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Handling-induced stress and mortalities in African wild dogs (*Lycaon pictus*)

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SUMMARY

Recently it was suggested that the handling of wild dogs (*Lycaon pictus*) by researchers in the Serengeti ecosystem created stress, resulting in the reactivation of latent rabies viruses in carrier animals. We present data from ongoing studies on free-ranging and captive wild dogs elsewhere in Africa which do not support this hypothesis. Cortisol profiles suggest that immobilization of wild dogs does not cause the chronic stress required for stress-reactivation of latent viruses. Furthermore, there is no evidence of handling-related mortalities in wild dogs: the survivorship of unhandled and handled free-ranging wild dogs did not differ and no captive animals died within a year of handling (immobilization and/or vaccination against rabies). We suggest that the mortalities observed in Tanzania were due to an outbreak of a disease which rabies vaccination was unable to prevent. Intensive monitoring and active management research programmes on wild dogs are essential as without these, critically endangered wild dog populations have little hope of survival.

1. INTRODUCTION

The African wild dog (*Lycaon pictus*), extinct in 19 of 34 subSaharan countries in which it once existed (Fanshawe *et al.* 1991), is an endangered species (Ginsberg & Macdonald 1990). Recently, wild dogs under study by researchers in the Serengeti ecosystem suffered a dramatic decline in numbers (Burrows 1992). Disease was implicated but the identity of the pathogen and the trigger of the disease outbreak were not known. Debate was sparked over the potential impact of handling by researchers on wild dogs. It was proposed that the stress experienced by wild dogs due to handling (immobilization and/or vaccination) caused immune suppression and the subsequent reactivation of latent rabies viruses in carrier animals, resulting in their deaths (Burrows 1992). The hypothesis was criticised on several counts (Creel 1992; Macdonald *et al.* 1992) but was recently reiterated (Burrows *et al.* 1994). Consequently, the Tanzanian National Parks Authority issued a ban on all handling and radio-tracking of wild dogs in national parks (*Lycaon* Working Party, personal communication).

If handling does result in mortalities, the ban will help to protect remaining packs of wild dogs in Tanzania. If the hypothesis is unfounded, however, the ban may endanger the future survival of these already threatened populations. The termination of active research programs will lead to less accurate monitoring and a reduced ability to assess threats to the populations, and thus a reduced probability of their protection. The seriousness of these implications makes it essential to examine the hypothesis that handling induces mortality.

Using data from ongoing field, captive and veterinary studies in southern and eastern Africa, we challenge two premises of the hypothesis: (i) that handling causes undue stress to wild dogs; and (ii) that handling (immobilization and/or vaccination) causes wild dog mortalities.

2. METHODS

During 1993 and 1994, the stress response of 14 captive and 11 free-ranging wild dogs to immobilization and blood sampling was measured. Captive animals were held at the De Wildt Cheetah Center and free-ranging animals were from the Kruger National Park, South Africa. The 13 male and 12 female study animals ranged from eight months to seven years of age.

Animals were immobilized between 09h30 and 12h30 by darting with fentanyl (0.1 mg kg^{-1}) and xylazine (1 mg kg^{-1}) (Kyron Laboratories (Pty) Ltd., Benrose, Johannesburg). After sampling, the effects of these drugs were reversed with yohimbine (0.125 mg kg^{-1}) and Narcan (1.2 mg per individual) (Boots Pharmaceuticals (Pty) Ltd., Isando, Johannesburg). Initial blood samples were drawn

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Table 1. *Initial plasma cortisol concentrations of wild and captive carnivores, either manually restrained or chemically immobilized*

species (common name)	wild/captive	description[a]	initial corticol mean ± s.d nmol l^{-1}	*n*	reference
manual restraint					
Canis familiaris dingo (dingo)	captive	M, B	140.7 ± 35.9	40	Corbett 1988
		M, NB	24.8 ± 5.5	40	
		F, B	80.0 ± 4.8	27	
		F, NB	30.3 ± 8.3	27	
Canis mesomelas (black-backed jackal)	captive	T	77.3 ± 65.1	6	van Heerden *et al.* 1982
		U	145.6 ± 42.5	5	
Mustela putorius furo (ferret)	captive	M, F	73.8 ± 7.0	48	Rosenthal *et al.* 1993
Canis familiaris (domestic dog)	captive	T	65.0 ± 22.0	25	Church *et al.* 1994
chemical immobilization					
Crocuta crocuta (spotted hyaena)	wild and captive	M	163.8 ± 28.0	33	van Jaarsveld *et al.* 1992
		F	168.0 ± 40.2	26	
Canis lupus (wolf)	captive	F	[41–126]	9	Packard *et al.* 1985
Panthera tigris (tiger)	captive	M, F	600[b]	6	Brown *et al.* 1988
Panther pardus japonensis (leopard)	captive	M, F	1100[b]	6	Brown *et al.* 1988

[a] M: males; F: females; B: in breeding season; NB: out of breeding season; T: tame; U: untamed.
[b] Estimates taken from graph (Brown *et al.* 1988).

8–12 min after darting, and serial samples were collected at 10 min intervals for 70 min after darting. Plasma was separated and frozen at −20 °C until analysis.

Plasma cortisol was assayed in duplicate using a validated human radioimmunoassay kit (Baxter Travenol Diagnostics CA-529). Antiserum specificity was determined by the suppliers. Cross reactivities with other steroids were as follows: Prednisolone 76 %, 11-Deoxycortisol 11.4 %, Prednisone 2.3 %, other steroids less than 1 %. The addition of cortisol (28, 138, 276, 552 and 1380 nmol l^{-1}) to a wild dog plasma pool resulted in recoveries which did not differ significantly from expected values ($t = -0.91$; d.f. = 4; $p > 0.05$) and parallelism between serial dilutions of wild dog plasma and a cortisol standard was evident over the entire range of the standard curve ($t = 0.173$; $v = 4$; $p > 0.05$). The interassay coefficients of variation were 9 % ($n = 8$) and 3 % ($n = 7$) for expected low and high cortisol plasma pools respectively, while the intra-assay coefficient of variation was 4 % ($n = 70$). Sensitivity of the assay was 5.52 nmol l^{-1}. Initial plasma cortisol concentrations of wild dogs were compared with those recorded by other authors for other wild carnivores and domestic dogs (*Canis familiaris*).

The survivorship of 40 captive and 135 free-ranging wild dogs one year after handling was compared to that of 305 unhandled free-ranging wild dogs one year after their first sighting. Handled animals were immobilized (various methods of anaesthesia used) and subjected to a variety of treatments including radio-collaring, implantation with radio-transmitters and blood sampling. Data pertaining to survivorship of free-ranging wild dogs (from Ginsberg *et al.* 1995) were from studies in five ecosystems (Kruger National Park, South Africa; Hwange National Park, Zimbabwe; northern Botswana; Selous Game Reserve, Tanzania; and Masai Mara, Kenya). Free-ranging animals which left the study area and were not sighted again were assumed to be dead.

Twenty-three captive animals were vaccinated against rabies using 1 ml Rabisin, an inactivated vaccine (Rhone-Poulenc Animal Health (Pty) Ltd., Halfway House, Johannesburg). Two animals were vaccinated by darting and the rest were vaccinated by hand, after immobilization. All other handled captive animals were immobilized as described above, implanted with transponders and/or subjected to blood sampling.

3. RESULTS

Wild dogs reacted to darting with little more than momentary alarm and disorientation was evident after about 5 min. Animals were usually fully sedated by 7 min post-darting and remained sedated throughout the 70 min sampling period. Occasionally, half-doses of drugs were administered after 60 min to animals which showed signs of recovery. Initial plasma cortisol concentrations of captive and free-ranging wild dogs (179.72 ± 41.16 nmol l^{-1}, $n = 10$ and 143.88 ± 76.38 nmol l^{-1}, $n = 7$, respectively) (see figure 1) did not differ significantly (Mann-Whitney, $Z = 0.830$, $p > 0.1$, $n = 17$) and were thus pooled.

The mean initial concentration (164.96 ± 58.92 nmol l^{-1}, $n = 17$) for wild dogs was higher than initial values recorded by other authors for several manually restrained carnivores (van Heerden & Bertschinger 1982; Corbett 1988; Rosenthal *et al.* 1993; Church *et al.* 1994) and for immobilized wolves (*Canis lupus*) (Packard *et al.* 1985) (see table 1). The initial plasma cortisol concentration of wild dogs was,

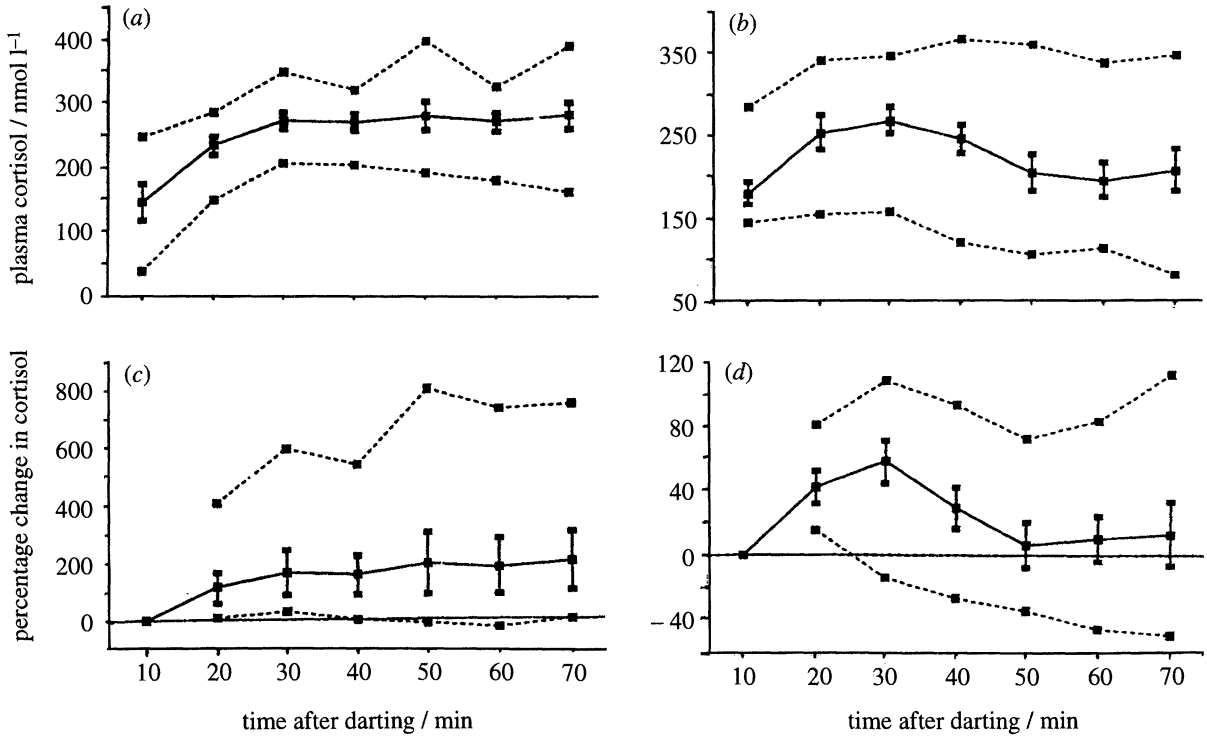

Figure 1. Cortisol response of wild dogs over 70 min after immobilization. Mean, s.e. of mean and range of plasma cortisol concentrations (nmol l^{-1}) in: (*a*) free-ranging and (*b*) captive animals. Percentage increase in cortisol concentrations over initial samples in (*c*) free-ranging and (*d*) captive animals. Solid line = mean + 1 s.e.; dashed line = range.

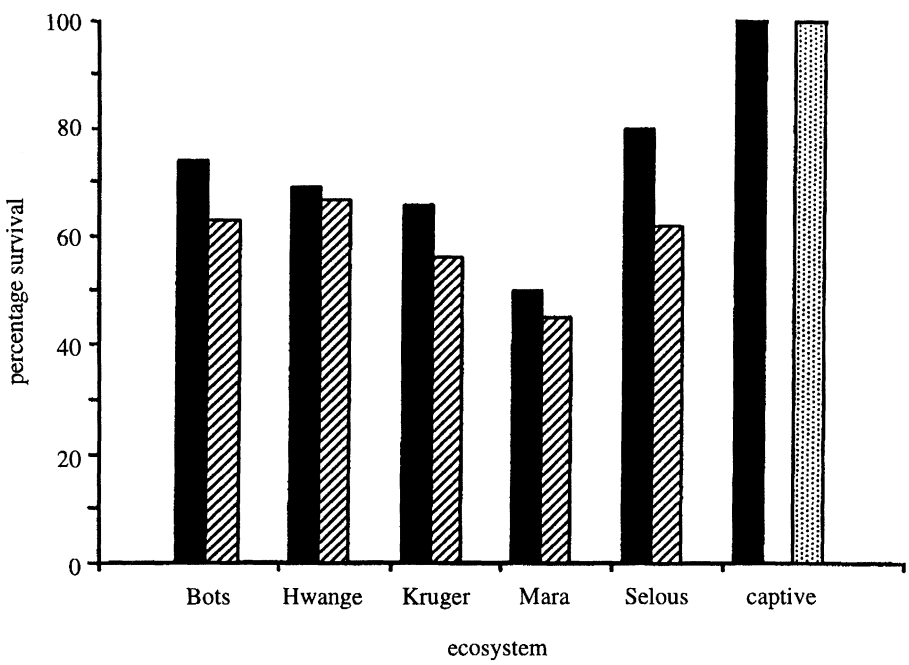

Figure 2. Percentage survival of unhandled (hatched bars), immobilized (solid bars) and immobilized and vaccinated (dotted bars) free-ranging wild dogs from five ecosystems (data from Ginsberg *et al.* 1995) and captive wild dogs. Bots: northern Botswana; Hwange: Hwange National Park, Zimbabwe; Kruger: Kruger National Park, South Africa; Mara: Masai Mara, Kenya; Selous: the Selous Game Reserve, Tanzania; Captive: captive animals, De Wildt Cheetah Breeding Center, South Africa.

however, comparable to that of immobilized wild and captive spotted hyaenas (*Crocuta crocuta*) (van Jaarsveld & Skinner 1992) (see table 1). It was also considerably lower than that measured for immobilized captive tigers (*Panthera tigris*) and leopards (*Panthera pardus*) (Brown *et al.* 1988) (see table 1).

From 10 min post-darting, the plasma cortisol profiles of the captive and free-ranging wild dogs differed markedly (see figure 1). Cortisol concentrations of free-ranging animals increased significantly from 10–20 min (Wilcoxon paired samples, $Z = 2.45$, $p < 0.01$, $n = 14$) and from 20–30 min after darting

(Wilcoxon paired samples, $Z = 2.98, p < 0.005, n = 22$) (see figure 1). Cortisol concentrations 70 min after darting were significantly higher than initial concentrations (Wilcoxon paired samples, $Z = 2.28, p < 0.05$, $n = 14$). Cortisol concentrations of captive animals also increased significantly from 10–20 min after darting (Wilcoxon paired samples, $Z = 2.45, p < 0.01, n = 14$). However, cortisol concentrations decreased significantly from 30–40 min (Wilcoxon paired samples, $Z = 2.00$, $p < 0.05$, $n = 24$) and from 40–50 min after darting (Wilcoxon paired samples, $Z = 2.63$, $p < 0.01$, $n = 24$). Thereafter, cortisol concentrations did not differ significantly from initial values (Wilcoxon paired samples, $Z = 0.67$, $p > 0.5$, $n = 24$).

The initial peak in cortisol concentration after darting was also recorded for each animal. These concentrations did not differ significantly for the free-ranging and captive animals (Mann Whitney, $Z = 0.57$, $p > 0.5$, $n = 25$) and represented a 119% (2.19-fold) increase over initial concentrations.

Ginsberg *et al.* (1995) have shown that handling has no effect on annual survival of free-ranging wild dogs in any of the five ecosystems investigated (see figure 2). Furthermore in the current study, 79 immobilizations of 40 captive wild dogs over the last two years did not result in any mortalities. Captive wild dogs which were vaccinated by darting ($n = 2$) or vaccinated by hand ($n = 21$) against rabies were all alive a year later.

4. DISCUSSION

(*a*) *Immobilization stress in wild dogs*

Animals subjected to a stressor respond by releasing a cascade of hormones, one of which is cortisol. This is an adaptive short-term response, preparing the animals to deal with the stressor. Regular exposure to severe stress will, however, be detrimental (Baxter & Tyrrell 1987). It has been postulated that exposure to chronic stress may cause the reactivation of latent viruses (see, for example, Soave 1964) and this forms the basis of the hypothesis that handling stress induces mortality in wild dogs (Burrows 1992). A single immobilization event is not a chronic but an acute stressor (Creel 1992). Nevertheless, aspects of the cortisol response to immobilization may reflect whether or not an animal routinely experiences high stress levels and how well it is able to cope with severe stressors.

Basal (unstressed) plasma cortisol concentration is one index of day-to-day stress (Sapolsky & Ray 1989). The stressfulness of the darting procedure probably results from the disorientation before unconsciousness rather than the pain of the intramuscular injection (Sapolsky 1982). In the current study, about 5 min elapsed between disorientation and collection of the first blood sample and this might be sufficient time for cortisol concentrations to alter from basal levels. Initial cortisol concentrations were thus merely an approximation of basal concentrations.

The considerable inter-individual variation in initial concentrations for wild dogs could be due to the extent of time lapse between disorientation and sampling, different individual stress histories, different levels of social stress experienced by different pack members (as found in baboon troops, see Sapolsky & Ray 1989) or the different conditions under which captive animals were held. Comparisons between studies are problematical because methodological differences (e.g. the type of drugs used for immobilization) will affect results. Nevertheless, initial cortisol concentrations of wild dogs were not excessively high when compared to those of most other chemically immobilized carnivores, particularly spotted hyaenas (van Jaarsveld & Skinner 1992). If stress results from the disorientation before unconsciousness (Sapolsky 1982), then it is unsurprising that the initial cortisol concentration of immobilized wild dogs was considerably higher than the basal concentrations of manually restrained carnivores.

Animals which lead stressful lives tend to have enlarged adrenal glands (Baxter & Tyrrell 1987) which, when a stressor is applied, will secrete greater amounts of cortisol. Peak cortisol concentration is therefore another index of the day-to-day stress experienced by an animal (we used the initial rather than the absolute peak as interpretation of the latter, usually recorded later in the sampling period, was more likely to be confounded by the effects of animals recovering from sedation). The similar peak cortisol concentrations of free-ranging and captive wild dogs may indicate that the two groups experience similar levels of day-to-day stress, although the stressors to which they are accustomed may differ. Alternatively, concentrations of free-ranging animals may have been excessively high because samples were drawn at the peak of the breeding season. The peak cortisol concentration for wild dogs represented a 2.19-fold increase in cortisol concentration over initial values. This was considerably less than the 6.8-fold increase above baseline reported for trapped dwarf mongooses (*Helogale parvula*) (Creel 1992) but similar to the greatest (more than two-fold) increase above initial values recorded for spotted hyaenas (van Jaarsveld & Skinner 1992). It therefore seems unlikely that either captive or free-ranging wild dogs experience chronically high levels of stress.

An indication of an animal's ability to cope with stress is the cortisol response over time after application of the stressor. Rats that were habituated to a particular stressor had a faster cortisol response and a faster return to basal cortisol concentrations than did rats which were unused to the stressor (Levine & Mullins 1966). A similar pattern was exhibited by the wild dogs in this study. The cortisol concentrations of free-ranging animals continued to increase significantly over a longer time period (30 min after darting) than did those of captive animals (20 min after darting). Furthermore, cortisol concentrations of captive wild dogs returned to initial levels by 50 min after darting whereas those of free-ranging wild dogs were still significantly higher than initial levels, 70 min after darting. Although the two groups of animals may experience similar day-to-day levels of stress, most captive animals had been immobilized previously. It would thus appear that the captive wild dogs were better habituated to immobilization stress than the free-ranging animals.

In this study, it was not possible to test the cortisol response of wild dogs to a chronic stressor. Instead, we attempted to assess whether study animals experienced chronic stress by measuring adrenal responsiveness to an acute stressor. The results indicate that captive wild dogs are better equipped to cope with immobilization stress than free-ranging wild dogs. However, even the continuous elevation of cortisol concentrations in free-ranging study animals does not appear to be a cause for concern (cortisol concentrations of olive baboons, *Papio anubis*, were still increasing 60 min after darting, see Sapolsky 1982). Furthermore, initial concentrations of captive and free-ranging animals were comparable. Neither group had unusually high initial cortisol concentrations or an excessive stress response after darting when compared to other immobilized carnivores. The cortisol response profiles of wild dogs after immobilization stress thus seem to be indicative of an adaptive acute response rather than the chronic stress response required by the hypothesis of stress-reactivation of latent viruses through immune suppression.

(*b*) *Handling and mortality in wild dogs*

Not only is there scant evidence that immobilization causes a stress response capable of reactivating latent viruses through immune suppression, there also appears to be no causal link between handling and mortalities of wild dogs. In southern Africa, handling of captive animals did not result in any mortalities. Furthermore, there was no effect of handling on the survivorship of free-ranging wild dogs in any of the five ecosystems investigated (Ginsberg *et al.* 1995). In fact, handled animals had a slightly higher survivorship than unhandled animals at every location.

Why should Serengeti be an exception? Contrary to Burrow's hypothesis, the population size and number of packs in Serengeti was the same in 1977 after several years of almost no handling as in 1990, after several years of handling (Burrows *et al.* 1994). The population crash in this area only occurred after the onset of the rabies vaccination program in September 1990. For this latter period, Burrows *et al.* (1994) reported that handled Serengeti wild dogs had significantly higher mortalities than unhandled wild dogs. The mortality of vaccinated animals was, however, significantly higher than that of immobilized but unvaccinated animals (Burrows *et al.* 1994). By combining these two types of treatment (immobilized and vaccinated) into one category, namely 'handled', a false impression may have been created. The high mortality of 'handled' animals may merely have been due to the high mortality of vaccinated individuals, with immobilization *per se* contributing little (if at all) to the mortality pattern.

The reason for the high mortality of vaccinated animals in Serengeti is unclear, as vaccination with an inactivated vaccine is not, in itself, harmful. This is evident from trials on captive wild dogs using Rabisin (D. G. A. Meltzer, personal communication) and Madivac, the vaccine used in Serengeti (Gascoyne *et al.* 1993). Because of the difficulty of performing challenge experiments on an endangered species, however, the efficacy of such vaccines is unknown. It may be that the mortalities experienced in the Serengeti study packs were due to an outbreak of disease which the vaccination program was unable to prevent (Creel 1992; Macdonald *et al.* 1992). This disease may have been rabies, as a case of rabies was confirmed in the Serengeti study population in 1990. Other diseases cannot be ruled out, however, and Macdonald *et al.* (1992) suggested distemper as one possibility. During 1993 and 1994, large numbers of lions (*Panthera pardus*) in the Serengeti region died from canine distemper virus (CDV) (Anon. 1994). Wild dogs are susceptible to this disease (van Heerden *et al.* 1989; Durchfeld *et al.* 1990) and the disappearance of wild dogs in Masai Mara was concurrent with an outbreak of CDV amongst domestic dogs (Alexander & Appel 1994). Moreover, exposure to disease may have been due to the increased influx of wild dogs into the study area from outside. No data have been presented which allow assessment of the state of this population of wild dogs. Their influx into the reserve is not necessarily indicative of a healthy population outside the study area, as suggested by Burrows *et al.* (1994), but could instead reflect increasing pressures on wild dogs in unprotected areas.

The final decimation of the Serengeti population of wild dogs, no doubt triggered by a stochastic event such as an outbreak of disease, may have been preempted by the decline in numbers over the previous years. This could have been due to the impact of other predator populations on wild dogs. Burrows *et al.* (1994) rejected the possible impact of lions because wild dog pup survival figures did not decrease with a corresponding increase in lion numbers. No census data for lions were provided, however, and the initial increase in lion numbers did correspond with a dramatic decline in pup survivorship (Burrows *et al.* 1994). Furthermore, the potential impact of lions on adult wild dogs was not considered. In the Kruger National Park, wild dog packs appear to avoid areas utilized by lions so that the distribution of wild dogs does not coincide with that of their preferred prey (Mills 1995). High lion density could thus also impact negatively on adult wild dog hunting success and survivorship.

5. CONCLUSIONS

Although an awareness of the possible negative impacts of research programs on study populations of endangered species is important, it should be borne in mind that without constant monitoring and active management there is little hope of ensuring the future survival of such populations. Neither the data of Burrows *et al.* (1994) nor the data presented here support the hypothesis that immobilization and/or vaccination with an inactivated rabies vaccine causes undue stress to wild dogs, or results in mortalities.

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