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RABIES IN AFRICAN WILD DOGS (*LYCAON PICTUS*) IN THE SERENGETI REGION, TANZANIA

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ABSTRACT: Rabies was confirmed as the cause of death of one African wild dog (*Lycaon pictus*) in the Serengeti region, Tanzania. One adult African wild dog in the same pack showed central nervous signs consistent with rabies infection. Inactivated rabies vaccine was administered intramuscularly to African wild dogs in two packs, by dart or by hand following anesthesia. These individuals comprised all known adults in the Serengeti National Park. In a limited study of seroprevalence of rabies antibody carried out at the time of vaccination, 3 of 12 African wild dogs sampled in the Serengeti had rabies serum neutralizing antibody titers before vaccination. Paired serum samples from two individuals sampled after vaccination showed increased antibody titers.

Key words: Rabies, *Lycaon pictus*, African wild dog, seroprevalence, Serengeti, wildlife vaccination, endangered species.

INTRODUCTION

The African wild dog (*Lycaon pictus*) is considered to be among the most endangered large carnivores in Africa and is declining over most of its range (Ginsberg and MacDonald, 1990; Fanshawe et al., 1991). In the Serengeti region of northern Tanzania, the known population of African wild dogs is comprised of <60 adults and thus is highly vulnerable to extinction from stochastic processes (Goodman, 1987).

Disease is one factor that may pose a severe extinction threat to small populations, as illustrated by an epizootic of canine distemper in black-footed ferrets (*Mustela nigripes*) in Wyoming (USA) in 1985, in which the population fell from 129 individuals to <20 individuals (Williams et al., 1988). For *Lycaon*, the risks associated with disease are further increased because of the intense social nature of the species. African wild dogs live in packs and all pack members frequently interact with each other, for example, by mouth-licking and by regurgitating food to pups (Skinner and Smithers, 1990). Once an infectious pathogen enters a group, the potential exists for transmission between all members of the pack.

In the 1970's, a recorded decline in the Serengeti plains African wild dog population from 110 adults to 26 adults was attributed largely to a high mortality in pups (Malcolm, 1979). Between 1985 and 1989 three packs disappeared entirely; but in other packs, all pups emerging from the den survived to adulthood (Laurenson, Lelo, and Borner, unpubl.). Some workers have suggested that disease has been a major cause of mortality in the Serengeti population (Schaller, 1972; Malcolm, 1979). In the late 1960's, clinical signs of disease, including weakness, incoordination, hindlimb paralysis, ocular and oral discharge were observed in three packs (Schaller, 1972) and in 1989, signs of disease were observed in one pack that subsequently disappeared (Fanshawe et al., 1991). However no disease epizootics were confirmed by laboratory diagnosis and little is known about the significance of different pathogens in the population.

Several pathogens have been identified in *Lycaon*, including *Ehrlichia canis* and *Encephalitozoon* sp. in captive animals (van Heerden, 1979; van Heerden et al., 1989), and anthrax in free-living animals (Turnbull et al., 1991). Based on clinical signs, post-mortem findings (Schaller, 1972; van Heerden et al., 1989) and histopathology (van Heerden et al., 1989), canine distemper may cause mortality in captive

and free-living African wild dogs. However, the disease has never been confirmed by virus isolation.

We describe a case of rabies in a pack of African wild dogs in the Serengeti region, Tanzania, in 1990, outline a rabies vaccination program, and give the results of a pre- and post-vaccination serological analysis.

MATERIALS AND METHODS

The Serengeti ecosystem (33°55′ to 35°50′E, 1°15′ to 3°3′S), is defined as the area traversed by the annual wildebeest migration, and comprises the Serengeti National Park (SNP) and areas within the Ngorongoro Conservation Area (NCA), Maswa Game Reserve, Loliondo Game Control Area, Ikorongo Controlled Area, and Grumeti Controlled Area in Tanzania and the Masai Mara Game Reserve, in Kenya. For the purposes of this paper, the term Serengeti will refer to the Tanzanian section of the Serengeti ecosystem.

An African wild dog monitoring project was set up in the Serengeti in 1985 by the Frankfurt Zoological Society, Federal Republic of Germany. Using aerial radiotelemetry, packs were located on an approximately monthly basis. Whenever possible, packs were subsequently observed on the ground. The unique coat pattern of each African wild dog allowed individuals to be readily identified and data were collected on pack movements, demography and behavior. In August 1990, the Serengeti African wild dog population comprised three known packs: the Salei Pack with a home range in the central and southeastern plains, the Ndoha Pack in the western corridor of the Serengeti National Park, and the Mountain Pack in the eastern plains.

On 24 August 1990, an adult male of the Mountain Pack which had been fitted with a radio collar on 24 May 1990 was located by aerial and ground radiotracking. On the same day the radio collar of another pack member, an adult female, was located nearby. The rest of the pack, comprising five adults and thirteen pups, could not be found. The radio-collared animal was thin and restless, and showed signs of a hind limb ataxia. The tail was held horizontally and the ears were less erect than normal. The dog did not pant, despite lying out in full sunshine in the middle of the day. He frequently chewed at skulls he encountered and yawned on several occasions. The dog was lost overnight. He was not observed after extensive aerial and ground radio-tracking over the subsequent two days, and he is presumed to have died underground.

On 24 August 1990, brain stem samples were collected from the head of an African wild dog carcass located by R. Burrows about 10 km from the radio-collared male. Brain stem samples were collected through the occipital foramen using World Health Organization (WHO) collection kits (Barrat and Blancou, 1988). Brain tissue was stored in a solution of 50% glycerol and 50% saline (0.85%). Samples of brain tissue were collected in 10% phosphate-buffered formalin and submitted for histopathological examination.

Rabies diagnosis was carried out at the WHO Collaborating Centre for Research and Management in Zoonoses Control, Malzeville, France. Diagnostic tests included direct immunofluorescence and mouse inoculation (Kaplan and Koprowski, 1973), and inoculation of murine neuroblastoma cells (Barrat et al., 1988). Monoclonal antibody typing of the isolated strain was carried out at the Central Veterinary Laboratory, United Kingdom, using a panel of antinucleocapsid monoclonal antibodies (King, 1991).

Fixed brain tissue was embedded in paraffin wax, cut at 5 μm and stained with hematoxylin and eosin (H&E) and Schleifstein's stain (Silverton and Anderson, 1961). Histopathological examination was carried out at the Institute of Zoology, Zoological Society of London. With the confirmation of rabies in the *Lycaon* population of both the Serengeti and the adjacent Masai Mara Game Reserve, Kenya (Kat, pers. comm.), the disease was identified as a threat to the survival of *Lycaon* in the Serengeti. Personnel of the Tanzania National Parks made the decision to implement a rabies vaccination program, which was considered an appropriate management intervention, based on guidelines set out by Hall and Harwood (1990). A parenterally administered inactivated rabies vaccine was used, because the risks associated with a live vaccine were considered to be too great for use in an endangered species.

A trial vaccination program was carried out on four seronegative captive African wild dogs at the Frankfurt Zoo using Madivak, an inactivated vaccine (Hoechst, Hanover, Federal Republic of Germany) known to induce an antibody response in foxes associated with protection against challenge infection 52 weeks after vaccination (von Neukirch et al., 1978). Following intramuscular injection of one ml of vaccine in this trial, no adverse effects were recorded in any individual. Five weeks after vaccination, serum samples were collected and serological analyses were carried out at the Veterinäruntersuchungsamt, Frankfurt, using an ELISA technique (Mebatsion et al., 1989). The four vaccinated dogs all seroconverted; three indi-

viduals had rabies neutralizing antibody titers of 2 International Units (IU)/ml and the other an antibody titer of 4 IU/ml (Frost, pers. comm.). Although challenge experiments are the only confirmation of vaccine efficacy, it was considered unacceptable by the Frankfurt Zoological Society to carry out such trials on an endangered species such as the African wild dog.

In September 1990, the two known African wild dog packs in the Serengeti were located by aerial and ground radio-tracking of collared individuals. Two dogs from each pack were anesthetized for fitting or removing radio collars. Doses of 62.5 mg xylazine (Rompun, Bayer, Bury St. Edmunds, Suffolk, United Kingdom), an average of 2.5 mg/kg, and 50 mg ketamine (Vetalar, Parke Davis & Company, Pontypool, Gwent, United Kingdom), an average of 2.0 mg/kg were administered using air-pressurized darts (New Softy, Distinject, Basel, Switzerland) fired from a Model 3/2 air-powered dart gun (Klinke, Birchenau, Federal Republic of Germany). Blood samples were taken from the saphenous vein into plain tubes and serum was separated and frozen within a few hours. One ml Madivak inactivated rabies vaccine (Hoechst) was administered by intramuscular injection in the quadriceps femoris. Intravenous injection of 0.5 mg of the alpha$_2$-antagonist RX821002A (Reckitt & Colman, Hull, United Kingdom) was administered to reverse anesthesia.

One ml of the vaccine was administered to each remaining dog in the pack >12-wk-old by darting into the shoulder muscle mass. Each dog was carefully identified at the time of darting to ensure that each individual was vaccinated only once. The site of impact of the dart was recorded. Pups <12 wk were not vaccinated because of the risk of impact injury and because of the possibility of inactivation of vaccine by maternally derived neutralizing antibodies. Pups in the Salei Pack were subsequently vaccinated after they were 12 wk of age.

Each pack was closely observed after vaccination for 48 h in the case of the Salei Pack and 15 hr for the Ndoha Pack; each animal was checked for signs of injection site reaction, lameness, injury, or systemic illness. After vaccination, attempts were made to locate and monitor each pack on an approximately monthly basis by aerial and ground telemetry. At both 28 and 59 days after vaccination, three individuals were again anesthetized with xylazine-ketamine for collection of post-vaccination blood samples to assess levels of rabies virus neutralizing antibodies. One of these dogs had been vaccinated by hand injection and two had been vaccinated by darting. Serum was separated the same day as collection. Serum samples were stored at −20 C and transported on dry ice.

Twelve pre-vaccination serum samples, from several wild dog packs, were analyzed to measure baseline rabies virus neutralizing antibody titers. These samples included sera from dogs anesthetized for fitting, replacing and removing radio collars during the previous two years, using the same protocol as described above. Neutralizing antibody titers were measured in three post-vaccination serum samples.

Serological analysis for rabies virus neutralizing antibody was carried out at the Central Veterinary Laboratory, Weybridge, England. Rabies virus neutralizing activity was measured using a modified rapid fluorescent focus inhibition test (Smith et al., 1973). Titers were expressed in IU/ml determined by comparison with a standard serum.

RESULTS

Direct immunofluorescence, inoculation of murine neuroblastoma cells and mouse inoculation tests were positive for rabies virus from brain stem samples collected from the African wild dog carcass from the Mountain Pack. Despite extensive post-mortem autolysis, there was a marked perivascular cuffing and a plasma lymphocytic infiltrate within the meninges. Schleifstein's stain was negative for Negri bodies.

Based on monoclonal antibody typing, the isolated virus was identified as serotype 1 and with a reaction pattern consistent with viruses isolated from domestic dog-associated rabies virus in eastern and southern Africa (King, 1991). The clinical signs exhibited by the radio-collared adult were consistent with central nervous system disturbance, such as that shown in clinical rabies infection.

Complications encountered during anesthesia included one animal which vomited on induction and one animal which started to recover prior to administration of the reversal agent. The reversal agent was subsequently given intramuscularly rather than intravenously. On recovery, animals quickly rejoined the pack.

Neither pack was greatly disturbed or disrupted by the darting procedure and the observed effect on each individual was minor. No lameness, injection site reactions, or systemic disease were seen in any

individual during a 5-mo follow-up period after vaccination. During this period three pups disappeared at sporadic intervals from the Salei Pack and a single adult disappeared from the Ndoha Pack. By 8-mo post-vaccination, at least four vaccinated adults died, but samples could not be retrieved and the cause of mortality was not determined.

Three of 12 unvaccinated African wild dogs, from two packs, had titers of rabies neutralizing antibody >0.5 IU/ml. These individuals were alive >15 mo after the blood samples were taken.

Post-vaccination samples all were >0.5 IU/ml. In two paired serum samples, there were increases in antibody titers subsequent to vaccination. The increase was from <0.21 to 0.55 IU/ml in a dart-injected animal and from 0.55 to 5 IU/ml in a hand-vaccinated animal.

DISCUSSION

In recent years, population biologists have become increasingly aware of the importance of disease in the dynamics of wild populations (Anderson and May, 1979). In small isolated populations that are highly vulnerable to extinction from chance environmental events, disease epidemics may play a particularly important role. The Serengeti African wild dog population is small and increasingly isolated within protected areas of Tanzania and Kenya.

Interpretation of results of the serological analyses should be treated with caution. Firstly, only a limited number of serum samples could be obtained, as logistical problems precluded extensive, controlled, and paired sampling of individuals. Secondly, the serological test was developed for measurement of serum neutralizing antibody levels in human sera and the specificity of the test has not been confirmed for African wild dogs.

In unvaccinated domestic dogs, antibody titers at levels of <0.5 IU/ml are considered to be nonspecific; but in rabies endemic areas, ≤17% of unvaccinated domestic dogs have specific serum rabies virus neutralizing antibody titers >0.5 IU/ml (Andral and Serie, 1957; Fekadu, 1991). If antibody titers >0.5 IU/ml in unvaccinated African wild dogs are specific for rabies, it appears that three African wild dogs, from two packs, previously have been exposed to rabies virus. The presence of serum neutralizing antibodies has been detected in other wild carnivore populations in rabies endemic areas, such as the Indian mongoose (*Herpestes auropunctatus*) (Everard and Everard, 1985), the raccoon (*Procyon lotor*) (McLean, 1972) and the striped skunk (*Mephitis mephitis*) (Carey and McLean, 1983). In foxes (*Vulpes vulpes*) in rabies-endemic areas of Europe (Baradel et al., 1988) and in jackals (*Canis adustus* and *Canis mesomelas*) in Zimbabwe (Foggin, 1988), a few animals have had detectable serum neutralizing antibodies.

If serum neutralizing antibody titers >0.5 IU/ml in unvaccinated African wild dogs are specific, they are likely to be indicative of previous nonfatal infection with rabies virus. In domestic dogs, non-fatal rabies infections have been well documented, with high serum neutralizing antibody levels occurring in dogs that have recovered from clinical rabies and in animals that have been exposed to a sublethal dose of rabies without showing clinical signs (Fekadu and Baer, 1980; Fekadu, 1991). However, the role of nonfatal rabies in the epizootiology of domestic dog and wildlife rabies is not known.

Most animals with high serum neutralizing antibody titers, including domestic dogs, cats (Bunn, 1991) and foxes (Blancou et al., 1986), usually resist subsequent challenge infection. However serum antibody levels are not the only protection against challenge (Fekadu and Shaddock, 1984). Without challenge experiments in *Lycaon*, it is not known what degree of protection is conferred by the antibody titers seen in the Serengeti African wild dog population, either pre- or post-vaccination. There is, however, no evidence that currently licensed vaccines fail to protect against chal-

lenge with different strains of rabies virus (Baer and Wandeler, 1987), such as the serotype 1 virus isolated from the Serengeti African wild dog. In contrast, commercial vaccines are known to afford little or no protection against infection by Mokola virus, a rabies-related virus (Foggin, 1988).

The source of infection and routes of transmission of rabies to Serengeti African wild dogs are not known. Between 1958 and 1978, rabies was not reported in the vicinity of the SNP (Rweyemamu et al., 1973; Magembe, 1985). However, in the late 1970's and 1980's there was a dramatic increase in the number of cases reported in the area, primarily in domestic dogs with occasional reports in other carnivore and ungulate species (Magembe, 1985). Among wildlife in the SNP, rabies was confirmed by laboratory diagnosis in bat-eared foxes (*Otocyon megalotis*) between 1986 and 1988 (Maas, pers. comm.).

The isolation of serotype 1 virus from the Serengeti African wild dog with a monoclonal reaction pattern consistent with domestic dog-associated rabies virus isolates supports the view that domestic dogs are a likely source of infection for African wild dogs. Both domestic dogs and African wild dogs may interact in pastoralist land surrounding the SNP, but the frequency of contact between these species is not known. Observations of encounters between African wild dogs and domestic dogs in Kenya and Botswana indicate that the two species show little hesitation in approaching each other and coming into close contact (Butynski, 1974). Interactions with many other wild carnivore species also occur frequently (Malcolm, 1979), but these have not been quantified. Although jackals are known to be important vectors of rabies in South African and Zimbabwe (Bengis & Erasmus, 1988; Foggin, 1988) no data are available for the incidence of rabies in jackals in the Serengeti region.

Further investigations are required to obtain an understanding of the ecology of rabies in the Serengeti ecosystem, to elucidate mechanisms of maintenance of the virus in wildlife species and to determine sources of infection and routes of transmission to African wild dogs.

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LITERATURE CITED

ANDERSON, R. M., AND R. M. MAY. 1979. Population biology of infectious diseases. Nature 280: 361–367, 455–461.

ANDRAL, L., AND C. SERIE. 1957. Etudes experimentales sur la rage en Ethiopie. Annales de l'Institute Pasteur (Paris) 93: 475–488.

BAER, G. M., AND A. I. WANDELER. 1987. *In* Virus infections of carnivores, M. Appel (ed.). Elsevier Science Publisher B.V., Oxford, England, p. 175.

BARADEL, J. M., J. BARRAT, J. BLANCOU, J. M. BOUTIN, C. CHASTEL, G. DANNACHER, D. DELORME, Y. GERARD, J. M. GOURREAU, U. KIHM, B. LARENAUDIE, C. LE GOFF, P.-P. PASTORET, P. PERREAU, A. SHWERS, E. THIRY D. TRAP, G. UILENBERG, AND P. VANNIER. 1988. Results of a serological survey of wild mammals in France. Revue Scientifique et Technique de l'Office International des Epizooties 7: 873–883.

BARRAT, J., AND J. BLANCOU. 1988. Simplified technique for the collection, storage and shipment of brain specimens for rabies diagnosis. WHO/Ra-

bies Research 88.27, World Health Organization, Geneva, Switzerland, 13 pp.

———, M-J. BARRAT, M. PICARD, AND M. F. A. AUBERT. 1988. Diagnostic de la rage sur cultures cellulaires. Comparison des resultats de l'inoculation au neuroblastome murin et de l'inoculation à la souris. Comparative Immunology Microbiology and Infectious Diseases. 11: 207–214.

BENGIS, R. G., AND J. M. ERASMUS. 1988. Wildlife diseases in South Africa: A review. Revue Scientifique et Technique de l'Office International des Epizooties 7: 807–821.

BLANCOU, J., M. P. KIENY, R. LATHE, J. P. LECOCQ, P. P. PASTORET, J. P. SOULEBOT, AND P. DESMETTRE. 1986. Oral vaccination of the fox against rabies using a live recombinant vaccinia virus. Nature 322: 373–375.

BUNN, T. O. 1991. Canine and feline vaccines, past and present. *In* The natural history of rabies, 2nd ed., G. M. Baer, (ed.), CRC Press, Boca Raton, Florida, pp. 415–425.

BUTYNSKI, T. M. 1974. An encounter between African wild dog and domestic dog. East African Wildlife Journal 12: 243.

CAREY, A. B., AND R. G. MCLEAN. 1983. The ecology of rabies: Evidence of co-adaptation. Journal of Applied Ecology 20: 777–800.

EVERARD, C. O. R., AND J. D. EVERARD. 1985. Mongoose rabies in Grenada. *In* Population dynamics of rabies in wildlife, P. J. Bacon (ed.). Academic Press, London, United Kingdom, pp. 43–69.

FANSHAWE, J. H., L. H. FRAME, AND J. R. GINSBERG. 1991. The African wild dog—Africa's vanishing carnivore. Oryx 25: 137–146.

FEKADU, M. 1991. Latency and abortive rabies. *In* The natural history of rabies, 2nd ed., G. M. Baer (ed.). CRC Press, Boca Raton, pp. 192–196.

———, AND G. M. BAER. 1980. Recovery from clinical rabies of two dogs inoculated with a rabies virus strain from Ethiopia. American Journal of Veterinary Research 41: 1632–1634.

———, AND J. H. SHADDOCK. 1984. Peripheral distribution of virus in dogs inoculated with two strains of rabies virus. American Journal of Veterinary Research 45: 724–729.

FOGGIN, C. M. 1988. Rabies and rabies-related viruses in Zimbabwe: Historical, virological and ecological aspects. D.Phil. Thesis. University of Zimbabwe, Harare, Zimbabwe, 254 pp.

GINSBERG, J. R., AND D. W. MACDONALD. 1990. Foxes, wolves, jackals and dogs; An action plan for the conservations of canids. IUCN World Conservation Union, Gland, Switzerland, 116 pp.

GOODMAN, D. 1987. The demography of chance extinction. *In* Viable populations for conservation, M. E. Soulé (ed.). Cambridge University Press, Cambridge, United Kingdom, pp. 11–34.

HALL, A., AND J. HARWOOD. 1990. The Intervet guidelines to vaccinating wildlife. National Environment Research Council, Sea Mammal Research Unit, Cambridge, England, 25 pp.

KAPLAN, M. M., AND H. KOPROWSKI. 1973. La Rage—Techniques de Laboratoire, 3rd ed. Monograph Series No. 23, World Health Organization, Geneva, Switzerland, pp. 75–87.

KING, A. A. 1991. Studies of the antigenic relationship of rabies and rabies-related viruses using anti-nuclear protein monoclonal antibodies. Ph.D. Thesis. University of Surrey, Guildford, United Kingdom, 272 pp.

MAGEMBE, S. R. 1985. Epidemiology of rabies in the United Republic of Tanzania. *In* Rabies in the tropics, E. Kuwert, C. Merieux, H. Koprowski, and K. Bogel (eds.). Springer-Verlag, Berlin, Federal Republic of Germany, pp. 392–398.

MALCOLM, J. R. 1979. Social organization and communal rearing of pups in African wild dogs (*Lycaon pictus*). Ph.D. Thesis. Harvard University, Cambridge, Massachusetts, 215 pp.

MCLEAN, R. G. 1972. Rabies in raccoons in the southeastern United States. The Journal of Infectious Diseases 123: 680–681.

MEBATSION, T., J. W. FROST, AND H. KRAUSS. 1989. Enzyme-linked immunosorbent assay (ELISA) using staphylococcal protein A for the measurement of rabies antibody in various species. Journal of Veterinary Medicine 36: 532–536.

RWEYEMAMU, M. M., K. LORETU, H. JAKOB, AND E. GORTON. 1973. Observations on rabies in Tanzania. Bulletin of Epizootic Diseases in Africa 21: 19–27.

SCHALLER, G. B. 1972. Serengeti lion: A study of predator-prey relations. University of Chicago Press, Chicago, Illinois, pp. 321–344.

SILVERTON, R. E., AND M. J. ANDERSON. 1961. Handbook of medical laboratory formulae. Butterworth & Co., London, England, pp. 445–446.

SKINNER, J. D., AND R. H. N. SMITHERS. 1990. The mammals of the southern African subregion. University of Pretoria, Pretoria, South Africa, pp. 429–433.

SMITH, J. S., P. A. YAGER, AND G. M. BAER. 1973. Rapid fluorescent focus inhibition test. Bulletin of the World Health Organization 48: 535–541.

TURNBULL, P. C. B., R. H. V. BELL, K. SAIGAWA, F. E. C. MUNYENYEMBE, C. K. MULENGA, AND L. H. C. MAKALA. 1991. Anthrax in wildlife in Luangwa Valley, Zambia. The Veterinary Record 128: 399–403.

VAN HEERDEN, J. 1979. The transmission of canine ehrlichiosis to the wild dog (*Lycaon pictus*) (Temminck) and black-backed jackal *Canis mesomelas* Schreber. Journal of the South African Veterinary Association 50: 245–248.

———, N. BAINBRIDGE, R. E. J. BURROUGHS, N. P. J. KRIEK. 1989. Distemper-like disease and encephalitozoonosis in wild dogs (*Lycaon pictus*). Journal of Wildlife Diseases 25: 70–75.

VON NEUKIRCH, M., O. JAEGER, B. LIESS, AND R.

BARTH. 1978. Parentrale Tollwutschutzimpfung von Füchsen mit Madivak. Deutsche Tierärztliche Wochenschrift 85: 77–112.

WILLIAMS, E. S., E. T. THORNE, M. J. G. APPEL, AND D. W. BELITSKY. 1988. Canine distemper in black-footed ferrets (*Mustela nigripes*) from Wyoming. Journal of Wildlife Diseases 24: 385–398.

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