 died after 67 days in captivity and was subjected to post-mortem examination by Game Division staff, and again a heavy worm burden was observed. On clinical grounds, a similar infestation was incriminated in the death of 4766 which died 29 days after capture.

In the third group (11 rhinoceroses: homidium bromide 0·75 to 2·25 g; pyrithidium bromide 1·50 to 4·50 g) there were another two deaths in late captivity. 3467 died after 43 days. This death was a result of mismanagement; the animal was pulled into a travelling crate. 4763 died 'en route' to Rubondo after 45 days in captivity. It was in poor condition and clinical indications were that helminthiasis was the precipitating factor in its death.

In the fourth group (9 rhinoceroses: homidium bromide 0·75 to 2·25 g; pyrithidium bromide 1·5 to 4·5 g; sprayed once with dioxathion. 1 rhinoceros: homidium bromide 2·75 g; sprayed once with dioxathion) there were nine deaths. Rhinoceros 5010 died at capture. Eight more rhinoceroses (3507, 5127, 5121, 5081, 5123, 3460, 5128 and 5766) died after 3, 5, 3, 15, 4, 11, 6 and 9 days in captivity, with symptoms and post-mortem features of what was at first thought to be myoglobinuria paralytica. Clinical symptoms were clear cut. There was noticeable muscular trembling, especially of the hind legs, and gait was unsteady. Within a few hours of the onset of symptoms, affected animals collapsed on their sides, showed distressed breathing and died in muscular spasms. Post-mortem examinations of 3507, 5121, 5127 and 5123 showed marked evidence of tissue damage in the muscles of the neck, shoulders, back and hind legs. The affected parts were pale and appeared par-boiled. The bladders of 3507, 5121 and 5123 were full of urine which was dark brown, albumenous and of the consistency of a thick soup. In 5127 the urine was normal. A comminuted supra-condylar fracture was noted at the distal end of the right humerus of 5121. The Hartley team reported muscle tissue damage in the skeletal structure of the other four dead rhinoceroses. The one animal which survived was translocated to Rubondo after 26 days in field captivity.

The fifth group (1 rhinoceros: homidium bromide 2·25 g; pyrithidium bromide 4·50 g) was not sprayed and was translocated to Rubondo after 30 days in captivity (5082).

Of the 14 rhinoceroses which were removed from the Lamai in 1965, one death was subsequently reported from Rubondo and two deaths from Kenya. The numbers of these animals were not known. No post-mortem examination was carried out on the rhinoceros which died at Rubondo. But a report from University College, Kenya, indicated that a severe enteritis was involved in

one of the Kenya deaths, although the cause of death in the other was indeterminate.

At the Lamai blood smears were prepared from 15 rhinoceroses at the time of capture. Trypanosomes were seen in the slides from three young animals. Brucei subgroup and *T. vivax* were seen in blood smears from 4769 and *T. vivax* in smears from 4678 and 3401. Small piroplasms, probably *Theileria* spp. were seen in slides from one young rhinoceros, 4678, and one mature, 3509.

Saanane 1964-67. In late 1964 two rhinoceroses, 3436 and 3479, were sent to Saanane for exhibition. 3436, a large pregnant cow from the Grumeti gave birth to a female calf in May 1965. Within three months the mother and calf developed corneal opacities. The condition in the cow became very severe, and suppuration and pointing of both eyeballs was apparent. Massive doses of vitamin A and supportive antibiotic treatments were administered. The calf died in late August. The cow remained permanently blind but, to outward appearances, the eyes were normal and there were no ocular discharges. In early February 1966, she developed severe diarrhoea and died in three days. Post-mortem examination showed that much of the mucosa of the large intestine was in the form of dried, hardened and raised diphtheritic plaques. Bacteriological examinations were negative. No haematozoa were seen in blood or tissue smears.

A young male from the Lamai, 3479, fared badly on arrival at Saanane and, although blood smears were negative, this animal looked as if it was about to die of active trypanosomiasis. It was treated with diminazine aceturate and homidium bromide and appeared to improve but then started to pass nematodes in the faeces. Phenothiazine was administered and recovery was followed by a long period of convalescence. It then showed signs of corneal opacities. However, this condition completely resolved after the oral administration of vitamin A, which also appeared to control recurrent attacks of diarrhoea. In order to try and reduce the incidence of these disorders it was decided, in 1966, to administer 10,000,000 i.u. vitamin A orally to the animal every three months. Unfortunately, in September 1967, it developed a severe attack of diarrhoea and died within 24 hours from the onset of symptoms. Post-mortem examination showed acute inflammation of the glandular part of the gastric mucosa and congestion of the mucosae of the small and large intestines. No haematozoa were seen in blood or tissue smears and bacteriological examinations of tissues were negative.

FEEDING AND MANAGEMENT

At both areas the bulk of the diet was composed of branches and leaves of the shrub *Grewia bicolor*. *Euphorbia candelabrum* was fed occasionally and sugar-cane *Saccharum officinale* was another favourite plant. The diet of young rhinoceroses was supplemented with sweetened maize meal gruel and they were also fed dried lucerne *Medicago sativa*. In early captivity, particular attention was paid to the evening feed. Foliage was sprayed with water, which encouraged sick animals to eat and ensured that the leaves remained fresh throughout the night–the period of maximum food intake. A few days before the animal was to be moved a crate was wired to the pen door and food was provided within the crate so that the animal became familiar with the structure. On the morning of translocation it was then possible, in most instances, to drop the sliding door and secure the animal as it was feeding. During movement, branches, lucerne and sugar-cane were thrown on the floor of the crate in front of the animal.

When travelling it was found that the lowering of buckets of water into the crate caused excitement and disturbance. On the other hand no reaction was caused by syphoning water into the crate and the animals quickly learned to suck water from the end of a hose-pipe.

Restraint of rhinoceroses in holding pens was avoided when possible. Prophylactic treatments against trypanosomiasis and chemotherapeutic treatments of poaching wounds and other injuries were generally given at capture. Further attention to wounds depended largely on washing with a saline solution by means of a stirrup pump. One rhinoceros, 3440, was cast to establish a drainage channel from an arrow wound. Other animals received ancillary treatments, such as oxytetracycline hydrochloride, sulphadimidine, betamethasone sodium phosphate, calcium borogluconate, magnesium sulphate and glucose saline. These drugs were administered to combat wound infection or to try to reduce the overall incidence of mortality.

DISCUSSION

Systematic protozoal infections in domestic animals are usually diagnosed by an assessment of clinical and/or post-mortem observations, in relation to microscopical findings. Biological and serological examinations provide only limited assistance in this respect. For instance, sub-inoculation of trypanosomes into rats is an aid to trypanosome identification, but is of no help in differentiating active from latent infection. As latent trypanosome infections are widespread in game animals in general, the diagnosis of active infection is difficult. It has to be based largely on clinical and/or post-mortem findings in relation to the characteristic features of active trypanosomiasis in domestic animals, described, for example, by Hornby (1952).

In our work with rhinoceroses the presence of latent trypanosome infection was established. Blood smears were prepared from 15 rhinoceroses at capture. Trypanosomes of the brucei subgroup and *T. vivax* were seen in blood smears from one young rhinoceros, and *T. vivax* in smears from another two young animals. The extent of latent infection was not established as blood slide examination is an unreliable method of assessment and latent infection in adults may have been overlooked.

At the Lamai, the deaths of five rhinoceroses were attributed to active trypanosomiasis. Three showed post-mortem features which were in accord with active infection with brucei subgroup. Two showed trypanosomes in blood and tissue smears, one showing brucei subgroup and the other mixed brucei subgroup and *T. vivax*. Nevertheless, we could not be sure that trypanosomes were the cause of the disease syndrome as there was no way of differentiating between primary infection, reinfection, latent infection and exacerbating latent infection. However, active trypanosome infection in wild-living game animals may be more widespread than is generally supposed. McCulloch (1967) drew attention to what he thought was probably natural and active infection of the brucei subgroup in wild and free-living zebras *Equus burchellii*. Nevertheless, the apparent problem of active trypanosomiasis at the Lamai was alleviated when the prophylactic cover was increased by about four to six times that initially provided at the Grumeti. Diminazine aceturate/pyrithidium bromide and homidium bromide/pyrithidium bromide appeared equally efficient.

Tsetse fly density was low at the Grumeti compared to the Lamai where flies were seen in the holding pens, in the tents and all about the immediate vicinity of the camp. In both areas *G. swynnertoni*, *G. pallidipes* and *G. brevipalpis* were seen although *G. swynnertoni* was the dominant species. Weitz (1963) reported on relevant tsetse feeding habits as follows: *G. swynnertoni:* 5531 meals, 65·4% from warthog, 6·7% from rhinoceros. *G. pallidipes:* 2688 meals, 19·2% from warthog, 0·6% from rhinoceros. *G. brevipalpis:* 1151 meals, nil from warthog, 1·8% from rhinoceros. At the Lamai, where warthogs *Phacochoerus aethiopicus* were fewer and rhinoceroses were more numerous than at the Grumeti, *G. swynnertoni* particularly may have been forced to feed to a large extent on rhinoceroses. It is probable that the apparent breakdown of the Grumeti prophylactic cover when used at the Lamai was associated with the disparity in tsetse fly density, and with the difference in the availabilities of warthogs and rhinoceroses as sources of fly food.

Two deaths were attributed to babesiasis. It was unfortunate that no post-mortem examinations were carried out, and that blood smears only were available for examination. On these, large forms of *Babesia* were seen. The parasites appeared to be very similar to those described by Brocklesby (1967) who is of the opinion that if piroplasm forms are present in very large numbers they may possibly be of pathogenic importance. Prophylactic control with diminazine aceturate appeared to be inadequate and an attempt to control vector ticks by spraying with dioxathion was followed by disastrous results. Unfortunately, it was not possible to establish unequivocally that dioxathion was responsible. Scott (1964) in a review article drawing attention to the toxic actions of pesticides on man and animals points out with regard to organo-phosphorus compounds, such as dioxathion, that copious salivation, hypermobility of the intestines and muscular twitchings are the most obvious signs of toxic activity. In our cases, the only outstanding symptoms were muscular twitching and weakness of muscle structures. Histopathological work on formalised tissue blocks from the affected rhinoceros has been reported by Mugera & Wandera (1967) who

observed a coagulative type of necrosis in kidney tissues; centro-lobular haemorrhagic hepatitis; pulmonary congestion, oedema and emphysema. They also reported granular and hyaline degeneration of skeletal muscle tissues. It might be worth trying the anti-babesiasis effect of a trypanosomidal complex of diminazine aceturate, homidium bromide and pyrithidium bromide.

It was noted that the Lamai rhinoceroses were essentially grass eaters, whereas those at the Grumeti browsed to a large extent on woodland bush. It is known that grazing, as distinct from browsing, encourages the propagation of intestinal parasites. Helminthiasis can be further aggravated by coprophagous habits. This vice, which has been reported amongst rhinoceros elsewhere (Klingel & Klingel, 1966), was particularly noticeable under captive conditions in the Lamai, and may have contributed to the worm build up there. *S. tremletti* was first identified in samples taken from Black rhinoceros in the Tsavo National Park, Kenya (Round, 1962). In this area elephant *Loxodonta africana* had caused widespread destruction of natural habitat and rhinoceroses were dying from malnutrition (Glover & Sheldrick, 1964). Round confirmed the identification of *S. tremletti* at Lamai and drew attention to concurrent *Kiluluma* infestation. Thus, in view of the experiences at the Tsavo and at the Lamai, there may be a case for regarding *S. tremletti* as a pathogenic nematode in Black rhinoceros.

In field captivity most of the food provided for the rhinoceroses was composed of branches and leaves of trees and shrubs normally eaten by them. The inclusion of browse in a diet seems to favour the intake of crude protein (Bredon & Wilson 1963; Wilson & Bredon 1963). We also found that without exception the animals were as fond of the irritant sap of *Euphorbia candelabrum* as they were of the sweet juices of sugar-cane. Nevertheless, in the presence of either active trypanosomiasis or heavy nematode infestations of the intestines, there was a rapid loss of weight despite the fact that there was no appreciable loss of appetite. Loss of weight in a rhinoceros is not readily recognised because of its thick skin. As a result, even though subcutaneous fat deposits are depleted, the animal's outward appearance is healthy. By the time the loss of weight is fully appreciated there is little prospect of recovery. Concurrent lethargy is not easy to recognise either, as rhinoceroses are quick to become docile in captivity. Within a few days they spend a lot of time asleep and are little perturbed by feeding, watering and cleaning activities.

The occurrence of ocular and digestive disturbance was a problem of prolonged captivity. To a large extent, the syndromes were thought to be due to vitamin A deficiency. One rhinoceros died after a little over a year on the island. Post-mortem examination showed lesions which indicated long standing intestinal damage. Despite supplementary feeding of vitamin A, the other specimen died after three years in the zoo and showed symptoms which were suggestive of an inadequate intake of this vitamin. Post-mortem features were indeterminate; bacteriological and parasitological examinations, and microscopical examinations of blood and tissue smears failed to assist in diagnosis.

For a variety of reasons we have been unable to continue with the programme of investigation and translocation. This is unfortunate as, although our observations were accompanied by heavy mortality and we were unable to examine every casualty, some interesting and important features have been brought to light.

In the 1964–65 period, a total of 59 rhinoceroses were captured and of these only three died of causes which were directly related to the method of capture. This low mortality rate (5·1%) reflects the efficiency of the Hartley team and compares favourably with wildlife losses related to immobilising techniques (Orr & Moore-Gilbert, 1964, 5·9%; Harthoorn & Bligh, 1965, 2·0%; Pienaar, *et al.* 1966, 2 to 3%; Short & Spinage, 1967, 9 to 29%).

In situations which warrant the translocation of endangered rhinoceroses to alternative and safer habitats the conventional method of chasing and noosing appears to be a satisfactory method of capture. Harthoorn (1965) recommends immediate translocation if the move to the new area can be carried out within 20 hours. However, translocations from the Lamai and the Grumeti to Rubondo take from two to three days to complete, and under these and similar conditions some time in field captivity appears unavoidable. In such circumstances it seems advisable to provide adequate trypanosomidal and vermicidal treatment.

SUMMARY

Black rhinoceroses were captured in the Grumeti and the Lamai game controlled areas of north-western Tanzania where there is considerable human pressure on the land. Rubondo Island and Saanane Island, in Lake Victoria, were respectively designated as a game reserve and a game reserve zoo. Rhinoceroses were translocated to Rubondo, exhibited on Saanane, and moved to Arusha in northern Tanzania or to Kenya, for overseas markets. Over the period 1962–5, a total of 77 rhinoceroses were captured by lassooing them from vehicles. Fifty-nine were captured in 1964 and 1965, and an account of the mortality over this latter period is given in some detail.

In 1964, eight rhinoceroses were caught at the Grumeti. One died as a result of mismanagement. The same year 17 rhinoceroses were caught at the Lamai of which seven died. One death was related to the method of capture, two were regarded as due to bad management and three were attributed to haematozoal infection. One rhinoceros died after it was taken to Kenya.

In 1965, capture activity was limited to the Lamai. Thirty-four rhinoceroses were caught. Mortality was high. Two deaths were attributed to the method of capture; one was due to bad management technique; four were associated with systematic haematozoal disturbance; four were attributed to helminthiasis, and eight followed closely after a spray treatment with an organo-phosphorus compound. One animal escaped, and, subsequently, one died in Rubondo and two died in Kenya.

Overall in 1964–65, a mortality rate of 5·1% was attributed to the method of capture; 6·8% to mismanagement; 8·5% to trypanosomiasis; 3·4% to babesiasis; 6·8% to helminthiasis; and 13·6% to spray toxicity.

Trypanosomes of the brucei subgroup, large forms of *Babesia* piroplasms and *Strongylus* nematodes were regarded as of pathogenic importance. Prophylactic treatments were introduced to try to reduce mortality from trypanosomiasis and babesiasis and it appeared that control of trypanosomiasis was secured. The position was less clear with regards to babesiasis and, unfortunately, the programme was adjourned before the helminthiasis problem could be adequately investigated.

The rhinoceroses were kept in holding pens, at the Lamai and the Grumeti, until it was convenient to move them to their various destinations. Periods in field captivity ranged from 3 to 121 days.

Over the whole series of operations (77 captures, 1962–65) two rhinoceroses escaped and thirty-nine (52·0%) were thought to have survived the first year of translocation or capture for trade purposes. Two rhinoceroses were exhibited on Saanane Zoo; one died after a year and the other after some three years on the island.

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PRODUCTS MENTIONED IN THE TEXT

Diminazine aceturate, Berenil: Hoechst A.G.
Pyrithidium bromide, Prothidium: Boots Pure Drug Co. Ltd.
Homidium bromide, Ethidium: Boots Pure Drug Co. Ltd.
Dioxathion, Delnav: Cooper, McDougal and Robertson, Ltd.
Phenothiazine, Phenovis: Imperial Chemical Industries, Ltd.
Oxytetracycline hydrochloride, Terramycin: Pfizer Ltd.
Sulphadimidine, Sulphamezathine: Imperial Chemical Industries, Ltd.
Betamethasone sodium phosphate, Betsolan: Glaxo Laboratories, Ltd.

Management of antelopes at Albuquerque Zoo

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INTRODUCTION

In September 1962 the State of New Mexico Department of Game and Fish imported one male and three female Siberian ibex *Capra ibex sibirica* as a nucleus for the Department's 'Exotic Game Programme' (Poglayen, 1963). The main purpose of the project is to establish captive breeding herds of several non-indigenous species of exotic hoofed animals, and eventually to release their progeny in the wild in order to increase the variety of New Mexico's big game animals. Owing to the United States Department of Agriculture's quarantine laws, only the offspring of imported hoofed animals can be released in the wild; the original animals have to remain permanently in a zoo approved by the United States Department of Agriculture (USDA).

Albuquerque Zoo is the only zoo in New Mexico approved by the USDA. It is also a municipal zoo and the State Department of Game and Fish therefore made a special agreement with the zoo, sanctioned by the Albuquerque City Commission, that the zoo would undertake the management of the Department's breeding groups of hoofed animals.

In August 1963 the Game Commission authorised the importation of two male and six female gemsbok *Oryx g. gazella*, which were followed in December 1963 by two male and six female Greater kudu *Tragelaphus strepsiceros*. Two additional Greater kudu arrived in September 1964, while one male and three more female Siberian ibex were imported in July and November 1964. More recently three species have been added to the project: one male and two female Bezoar wild goats *Capra hircus aegagrus* arrived in April 1965, and three more females in May 1967; one male and one female Elburz red sheep *Ovis o. orientalis* in April 1965; and two male and three female Persian or Goitred gazelles *Gazella subgutturosa* in May 1967. All six species are now breeding in Albuquerque Zoo.

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