Cause of wild dog deaths

SIR — African wild dogs (*Lycaon pictus*) are highly endangered, and their conservation is a top priority of the IUCN canid specialist group[1]. The wild dog population of Serengeti National Park has recently undergone a drastic, disease-driven decline. Burrows[2] has suggested that the population's demise is attributable to activation of latent rabies infections as a result of the stress of handling (darting, anaesthesia, radiocollaring or vaccination). He hypothesizes that handling may stimulate the production of adrenocorticosteroids, leading to immune suppression and thus to increased vulnerability to disease.

Because post-vaccination immunosuppression can lead to clinical distemper in captive African wild dogs[3], the hypothesis warrants careful testing. Furthermore, the hypothesis calls into question fundamental methods of studying wildlife; if typical handling is capable of inducing immune suppression, substantial changes in methods would be needed for many studies. In this light, several points are relevant.

Citing ref. 4, Burrows states: "handling-induced stress, as measured by highly elevated peripheral serum cortisol concentrations, results from immobilization of captive wild dogs". Four features of these data should be noted. (1) Cortisol was measured with a human cortisol radioimmunoassay kit[4], with no validation for *Lycaon*. (2) The wild dogs in this study were darted and anaesthetized 13 times in 51 days. As a result, they showed panic when darting was attempted, running directly into chain-link fences[4]. This is in sharp contrast to anaesthesia in the field, where darted wild dogs react little. (3) After handling, mean serum cortisol levels were 181 nmol l^{-1} (n=35). Baseline plasma cortisol levels are not known for *Lycaon*, but assuming a baseline similar to that of domestic dogs, repeated darting caused short-term cortisol peaks that were 1.4-fold above baseline; this is a mild elevation in comparison to corticosteroid peaks in other species. For example, in wild dwarf mongooses (*Helogale parvula*) captured in livetraps, urinary free cortisol increased 6.8-fold above baseline (t_{242} = 14.15, $P < 0.001$; S. C. and S. Monfort, manuscript in preparation).

(4) Most important, corticosteroid immune suppression results from chronically elevated adrenocorticoid levels, and not from adaptive short-term increases. No data allow us to address whether handling induces a long term stress response in *Lycaon*. Indeed, no previously published data have tested for chronic corticosteroid elevations in response to typical handling in the field, for any species. In Serengeti dwarf mongooses, trapping, anaesthesia and handling induced a significant short-term increase in urinary free cortisol. But in the month following trapping, mean urinary cortisol levels dropped to 10.1 ± 4.9 ng per mg creatine (mean ± s.e., n = 23), below the baseline (27.2 ± 1.8 ng per mg creatinine, n = 234). Thus, for the only species for which data are available, handling does not induce chronic corticosteroid elevation. For wild dogs, we have no data to address the question, and an answer awaits endocrine data from the field (now under way).

Are there alternative explanations to be considered? Wild dogs are susceptible to several fatal diseases, including distemper, rabies and anthrax[2,5]. The history of the Serengeti population shows that this disease outbreak is the latest in a series. In 1967–68, the population was not handled, yet three packs were noted to have been more than halved by disease (probably distemper)[6]. In 1971–73, the population was not handled, but the population fell from 96 to 49, with 5 of 12 packs disappearing; again, disease was implicated[7].

Studies of four major wild dog populations so far show no effect of darting and radiocollaring on mortality (vaccination has not occurred in these populations). 'Handling' is common to stable populations and the rollercoaster Serengeti population. Other factors differ, such as Serengeti's history of low population density and repeated bottlenecks[7]. These factors could be involved in the recent crash, and should be given equal consideration.

Scott Creel
Wildlife Conservation Research Unit, Department of Zoology, Oxford University, Oxford OX1 3PS, UK

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SIR — Burrows's Scientific Correspondence draws attention to the controversy over the possible connection between rabies vaccination and associated handling of the African wild dog, *Lycaon pictus*, and their catastrophic mortality in 1990–91 in the Serengeti/Masai Mara ecosystem[1]. *Lycaon* is a highly endangered, flagship species numbering fewer than 5,000 individuals distributed in fragmented populations[2]. Any concerns regarding the effectiveness of research on, or conservation of, this species demand close scrutiny.

Burrows raises misgivings about the need for and results of the vaccination programme. His simple question, "why vaccinate?", has a complex answer. Rabies characteristically causes high mortality in most canids[3,4]. Many countries in eastern Africa, including Kenya, have record numbers of confirmed cases, mainly in dogs[5]. Rabies has been confirmed in Serengeti bat-eared foxes (B. Maas, personal communication), and a reportedly rabid hyaena in the Mara has recently killed a child. Rabies was confirmed in five *Lycaon* individuals in the Serengeti/Mara ecosystem; the virus isolated from one of these is similar to the strain found in domestic dogs in Africa. Blood samples were drawn from 12 *Lycaon* in the Serengeti, three of which showed >0.5 IU per ml of rabies serum-neutralizing antibodies (RSNA)[6]. The measured presence of antibodies in unvaccinated animals could be caused by non-specific neutralizing substances, but titres of >0.5 IU per ml in domestic dogs are generally considered specific to prior exposure to rabies virus (M. Fekadu, personal communication). These antibodies do not necessarily mean that an animal is protected; it may be incubating the disease. But because all except one of the sampled *Lycaon* were seen 5 months later it is more likely that those with >0.5 IU per ml RSNA had survived earlier exposure. Protection from natural rabies virus infection requires both humoral (antibodies) and cell-mediated (activated lymphocytes) responses; it does not follow from the presence of humoral antibody that an animal is protected. Even if some individuals were immune, near-total population immunity to rabies has never been reported for any canid. For all these reasons, and because rabies (and distemper) was observed in domestic dogs within and adjacent to the ecosystem (K. Alexander, personal communication), rabies is clearly a disease risk for *Lycaon*, and one with potentially devastating consequences.

Why did rabies vaccination fail? The most plausible reason for so many *Lycaon* dying despite vaccination against rabies is that they did not die of rabies. The data are too few to determine if rabies caused the deaths in *Lycaon* up to 8 months after vaccination, but if it did either they were incubating the disease before vaccination or vaccination failed to protect them against subsequent challenge. The incubation period is unknown in *Lycaon*, but may exceed 12 months in domestic dogs[7] and in some wild African carnivores it may exceed 6 months (J. Bingham, personal communication). Vaccination could fail if: (1) the vaccine